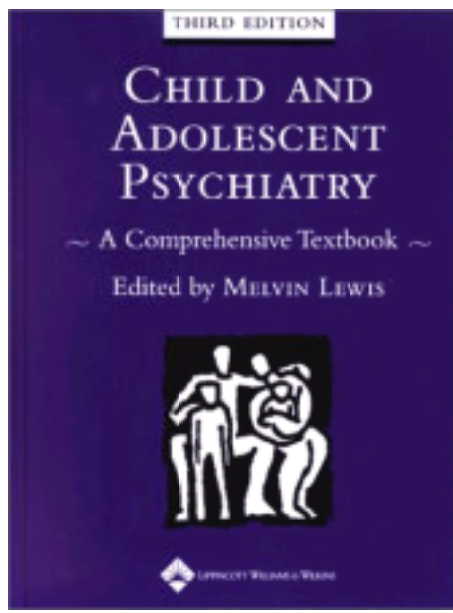


Child and Adolescent Psychiatry: A Comprehensive Textbook 3rd edition (May 15, 2002): By Melvin Lewis (Editor) By Lippincott Williams & Wilkins Publishers



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# Child and Adolescent Psychiatry: A Comprehensive Textbook

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## FOREWORD TO THE FIRST EDITION

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On two previous occasions, including my Presidential Address to the American Psychiatric Association (1984), I delineated the requirements for a field of medical practice to be identified as an independent specialty. Among a number of requirements, I emphasized the need for a scientific base that is specific to the specialty, even though substantial reliance might be placed on the basic sciences of other specialties. I went on to point to the importance for clinicians in the specialty to have a unique set of knowledge and skills. I also stated that both the scientific base and the specific skills must have a degree of conceptual clarity to form the framework of a required educational curriculum. Child and adolescent psychiatry now has such an educational curriculum.

A specialty such as child and adolescent psychiatry also needs its own comprehensive textbook that conceptualizes the whole field for the reader. Such a textbook should combine information on the scientific bases and the techniques into an amalgam that enables the reader to acquire the up-to-date knowledge necessary for state-of-the-art practice. No easy task!

Approaching Lewis' volume precisely in the context of the above concepts, readers will acquire equal knowledge about what to do, when to do it, and, most importantly, why to do it. They will also become better practitioners whose actions will be based not on empiricism alone but on scientific evidence as well. Best of all, *Child and Adolescent Psychiatry* has the content and conceptual clarity needed for the education of new generations of students.

In addition to students of child and adolescent psychiatry, others concerned with the mental health of children and adolescents will find the text of great value. *Child and Adolescent Psychiatry* will be useful for experienced practitioners, pediatricians, psychologists, and neurologists, as well as allied professionals such as educators, nurses, social workers, lawyers, and policy planners. Some will find the scientific expositions of greater interest, others will focus on the practical clinical issues. All will gain a new opportunity for enlarging their knowledge and rounding out their clinical expertise through reading this intellectually satisfying and clinically informative book.

*Child and Adolescent Psychiatry* is more than a detailed primer for students of child and adolescent psychiatry. It is, in fact, a truly comprehensive reference manual for those who labor for the advancement of mental health for children and adolescents. Conceptually, scientifically, clinically, and pedagogically this book fills an important need in child and adolescent psychiatry. This was the goal for which Mel Lewis and the contributors strive for with the hope of contributing to the greater achievements and happiness of new generations of our young. They have succeeded admirably in this goal.

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## PREFACE TO THE THIRD EDITION

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This is a big book, bigger than its first and second editions. Its growth reflects that of the field of child and adolescent psychiatry. Our understanding of normal child development and of the roots of psychopathology has advanced from a primarily psychodynamic paradigm to a sophisticated appreciation of the ways in which genetic, neurophysiological and environmental variables interact to influence adaptation. Advances in neuroimaging, psychoneuroendocrinology and psychoneuroimmunology are beginning to shed light on the different ways in which human development can go awry. As we literally and figuratively unravel the human genome and compare ourselves to the other denizens of our planet, we begin to appreciate those characteristics we share and those that distinguish us from more primitive species.

Our increasing recognition of the differences between the neuroanatomy, neurophysiology and psychoneuroendocrinology of males and females from infancy through adulthood has moved us beyond simplistic social explanations for behavioral differences. Scientific advances in the understanding of gender identity and behavior have encouraged not only a reexamination of the differences between the ways that boys and girls think and feel, but also a reassessment of the ways that we measure intelligence and even morality.

In tandem with our increasing knowledge regarding the ways that genetic and neurophysiological variables affect behavior has come an increasing appreciation of the numerous ways in which social and cultural variables influence adaptation. Mental illness is understood and expressed in different ways in different settings. We are just beginning to recognize our own prejudices and the ways that they, and the availability or lack of treatment resources influence the diagnoses and dispositions we make. Most important, the devastating effects of poverty, grinding poverty, on the growth, development, mental health and adaptation of children of every race and culture has been well documented. We can now identify its effects at the biochemical, neurophysiological, neuroanatomical and behavioral levels. Unfortunately we are less skilled at preventing and reversing these consequences.

This big purple book contains an account of where we stand clinically and scientifically. It confronts us with the chasm that separates what we know about the causes and treatments of children's psychiatric disorders and what we as a society actually provide. It reminds us that the current constraints imposed on caregivers, the extreme criteria for admission to hospital, and the absurdly limited lengths of stay permitted under our current delivery systems, deprive the majority of mentally ill children in our country of the optimal care we know how to provide. At this point in our own development as a society our social values and priorities have a long way to go to catch up with our scientific understanding and our sophisticated technology. We must keep in mind that in the face of all that we know about human development and the ongoing myelination of the frontal lobes into the third decade of life and beyond, we nevertheless allow our children to be tried as adults and executed for crimes committed before their eighteenth birthdays. Let us hope that before the fourth edition of this book appears, our ethical values will have caught up with our scientific knowledge.

This purple book contains the knowledge and wisdom of 200 experts in their respective clinical and research disciplines. To thank them individually would add enormously to the length of this volume. Therefore I must thank them together for their enthusiasm, dedication and especially for the time spent sharing their expertise with the rest of us in clear, readable, plain English. I am grateful too to the members of the advisory committee for their suggestions and wisdom.

Lippincott, Williams & Wilkins have collaborated with me to make what at first seemed an insurmountable task into an exhilarating challenge to be met. I particularly want to thank Joyce Murphy, managing editor, and Charley Mitchell, executive editor, for their warmth, common sense and ongoing encouragement and Allison Risko, project editor, for her untiring striving for perfection in correcting the page proofs.

I am especially grateful to Ann Chieppo who has assisted me through all three editions of the purple book and who, unfazed by 133 chapters and 200 authors, helped bring it all together.

Finally, I thank my family, Dorothy, Gillian, and Eric, for rejoicing with me when chapters were flowing in, commiserating with me when authors requested "just one more week" in order to include their newest data, and for being there to share the frustration and exhilaration of putting together this big purple book.

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## PREFACE TO THE FIRST EDITION

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*Child and Adolescent Psychiatry* presents an up-to-date, comprehensive, and useful account of child and adolescent psychiatry today. The book covers the field completely, from the migration of cells in the embryonic cortex to issues of world mental health. The book encompasses normal development, psychopathology, diagnosis, and treatment. International authorities have contributed scholarly chapters on virtually the complete syllabus for the modern child and adolescent psychiatrist.

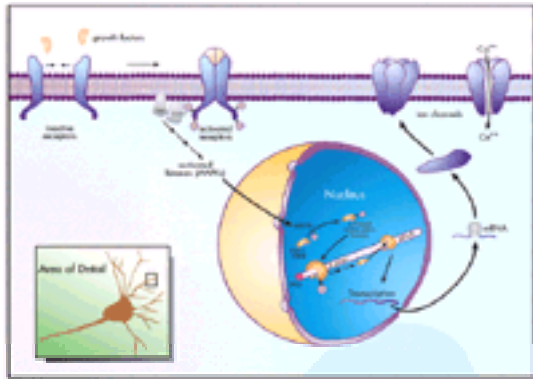
The book contains nine sections: Normal Development; Development of Symptoms; Overview of Etiological Influences; Nosology and Classification; Diagnostic Assessment; Syndromes; Treatment; Child Psychiatry and Allied Professions (pediatrics, education, law, and public health); and Training and Research.

A unique feature is Section II, Development of Symptoms. Here, authorities were invited to describe their understanding of the actual development of cardinal symptoms encountered in child and adolescent psychiatry. Symptoms such as anxiety, depression, psychotic thinking, and violence, seen in a variety of different kinds of disorders, are explored in a manner similar to the way internal medicine would approach symptoms such as fever and pain. Another unusual feature of the book is its emphasis on the relevance of research findings to clinical experience. Clinical case illustrations highlight important aspects of the syndrome being described. The book also draws on many theories of development, which are discussed in terms of their clinical relevance. The book will be useful to all specialists who try to help children and their families. There are subsections on child psychiatry and pediatrics, child psychiatry and education, child psychiatry and law, and child and adolescent psychiatry and public health. For the child psychiatry trainee and the young researcher, there are specific chapters on training and certification in child and adolescent psychiatry, new research techniques, and the ethics of research involving children.

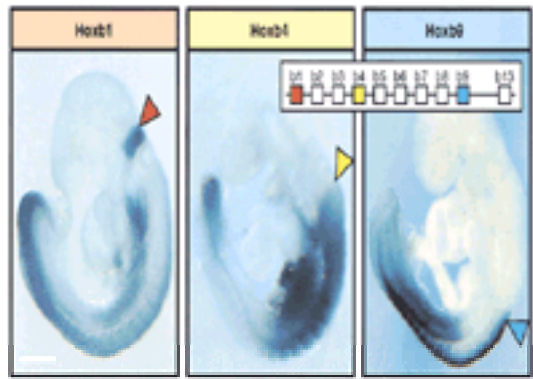
The enthusiasm of the authors and their commitment to excellence is a source of great pleasure and pride to me. I am indebted to each of them. I wish to express special appreciation and gratitude to Doctors Dennis Cantwell, Stella Chess, James Leckman, Dorothy Otnow Lewis, David Tomb, Elizabeth Weller, and Gabrielle Weiss who comprised the Advisory Committee and whose advice was invaluable. I wish to thank the staff at Williams & Wilkins, including Nancy Collins, Carol Eckhart, Michael Fisher, John Gardner, Rebecca Marnhout, Anne Stewart Seitz, Steve Siegforth, Deborah Tourtlotte, and Chuck Zeller for their special combination of competence and enthusiasm. I also thank my secretary, Ann Chieppo, for her steadfast support and assistance in this immense task. Finally, I wish to thank my wife Dorothy for her clarity of thought and loving support.

Melvin Lewis

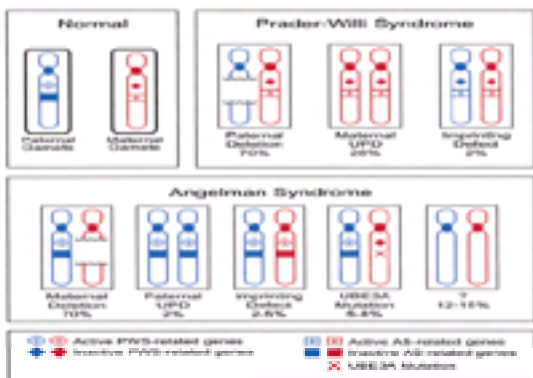
## Color Plates



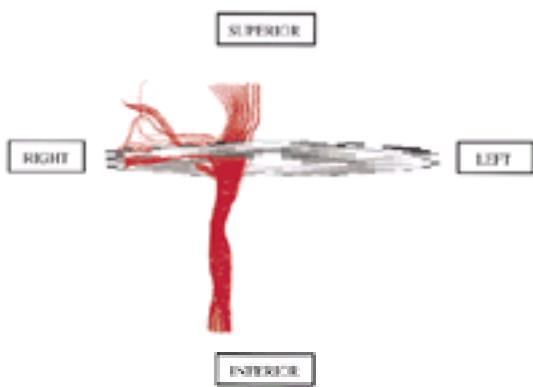
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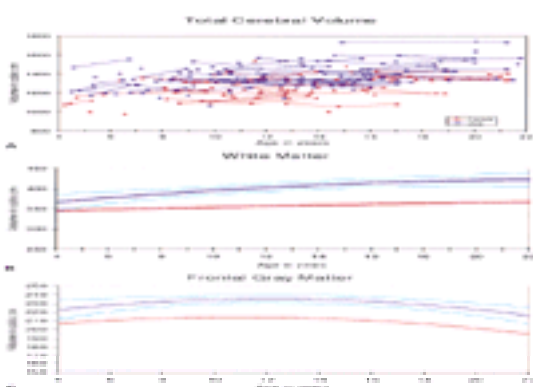
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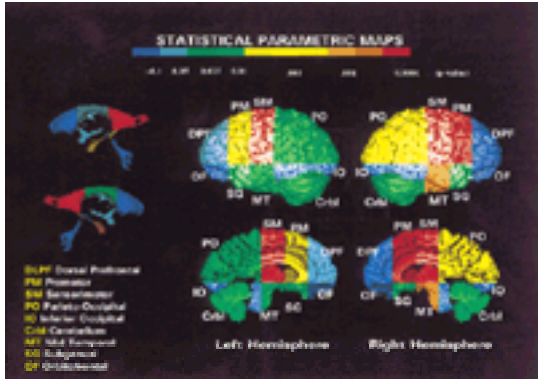
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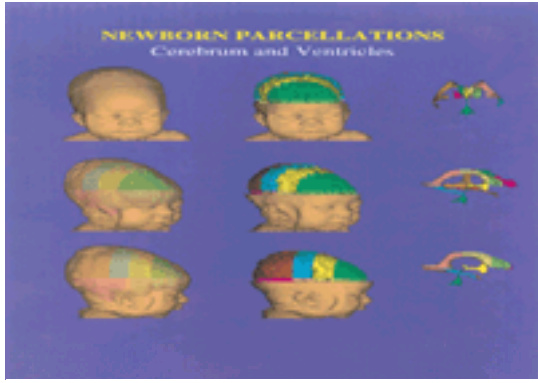
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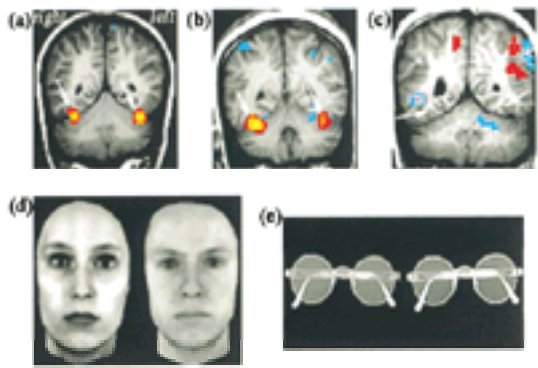
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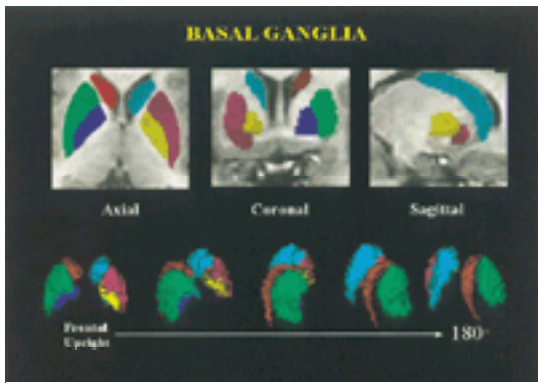
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Color Plate Figure 9.6: (See Black and White [Figure 9.6.](#))



Color Plate Figure 9.8: (See Black and White [Figure 9.8.](#))

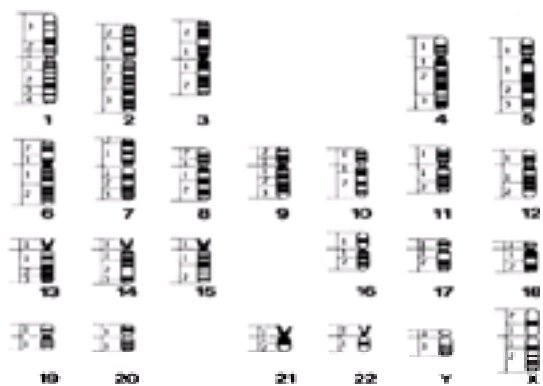
# 1 GENES AND DEVELOPMENTAL NEUROBIOLOGY

James F. Leckman, M.D., Flora M. Vaccarino, M.D., and Paul J. Lombroso, M.D.

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Human beings are complex living organisms that can be characterized by their appearance and behavior at each point in the life cycle. Many of these characteristics are uniquely human, such as the array of languages that facilitate interpersonal communication and permit a meaningful interplay of ideas and emotions. Other characteristics, such as affection and aggression, are less distinctive and place our species as one among many that populate the earth. Scientific advances over the past 150 years clearly indicate that hereditary factors are transmitted from generation to generation and account for much of the observed variation among and within species. Although the complexities of human existence cannot be reduced simply to the effects of genes, it is inescapable that genetic factors provide the biological basis for many of our potentialities and vulnerabilities as human beings ([Leckman and Mayes, 1998](#)).

Our genetic endowment, as a species, is a unique collection of discrete units of heredity (genes) that for the most part are linearly arranged on 46 chromosomes (22 pairs of homologous chromosomes and two sex chromosomes) ([Fig. 1.1](#)). This collection of genes makes us both alike and different from other organisms. Although the precise genetic determinants of our interspecies similarities and differences are largely obscure, it is probable that many of the responsible genetic factors will be identified. For example, investigators are in the midst of discovering the cascade of genes that have contributed to the remarkable neuroanatomic and functional evolution of the cerebral neocortex across different mammalian species over the past 50 million years ([Northcutt and Kaas, 1995](#)).



**Figure 1.1.** Depiction of high-resolution banded human chromosomes. (Adapted from Yale-HHMI Human Gene Mapping Library Chromosome Plots, Number 5. New Haven, CT, Howard Hughes Medical Institute, 1989.)

Genetic factors also contribute to variations within species. A large number of physical and psychological traits, including gender, height, and intelligence, have been shown to be under at least partial genetic control. One need only examine the striking physical and psychological similarities between monozygotic (genetically identical) twins reared apart to recognize the powerful influence of genes in determining who we are ([Juel-Nielsen, 1980](#); [Shields, 1962](#)). Some of these intraspecies differences, such as gender, are owing to actual differences in the number and type of genes present in the individual. For example, any gene on the Y chromosome is present only in males and is transmitted exclusively from father to son. Other intraspecies differences are owing to there being multiple forms (polymorphic alleles) of specific genes that are distributed within the population. Some of these allelic variants contribute to traits such as blood type, height, or eye color. In others, the changes within the genes are more subtle and make the individual more susceptible to additional factors, genetic or environmental, that lead to phenotypic expression of clinical significance.

Some allelic variations are so significant that we use the term "mutation" to signify that the changes will usually lead to disease states such as Rett's syndrome, Huntington's disease, Marfan's syndrome, or sickle cell anemia, disorders in which other ameliorating factors will have little effect. Finally, some intraspecies differences may depend on the sex of which parent passed on a particular piece of genetic material through the process of genetic imprinting ([Moore and Haig, 1991](#)). Briefly, imprinting is an epigenetic process that effects the shape of the DNA molecule and its accessibility to proteins that regulate gene expression, such that the genes within an imprinted chromosomal region from one parent are effectively silenced. The most dramatic example of imprinting concerns two distinctively different developmental disorders, Prader-Willi and Angelman's syndrome, that are caused by alterations of DNA in the same general chromosomal region on chromosome 15 ([Knoll et al., 1993](#)) ([Chapter 2](#)).

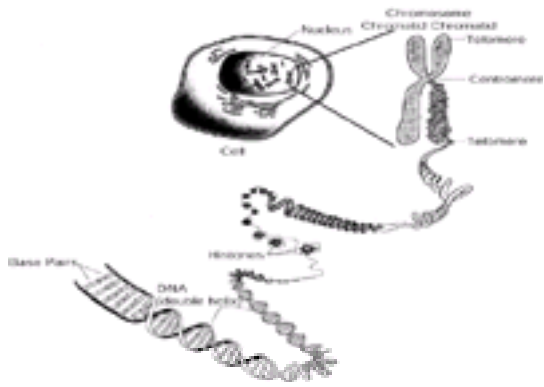
Apart from interspecies and intraspecies variations, it is also important to recognize that maturational differences within an individual member of a species can also result from genetic factors. Simply put, this means that not all genes are active at the same time. For example, the hemoglobin genes active during fetal life are different from those active in adults. As the temporal patterns of gene expression during human development become known, we may come to be able to better predict each individual's genetic potential.

The next two sections, Genes, and Regulation of Gene Function, present a condensed summary of some of the fundamental aspects of the structure and function of genes and gene products. Several excellent general references cover this material in greater depth ([Alberts et al., 1995](#); [Watson et al., 1988](#)).

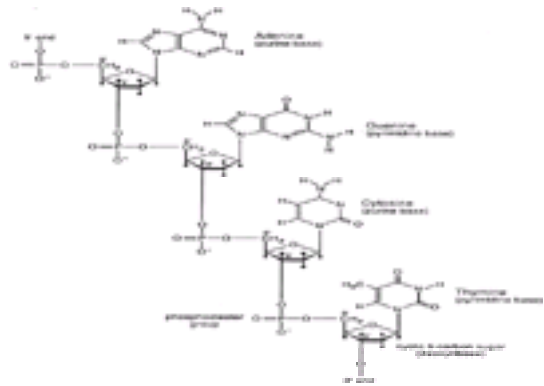
## GENES

The existence of discrete hereditary factors or genes was initially postulated by Mendel in 1865 ([Mendel, 1965](#)), but their importance was not appreciated until the early 1900s. Genes are linearly arranged on chromosomes found in the nuclei of cells ([Fig. 1.2](#)). They are composed of deoxyribonucleic acid (DNA). DNA consists of a string of nucleotides (complex molecules that contain a sugar moiety, a phosphate group, and either a purine or pyrimidine base) that are linked end to end. These linkages involve connecting the carbon atom of one sugar moiety to the carbon atom of the next nucleotide through a phosphate group. Every nucleic acid chain has a direction determined by the orientation of its sugar phosphate backbone. The two ends are designated 5' and 3' to indicate this orientation. Four separate nucleotides are found in DNA. Two contain purine bases (adenine and guanine), and two contain pyrimidine bases (thymine and cytosine) ([Fig. 1.3](#)).





**Figure 1.2.** The structure of chromosomes. Chromosomes are thread-like packages of DNA in the nucleus of a cell. This drawing also depicts a diagram of the DNA double helix in its common form showing the antiparallel orientation of the complementary strands and the wrapping of DNA around histone cores. (Adapted from the Glossary of Genetic Terms and associated illustrations found on the web site of the National Genome Research Institute, 2000.)



**Figure 1.3.** Chemical structure of DNA, showing the 3'-5' phosphodiester linkages that connect the nucleotides. (Adapted from Watson JD, Hopkins NH, Roberts JW, et al.: *Molecular Biology of the Cell*. Menlo Park, CA, Benjamin/Cummings, 1987.)

Genetic information is conveyed by the specific nucleotide sequence of a particular DNA molecule. Most DNA molecules exist in a double helical structure composed of two polynucleotide chains held together by a series of hydrogen bonds between complementary base pairs (adenine is bonded to thymine and guanine to cytosine) (Fig. 1.3). This structure confers stability on the molecule and provides the basis for replication. Knowing the sequence of one strand immediately provides precise knowledge of the sequence of the other because each strand of DNA in the double helix is exactly complementary to the other.

The sequence of nucleotides on a DNA molecule determines the order of the 20 different amino acids in proteins. As a consequence, the information contained in DNA provides the instructions that direct cells to grow and divide, set in motion developmental sequences that lead to orderly differentiation of cell types, and provide for the maintenance of a diversified population of cells necessary for the successful functioning of complex organisms.

The nucleotide sequence, however, does not provide a direct template for protein synthesis. Instead, there is a series of intervening steps that requires the DNA to be transcribed into messenger ribonucleic acid (mRNA). Messenger RNA molecules are very similar in composition to DNA and can hybridize with complementary DNA sequences. All mRNA chains grow in a 5' to 3' direction and are single stranded. Mature mRNA molecules are rapidly transported out of the nucleus and into the cytoplasm where they serve as the template for protein synthesis.

The translation of a part of the genetic code into a specific amino acid sequence occurs at ribosomes located in either the cytoplasm or attached to the endoplasmic reticulum. The genetic code is determined by the sequence of bases, with sets of three bases constituting one coding unit, or codon. At the ribosome, codons of an mRNA molecule bind to complementary anticodons of transfer RNA (tRNA), which then transfer specific amino acids to a growing protein chain. The basic elements of this "central dogma" of protein production were first proposed by Crick in 1956 and remain fundamental to our understanding of these basic molecular events (Crick et al., 1961).

Genes are normally extremely stable and are precisely copied during the chromosomal duplications that precede cell divisions (mitosis). Obviously, any mistakes that occur have the potential of disturbing the normal sequence of amino acids in a protein. There are a number of proteins within the nucleus whose functions are to recognize and repair any errors within the DNA sequence. Very rarely, mistakes go uncorrected and result in mutations. The majority of such changes have no effects as they occur in regions on the DNA molecule that do not encode for protein; however, when mutations occur within the sequence that encodes for protein, they may have several effects. First, they may have no effect or slightly change the function of the protein, leading to the allelic variations discussed in the preceding. Second, they may be deleterious to the function of the encoded protein and, ultimately, to the organism. Finally, this capacity for change can, in rare instances, lead to positive consequences that serve as the basis for evolution.

## REGULATION OF GENE FUNCTION

According to some estimates, only about 1% of the genome is being expressed at a given time in higher eukaryotic cells (Mendel, 1965). During the course of development and in any particular tissue, different but overlapping sets of genes are active. Some genes are constitutively expressed, whereas others are highly responsive to environmental changes. Regulation of gene function can occur at any one of the many steps required for gene expression (Fig. 1.4).



**Figure 1.4.** Sequence of events leading to gene expression. A protein-coding gene comprises a stretch of genomic DNA that contains an open reading frame. This region contains instructions for making the protein, as well as adjacent control regions—promoters and enhancers—where the gene's transcriptional mechanism is switched on or off. The promoter region is the site at which RNA polymerase binds and starts transcribing. The enhancer regions may be thousands of base pairs distant from the promoter. Transcription of the gene into mRNA may be either stimulated or inhibited by transcription factors that bind to promoter and enhancer regions. The mRNA formed by transcription is spliced to remove introns and processed within the cell nucleus to produce mRNAs that are exported to the cytoplasm for translation into protein at the ribosomes. Some proteins go through posttranslational modification to become biologically active. The four examples depicted include: cleavage of precursor proteins, conformational change through covalent cysteine-cysteine (C-C) bonds, phosphorylation of serine (S) or tyrosine (Y) (black squares), and glycosylation of asparagines (N, branching motif).

## Transcriptional Factors

Transcription depends on a complex series of events that lead to the formation of mRNA. A central event is the binding of RNA polymerase II in combination with other general transcription factors to specific region of the DNA, termed core promoter sites ( [Gumucio et al., 1996](#)). These core regions are usually found immediately upstream of the transcriptional start site, the most common being the “TATA” box. This is a sequence of highly conserved nucleotides within the promoter that serves as a signal and allows the active RNA polymerase II complex to bind tightly to the double-stranded DNA. A series of steps is initiated that unravels the sense strand and makes it more accessible to DNA polymerase II, which is able to initiate transcription. In addition to these factors, other highly conserved DNA sequences have been identified that either enhance or repress the transcription of target genes. These sequences are usually found within the promoter region and are capable of forming a wide variety of unusual DNA structures with simple and complex loop folding patterns. Enhancers function by interacting with transcription factors to form complexes of proteins that allow the transcription machinery to bind more efficiently to the underlying gene. By way of contrast, repression factors function by making it even more difficult for DNA polymerase II to gain access to the gene in question. In this way, genes are expressed or repressed depending on the precise mixture of enhancers, repressors, and transcription factors present in the cell. Hence, the configuration of transcriptional elements and their interplay confer on each gene a unique spatial and temporal transcription program.

Some of these highly conserved repetitive DNA sequences in the promoter regions of genes serve as “phylogenetic footprints,” reliable guides to important regulatory regions ( [Mitchell and Tjian, 1989](#)). As such, their presence is being used as one element of algorithms to identify putative novel genes within the vast sequence data bases generated by the Human Genome Project and related commercial efforts ( [Hardison et al., 1997](#)).

Several classes of DNA-binding proteins exist that regulate the transcription of most genes. The best characterized of these transcription factors contain conserved amino acid regions that in turn bind to specific DNA sequences. For example, homeodomain-containing genes have a highly conserved DNA sequence of 180 nucleotides that encode a polypeptide with a helix-turn-helix structure. This structure contains two a helices that bind in the major groove of the double helix of DNA ( [McGinnis et al., 1984](#)). Other transcriptional factors have different tertiary structures known as *zinc fingers* and *leucine zippers* that also bind to DNA regulatory sequences within the promoter and facilitate the initiation of transcription ( [Landschultz et al., 1988](#); [Miller, 1985](#)).

Another major issue concerns the mechanisms by which genes remain quiescent in some cell lineages or in a repressed configuration after a period of activity ( [Davidson, 1986](#)). This is a critical issue during the complex cascade in time and space of the genes necessary for the formation of the brain. During specific points in development, different genes are sequestered within heritable forms of chromatin complex that preclude transcription. Developmental repression of adult somatic cell genes is remarkably efficient. Ratios of 1:10,000,000 or more have been estimated for the level of globin and growth hormone transcripts in cells not expressing these genes compared with those that are ( [Groudine and Weintraub, 1975](#); [Ivarie et al., 1983](#)).

One way to achieve gene repression is for transcription factors to act on the acetylation of histones. In normal transcription, histone acetylation causes an unwinding of the DNA allowing the transcriptional machinery to bind. Consequently, factors that activate histone acetylases act as gene promoters. Another important mechanism of repression is achieved through “CpG islands” that are present within the 5' control regions of most genes. “CpG islands” refer simply to the presence of regions rich in C+G nucleotides. Intriguingly, the methylation status of these islands has emerged as being an important factor in two neurodevelopmental disorders, Rett's and fragile X syndrome, as well as the molecular events leading to various cancers. (See [Chapter 2](#) for a more detailed examination of these two syndromes.) DNA in somatic tissue is characterized by a bimodal pattern of methylation, which is established through a series of developmental events ( [Greally and State, 2000](#)). Very early in development, most DNA is unmethylated, but after implantation, a wave of *de novo* methylation modifies most of the genome, excluding the majority of CpG islands, which are mainly associated with housekeeping genes. These genomic methylation patterns are broadly maintained during the life of the organism by maintenance methylation and generally correlate with gene expression.

Beyond the complete silencing of genes, “gene dosage” is another crucial issue. Under normal circumstances in adults, there are large classes of genes where only one of the two inherited copies is active. Two prominent examples include X-inactivation and genomic imprinting. X-inactivation is the early coordinated silencing of nearly all genes on one of the two X chromosomes in females. Typically, imprinting also involves the coordinated silencing of contiguous genes in certain chromosomal regions coming either from the father or mother. Although the mechanisms that underlie such events are poorly understood, significant progress is being made to understand these processes at the molecular level ( [Marahrens, 1999](#); [Sleutels et al., 2000](#)).

It is evident from this description that a complex combination of regulatory proteins exists. A different pattern of gene expression emerges depending on which nucleotide sequences are present within a promoter region, as well as which transcription factors, enhancers, or repressor proteins are present within the cell. Moreover, this interplay of regulatory factors determines whether and how much of a specific protein is transcribed.

A species' genetic program unfolds in a largely predictable fashion despite its formidable complexity from the earliest gene expression in the zygote through the entire morphogenesis of the organism. This uniformity may depend, in part, on the presence of redundant pathways and in part from the fact that development proceeds largely in the direction of increasing complexity, but lesser potential. We return to these concepts in the following when we describe exactly how certain growth factors, as well as environmental factors, interplay with transcription factors to either initiate or repress expression of genes within the brain.

## Posttranscriptional Events

Transcribed RNA typically goes through a number of modifications before it is ready for export from the nucleus. These steps include the excision of intervening regions of the message, termed introns, that do not encode for protein. This processing of immature RNA molecules occurs through a mechanism called splicing. The end result is a mature mRNA that contains an uninterrupted sequence of nucleotides that encode the amino acid sequence for the protein. In addition, long stretches of adenine nucleotides, the poly(A) tail, are added to the mRNA message prior to its being shuttled out of the nucleus and into the cytoplasm.

Several splice sites might be present within a gene and allow for different portions of the gene to be brought together. This flexibility in mRNA formation has several important functions. A single gene may produce several nearly identical proteins that differ in certain critical amino acid sequences. The resulting proteins may have different enzymatic functions or unique binding affinities for novel proteins. Alternatively spliced messages are particularly enriched within the central nervous system (CNS), where they are often expressed at different developmental periods.

## Translational Factors

The process of translating mature mRNA molecules into proteins occurs in the cytoplasm. Ribosomes bind to the mRNA messages and initiate a complex series of events, many of which are themselves under regulatory control. Although regulatory mechanisms at this level may seem unnecessary, they do provide a means of rapidly controlling synthesis of gene products by cells.

The stability of a mature mRNA that has entered the cytosol is a critical determinant of how many copies of a protein will be synthesized by the ribosomal apparatus. Certain base sequences in the 3' untranslated region, as well as the poly(A) tail, are thought to influence the stability of many mRNA molecules and their rate of degradation.

## Posttranslational Processing

Once a protein has been formed, it often undergoes further modification. For example, sulfhydryl groups in two cysteine residues may form a covalent bond with each other; this provides stability for the protein, as well as helping to fold it into its final tertiary structure. Several other chemical modifications can occur also, such as the phosphorylation of serine, tyrosine, and threonine residues; glycosylation of the amino acid asparagine; acetylation of the NH<sub>2</sub> terminal amino acids; or hydroxylation of proline and lysine residues. Glycoproteins are formed by the addition of various sugar moieties to the free hydroxyl group of serine or threonine ( [Freifelder, 1987](#)).

The removal of certain amino acid stretches from a protein is another posttranslational process that occurs. One example is the formation of polypeptide hormones and neuropeptides that often involves the cleavage of precursor proteins at specific sites. For example, the processing of prodynorphin molecules leads to a variety of dynorphin moieties and leu-enkephalin. Intriguingly, differential processing of prodynorphin, as well as other precursor proteins, in different tissues is commonplace, so that the form of dynorphin found in the anterior lobe of the pituitary is different from the form of dynorphin found in the neurointermediate lobe ( [Molineaux et al.,](#)



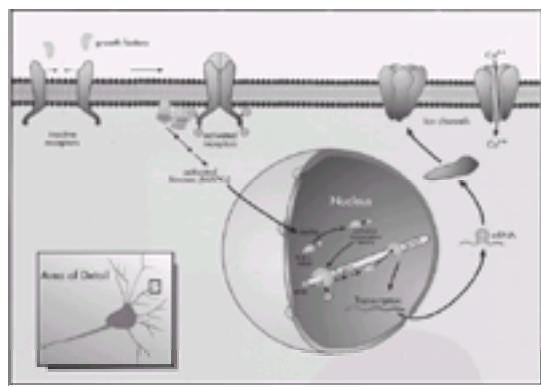
## ENVIRONMENTAL EFFECTS ON GENE TRANSCRIPTION

### Growth Factors

Growth factors have been implicated in a broad range of developmental processes in which cell specification, growth, and survival have to be coordinated across tissues or germ layers. For example, members of the transforming growth factor family, such as the polypeptide, nodal, are necessary for the initial specification of mesoderm within the developing embryo. In contrast, members of the fibroblast growth factor (FGF) family are required for the proliferation of tissues that form the primordia of the placenta and connect the mammalian embryo with its mother. Members of the FGF family are also required during the earliest phases of anterior-posterior patterning of the embryo, possibly through the regulation of downstream homeodomain-containing genes. More recently, basic fibroblast growth factor (Fgf2) has been determined to be necessary for cell proliferation and neurogenesis in the developing cerebral cortex ( [Raballo et al., 2000](#); [Vaccarino et al., 1999](#)). Later, in brain development, growth factors are thought to play a critical role during periods of development when external events and environmental factors influence axonal connectivity, and the development and pruning of synapses, as well as apoptosis (cell death) ( [Klintsova and Greenough, 1999](#)). Thus growth factors are required for multiple steps during development ( [Vaccarino et al., 1999](#)).

Although many different growth factors are produced within the CNS, only minute amounts of these molecules are secreted. The end result is that neurons compete for the small amounts of trophic factors present. The receptors for the various growth factors lie on the outer membrane and bind their specific growth factor. Once bound, a cascade of signals is initiated that promotes their growth and differentiation, as well as the development of synaptic connections and their long-term survival.

Most receptors have two functional domains: an extracellular portion that binds to the signaling molecule, and an intracellular domain that passes along the signal. The binding of a growth factor to its receptor often results in two ligand–receptor complexes coming together on the membrane and association with each other to form dimers ( [Fig. 1.5](#)). The catalytic domains of the receptors are protein kinases, and the dimerization leads to the phosphorylation of each.



**Figure 1.5.** Signal transduction and the action of growth factors. Growth factors generate signals in a cell by binding to specific receptors on the plasma surface and initiating transcription of needed proteins. Two molecules of a growth factor are shown binding to their receptor. The receptors are transmembrane tyrosine kinase receptors that associate with each other after ligand binding and become phosphorylated. In their phosphorylated state, the receptors attract other signaling proteins, including the adapter protein SOS and the enzyme, Ras. The newly formed complex of proteins activates several kinase pathways, one of which is shown here (MAPKs). In this pathway, transcription factors (Elk-1, Jun) are activated by phosphorylation within the nucleus and initiate the transcription of genes that are themselves transcription factors (immediate early, or IE, genes). The IE transcription factors initiate transcription of additional genes, and their mRNA messages are transported back into the cytoplasm and translated into proteins, such as the ion channels shown. In this example, an ion channel is being synthesized and leads to an increased level of the second messenger,  $\text{Ca}^{2+}$ . (Adapted from Vaccarino FM, Lombroso PJ. Growth factors. *J Am Acad Child Adol Psychiatry* 37:789–790, 1998.) (See [color plate](#).)

Several events then occur in rapid succession. A number of proteins are recruited from the cytoplasm to form complexes of signaling proteins at the plasma membrane. These associated proteins are often themselves phosphorylated and thereby activated by the receptors. For example, one of the best studied signaling pathways leads through a series of successive phosphorylations to the activation of a protein called “mitogen activated protein kinase” (MAPK). MAPK is transported to the nucleus where it phosphorylates specific transcription factors and initiates expression of genes important for cell proliferation or differentiation.

In summary, the binding of growth factors to their receptors on the cell surface leads to the rapid transmission of the signal into the neuron. It is not surprising that gain-of-function mutations in some of these receptor proteins disrupt the normal transmission of intracellular signaling, leading to a number of developmental disorders affecting the structure and function of the CNS ( [Passos-Bueno et al., 1999](#)).

### Hormones

Hormones use a related mechanism to transmit signals into cells ( [Evans, 1988](#)). There are many different types of hormones within our bodies. We review one of these that has an important role within the CNS. Hydrocortisone is a glucocorticoid hormone secreted in response to stressful events. Although stress responses are critical for our survival as a species, too much stress has adverse effects on our physiology and may damage neuronal structure and function ( [Post et al., 1998](#)).

Hydrocortisone is synthesized in the cortex of the adrenal gland where it is released into the blood stream. Similar to most steroid hormones, hydrocortisone is a water-insoluble molecule. This fact leads to several important differences between this family of signaling proteins and the growth factors.

Growth factor receptors lie on the plasma membrane where they are able to directly bind their ligand. In contrast, the receptors for most hormones are concentrated in the cytosol or nucleus. Hormones are able to pass directly across the membrane because they are water-soluble molecules and bind to their receptors to initiate specific cascades of signals.

A second difference lies in the fact that hormone receptors can act directly as transcription factors. Prior to binding their respective hormones, the receptors are in an inactive state because they are tightly associated with an inhibitory protein. The inhibitory protein is released when the hormone binds to the receptor and a DNA-binding domain on the receptor–hormone complex becomes unmasked. The complex is now able to interact with specific DNA sequences within the promoter region of genes. In this way, hormone receptors initiate the transcription of genes required by the cell at that moment.

Most hormones exert their effects in two stages. Initially, the hormone receptor induces the transcription of a small number of genes. This effect usually occurs within 30 minutes and is known as the primary hormonal response. Because many of these genes are themselves transcription factors, a cascade of transcription is begun. Steroid hormones thus are able to exert much longer-lasting responses than growth factors. Typically, the immediate effects of growth factors disappear within seconds or milliseconds, although their action can be prolonged by the activation of downstream signaling. In contrast, hormones such as hydrocortisone persist in the blood for hours, whereas the thyroid hormones last even longer and their actions persist for days. The newly synthesized transcription factors activate additional genes in a delayed or secondary hormonal response. A single hormone, therefore, can initiate complex patterns of gene expression.

## GENE AND THE CREATION AND MAINTENANCE OF THE NERVOUS SYSTEM

Although the precise number of genes that regulate the growth and development of the CNS is unknown, some investigators have estimated that at least one-third of the mammalian genome is devoted exclusively to this task ( [Hahn et al., 1982](#)). Recent estimates that there are over 80,000 genes in the human genome come from the human genome sequencing project. Approximately 30,000 of these are specifically expressed within the human CNS. This figure does not include constitutively expressed genes that maintain the basic functions of cellular life, including the regulation of the cell cycle, production of subcellular organelles, and maintenance of



the cellular structure.

The process of development is a heritable feature of all organisms. Genetic factors must contain virtually all of the information necessary to guide the orderly succession of events that will transform portions of the fertilized egg into a fully developed CNS. The morphogenesis of the nervous system involves at least five major processes that are regulated in part by genetic factors: the birth of specific cell types, their migration to their final destination, their growth, the development of neural connections, and cell death (Jacobson, 1970; Vaccarino and Leckman, 1998). A fundamental understanding of these processes would require precise knowledge of: (a) the developmental information relevant to the CNS that is encoded in the human genome, (b) how this information is regulated and utilized in (morphogenetic" time and space, and (c) how the products of these genes endow the differentiating and differentiated cells of the embryonic CNS with their functional characteristics (Davidson, 1986). Although we remain largely ignorant of the critical determinants of these processes, remarkable progress toward understanding them has been made during the past decade (Rubenstein and Rakic, 1999). Genetic studies of the development of the CNS in flies, worms, and other model systems have led the way to many of our most fundamental discoveries.

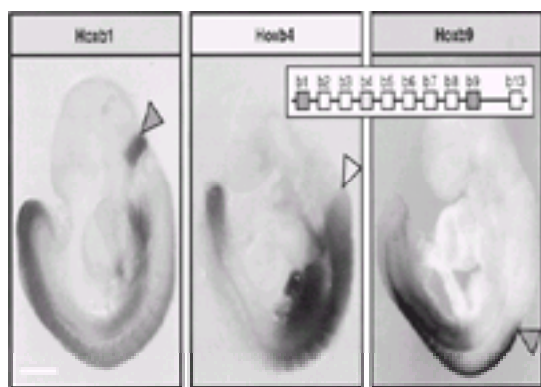
### Spatial Differentiation and Determination of Specific Cell Lineages

Interestingly, RNA synthesized by the mother during oogenesis provides the basis for most of the biosynthetic capacity of early embryos. By the time of fertilization, the egg has already been programmed to ensure the development of spatially distinct embryonic cell lineages. Literally, thousands of mRNAs are formed during oogenesis and remain dormant within the egg until fertilization occurs. One of the reasons for this is that much of the machinery required for the proper transcription, translation, and processing of mature proteins is not yet available in the very early embryo. In some species, the set of maternal genes constitutes the majority of all genomic loci active during ontogeny (Davidson, 1986).

The maternal mRNAs persist until the blastula stage, when they are replaced by transcripts produced by the developing organism. Many of these transcripts are homologous with the maternal transcripts, but some are novel genes not included in the maternal set. An instructive example of this transition from maternal to zygotic transcription concerns several evolutionarily conserved proteins that act as a trigger for cell divisions. In this case, studies have shown that the accumulation of the maternally derived protein cyclin serves as the mitotic trigger for early cellular divisions, whereas later cell divisions depend on the production of a different trigger protein produced by the developing zygote (O'Farrell et al., 1989).

Two other important classes of genes active at this point in development are segmentation and homeotic genes. These genes have been best characterized in *Drosophila*, where they determine the number and polarity of body segments and the identity and sequence of body segments (Gehring, 1987). Many of these genes are evolutionarily conserved and present in the human genome.

Although the precise functions of these genes in mammalian development have not been fully established, they are known to be differentially expressed in the central and peripheral nervous systems, as well as in mesodermal derivatives (Graham et al., 1989). Intriguingly, some of these genes exist in multigene clusters related by duplication and divergence. In some instances, the relative chromosomal position of the gene in a cluster corresponds to the domain of expression in the developing embryo (Fig. 1.6). Many of these genes code for transcription factors. As a consequence, attention has focused on their roles in organizing the differential expression of other genes that are ultimately responsible for establishing distinctive cellular phenotypes. Indeed, their expression patterns provided new evidence for the segmental organization of the rostral CNS (Puelles and Rubenstein, 1993; Rubenstein et al., 1994).



**Figure 1.6.** Homeodomain-containing genes and their expression in the neural axis. Hox gene expression in the mouse embryo. The three panels show lateral views of 9.5 day old mouse embryos stained with antibodies specific for the protein products of *Hoxb1*, *Hoxb4*, and *Hoxb9* genes. (Adapted from Wolpert L, Beddington J, Brockes J, et al.: *Principles of Development*. Oxford, Oxford University Press, 1998, p. 103.) (See [color plate](#).)

Dozens of regulator genes have been identified during the past decade that appear to play crucial roles in the development of the vertebrate CNS. These include the vertebrate homologs of *Drosophila* segment polarity genes *engrailed* and *wingless* (Joyner, 1996). The first forebrain-specific homeodomain-containing genes including the *distalless*, *gastrulation and brain specific gene*, *orthodenticle*, and *emptyspiracle* gene families were isolated and characterized by the early 1990s (Boncinelli et al., 1993; Murtha et al., 1991; Rubenstein and Puelles, 1994; Simeone et al., 1992). Subsequently, vertebrate genes encoding for secreted morphogens such as sonic hedgehog were found to be expressed in stripes along the entire neural axis (Tanabe and Jessell, 1996). The intricate cascade of genetic and molecular events that lead to the development of the mammalian CNS is one of the most exciting stories in the developmental neurosciences.

Not surprisingly, defects in genes controlling neuronal proliferation are being recognized with increasing frequency as a cause of cortical malformations (Raymond et al., 1995). Examples of this phenomenon include tuberous sclerosis (Vinters et al., 1998) and schizencephaly (Brunelli et al., 1996).

Finally, it has also become clear that neurogenesis occurs in adults as well as in the prenatal period. New neurons have been identified that originate in the subventricular zone and then migrate through the white matter to the neocortex, where they extend axons, and become functionally active (Gould et al., 1999).

### Migration of Neurons

The early embryonic development of the nervous system is characterized in part by the migration of populations of neurons. Examples of this phenomenon include the migration of neural crest cells to form elements of the peripheral nervous system (autonomic and sensory ganglia, glial cells, and adrenomedullary cells) and the migration of neurons born within the ventricular zone to their final destination within the cortical laminae. A range of factors mediate these events, and some have been identified, including proteins that contribute to inherent directional preferences, chemotaxis, and differential adhesion of cells as they migrate (Purves and Lichtman, 1985).

In the case of corticogenesis, it has been established that the newly formed glutaminergic projection neurons migrate along radial glial guides that stretch between the ventricular surface and outer cortical surface (Rakic, 1988a) (Chapter 3). It is likely that certain recognition and adhesion molecules are necessary for this migratory process to occur (Hatten and Mason, 1986; Rakic, 1981; Rakic, 1988b). The differential expression for some of these molecules has been established, and genetically mediated defects in their structure and function are now known to result in aberrant migratory patterns (as is seen in the "reeler" and "weaver" mouse mutants) (Rakic and Sidman, 1973a,b). Intriguingly, GABAergic interneurons in the cerebral cortex arise in the primordia of the basal ganglia and migrate tangentially into the cerebral cortex (Ware et al., 1999).

Genetically based abnormalities in the genes that govern neuronal migration have been implicated in a number of human disorders, including X-linked lissencephaly, focal pachypolymicrogyria, and the "double-cortex" syndrome (Gleeson et al., 1998) (Chapter 2). In some instances, the genes responsible for the abnormal migration have not yet been identified (Yoshimura et al., 1998).

### Neural Connectivity and Survival

Developing nerve cells have the remarkable characteristic of being able to maintain contact with literally thousands of other nerve cells by extending cellular

processes over substantial distances. These contacts are of crucial importance in establishing and maintaining the functional integrity of the nervous system. These processes initially develop by way of local extension and retraction of specialized areas on the surface of the neurons called growth cones. A variety of external signals regulate the formation, maintenance, and/or degradation of these neuronal connections, including mechanical guides, differential adhesiveness, the influence of electrical fields, and interaction with gradients of trophic substances ( [Kalil et al., 1986](#); [Purves and Lichtman, 1985](#)).

Once neuronal processes reach their target field, the neurons acquire obligatory trophic dependencies. Target fields contain growth factors such as nerve growth factor that bind to the axons projecting to that field and are retrogradely transported to the afferent cell body. A given target field, however, is able to support only a limited number of neurons, and the “extra” neurons are lost. For example, although nerve growth factor (NGF) does not attract sensory nerve fibers to their target-fields, it is intimately involved in the target-mediated survival of neurons. Indeed, from the first appearance of the neuronal processes in the target-field, there is a marked increase in the rate of transcription of the NGF gene and the amount of NGF in the target-field. There is also a rapid appearance of cell surface receptors for NGF on the sensory neurons (mediated by the transcription of the NGF receptor gene) ( [Davies et al., 1987](#) ). We discuss in the following how these growth factors send their signals from the neuronal surface to the inside of the cell to effect changes in the transcription of specific genes that are required at that moment.

Genetic factors are likely to play an important facilitatory role in neurite outgrowth and synaptogenesis throughout life. The dynamic equilibrium between neurite outgrowth and synaptogenesis versus neurite pruning and synapse withdrawal may well be a crucial mechanism that allows organisms to modify their behavior or “learn” as a result of experience. Although the precise genetic mechanisms that underlie these complex processes are not well understood, one mechanism may involve those genes that regulate the expression of molecules involved in neuronal communication. For example, *in vitro* and *in vivo* studies have indicated that many neurotransmitters and neuromodulators, in addition to their communicative function in mature neural systems, play important roles in the development and maintenance of these systems by promoting or inhibiting the growth of neural processes ( [Lipton and Kater, 1989](#) ).

Recently, a carboxypeptidase A inhibitor, latexin, has been identified that appears to play a role in cortical projection neurons that are fated to establish links between the primary sensory motor cortex and secondary association areas ( [Arimatsu et al., 1999](#) ). A number of mouse mutants are also providing valuable insights into the genes, molecules, and mechanisms that mediate the connection of cortical neurons to cortical and subcortical targets ( [O'Leary et al., 1999](#) ).

## THE ROLE OF EARLY LIFE EXPERIENCE

The development of the nervous system depends on epigenetic and environmental factors, as well as genetic influences. At each level and stage of development, the microenvironments and macroenvironments of the organism play a crucial role. The unfolding of the genetic program, with its complex sequences and patterns of gene expression, depends in large measure on the presence of transcriptional factors in the microenvironment of the nucleus. This means that our ability to separate out the relative contributions of genetic and epigenetic influences is difficult because the genes influence the environment through the production of various proteins and the environment, in turn, alters the expression of genes ( [Fig. 1.7](#) ).

**Figure 1.7.** Gene-environment interactions. (Adapted from Purves D, Lichtman JW. *Principles of Neural Development*. Sunderland, MA, Sinauer, 1985.)

This reciprocal relationship between genes and the environment is played out over the entire course of development. Developmental biologists have long been interested in characterizing the sequence of microenvironmental changes occurring within the CNS that allow for the normal unfolding of these processes. In fact, a great deal has been learned regarding the orderly turning on of various transcription factors that organize the body plan, including the brain ( [Fig. 1.5](#) ). In addition to transcription, a growing list of growth factors and cell surface molecules plays a critical role in the early growth and differentiation of neurons, migration, and survival of neurons by activating signaling pathways that were discussed in the preceding.

This is not to say that external environmental factors can not influence some of these early developmental events. It is well established that drugs, alcohol, altered nutrition (including a lack of oxygen, or hypoxia), and certain viral illnesses disrupt the orderly progression of neuronal growth during critical periods of brain development. However, the brain of a healthy fetus in a “normal” environment will develop because of the directions contained in its genetic code.

The situation changes significantly during the latter part of gestation onward as the CNS becomes functionally active. In humans as well as other mammals, there is a tremendous growth in the number of synaptic connections ( [Bourgeois and Rakic, 1993](#) ). Moreover, as neuronal connections form, the proper ones are strengthened throughout early infancy by the expansion of functioning synaptic contacts. Although neuronal connections can be made throughout life, it is during the early childhood years and again during puberty that the greatest explosion occurs in synapses throughout the nervous system.

A great deal of experimental evidence has indicated that neuronal activity is required for the proliferation of these connections, as well as their later refinement ( [Edelman, 1987](#); [Shatz, 1990](#) ). Neuronal activity is triggered by interactions of the developing organism with its intrauterine and postnatal environments. Ultimately, genes within the brain must be activated while others must be repressed to permit the normal development of axonal growth cones and the elaboration of synapses, as well as the pruning back of unwanted or unnecessary connections. Much of what developmental neurobiologists have learned regarding these processes began with the seminal work of Hubel and Wiesel over 30 years ago. These investigators were interested in determining how the visual cortex develops and organizes itself. The visual cortex is a region of the brain that encompasses approximately 30% of the cortex when its associated regions are included. Their work clearly established that the connectivity of the adult visual cortex is critically dependent on early synaptic input ( [Hubel, 1988](#); [Wiesel, 1982](#) ). Visual experiences are required for the development in the primary visual cortex of what are termed ocular dominance columns. In order to function properly, visual input from the right or left eye must be separated into alternating columns within layer IV. Prior to and immediately following birth, inputs from the eyes are not separated but, rather, make overlapping synaptic contact with neurons in the visual cortex. Once visual activity is initiated after birth, there is a progressive segregation of the visual inputs into orderly columns. This reshaping of the axonal connections within the cortex by visual experiences is referred to as activity-dependent development ( [Purves, 1994](#) ).

What was perhaps unexpected from the work of Hubel and Wiesel was the discovery that the orderly segregation requires visual experience during a critical period of time. Normal ocular dominance columns will not develop if an animal, such as a kitten, is raised for the first 6 weeks with vision permitted only through one eye because the occluded eye will become fundamentally disconnected from the cortex. Consequently, if vision is then restored to the deprived eye, a “catch-up” does not occur, and the failure to develop proper segregated columns becomes permanent. The visual cortex remains abnormally organized throughout the animal's life ( [Katz and Shatz, 1996](#) ).

Similar deficits are seen in humans who do not experience normal visual inputs during their first years of life. The clinical examples most commonly described are the abnormalities that occur occasionally in an infant born with either unilateral or bilateral cataracts. If the cataract is not diagnosed and surgically removed early in life, the child will never develop vision in the affected eye(s). Moreover, there appears to be a critical period in human infants when proper synaptic connections within the visual cortex must be formed. This is in contrast to the situation in adulthood, where cataracts are sometimes present for years; however, once removed, the individual regains visual acuity.

The preceding discussion raises several related issues. One is whether other regions of the brain show a similar reliance on environmental input for their growth and development. The visual system remains the best studied cortical area that has addressed this issue. However, it is clear that other regions of the cortex involved in sensory perception must have neuronal activity to develop proper synaptic outgrowth ( [Katz and Shatz, 1996](#) ). One example comes from the work of investigators interested in what happens to cortical regions after amputation of a finger ( [Merzenich et al., 1983](#) ). The earlier this occurs, the more plastic is the cortex in remodeling. Regions that used to subserve the amputated finger regress and nearby digits expand their representations into the region.

An extreme example of neural plasticity and the capacity for the brain to reorganize connections involves the rewiring of visual projections to the auditory cortex early in life that allows the animal to “see” using its auditory cortex ( [von Melchner and Pallas, 2000](#) ).

Another example relates to how much easier it is to learn a second language early in life. Recent work in this area suggest that other regions of the brain are profoundly influenced by early neuronal activity. Functional magnetic resonance imaging was used to determine the areas of the brain that are metabolically activated during language acquisition in individuals who learn both native and second languages. When a child learns two languages, both languages are



represented in a single language center; however, when an adult attempts to learn a second language, a new language center is established that is clearly distinguishable from the primary language center ( [Kim et al., 1997](#)).

At the macroenvironmental level, manipulation of sensory input can alter the structure and function of cytoarchitectonic areas of the cortex ( [Kalil et al., 1986](#); [Rakic, 1988b](#)). Although these external events are unquestionably translated into internal events that alter the microenvironment and disturb the balance of factors that permit neuronal outgrowth and survival, they underline the exquisite sensitivity of the developing nervous system to its external environment.

The sphere of influences acting on the nervous system continually enlarges during development ( [Purves and Lichtman, 1985](#)). Assuming a “good enough” environment to support development, the cytoplasm of the egg, with its maternally derived biosynthetic apparatus, provides the initial milieu. Later local cellular interactions between neurons and glia and the reciprocal activity of more distant neurons influence the multiple microenvironments of the discrete cell lineages and regulate morphogenesis. Finally, the nervous system is also shaped by wholly external events in the prenatal and postnatal world.

Consistent with this view, evidence for a critical period in the establishment of stress responsivity and maternal behavior has recently been presented ( [Francis et al., 1999](#)). In these animal studies, the level of maternal care as measured by licking and grooming by the mothers profoundly influenced the quality of maternal care offered by the female adult offspring. It also established an enduring pattern of stress response in the pups as they matured. The pups raised by the attentive dams were more likely to show less stress in response to novel environments. These findings are also consistent with other studies of the enduring impact of maternal stress in the perinatal period ( [Ladd et al., 2000](#); [Vallee et al., 1999](#)). Importantly, many of these effects appear to result from environmental and not genetic effects in large measure. It is also likely that alterations in these neurobiological systems may alter an individual's vulnerability to latter psychopathology, including mood and affective disorders ( [Heim and Nemeroff, 1999](#)).

Finally, it is likely that events in early family life, interactions with peers, and educational opportunities shape the course of development just as surely as the developing individual profoundly influences his or her environment. The developmental perspectives of child psychiatry echo in the study of developmental neurobiology.

## FUTURE PROSPECTS

Genetics and the developmental neurosciences are on the threshold of a new era in which the sequence of the human genome will be known and widely available, and the intricate cascade of molecular events that govern brain development will be established. “DNA chips” containing fragments of virtually all the mRNAs coded for by the human genome will also rapidly become available to investigators in commercial, as well as academic settings. Using these tools, animal models of even complex human diseases are likely to be successfully developed.

Although the scientific, information management, and logistical challenges to understanding the complexity of the human genome vis á vis brain development are daunting, they are likely to pale in comparison to the ethical dilemmas to be faced as information about our vulnerability genes becomes accessible. The ethical problems will multiply as commercial firms develop even more potent pharmacologic tools to influence these developing neurobiological systems. We also predict that this new knowledge will emphasize the importance of early life events in shaping the CNS and will point the way to the profound and enduring importance of selective early interventions for families at high risk ( [Harris, 1996](#); [Olds et al., 1998](#)).

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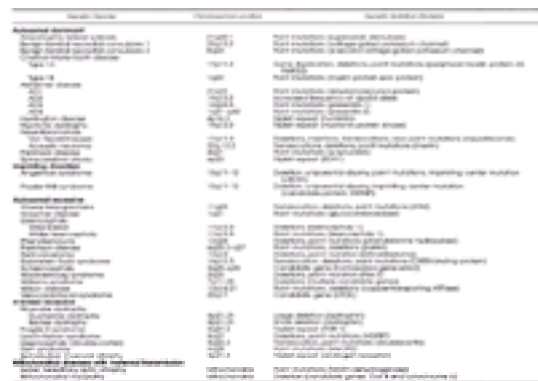
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## 2 MOLECULAR BASIS OF CHILDHOOD PSYCHIATRIC DISORDERS

Paul J. Lombroso, M.D. and James F. Leckman, M.D.

[Rett Syndrome](#)  
[Fragile X Syndrome](#)  
[Lissencephaly](#)  
[Prader-Willi and Angelman Syndromes](#)  
[Williams Syndrome](#)  
[Common Child Psychiatric Disorders](#)  
[Conclusion](#)  
[Chapter References](#)

The past decade has seen remarkable progress in the application of molecular genetic strategies to the study of neurologic and neuropsychiatric disorders ( [Martin, 1989](#)). The approximate chromosomal location for a number of autosomal-dominant, autosomal-recessive, and X-linked recessive disorders has been accomplished ( [Table 2.1](#)). The genes that are actually mutated in many of these illnesses have been identified, and determining the function of both the normal and mutated proteins has begun. This chapter reviews some of the important accomplishments in this area and closes with a brief consideration of the molecular basis of more common child psychiatric disorders.



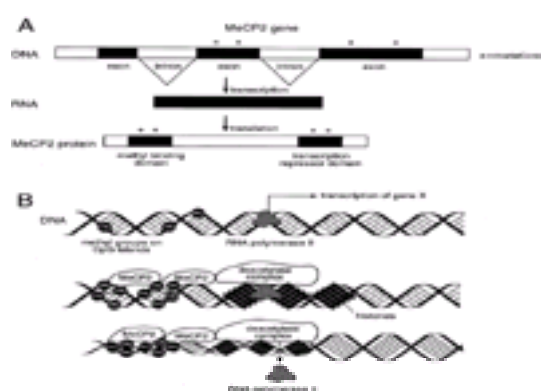
**Table 2.1. Chromosomal Localization and Gene Abnormalities in Selected Psychiatric and Neurologic Disorders**

### RETT SYNDROME

Rett syndrome is a disorder within the autism spectrum ( [Rett, 1966](#)). It has a prevalence of 1 in 10,000. Affected children develop normally and reach developmental milestones within the first year of life; parents are unaware of any abnormalities. One of the first clinical symptoms is the loss of purposeful hand movements. Other clinical findings soon emerge, including loss of speech, growth retardation with microcephaly, ataxia, and a severe disruption of normal cognitive functioning. Clinical symptoms stabilize for the next several decades of life after this initial rapid regression ( [Naidu, 1997](#)).

One unusual feature of the disorder is that females are almost exclusively affected. An early explanation for the preponderance of affected females is that the mutated gene is located on the X chromosome. There are many examples of X-linked disorders in which males die *in utero* because males only have one X chromosome and only a single copy of the gene in question. Females have a second copy of the gene on their other X chromosome, and it appears that transcription of this gene in a significant subset of cells provides some degree of protection during fetal development and early postnatal life. However, symptoms may eventually emerge because haploinsufficiency, a single functional copy of a gene, is unable to confer lasting protection.

The chromosomal location for the gene was facilitated by analysis of affected sisters, and its approximate position was identified at Xq28 ( [Webb et al., 1998](#)). Candidate genes known to be present in this region were carefully studied. A number were systematically excluded as analyses of their nucleotide sequences revealed no mutations compared to the identical sequences from normal individuals; however, a gene was recently discovered that is mutated in several affected individuals ( [Amir et al., 1999](#)). The protein encoded by this gene is methyl CpG-binding protein 2 (MeCP2), and mutations in two of its functional domains have now been characterized ( [Fig. 2.1](#)).



**Figure 2.1.** Rett syndrome. *A:* The *MeCP2* gene consists of three exons separated by two introns. The exons are spliced together to produce a mature RNA message that is translated into MeCP2 protein. The gene has been implicated in Rett syndrome after several mutations were found within the coding regions in a number of patients. These are indicated by asterisks (\*) in the nucleotide sequence and protein. Mutations to date have been found to change the amino acid sequence within two functional domains of the protein. *B:* MeCP2 protein, through one of its functional domains, binds to methylated cytosine nucleotides present in CpG islands that are enriched within regulatory regions of many genes. After binding to DNA, the second functional domain is activated and recruits a deacetylase complex that chemically modifies nearby histone molecules. The chemical modification of histones leads to a further compacting of the DNA into chromatin. The transcriptional machinery, including DNA polymerase II, no longer has easy access to the underlying DNA and is unable to initiate transcription. (Adapted with permission from Lombroso PJ: Rett syndrome *J Am Acad Child Adol Psychiatry* 2000.)

How are mutations in this gene related to clinical symptoms? As discussed in the previous chapter, many genes are specifically expressed in certain tissues and not in others. Of the approximately 45,000 to 100,000 genes on the human genome, only about one-third are expressed exclusively within the central nervous system (CNS). Some of these genes and their protein products are important for normal development during critical periods of brain development. Others are needed only after birth, whereas others must be expressed at all times because they are involved in normal housekeeping functions required of the cell. This illustrates how carefully gene expression must be regulated for normal development and maintenance of tissues to proceed.

The previous chapter reviewed how transcription factors and other regulatory proteins such as enhancers and repressors bind to promoter regions of genes and either initiate transcription of specific subsets of genes or maintain their stable repression. The proteins involved in these regulatory events bind to specific deoxyribonucleic



acid (DNA) sequences within the promoter regions that control gene expression.

The protein that is mutated in Rett syndrome plays a critical role in regulating gene expression. The accessibility of DNA sequences depends to a large extent on the degree of methylation present within the regulatory regions ( [Kass et al., 1997](#)). Methylation is a chemical modification to DNA that occurs when methyl groups are added to cytosine nucleotides. It is particularly prevalent in regions of DNA that contain a high content of cytosine and guanine pairs, so-called CpG islands. Although CpG islands are present throughout the genome, they are most enriched within the promoter regions of genes. In fact, one approach to identifying transcriptional start sites is to look for CpG islands because they are often found immediately upstream of the transcriptional start site.

It was thought that DNA methylation alone was capable of repressing gene expression. DNA polymerase II is the enzyme that transcribes most DNA into RNA. Methylation of CpG islands within promoter regions was initially believed to be sufficient to prevent the enzyme from gaining access to these regions and initiating transcription; however, what actually occurs is somewhat more complicated.

The protein implicated in Rett syndrome, MeCP2, has two functional domains. One end of the protein recognizes methylated cytosines and binds tightly to them. The second domain is then activated and functions by recruiting another set of proteins, the histone deacetylase complex, to the immediate vicinity. Histones are a family of proteins present within the nuclei of all cells. Their role in modifying the secondary structure of DNA has been recognized for some time because they provide a core of protein around which the chromosomal DNA is wrapped ( [Nan et al., 1997](#)). The histone deacetylase complex that has been recruited by MeCP2 chemically modifies histones. The result is a compaction of the DNA surrounding the promoter region such that the transcriptional machinery of the nucleus is no longer able to gain efficient access to the gene. This effectively silences it.

The initial report on Rett syndrome found six distinct mutations among the patients who had a mutation of their *MeCP2* gene ( [Amir et al., 1999](#)). Several of these are missense mutations that replace critical amino acids within the protein. Mutations of this type are located within the methyl-binding domain and disrupt its ability to recognize and interact with methyl groups. The remaining mutations are found in the second functional region, the domain that recruits the deacetylation complex. Two types of mutations are present in this second domain. One is the insertion of a single nucleotide that leads to a shift in the downstream codons. A shift such as this results in the translation of a new amino acid sequence downstream of the point of mutation. The second mutation results in a novel stop codon. This type of mutation leads to the production of a shortened or truncated protein. Each of the mutations results in an impaired or nonfunctional protein ( [Amir et al., 1999](#)).

So far, mutations of the *MeCP2* gene were found in only a minority of Rett syndrome patients. There are several possible explanations for this. Only certain regions of the *MeCP2* gene were actually sequenced in the initial study. The regions sequenced were those that contain the open reading frame or that portion of the DNA that encodes for protein. These regions are often analyzed first because they require far fewer nucleotides to be sequenced compared to an analysis of the entire gene with its multiple introns and regulatory regions. Mutations in open reading frame, therefore, are the first to be detected, although regulatory regions of a gene also may be disrupted by mutations. For example, mutations at a promoter area may interfere with the proper initiation of transcription, whereas mutations in introns may interfere with the proper splicing together of exons by disrupting a splice site regulatory sequence.

*Allelic heterogeneity* refers to the presence of different mutations within a single gene. In the majority of disease-causing mutations studied so far, the same or a very similar clinical phenotype is seen when different functional domains of a single gene are mutated. This type of allelic heterogeneity was demonstrated in the initial study on Rett syndrome ( [Amir et al., 1999](#)). Six distinct mutations were found in different regions of the same gene, and all the patients had the same disorder. It is likely that additional mutations will be found within regulatory sequences of *MeCP2* that result in a similar phenotype. On the other hand, mutations to a single gene occasionally cause very different clinical presentations. For example, several mutations within the receptor for the fibroblast growth factor are responsible for a number of distinctly different skeletal and growth abnormalities ( [Park et al., 1995](#)).

By way of contrast, *locus heterogeneity* refers to mutations in different genes that result in a similar clinical presentation among affected individuals. This can happen, for example, when several proteins are involved in a series of related enzymatic reactions. For example, *MeCP2* encodes for one member of a larger family of proteins. At least two other members have been discovered that also bind to DNA and are involved in the recruitment of the histone deacetylation complex. Each of these proteins is now being examined to determine whether mutations in their genes also cause Rett syndrome. Moreover, as discussed, a number of proteins besides MeCP2 are required to properly repress target genes. Mutations of any of these other genes may result in a related clinical phenotype.

The recent findings with Rett syndrome raise a number of interesting questions. One relates to the predominance of neurologic symptoms in the clinical examination. *MeCP2* is not uniquely expressed in the brain but is found in many other tissues. Nevertheless, symptoms outside of the brain are not a predominant part of the disorder. It appears that the CNS is particularly vulnerable to disruptions of this gene. A similar situation occurs in Huntington's chorea, where neurologic symptoms are also the central part of an illness, although the mutated gene is expressed in many tissues.

A second related question concerns the normal developmental trajectory early in life that precedes the development of symptoms. Much longer delays are seen with a number of neurodegenerative disorders where symptoms are not detected until the fourth or fifth decade of life. One explanation put forward is that toxic compounds are slowly produced over time and must accumulate before neuronal damage occurs. Free radicals are examples of toxic compounds implicated in a number of neurodegenerative disorders, including Huntington's chorea. Normally, enzymes are present that detoxify free radicals within cells. If these enzymes lose their full functional activity through a mutation or are expressed in smaller amounts, then the toxins will accumulate over time and eventually interfere with normal neuronal function.

This may be what happens in the brains of Rett syndrome patients. As discussed, the normal function of the MeCP2 protein is to repress transcription of several additional genes. These downstream target genes are not yet known, but it is reasonable to assume that mutations to *MeCP2* lead to the inappropriate expression of these genes. It is possible that the products of these genes are themselves toxic, or interfere with the normal signaling pathways that function during this developmental period. Neuroanatomic studies might clarify this hypothesis. The literature contains a single autopsy report ( [Belichenko et al., 1994](#)), and abnormalities were detected in pyramidal neurons in layers II–III of the cerebral cortex. They had fewer dendrites than normal and many fewer dendritic arborizations. Additional neuroanatomic studies are necessary to help clarify the neuropathologic process that occurs in Rett syndrome.

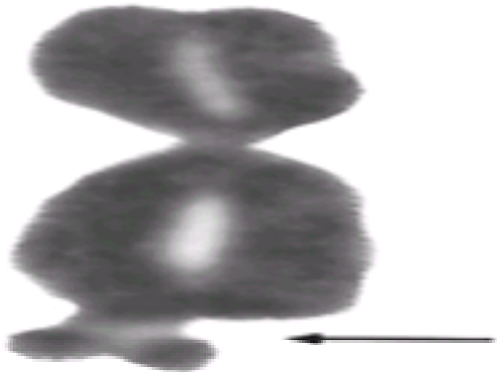
## FRAGILE X SYNDROME

The application of molecular biology to child psychiatric disorders has also advanced our knowledge of fragile X syndrome ( [Nelson, 1995](#)). This disorder is the second most common cause of mental retardation and affects as many as 1 in 750 to 1,000 males and 1 in 500 to 750 females ( [Dykens et al., 1993](#)) ( [Chapter 48](#)).

Children with fragile X syndrome are born with mild to severe mental retardation. Additional clinical symptoms include facial, testicular, and connective tissue abnormalities ( [Baumgardner et al., 1995](#); [Martin and Bell, 1943](#)). Abnormal speech patterns are present in the majority of cases and include echolalia and high-pitched speech, as well as poor articulation, dysfluency, and dyspraxia. There is often gaze aversion among these children, as well as stereotypic behaviors, hyperactivity, and attentional difficulties. Aggressive and self-injurious behaviors are prominent features in some cases ( [Bregman et al., 1987](#)). Although there has been some interest in identifying specific linguistic, cognitive, or behavioral deficits among affected individuals, these have not been found and a wide variation exists in neuropsychiatric results ( [Dykens et al., 1993](#); [Freund et al., 1992](#); [Fryns, 1986](#); [Hodapp et al., 1992](#)).

Fragile X syndrome is transmitted from one generation to the next as an X-linked disorder. It has been known for many years that the phenotype of fragile X syndrome often co-segregates with what appears to be a "fragile" site on the X chromosome. In a certain proportion of cells grown in the absence of the nutrient, folic acid, a break point becomes visible on one of the X chromosomes ( [Fig. 2.2](#)) ( [Lubs, 1969](#)). These findings suggest that the gene or gene(s) involved in the disorder might lie near the disrupted site.

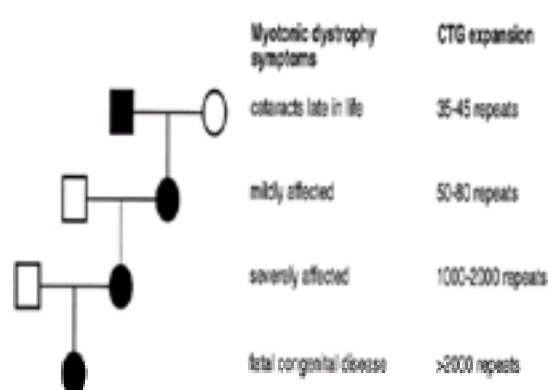




**Figure 2.2.** Fragile X site. The fragile site is shown on an affected chromosome of an individual with fragile X syndrome. (Adapted with permission from Lubs H: A marker X chromosome. *Am J Hum Gen* 21:231–244, 1969.)

Several unusual aspects of the disorder also were noted before the gene was actually cloned. Approximately 20% of the males who carry the abnormal gene are not mentally impaired. If the gene lay on the X chromosome, then why were these individuals not affected? They have no normal copy of the gene to compensate for the mutated gene. These men, called “normal transmitting males,” pass the abnormal gene to their daughters, who typically are also not affected. However, their grandsons are at high risk for the full syndrome. This progression in severity over several generations is known as *anticipation*.

The molecular basis for anticipation is now understood, and relates to the molecular defect found in the affected *FMR-1* gene (Fu et al., 1991). A novel type of mutation has been identified with the isolation of this gene. Triplet repeats refer to any three bases in the normal nucleotide sequence that is repeated several times one after the other. In the case of fragile X syndrome, the three repeated nucleotides are cytosine-guanine-guanine (CGG). Normal individuals have between six to 50 repeats of these bases in their *FMR-1* gene, with 29 repeats being the most frequent number seen (Fu et al., 1991). Affected individuals, however, have a dramatic increase in the number of repeated sequences, with 200 to 1,000 repeats typically. The extreme amplification of this CGG repeat sequence is responsible for the fragile X mutation site in the great majority of cases. Mothers of affected probands often have numbers of CGG repeats that fall in between those seen in normal individuals and those affected with the full fragile X syndrome. They typically have between 50 and 200 CGG repeats (Fu et al., 1991); repeats in this range are called *premutations* (Fig. 2.3).



**Figure 2.3.** Anticipation. Anticipation refers to the increase in severity for a disorder over several generations. In myotonic dystrophy, for example, the increase in symptoms ranges from cataracts late in life, to mild and then more severe muscle disorder, to fatal congenital illness. A corresponding increase is found in the triplet repeat expansion for cytosine-thymidine-guanine (CTG).

Some individuals who carry the premutation of the *FMR-1* gene often have mild cognitive and behavioral symptoms (Hagerman et al., 1996). Heterozygotic fragile X females perform poorly on visuospatial and/or memory subtests (de von Flindt et al., 1991; Kemper et al., 1986). Moreover, magnetic resonance imaging (MRI) and positron emission tomography (PET) analysis have shown premutation female carriers to have significant decreases in total brain volumes, as well as metabolic increases in the hippocampus and cerebellum (Murphy et al., 1999). These cognitive and behavioral symptoms are thought to result from the intermediate length of their CGG triplet repeat. Mothers who have the premutation are at a much higher risk of expanding the triplet repeat in the next generation to produce affected children with the full mutation. Although why it happens remains unclear, the expansion change from premutation to the full mutation is the molecular basis for the phenomenon of anticipation.

In addition to the 200 to 1,000 CGG repeats in affected individuals, other abnormalities in the DNA have been noted. As described, CpG islands are normally modified chemically through a process known as methylation. The triplet repeat expansion produces a large increase in the number of CpG islands. It is not surprising that abnormal methylation patterns are often found in patients with fragile X who have thousands of CGG repeats (Oberle et al., 1991), and these regions become highly methylated. In addition, the extent of methylation correlates directly with the loss of functional protein (Pieretti et al., 1991). The variations in the CGG repeat sequence induce abnormal methylation patterns adjacent to the promoter region of the *FMR-1* gene. The normal transcription of the gene is interfered with, ultimately leading to the absence of functional protein seen in most affected individuals.

What is the normal function of the FMR-1 protein and how does its absence lead to clinical symptoms? Soon after the gene was isolated in 1991, researchers noticed that the protein contained two domains with a highly conserved amino acid pattern. These domains are homologous to a motif found in other proteins known to bind tightly to ribonucleic acid (RNA) molecules. These proteins are called RNA-binding proteins and are often involved either in processing messenger ribonucleic acid (mRNA) transcripts or translating these transcripts into proteins (Eichler et al., 1993; Siomi et al., 1993, 1994). A mutation in an RNA-binding protein would seriously impair the ability of cells to produce mature messages or translate those messages into protein.

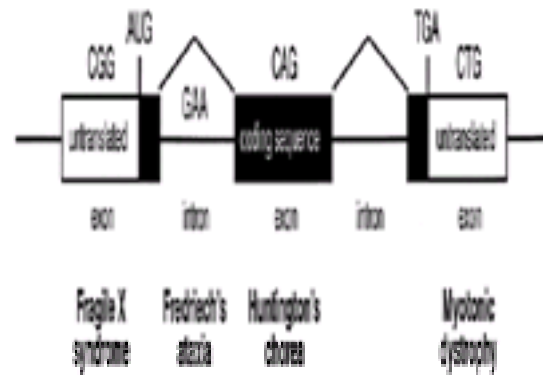
Knowledge of the intracellular location of the FMR-1 protein has extended our understanding of its normal function. The fragile X protein binds to ribosomes, the organelles within cells that are made from ribosomal RNA and various RNA-binding proteins and function as factories that translate mRNA transcripts into proteins (Khandijian et al., 1996). The fragile X protein is normally found in cells that produce large volumes of protein for export, such as neurotransmitter-producing neurons and androgen-producing testicular cells.

In the majority of cases, fragile X syndrome is caused by a triplet repeat expansion; however, any mutation that disrupts the functional activity of the FMR-1 protein could reasonably lead to a similar clinical syndrome. This was in fact described in a fragile X patient who did not have the expected triplet repeat expansion, but instead had a point mutation that changed a single highly conserved amino acid normally present within one of its functional domains, the RNA-binding motif (De Boulle et al., 1993; Musco et al., 1996; Siomi et al., 1994). The domain is critical for the proper function of FMR-1 protein, and mutations in this domain interfere with its ability to bind RNA molecules and to process them correctly. It is another example of allelic heterogeneity, where mutations in different parts of a single gene lead to the identical phenotype.

Studies of the pattern of expression of the *FMR-1* gene during development show the areas of the brain that normally have the highest level of expression. These include the basal forebrain and hippocampus (Abitbol et al., 1993). Both are involved in short-term memory and sequential processing of information, and are affected in some neurodegenerative disorders, such as Alzheimer's disease. The results of these developmental expression studies are consistent with both clinical and phenomenologic investigations that have demonstrated the cognitive, speech, and language strengths and weaknesses seen in fragile X syndrome. In addition, clinical neuroimaging studies have detected age-related volumetric changes in fragile X individuals in the cerebellar vermis, fourth ventricle, and hippocampus (Mostofsky et al., 1998a,b; Riess et al., 1994). Finally, animal models, such as knockout mice, have been developed by disrupting the normal expression of *FMR-1*. Some of the clinical features seen in fragile X syndrome are present in these knockout mice, setting the stage for even more detailed examination of the

neurobiological consequences of the abnormal *FMR-1* gene ([Dutch-Belgian Fragile X Consortium, 1994](#)).

Expansion of the *FMR-1* gene through triplet repeats was originally thought to be a genetic anomaly unique to fragile X syndrome. More than a dozen other triplet repeat disorders have now been identified ([Fig. 2.4](#)), including Huntington's chorea, Friedreich's ataxia, and myotonic dystrophy ([Caskey et al., 1992](#); [Nelson and Warren, 1993](#); [Warren, 1996](#)). The phenomenon of anticipation was apparent in most of these disorders but early investigators dismissed it as the result of ascertainment bias. The discovery of triplet repeat expansions, however, provides the molecular explanation for this phenomenon.



**Figure 2.4.** Triplet repeat disorders. A hypothetical gene is shown prior to the splicing together of exons and the removal of introns to produce a mature RNA message. Triplet repeat expansions have been discovered in all regions of a gene. In fragile X syndrome, the cytosine-guanine-guanine (CGG) repeat lies in the 58 untranslated region immediately adjacent to the promoter region. The expansion to 1,000 to 2,000 repeats leads to abnormal methylation patterns and disrupts normal transcription of the gene. In myotonic dystrophy, the cytosine-adenine-guanine (CAG) repeat is found at the other end of the gene within the 38 untranslated region. It is believed that the expansion there results in an unstable mRNA prone to degradation, as well as perhaps affecting the transcription of a second nearby homeobox gene. In Huntington's chorea, the triplet repeat expansion occurs in the open reading frame of the gene and leads to the inappropriate incorporation of an amino acid, in this case glutamine, within the protein sequence. Triplet repeat expansions have also been found in introns, where they presumably interfere with the proper splicing of the message. Friedreich's ataxia is an example of this type of expansion.

The degree of anticipation can be quite remarkable ([Fig. 2.3](#)). In myotonic dystrophy, for example, expansions that are just above the threshold for disease may only result in individuals who develop cataracts late in life. Several generations later, descendants have the full expansion with long repeats and fatal congenital illness ([Furuta et al., 1992](#)). Interestingly, anticipation has been described in families with schizophrenia and bipolar disorder, and some investigators believe triplet repeat expansions may account for a subset of affected patients ([McInnis et al., 1993](#); [Morris et al., 1995](#); [O'Donovan et al., 1995](#)).

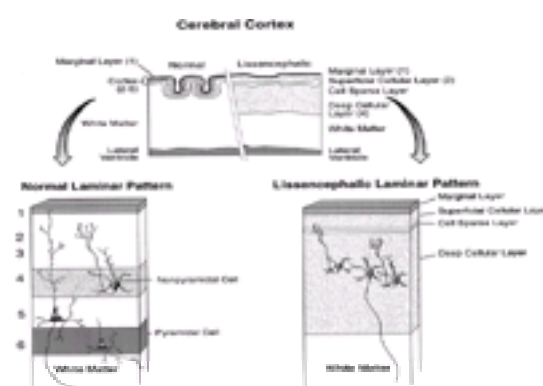
## LISSENCEPHALY

The cerebral cortex is formed of billion of cells that are born during just a few months of embryonic life by a small population of progenitor cells. The cells that are born must differentiate and migrate to reach their final resting place in the appropriate layer of the cortex. These events are discussed at greater length in the following chapter.

However, a number of disorders are caused by mutations in genes whose proteins are required for the orderly progression of events from birth, differentiation into appropriate neuronal subtypes, migration, and eventual elaboration of synaptic connections.

Neurogenesis occurs in a region called the ventricular zone, which lies adjacent to the lumen of the neural tube. Although neurogenesis primarily occurs during the development of the brain *in utero*, some degree of neurogenesis occurs throughout life ([Gould and Tanapat, 1999](#)). In the cerebral cortex, neurons must travel through layers of earlier born cells to reach their final destinations. Those born at later periods have more complex migratory routes. Many employ a guidance system composed of radial glial cells ([Rakic, 1972](#)). The radial glial cells are thought to send a process that spans the developing cortex and serves as scaffolding along which certain neurons migrate. We believe that chemotactic signals are produced that keep the neurons moving along the radial glial process. When it reaches its final destination, other signaling molecules are produced that instruct the neuron to stop migrating, enter the proper lamina, and begin to elaborate its processes ([Rakic et al., 1994](#)).

Disruption of the normal migration of neurons causes several human disorders. The most common of these are the lissencephalies that consist of several syndromes: isolated lissencephaly (ILS), Miller-Dieker syndrome (MDS), and X-linked lissencephaly ([Dobyns and Truwit, 1995](#)). In each, the disruption to the normal neuronal migration is reflected in a smooth cortical surface that lacks the normal pattern of gyri and sulci. In addition, although the cerebral cortex normally has six layers, cortices of affected individuals have only four ([Fig. 2.5](#)).



**Figure 2.5.** Lissencephaly. The normal cerebral cortex is a highly organized structure, and its six layers are shown on the left (1–6). In contrast, the lissencephalic brain lacks the normal pattern of sulci and gyri, and there are only four layers. (Adapted with permission from Reiner O, Lombroso PJ: Lissencephaly. *J Am Acad Child Adol Psychiatry* 37:231–232, 1998.)

In 1993, a large deletion on chromosome 17 was found in two patients with MDS. The search then began to clone the gene responsible for the disorder, and it was soon isolated and termed *LIS-1* ([Reiner et al., 1993](#)). This gene and genes on either side of it were absent owing to the deletion and is another example of a contiguous gene syndrome. The absence of several genes is believed to be responsible for the more severe MDS phenotype, which also has numerous congenital abnormalities. Point mutations in the same *LIS-1* gene were soon found in other affected individuals. These individuals had the milder form of lissencephaly (ILS).

How does a mutation in *LIS-1* cause the observed cortical abnormalities? *LIS-1* encodes for a regulatory protein that controls the activity of a second protein, termed platelet activating factor (PAF)-acetyl hydrolase ([Hattori et al., 1994, 1995](#)). PAF acts as a signaling protein that binds to the surface of neurons. A cascade of signals is initiated required for the normal migration of neurons ([Reiner et al., 1995](#)). *LIS-1* is expressed at its highest levels in the developing cortex, consistent with the protein's putative role signaling. Exactly how mutations in *LIS-1* interfere with normal migration is not well understood, although one model suggests that a disruption occurs to cytoskeletal proteins that need to rearrange themselves at the growing tip of the migrating neuron ([Sapir et al., 1999](#)).

A second disorder that affects neuronal migration is termed subcortical band heterotopia ([Walsh, 1999](#)). This disorder, also known as X-linked lissencephaly or double cortex, is associated with mental retardation and epilepsy. The histologic findings are made of bilateral bands of gray matter consisting of disorganized neurons present in the central white matter between the cortex and ventricular wall. The degree of mental retardation is directly related to the thickness of the extra



neuronal tissue.

The gene that causes double cortex was recently cloned ([des Portes et al., 1998](#); [Gleeson et al., 1998](#)). The gene encodes for the protein double cortin (*DCX*). Structurally, this protein is highly homologous to a family of kinases, called calcium calmodulin-dependent kinases. Although its exact role in neuronal migration remains to be clarified, recent studies indicate that it binds to and appears to stabilize cytoskeletal proteins required for the normal movement of neurons ([Francis et al., 1999](#); [Gleeson et al., 1999](#)).

It is interesting to note that 20% of individuals with lissencephaly have no detectable mutation in *LIS-1*, whereas a similar number of individuals with subcortical band heterotopia have no detectable mutation of *DCX*. It is possible that additional studies will detect novel mutations within these genes; however, it is likely that different genes are mutated and lead to similar cortical abnormalities—another example of locus heterogeneity.

## PRADER-WILLI AND ANGELMAN SYNDROMES

Half of our 46 chromosomes derive from our mothers and the other half from our fathers. For years, it had been assumed that genes that lay on either chromosomal pair were equivalent and that each produced the same amount of functional protein. Mutations that lay in one gene often could be overcome through the actions of the normal protein derived from the homologous gene.

This is an accurate description of the molecular events for the majority of genes; however, recent studies over the past decade have demonstrated that some gene pairs are not functionally equivalent. Instead, the production of functional proteins depends on whether the gene that encodes it lies on the chromosome derived from the mother or the one from the father. In some cases, the maternal gene is expressed; in others, the paternal gene. This phenomenon is termed *genomic imprinting*.

For the majority of disorders, mutations within a gene change the specific nucleotide sequence and thereby disrupt protein expression. For a smaller group of disorders, factors other than the underlying nucleotide sequence determine whether the DNA produces useful patterns of gene expression. These factors are known as *epigenetic* phenomena to distinguish them from events that have an effect on the actual nucleotide sequences. We have discussed some epigenetic factors earlier in our discussions of Rett syndrome and fragile X syndrome. In these disorders, the extent of chromatin packaging or DNA methylation influences gene expression.

Approximately 30 genes have been discovered to date that are imprinted. Many of them play a key role in the growth and differentiation of various tissues. Disruption of these genes is implicated in a number of cancers and developmental disorders. These advances are reviewed in the following with an emphasis on two developmental disorders caused by defects in the imprinting mechanism—Prader-Willi and Angelman syndromes.

Prader-Willi syndrome (PWS) is a rare disorder with a prevalence of 1 in 10,000 births. A constellation of symptoms arises shortly after birth ([Prader et al., 1956](#)). Infants are often hypotonic and fail to thrive; however, their dietary habits change within the first year or two of life, and these individuals typically become hyperphagic and obese ([Holm et al., 1993](#)). They are often mildly to moderately mentally retarded and have a number of additional behavioral problems that include temper tantrums, aggressive behaviors, and obsessive-compulsive symptoms outside of the compulsive food-related behaviors ([Dykens et al., 1996](#); [Dykens and Cassidy, 1995](#); [State et al., 1999](#)).

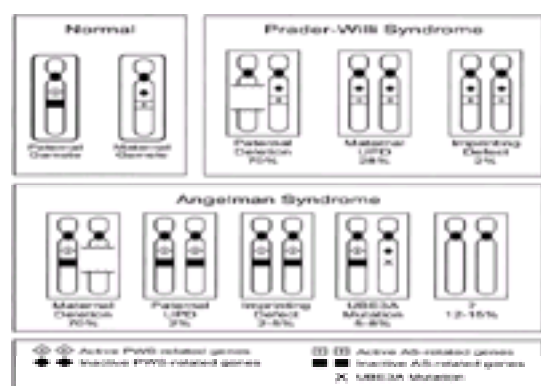
Angelman syndrome is also a relatively rare disorder with a prevalence of 1 in 10,000 births. Individuals with the disorder are often hypotonic as infants and then develop motor delays and moderate to severe mental retardation. They have a characteristic facies with a large mandible with an open-mouth expression, abnormal gait, and puppet-like limb movements; they rarely develop speech. Epilepsy develops soon after birth and affected individuals have an abnormal electroencephalogram ([Angelman, 1965](#)).

By the mid-1980s, it was known that both disorders are caused by a deletion on chromosome 15 (15q11–q13). Cytogenetic and molecular techniques show that the same region is deleted in individuals with each syndrome. This result was somewhat puzzling because the disorders are very different, yet the identical deletion seemed to cause PWS in some cases and Angelman syndrome in others ([Ledbetter et al., 1981](#); [Magenis et al., 1987](#)).

It was then discovered that the clinical symptoms depend on which parent donates the deleted chromosome ([Cassidy and Schwartz, 1998](#)). The deletion derives from the father in most cases of PWS. In contrast, most individuals with Angelman syndrome have a deletion on the chromosome that derives from the mother.

A further clarification of the underlying mechanism came when the region for each disorder was pinpointed on chromosome 15. The section of DNA responsible for PWS is distinct but very close to the area responsible for Angelman syndrome. Part of the puzzle was, thus, clarified. A large deletion may occur that spans both regions. The child will develop PWS only if the deletion occurs on the paternal chromosome, and Angelman syndrome if the deletion occurs on the maternal copy.

Chromosome 15 contains two nearby regions that are differentially imprinted. Among unaffected individuals, genes within the PWS region are expressed on chromosomes that derive from the father. Immediately downstream is a second set of genes within the Angelman-critical region. These genes are imprinted and not expressed on the paternal chromosome. The opposite expression pattern is found on the maternal chromosome. Genes within the Angelman region are expressed, whereas genes within the PWS region are imprinted ([Cassidy, 1997](#)) ([Fig. 2.6](#)).



**Figure 2.6.** Imprinting in Prader-Willi and Angelman syndromes. Imprinting refers to the silencing of certain genes. It is a stable and reversible event that depends on the parental origin of the chromosome on which the gene lies and results in repression of a gene. For some genes, the paternal gene is silenced, whereas for other genes, it is the maternal gene. On chromosome 15, two adjacent regions are imprinted. In the normal situation (shown in the left, top panel), genes in the Prader-Willi syndrome (PWS) region are expressed on the paternal chromosome, whereas genes in the Angelman region are imprinted. The opposite situation exists on the maternal chromosome: Genes in the PWS region are repressed, whereas genes in the Angelman region are expressed. Three mechanisms for developing PWS are shown, whereas four mechanisms have been discovered for Angelman syndrome (AS). The key at the bottom indicates genes, imprinting patterns, and mutations in both disorders. The relative proportion of each type of genetic abnormality in each syndrome is also shown. UPD, uniparental disomy. (Adapted with permission from Everman D, Cassidy S: Genomic imprinting: Breaking the rules. *J Am Acad Child Adol Psychiatry* 39:386–389, 2000.) (See [color plate](#).)

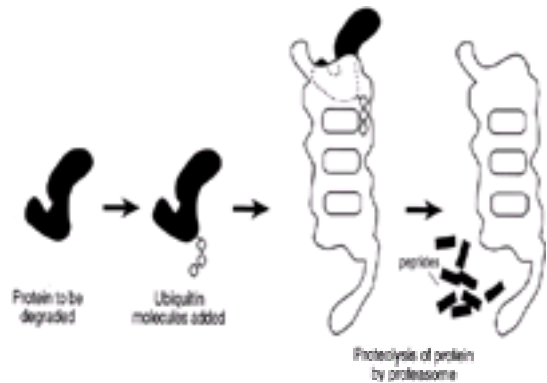
Deletions are responsible for approximately 70% of cases of both PWS and Angelman syndrome. A second mechanism exists and provides further insight into disorders caused by disruptions of the normal imprinting mechanism. Both illnesses also may be caused when children receive two copies of this region of chromosome 15 from one of their parents. This unusual mechanism is called uniparental disomy (UPD). It occurs after an initial trisomy event in which two chromosomes from one parent are inappropriately passed along with one copy of the chromosome from the other parent, resulting in a total of three (rather than two) chromosomes. One chromosome is lost during gamete formation. If the initial trisomy involves two maternal and one paternal chromosome, then loss of the extra paternal copy would result in two maternal chromosomes, or maternal UPD. If the initial trisomy involves two paternal and one maternal chromosome, then loss of the extra maternal copy would result in paternal UPD.

Maternal UPD is a second mechanism by which PWS occurs ([Nicholls et al., 1989](#)). In this situation, the genes that lie within the PWS region are present but on two maternal chromosomes. In this case, all the genes within the critical region are imprinted and no functional proteins are produced. Paternal UPD represents the

opposite situation. PWS genes are present on both chromosomes and both are expressed. However, the genes that lie immediately adjacent and within the Angelman region are imprinted; therefore, even though there are two copies, both are repressed and no protein is expressed.

A third mechanism that leads to these disorders involves a region of DNA called the imprinting center. This center controls imprinting by regulating the extent of methylation and chromatin compaction for hundreds of kilobases of DNA on either side. It appears that one center determines the state of imprinting within both the PWS- and Angelman-critical regions. Mutations within the imprinting center have been discovered that lead to inappropriate imprinting or a lack of imprinting where it should normally occur. These types of mutations cause PWS or Angelman syndrome in about 2% to 3% of affected individuals ( [Ohta et al., 1999](#)).

Finally, a fourth mechanism exists that causes Angelman syndrome in approximately 25% of the cases. This is a mutation within a single gene lying within the Angelman-critical region called *UBE3A* ([Kishino et al., 1997](#); [Matsuura et al., 1997](#)). The protein encoded by this gene normally regulates the life span of other proteins within the cell by regulating their degradation or proteolysis ( [Fig. 2.7](#)).



**Figure 2.7.** Angelman syndrome. It is critical to remove unwanted proteins from within cells before they disrupt cellular metabolism. Several steps are involved in this process and lead to the addition of one or more ubiquitin molecules to proteins targeted for degradation. The addition of ubiquitin to the protein serves as a signal to the proteasome, an organelle that breaks down proteins and serves as the cellular equivalent of a garbage disposal for a cell's unwanted proteins. (Adapted with permission from Lombroso PJ: Genetics of childhood disorders: XVI Angelman syndrome. *J Am Acad Child Adol Psychiatry* 2000.)

Certain proteins must be quickly degraded and removed from the intracellular environment to ensure proper cell function. These include signaling proteins (such as neurotransmitters) in which rapid turnover permits repeated signaling. It is also critical to remove certain enzymes that have become damaged before they interfere with normal intracellular signaling pathways. A small molecule is added to many proteins destined for degradation. This molecule, ubiquitin, acts as a flag to target the protein for degradation by other chemicals. The *UBE3A* gene is required in this process. It is one of several proteins that are needed in sequential order to attach ubiquitin molecules to the target proteins. Intracellular organelles called proteasomes recognize ubiquitinated proteins, bind to them, and activate proteases that cut the protein into its constitutive amino acids. When this protein is mutated and unable to function properly, it is thought that an inappropriate accumulation of proteins occurs within CNS neurons.

Several enzymes in addition to *UBE3A* are required in the series of enzymatic reactions that ends with the addition of ubiquitin molecules to proteins targeted for destruction. It is possible that mutations in these other genes will be found and lead to similar clinical problems. These mutations would represent other examples of locus heterogeneity in which mutations in distinctly different genes produce the same phenotype. A number of laboratories are actively investigating this possibility. On the other hand, no single gene mutation has yet been found in patients with PWS, and it remains likely that deletions of several genes within the critical PWS region are necessary for the disorder.

One final note should be made regarding Angelman syndrome. One of the genes within the Angelman region encodes for a subunit of the GABA<sub>A</sub> receptor ([DeLorey et al., 1998](#)). The absence of this gene and a related disturbance in GABA transmission is believed to be responsible for the seizure disorders present in individuals with the full deletion syndrome. As expected, individuals with a point mutation within only the *UBE3A* gene do not have epilepsy.

## WILLIAMS SYNDROME

Deletions that span a number of genes and cause a constellation of symptoms are called *contiguous gene syndromes*. Williams syndrome is one such disorder caused by a deletion on chromosome 7. This developmental disorder is characterized by distinct facial features, a variable degree of mental retardation, cardiovascular disease, and a very distinctive cognitive profile ( [Pober and Dykens, 1996](#)). The cognitive problems consist of visual-spatial deficits, which are difficulties in visualizing a complete picture. Affected children are unable to integrate the parts of a picture into a whole pattern ( [Ewart et al., 1993](#)). Interestingly, these children frequently exhibit strengths in other cognitive areas, including verbal skills. Indeed, some elements of their speech are normal, such as the quality of their vocabulary, auditory memory, and social use of language. Moreover, many patients sing or play musical instruments with considerable talent. The disorder, first described by Williams in 1961, has a rather low prevalence of 1 per 20,000 live births, but remains a topic of considerable interest because of the striking disparity in cognitive strengths and weaknesses ( [Bellugi et al., 1990](#); [Wang et al., 1995](#)).

This disparity raises the possibility that the gene(s) responsible for Williams syndrome influences the development of specific cortical regions and related cognitive abilities. This is in contrast to other forms of mental retardation, such as fragile X syndrome, where a more uniform depression is seen across many cognitive skills. As such, Williams syndrome has the potential to help identify factors that are important for acquisition of cognitive abilities during normal development.

Imaging and neuropathologic studies have helped clarify some of the underlying neuropathologic mechanisms in Williams syndrome. [Jernigan and Bellugi \(1990\)](#) conducted MRIs on IQ- and age-matched subjects with Williams and Down syndromes. The cerebral cortices of both groups showed an overall decrease in volume. Cerebellar size, on the other hand, differentiated the two conditions. It was normal among the Williams syndrome subjects but significantly hypoplastic among the Down subjects.

Jernigan and coworkers extended this study by focusing in greater detail on the morphologic abnormalities in the cortices of Williams subjects ( [Jernigan et al., 1993](#)). The overall volume of the cerebral cortex was once reduced compared to normal subjects, but there was a relative sparing of the frontal areas and limbic structures, as well as a greater degree of hypoplasia in more posterior cortical structures. Different cortical regions are apparently affected to different degrees in Williams subjects.

Additional morphologic data comes from the autopsy of a Williams subject. Galaburda and colleagues found abnormal organization among the neurons in posterior cortical regions (area 17), an increase in cell packing density throughout cortical regions, and abnormal neuronal clustering ( [Galaburda et al., 1994](#)). It is a single autopsy case, however; additional work is necessary to clarify the underlying pathologic process.

Taken together, these findings suggest that abnormal, nonuniform development of the cerebral cortex may be responsible for the nonuniform cognitive findings that characterize these patients. The relatively intact cognitive skills related to affective recognition, face processing, and linguistic skills may be a consequence of the relatively normal development of limbic and frontal cortices and perhaps the relatively normal cerebellar structures. Poor functioning in visuospatial skills may be owing, at least in part, to the abnormalities in the posterior cortical structures, such as the parietal and occipital cortices known to be involved in visual processing. It is reasonable to suggest that the gene or genes affected in Williams syndrome may have their greatest impact on the development or normal functioning of more posterior regions of the cerebral cortex.

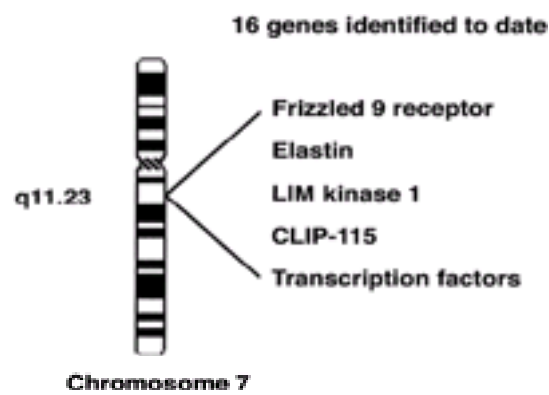
Nearly all patients with Williams syndrome have a large deletion of a segment of chromosome 7 ( [Lowery et al., 1995](#); [Nickerson et al., 1995](#)). A number of researchers note that the cardiovascular symptoms seen in many patients co-segregate with a gene in the deleted region called elastin. Elastin is a major component of skin, blood vessels, and lung tissues. The haplodeficiency caused by the deletion of elastin is likely to cause the vascular abnormalities and characteristic facies in these patients ( [Lowery et al., 1995](#)). Elastin, however, is not detectable in fetal or adult nervous tissue and probably does not contribute to the cognitive abnormalities



(Frangiskakis et al., 1996).

Tassabehji and colleagues (1996) were the first to report the deletion of a second gene in 20 out of 20 Williams syndrome patients. The sequence of this gene is nearly identical to a previously characterized gene called *LIM kinase 1*. Because it is expressed in high concentrations in the brain (Mizuno et al., 1994; Proschel et al., 1995), mutations in this gene are being searched for and are possible candidates responsible for the cognitive deficits seen in Williams syndrome patients.

For a brief period, it was thought that elastin and LIM kinase 1 were the only genes within the deleted region. It is now known, however, that at least 16 genes are present within the Williams syndrome critical region (Fig. 2.8). In addition to elastin (Ewart et al., 1993; Nickerson et al., 1995) and the LIM kinase 1 (Frangiskakis et al., 1996; Tassabehji et al., 1996), other genes include syntaxin 1A (Osborne et al., 1997), CLIP-115 (Osborne et al., 1996), and Frizzled 9, a receptor for Wnt signaling (Wang et al., 1997), the replication factor C subunit (Peoples et al., 1996), and the general transcription factor 2I (Perez Jurado et al., 1998). Several laboratories are currently investigating the function of numerous other genes.



**Figure 2.8.** Williams syndrome. The deletion at 7q11.23 in patients with Williams syndrome contains at least 16 genes. Several of these genes are expressed within the central nervous system. These are candidate genes for contributing to the neuropsychiatric symptoms observed in Williams subjects. When several genes contribute to the expression of a disorder through their deletion, the illness is termed a contiguous gene syndrome.

## COMMON CHILD PSYCHIATRIC DISORDERS

Child psychiatry has yet to establish the molecular basis of many of the most common child and adolescent mental disorders. The reasons for this failure lie beyond the scope of this chapter, but genetic complexity is one culprit. Many of the disorders described in the preceding exhibit mendelian patterns of inheritance, such as the X-linked transmission of Rett and fragile X syndrome; however, pedigree studies of most common child and adolescent mental disorders fail to show such a pattern of vertical transmission across generations. The presence of non-mendelian patterns of transmission does not necessarily imply that genetic factors are unimportant, only that their role in the transmission and expression of disease phenotypes is complex. Examples include polygenetic transmission in which a multiplicity of genetic and environmental factors is causative. Height and intelligence are good examples of polygenetic traits and it may be that some forms of childhood-onset anxiety disorders and attention deficit hyperactive disorder will fall into this category similarly. Interestingly, the alleles that contribute to the vulnerability to develop these disorders may be common in the population and be seen as normal variants when they act in concert with other vulnerability alleles or adverse environments that they produce the syndrome in question.

Understanding the genetics of complex disorders is relevant to other fields of medicine and significant progress has been made in areas such as breast cancer and hypertension (Lifton, 1996; Miki et al., 1994) and our field is poised to take advantage of their success. If child psychiatric disorders follow in a similar course, we can expect that in rare instances single gene mutations will be responsible for common disease phenotype. Such observations will then set the stage for animal studies that will serve a valuable heuristic role in elucidating additional genes and relevant neurobiological pathways.

One example is resistance to thyroid hormone (RTH) and attention deficit hyperactivity disorder (ADHD). RTH is a heritable condition defined by normal or elevated levels of thyroid-stimulating hormone (TSH) in the presence of high levels of serum triiodothyronine and thyroxine, and resistance of tissues to the actions of thyroid hormone (Refetoff et al., 1993). RTH is caused by mutations in exons 9 and 10 of the *thyroid hormone B receptor gene (TRB)*, which codes proteins in the T3-binding domain of the thyroid hormone receptor (Parilla et al., 1991). ADHD is a common symptom of RTH (Hauser et al., 1993). Further, transgenic studies of mice bearing the human mutant *TRB* gene show that they have similar phenotypic features as children with ADHD, including motoric hyperactivity and impaired learning (McDonald et al., 1998). More extensive studies of the role of the thyroid hormone receptor in mediating attentional mechanisms over the course of development are needed and may lead to more profound insights into the molecular basis of ADHD.

## CONCLUSION

Advances in genomics, molecular genetics, and developmental neuroscience provide an impressive array of accomplishments that are laying the foundation for a deeper understanding of the biological basis of some childhood onset neuropsychiatric disorders. Success in these areas will likely herald success in other rare diseases, such as autism and other pervasive developmental disorders. Therapeutic advances also can be anticipated as therapeutic agents are developed that specifically target the molecular and cellular consequences of specific genetic mutations. Perhaps even the promise of gene therapy can be realized for some of the single gene conditions. Nevertheless, the road ahead, particularly for common disorders, will not be an easy one given the complexities involved and the crucial role of the environment in shaping and reshaping the CNS within the constraints of our genetic endowment.

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## 3 GENESIS OF NEOCORTEX IN HUMAN AND NONHUMAN PRIMATES

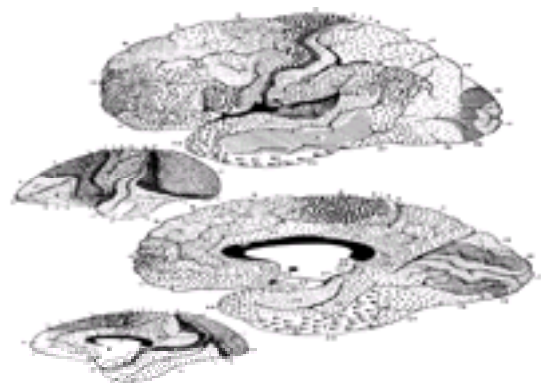
Pasko Rakic, M.D., Ph.D.

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The human cerebral cortex comprises about two-thirds of the neuronal mass of the brain and contains almost three-quarters of all our synapses. It is also the structure that most distinctively sets us apart from other species. Therefore, the principles governing the development of the cerebral cortex may provide the key to explaining our cognitive capacity, as well as the evolution of human intelligence and creativity. Modern developmental neurobiology provides insight into how this complex structure may have evolved, how it develops, and how this finely tuned process may go astray in various neuropsychiatric disorders.

One of the most prominent features of the cerebral cortex in all species, particularly primates, is its parcellation into distinct laminar, radial, and areal domains ([Eccles, 1984](#); [Goldman-Rakic, 1987a](#); [Mountcastle, 1997](#); [Rakic and Singer, 1988](#); [Szentagothai, 1978](#)). However, although the surface of the neocortex has expanded enormously during phylogeny, its thickness and basic cellular organization have undergone relatively minor change ([Rakic, 1995b](#)). Comparison of the cytoarchitectonic cortical maps in the three mammalian species in [Fig. 3.1](#) illustrates some differences among species that have profound functional significance. First, the surface of the neocortex has expanded enormously during our common ancestral evolution (e.g., the human neocortex surface is 10 times larger than that of a macaque monkey and 1,000 times larger than that of a rat). Second, during evolution, cytoarchitectonic areas do not expand uniformly (e.g., the primary visual cortex occupies one-fifth of the neocortical surface in monkeys and only one-thirtieth that of humans). Third, new functional and anatomic areas are introduced during evolution (e.g., Broca's language areas in humans). Fourth, there are large variations in the size of cortical areas among individuals of the same species and between both hemispheres in the same individual (e.g., the planum temporale is larger on the left side in most right-handed humans). However, despite these enormous differences in size of cortical surfaces and pattern of cytoarchitectonic maps, the thickness of the cortex relatively has not changed in phylogeny.



**Figure 3.1.** Lateral (*upper figures*) and medial (*lower figures*) views of the cerebral hemisphere in human (*right*), macaque monkey (*left top*), and rat (*left bottom*), with schematic designation of individual cytoarchitectonic areas. (Reproduced to approximate scale from Brodmann K: *Vergleichende Localisationslehre der Grosshirn-hinde*. Leipzig, Barth, [1909](#).)

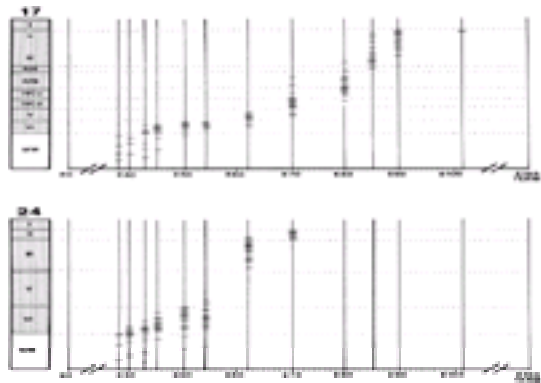
Understanding the development of the cerebral cortex and its expansion in size and complexity critically depends on the answer to some fundamental questions: When and where are cortical neurons generated? How is their number determined in each species and individual? When and how are cell phenotypes determined that constitute the cortex? How do postmitotic cells attain their proper laminar and areal position within the cortex? Why does the surface of the cortex expand so enormously during evolution, whereas its thickness remains relatively constant? How are interspecies and intraspecies differences in cortical parcellation generated? To what extent is the size of cytoarchitectonic areas determined innately and can this size be changed by external stimulation? What are the mechanisms for the formation of complex connections between areas and with various subcortical structures? What is the role of various environmental factors, including functional activity and experience, in this process? How is the number and proportion of various classes of synapses in the cerebral cortex established? When does the critical period for structural and functional modifiability of cortical connections and cytoarchitectonic cortical maps end? Are there additions and/or turnover of neurons in the adult neocortex? Elucidation of these issues is an essential prerequisite for understanding the etiology of genetic and acquired developmental disorders of the cerebral cortex.

This chapter is based mainly on experimental studies of neocortical development in the macaque monkey. The large size of the cerebrum and presence of visible landmarks on the convoluted surface in this primate species allow precise delineation of cortical areas, whereas its protracted development enables accurate timing of cellular events. Furthermore, the macaques, similar to the human, belong to a group of Old World primates who share many aspects of cortical organization and developmental mechanisms. Therefore, the data on the basic cellular and molecular events in these two primate species are reviewed whenever available.

### TIME OF NEURON ORIGIN

The cerebral cortex in the newborn child has well-delineated cortical layers. Based on studies using classical methods, it has been suspected that most cortical neurons in the human cerebrum were generated before birth ([Poliakov, 1949](#), 1959, [1965](#)). The <sup>3</sup>H-thymidine autoradiography, which denotes the time of final deoxyribonucleic acid (DNA) replication, that is, the "birthday of a neuron" ([Angevine, 1965](#); [Sidman and Rakic, 1973](#)), provides an opportunity to obtain precise data on the onset and termination of specific neuronal subclasses during corticogenesis in primates. The examination of a series of adult macaque monkeys exposed to <sup>3</sup>H-thymidine during prenatal and postnatal ages revealed that genesis of cortical neurons occurs during the middle of the 165th day gestation in this species ([Rakic, 1974](#), [1988a](#)). Neurogenesis of cortical neurons starts around the 40th embryonic day (E40) and lasts between 1 and 2 months, depending on the cytoarchitectonic area. For example, in the anterior cingulate cortex (Brodmann's area 24), neurogenesis stops at E70, whereas in the primary visual cortex (area 17), it stops at E100 ([Fig. 3.2](#)). Analysis of DNA labeling with <sup>3</sup>H-thymidine in brain sections of animals exposed to <sup>3</sup>H-thymidine after birth revealed no radiolabeled cells with clear neuronal morphology, suggesting that neocortical neurons in macaque monkeys are not produced during the 30-year life span of this species ([Rakic, 1985](#)).

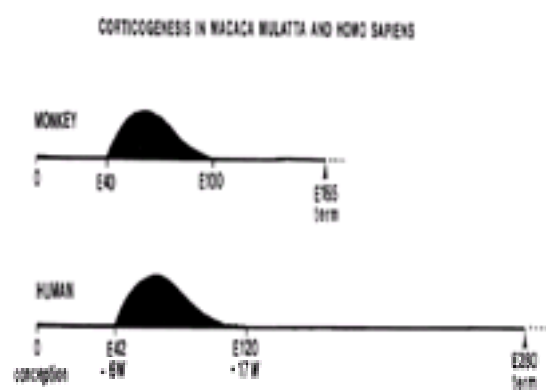




**Figure 3.2.** Diagrammatic representation of the positions of heavily labeled neurons in the cortex of juvenile monkeys, each of which had been injected with  $^3\text{H}$ -thymidine at selected embryonic days. *Top*: area 17; *bottom*: area 24 of Brodmann. On the left side of each diagram is a drawing of the cortex from cresyl violet-stained sections, in which subdivisions into cortical layers are indicated by roman numerals. Embryonic days ( $E$ ) are represented on the horizontal line, starting on the left with the end of the first fetal month ( $E27$ ) and ending on the right at term ( $E165$ ). Positions of vertical lines (*left to right*) indicate the embryonic day on which one animal received a pulse of  $^3\text{H}$ -thymidine. On each vertical line, short horizontal markers indicate positions of all heavily labeled neurons encountered in one 2.5-mm long strip of cortex. WM, white matter. (From Rakic P: Neurons in the monkey visual cortex: Systematic relation between time of origin and eventual disposition. *Science* 183:425–427, 1974 (*top*); Rakic P: Prenatal genesis of connections subserving ocular dominance in the rhesus monkey. *Nature* 261:467–471, 1976a (*bottom*).

The claim that the primate neocortex continues daily to acquire a large number of new neurons throughout the entire adult life (Gould et al., 1999) has been met with skepticism (Nowakowski and Hayes, 2000; Rakic, 2001). These data were obtained by more sensitive, but less reliable methods for detection. The Gould and associates data contradicts previous studies conducted in species ranging from rodents to carnivores and primates, all indicating that cortical neurons originate during the prenatal and/or early perinatal period (Angevine and Sidman, 1961; Luskin and Shatz, 1985b; Rakic, 1974). These data were obtained by more sensitive, but less reliable methods for detection. Furthermore, the suggestion that “new cortical neurons” are transient and die within a few weeks after their genesis was not supported by the finding of pyknotic cells that is commensurate with the massive apoptosis suggested to occur. Thus, until credible evidence is provided, neurons of the primate neocortex all appear to be generated prenatally.

Autoradiographic data cannot be obtained for the human fetal brain, but comparative cytological analysis indicates that production of cortical neurons starts around E45 (6.5 gestational weeks), proceeds in two large waves until about E125 or 17 GA weeks (Fig. 3.3) and then continues on a much smaller scale for another 2 months (Judas et al., 1999; Rakic, 1978, 1988a; Sidman and Rakic, 1973). At the time of birth, the proliferative ventricular and subventricular zones that generate the cortical neurons are exhausted, and the subependymal layer begins to produce a massive number of nonneuronal cells such as the astrocytes and oligodendrocytes that populate the large white matter of the human cerebrum (Yakovlev and Lecours, 1967). In the human, these nonneuronal cells outnumber neurons by severalfold and are responsible, in part, for the postnatal growth of the cerebrum. The claim of a large addition and fluctuation in the number of cortical neurons during infancy, based on the old Conel (1939) photographs and data, has been considered unfounded (Korr and Schmitz, 1999). Thus, like most mammalian species that have been examined so far by using stringent criteria and appropriate methods (see the preceding), the neocortex in primates, including humans, also acquires its cortical neurons before birth, mostly during the middle third of gestation (Fig. 3.3).



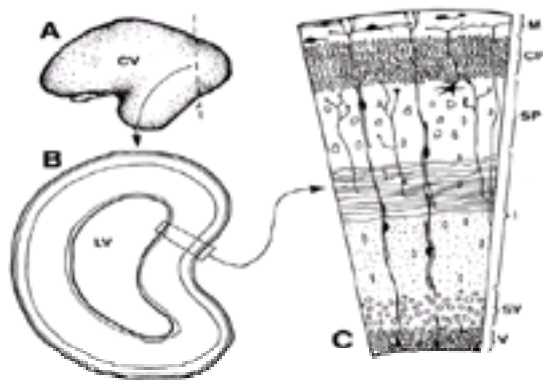
**Figure 3.3.** Diagrammatic representation of the time of neuron origin in rhesus monkey (*top*) and humans (*bottom*). The data for monkeys are obtained by  $^3\text{H}$ -thymidine autoradiography, whereas the data for humans are based on the number of mitotic figures in the ventricular zone, supravital DNA synthesis, and the presence and density of migrating neurons in the intermediate zone of the fetal cerebrum. (From Rakic P: Neurons in the monkey visual cortex: Systematic relation between time of origin and eventual disposition. *Science* 183:425–427, 1974; Rakic P: Neuronal migration and contact interaction in primate telencephalon. *Postgrad Med J* 54:25–40, 1978; Rakic P, Sidman RL: Supravital DNA synthesis in the developing human and mouse brain. *J Neuropathol Exp Neurol* 27:246–276, 1968; Sidman RL, Rakic P: Development of the human central nervous system. In: Haymaker W, Adams RD (eds): *Histology and Histopathology of the Nervous System*. Springfield, IL, Charles C Thomas, 1982, pp. 3–145.)

Considering the existence of adult neurogenesis in many central nervous system (CNS) structures of lower vertebrates, the extent and capacity for adult neurogenesis has evidently diminished in mammalian evolution (Rakic, 1985). In the adult primate, brain neurogenesis has conclusively been shown only for the small interneurons of the olfactory bulb and dentate gyrus, where it is less prominent than in rodents (Kornack and Rakic, 1999; Rakic 2001). Cerebellar granule cells also continue to be generated during the first few postnatal years (Rakic and Sidman, 1977; Zecevic and Rakic, 1976). It has been proposed that this stable population of cortical neurons during adulthood may be a biological necessity, so that individual cognitive experience can be preserved in the synaptic assemblies through decades of the primate life span (Rakic, 1985; Rakic and Kornack, 1993). The resistance of mature neurons to cell division seems to be so powerful that no malignancy of the neurons has been recorded on the adult human cerebrum, although neuroblastoma in children and gliomas are frequent in the adult cortex (Russell and Rubenstein, 1963).

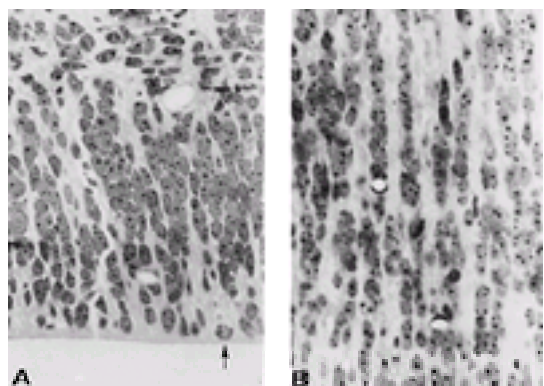
## SITE OF NEURON ORIGIN

It has been suspected, based on the high density of mitotic figures observed near the ventricular surface in the human fetal cerebrum, that most cortical neurons might be produced there, rather than in the cortex itself (His, 1904). However, again, the direct evidence for this hypothesis comes from  $^3\text{H}$ -thymidine autoradiographic analysis. Examination of a series of macaque monkey embryos killed shortly after injection of the DNA precursor revealed that all neurons destined for the neocortex are produced in the layer of germinal cells situated near the cerebral ventricle (Rakic, 1975). This layer, initially consisting of asynchronously dividing precursor cells, was named the ventricular zone (Fig. 3.4) (Boulder Committee, 1970). The nuclei of the precursor cells move away from the ventricular surface to synthesize DNA and then return back to the surface to undergo mitotic division (Sidman and Rakic, 1973). Golgi, electron microscopic, and immunocytochemical analyses reveal that neuronal and glial cell lines coexist in the ventricular zone from the onset of corticogenesis (Levitt et al., 1981; Rakic, 1971, 1972). The early divergence of these two basic cell types has been confirmed using retroviral gene transfer, which labels separate lineages of the precursor cells in the developing telencephalon (Cameron and Rakic, 1991; Luskin et al., 1988; Nakatsuji, et al., 1991; Tan et al., 1998). Furthermore, as illustrated in Fig. 3.5, the ventricular zone in primates is divided by glial septa into columns of precursor or stem cells, termed “proliferative units” that generate generations of neurons destined to form corresponding radial units (Fig. 3.5B) (Rakic, 1978, 1988a). The use of the retroviral gene transfer method in the monkey fetus has confirmed this radial organization that is particularly prominent in primates (Kornack and Rakic, 1995; Rakic, 1995a). Initially, the number of stem cells in a proliferative unit is only three to five, but increases to more than 10 at later developmental stages (Rakic, 1988b).  $^3\text{H}$ -Thymidine autoradiographic analysis indicates that around E40 proliferative units start producing postmitotic neurons, which migrate to their prespecified areal and laminar positions in the cortex (Rakic, 1974). The same method of analysis cannot be applied to human embryonic material, but the use of various cytologic criteria (Sidman and Rakic, 1982) and the application of supravital DNA synthesis to slices of fetal brain tissue (Rakic and Sidman, 1968) indicate that the corresponding time point for the onset of corticogenesis in humans is 6 fetal weeks, or about E42. At later stages of cortical development, there is another proliferative zone, termed the subventricular zone, which mainly produces glial cells in rodents but also becomes a source of interneurons in both primates

and humans ([Rakic, 1975](#); [Sidman and Rakic, 1982](#)).



**Figure 3.4.** Cytologic organization of the primate cerebral wall during the first half of gestation. *A*: The cerebral vesicle of 60- to 65-day-old monkey fetuses is still smooth and lacks the characteristic convolutions that will emerge in the second half of gestation. *B*: Coronal section across the occipital lobe at the level indicated by a vertical dashed line in (*A*). The lateral cerebral ventricle at this age is still relatively large and only the incipient calcarine fissure (CF) marks the position of the prospective visual cortex. *C*: A block of the tissue dissected from the upper bank of the calcarine fissure. At this early stage, one can recognize six transient embryonic zones from the ventricular surface (*bottom*) to the pial surface (*top*): CP, cortical plate; I, intermediate zone; M, marginal zone; SP, subplate zone; SV, subventricular zone; V, ventricular zone. Note the presence of spindle-shaped migrating neurons moving along the elongated radial glial fibers, which span the full thickness of the cerebral wall. The early afferents originating from the brain stem, thalamus, and other cortical areas invade the cerebral wall and accumulate initially in the subplate zone where they make transient synapses before entering the overlying cortical plate. (From Rakic P: A small step for the cell—a giant leap for mankind: a hypothesis of neocortical expansion during evolution. *Trends Neurosci* 18:383–388, [1995b](#).)



**Figure 3.5.** The radial organization of both ventricular zone and fetal cortical plate is best visible in cresyl violet-stained sections. *A*: Photomicrograph of an array of proliferative units within the ventricular zone of the occipital lobe in a 91-day-old monkey embryo. Most mitotic figures are located directly at the ventricular surface (*arrow*), although at this age some can be found in the subventricular zone (*crossed arrow*). *B*: Cortical plate in the occipital lobe of the same animal, showing ontogenetic columns composed of neurons that have originated from the set of proliferative units illustrated in (*A*). Epon-embedded tissue, cut at 1  $\mu$ m, stained with cresyl violet. (From Rakic P: Specification of cerebral cortical areas. *Science* 241:170–176, [1988a](#).)

## DEVELOPMENT OF THE TRANSIENT EMBRYONIC ZONES

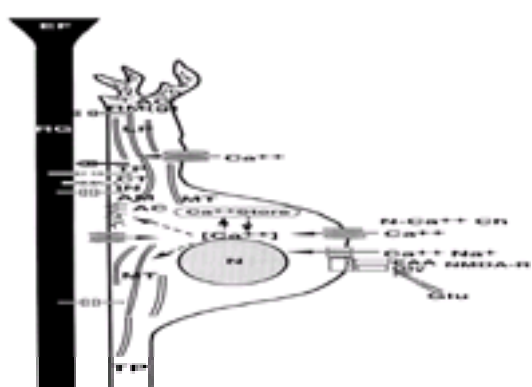
During embryonic and fetal development, the telencephalic wall consists of several cellular layers or zones that do not exist in the mature brain: ventricular zone, subventricular zone, intermediate zone, subplate zone, cortical plate, and marginal zone ([Fig. 3.4](#)). The role of ventricular and subventricular zones was described in previous sections, whereas the present section focuses on the marginal and subplate zones.

The marginal zone, appearing early beneath the pial surface of the cerebral vesicle, is transformed into layer I after arrival of large Cajal-Retzius neurons ([Cajal, 1911](#); [Retzius, 1893](#)). The cellular composition of layer I in primates is distinct in several respects, including presence of the transient subpial granular layer that is absent or minimal in other mammals ([Brünn, 1965](#)). In the macaque monkey, neurons of layer I are generated during the period of corticogenesis ([Zecevic and Rakic, 2001](#)). The classic Cajal-Retzius (C-R) cells are generated first and their processes form a broad, rectangular network oriented parallel to the pia. Genesis of smaller, GABAergic neurons of the primate layer I begins slightly later and continues until completion of corticogenesis ([Zecevic and Rakic, 2001](#)). These late generated cells are imported from outside sources, such as the olfactory primordium, subventricular zone, and ganglionic eminence, via a massive subpial granular layer ([Lavdas et al., 1999](#); [Meyer et al., 1999](#); [Wichertle and Alvarez-Buylla, 2001](#); [Zecevic and Rakic, 2001](#)).

The hypothesis that C-R cells control layering in the cortical plate has gained support through the finding that they produce reelin, a glycoprotein missing in reeler mice, which show defective lamination ([D'Arcangelo et al., 1995](#); [Ogawa et al., 1995](#); [Rakic and Caviness, 1995](#)). A suggestion that C-R cells hold positional information essential for development of initial cortical connections was based on a variety of approaches ([Hevner et al., 2001](#); [Marin-Padilla and Marin-Padilla, 1982](#); [Soria and Fairén, 2000](#)) and is in harmony with the existence of an early primordial protomap in the developing cerebral wall (see the following). The remarkable perpendicular orientation of C-R cell processes has been observed in other species, and the geometrically precise pattern is more pronounced in primates ([Zecevic and Rakic, 2001](#)), which may be related to sharper laminar and columnar organization in this species ([Mountcastle, 1997](#); [Rakic, 1995a](#)).

Although many C-R cells disappear after birth, some survive or change their morphology ([Edmunds and Parnavelas, 1982](#); [Marin-Padilla, 1978](#); [Meyer et al., 1999](#); [Parnavelas and Edmunds, 1982](#); [Poliakov, 1965, 1969](#)), suggesting a more protracted additional role. Identification of subpopulations of layer I cells may help in elucidating their role in congenital malformations of the human cerebral cortex such as lissencephaly ([Hong et al., 2000](#)).

The main embryonic zones were described in classical literature ([His, 1904](#)), but the subplate zone has been recognized as a separate entity much later ([Kostovic and Rakic, 1990](#)). This zone consists of early-generated subplate neurons scattered among numerous axons, dendrites, glial fibers, and migrating neurons ([Fig. 3.4](#)). Most of the subplate neurons eventually degenerate, but some persist in the adult cerebrum as a set of interstitial cells ([Kostovic and Rakic, 1980](#); [Luskin and Shatz, 1985a](#)). Migrating neurons traversing this zone remain attached to radial glia cell guides before entering the cortical plate to form radially oriented columns ([Fig. 3.5](#) and [Fig. 3.6](#)). Although the existence of the subplate zone may provide an opportunity for interaction between migrating neurons, incoming afferent fibers, and early-generated neurons, the significance of these transient contacts is not fully understood.





**Figure 3.6.** Model of a proposed cascade of molecular events that take place during the migration of postmitotic cells in the developing cerebral wall. After their last mitotic division in the ventricular zone, migrating cells extend a leading process (LP) that follows the contours of the radial glial fiber (RG) as it spans the expanding cerebral wall. The cytoskeleton within the LP and trailing process (TP) contain microtubules (MT) and actin-like contractile proteins (AC) that are involved in translocation of the cell nucleus (N) and the surrounding cytoplasm within the leading process until the cell enters the cortical plate. This system, maintained *in vitro* in slice preparations or imprint culture provides an opportunity to examine the role of the various molecules that are engaged in recognition, adhesion, transmembrane signaling, and motility that underlies directed neuronal migration. The voltage-gated (N-type) and ligand-gated (NMDA-type) receptors/channels are thought to control calcium influx, which serves as messengers for execution of this movement. Abbreviations: AM, homotypic adhesion molecule; EAA, excitatory amino acid; EF, end foot of the radial glial fiber; Gly, glycine; RM(g), glycophilic recognition molecule; TP, tyrosine phosphorylation. (Modified from Rakic, 1997)

One possibility, suggested soon after discovery of the subplate zone, was that it serves as a “waiting” compartment for afferents that are generated ahead of their neuronal targets and for cellular substrata competed for among cortical afferents (Rakic, 1977). Subsequent autoradiographic, electron microscopic, and histochemical studies in primates indicate that axons observed in the subplate zone originate sequentially from the brain stem, basal forebrain, thalamus, and the ipsilateral and contralateral cerebral hemispheres (Kostovic and Rakic, 1990). After a variable and partially overlapping period, these diverse fiber systems enter the cortical plate, whereas the subplate zone disappears, leaving only a vestige of cells scattered throughout the subcortical white matter that are known as interstitial neurons (Chun and Shatz, 1989; Kostovic and Rakic, 1980).

In various species, examination of the developing cerebrum reveals that the size and relative duration of the subplate zone increases during mammalian evolution and culminates in human fetuses concomitantly with enlargement of the cortico-cortical fiber systems (Kostovic and Rakic, 1990). The regional difference in size, pattern, and resolution of the subplate zone also correlates with the pattern and size of cerebral convolutions (Goldman-Rakic and Rakic, 1984; Rakic, 1988b). Studies in developing macaque monkeys indicate that the prevailing view of the subplate zone as a vestige of the phylogenetically old neural network should be modified (Marin-Padilla, 1978; Shatz et al., 1988). This transient embryonic zone actually expanded during evolution of the cerebral cortex, most likely as result of the increasing number of cortico-cortical connections and the elaboration of cerebral convolutions (Kostovic and Rakic, 1990). An abnormally large vestige of subplate neurons may form heterotopic masses in the form of a double cortex, and these are believed to be the source of intractable epileptic discharges in children (Palamini et al., 1991b). It was also suggested that migratory defects, which leave an abnormal cell population within the subplate zone, might be a source of abnormalities in some form of schizophrenia (Akbarian et al., 1993).

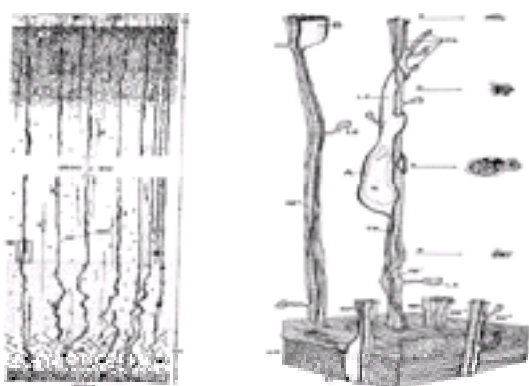
The concept that layer I and subplate neurons are remnants of the initially common “preplate” zone, bisected by the developing neocortex (Aboitiz, 1999; Marin-Padilla, 1978), needs modification in light of the findings in both human and monkey, where the majority of layer I neurons are generated long after the split of the preplate (Kostovic and Rakic, 1980).

## CONTROL OF CORTICAL SIZE

Based on data on the time of cell origin and cell proliferation kinetics in the ventricular zone, we proposed that the number of cortical cells in a given species depends on the timing of cell production, mode of mitotic division (symmetric versus asymmetric), duration of the cell cycle, and the degree of programmed cell death in the proliferative zones (Rakic, 1988a, 1995b). In the past few years, technological advances have allowed analysis of these cellular events in transgenic mice as well as in nonhuman primates using the retroviral gene transfer method (Kornack and Rakic, 1995, 1998; Kuida et al., 1996, 1998; Zhong et al., 1996). Because most genes and their products involved in cell production and fate determination seem to be preserved during evolution, one might expect that control of neuronal number and differentiation would be basically similar in all species (Williams and Herrup, 1988). For example, one mechanism that regulates the number of cells produced in the ventricular zone is programmed cell death (PCD) or apoptosis. Although PCD has been considered a major factor contributing to the formation of the vertebrate brain (Glucksmann, 1951), contemporary research has focused mainly on histogenetic cell death involved in the elimination of inappropriate axonal connections at later stages of development (Oppenheim, 1991; Rakic and Riley, 1983a,b). However, the discovery of several classes of genes involved in PCD, which were initially identified in invertebrates, created the opportunity to study this phenomenon in the mammalian cerebrum. For example, a family of enzymes, called caspases, has been shown to play an important role in PCD in a variety of organs and tissues (Ellis and Horvitz, 1991). We have recently demonstrated that in mouse embryos deficient in caspase 9 and 3, fewer cells are eliminated than in their littermates (Kuida et al., 1996, 1998). Reduction of apoptosis in the knockout mice results in the formation of supernumerary founder cells in the cerebral ventricular zone. As a consequence, these mice form ectopic cells in the intermediate zone, as well as a larger cortical plate with more radial units. Remarkably, the cerebral cortex in these transgenic mice has a larger surface and begins to form convolutions. These new approaches provide an example of how mutation of a few genes that control the reduction of cells could result in the expansion of the cortex during evolution (Rakic, 1995b).

## NEURONAL CELL MIGRATION

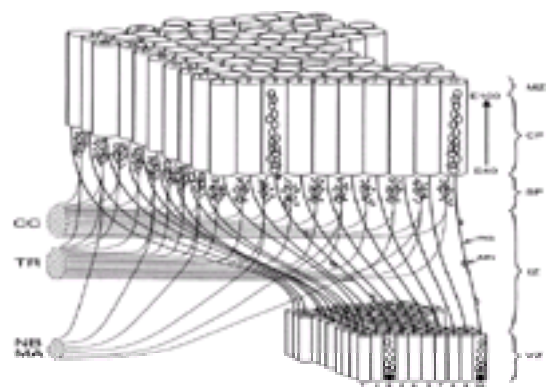
Because all cortical neurons originate near the ventricular surface of the cerebral vesicle, they must all move to their final positions in the cortex, which develops in the outer regions of the cerebral wall, just below the pia. Initially, although the cerebral wall is still thin, cells move only a short distance. However, the length of their pathway increases with the enlargement of the cerebral hemispheres, particularly in the large primate cerebrum in which, during midgestation, a massive migration of neurons occurs concomitantly with the rapid growth of the cerebral wall (Rakic, 1988a; Sidman and Rakic, 1982). This magnitude of cell movement is perhaps the reason that neuronal cell migration was first observed in human embryos (His, 1874). In the early 1970s, advances in methods enabled the discovery that postmitotic neurons find their way to the cortex by following the elongated shafts of radial glial cells (Fig. 3.4, Fig. 3.6, and Fig. 3.7) (Rakic, 1972). These elongated cells, which span the fetal cerebral wall from the beginning of corticogenesis, stop transiently to divide during midgestation at the peak of neuronal migration (Schmechel and Rakic, 1979). The radial glial cells are not only much longer, but also have different structural and biochemical properties than in small rodents (Cameron and Rakic, 1991; Levitt et al., 1981).



**Figure 3.7.** *Left:* Camera lucida drawing of the occipital cerebral wall of the monkey fetus at midgestation. The composite illustration is derived from a Golgi-impregnated section (*black profiles*) and an adjacent section counterstained with toluidine blue (*outline of small nuclei*). The middle 2,000  $\mu\text{m}$  of the intermediate zone, similar in structure to the sectors drawn, is omitted. The rectangle marked with an asterisk shows the approximate position of the cell reconstruction seen in (*B*). *C*, cortical plate; *I*, intermediate zone; *M*, molecular layer; *MN*, migrating neuron; *RF*, radial fiber; *SV*, subventricular zone; *V*, ventricular zone. *Right:* Three-dimensional reconstruction of migrating neurons, based on serial electron micrographs made at the level of the intermediate zone, indicated by the rectangle in (*A*). The lower portion of the diagram contains parallel fibers of the optic radiation (OR), and the remainder is occupied by a more disposed fiber system. Except at the lower portion of the figure, most of these fibers are deleted from the diagram to expose the radial fibers (*striped vertical shafts RF<sup>1-6</sup>*) and their relations to the migrating cells A, B, and C. The soma of migrating cell A, with its nucleus (N) and voluminous leading process (LP), is situated within the reconstructed space, except for the terminal part of the attenuated trailing process and the tip of the vertical ascending pseudopodium. Cross-sections of cell A in relation to the several vertical fibers in the fascicle are drawn at levels a to d at the right side of the figure. The perikaryon of cell B is cut off at the top of the reconstructed space, whereas the leading process of cell C is shown just penetrating between fibers of the optic radiation (OR) on its way across the intermediate zone. LE, lamellate expansions; PS, pseudopodia. (From Rakic P: Mode of cell migration to the superficial layers of fetal monkey neocortex. *J Comp Neurol* 145:61–84, 1972.)

While moving along the glial surface, migrating neurons remain preferentially attached to glial fibers, which suggests a “gliophilic” mode of migration ( [Rakic, 1990](#)) that is mediated by heterotypic adhesion molecules ( [Rakic et al., 1994b](#)). However, some postmitotic cells do not obey glial constraints and move along tangentially oriented axonal fascicles (e.g., black horizontally oriented cell aligned with TR in [Fig. 3.6](#)). We suggested the term “neurophilic” to characterize the mode of migration of this cell class ( [Rakic, 1990](#)). Although lateral dispersion of postmitotic neurons was initially observed in Golgi-stained preparations ( [Boulder Committee, 1970](#)), it has attracted renewed attention after the discovery that a specific, presumably neurophilic, cell class moves from the telencephalon to the olfactory bulb ( [Menezes and Luskin, 1994](#)) and after studies in rodents suggested more widespread dispersion of clonally related cortical cells ( [Rakic, 1995a](#); [Tan et al., 1998](#)). However, it should be underscored that application of the same methods in the convoluted primate cortex revealed that the majority of migrating cells obey the radial constraints imposed by the radial glial scaffolding ( [Kornack and Rakic, 1995](#); see also section on Radial Glial Hypothesis).

Considerable progress has been made in understanding the molecular mechanisms behind neuronal migration and the physical displacement of cell perikarya during translocation of the cell nucleus and soma across the densely packed tissue. Initially, based on an observation *in situ*, it was proposed that a single pair of binding, complementary molecules with glycophilic properties can account for the recognition of glial guides ( [Rakic, 1981a](#)). In the last decade, however, several candidates for recognition and adhesion molecules have been discovered and are being tested ( [Anton et al., 1996](#); [Cameron and Rakic, 1994](#); [Edelman, 1983](#); [Hatten and Mason, 1990](#); [Schachner et al., 1985](#)). Recently, it was also shown that voltage- and ligand-gated ion channels on the leading process and cell soma of migrating neurons regulate the influx of calcium ions into migrating neurons ( [Komuro and Rakic, 1992, 1993, 1996](#); [Rakic and Komuro, 1995](#)). Calcium fluctuations, in turn, may trigger polymerization of cytoskeletal and contractile proteins essential for cell motility and translocation of the nucleus and surrounding cytoplasm ( [Rakic et al., 1996](#)). These studies indicate that neuronal migration is a multifaceted developmental event, involving cell–cell recognition, differential adhesion, transmembrane signaling, and intracytoplasmic structural changes ( [Rakic et al., 1996](#)). A simple model of molecular components involved in cell migration is provided on the diagram in [Fig. 3.8](#). The discovery of the glial-guided radial migration led to the proposal of the *radial unit hypothesis* ( [Rakic, 1988a](#)), which has served as a useful working model for research on the cellular and molecular mechanisms involved in normal and abnormal cortical development.



**Figure 3.8.** The relation between a small patch of the proliferative ventricular zone (VZ) and its corresponding area within the cortical plate (CP) in the developing cerebrum. Although the cerebral surface in primates expands and shifts during prenatal development, ontogenetic columns (outlined by cylinders) remain attached to the corresponding proliferative units by the grid of radial glial fibers. Neurons produced between E40 and E100 by a given proliferative unit migrate in succession along the same radial glial guides (RG) and stack up in reverse order of arrival within the same ontogenetic column. Each migrating neuron first traverses the intermediate zone (IZ) and then the subplate (SP) that contains interstitial cells and “waiting” afferents from the basal forebrain (NB), monoamine subcortical centers (MN), thalamic radiation (TR), and ipsilateral and contralateral cortico-cortical connections (CC). After entering the cortical plate, each neuron bypasses earlier-generated neurons and settles at the interface between the CP and marginal zone (MZ). As a result, proliferative units 1 through 100 produce ontogenetic columns 1 through 100 in the same position relative to one another without a lateral mismatch. Thus, the specification of cytoarchitectonic areas and topographic maps depends on the spatial distribution of their ancestors in the proliferative units, whereas the laminar position and phenotype of neurons within ontogenetic columns depends on the time of their origin. (Modified from Rakic P: Specification of cerebral cortical areas. *Science* 241:170–176, [1988a](#).)

## DETERMINATION OF NEURONAL PHENOTYPES

Immunocytochemical analyses in fetal monkeys provided the initial evidence that proliferative cells in the embryonic ventricular zone simultaneously produce both neurons and glial cell clones ( [Levitt et al., 1981](#)). Furthermore, examination of the fate of <sup>3</sup>H-thymidine-labeled cells suggests that multiple neural phenotypes are simultaneously produced and terminate either in the single or adjacent cortical layers ( [Rakic, 1988a](#)). Therefore, after their last cell division, postmitotic neurons seem to become restricted in the repertoire of their possible fates. Several lines of evidence support this. For example, if neurons remain in ectopic positions near the cerebral ventricle (as a consequence of x-irradiation during embryonic stages), they, nevertheless, acquire the morphology and connections expected from the time of their origin ( [Jensen and Killackey, 1984](#)). Likewise, ventricular cells transplanted from the embryos into the telencephalic wall of a newborn ferret migrate to the host cortex and assume laminar cortical positions, morphologic characteristics, and a pattern of connections appropriate for the stage that they have achieved in the donor ( [McConnell, 1988](#)). Callosal neurons send their axons to the contralateral hemisphere before their soma have entered layer II of the cortical plate ( [Schwartz et al., 1991](#)). This early laminar commitment of cortical neurons is also evident in the mutant *reeler* mouse in which sequentially generated neurons assume position in the cortical layers that is reversed compared to the normal mouse, but, nevertheless, differentiates into phenotypes expected from the time of their origin, rather than from their ectopic location ( [Caviness and Rakic, 1978](#)). Recent analysis, in which ribonucleic acid (RNA) retrovirus-mediated gene transfer was used to mark the progeny of dividing cells, revealed that neurons of a different type originate from separate clones ( [Parnavelas et al., 1991](#)). Collectively, these findings indicate that the range of morphologies and patterns of synaptic contacts of cortical neurons may be specified, in considerable measure, before they reach their final positions.

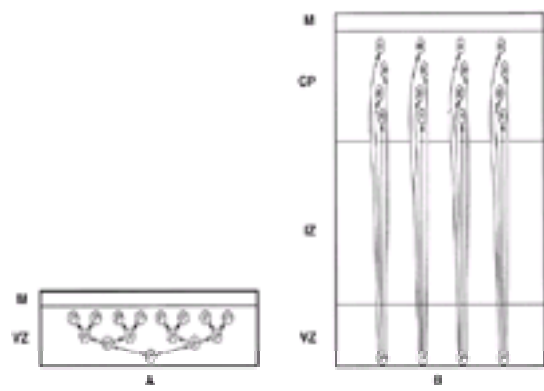
A survey of the emergence of the various transmitters and their receptors in the embryonic monkey telencephalon showed that, contrary to our expectations, most of them appear very early, prior to the formation of synapses. For example,  $\alpha_1$ -adrenergic receptors are prominent in the ventricular and subventricular zones, up to midgestation, in conjunction with the intensive proliferation of cortical neurons, whereas, in contrast,  $\beta$  receptor subtypes emerge in these zones only after the proliferation subsides and corticogenesis is completed ( [Lidow and Rakic, 1994](#)). This sequence of developmental events suggests that  $\alpha_1$  sites may participate in promotion of cell proliferation, whereas the  $\beta$  sites may participate in suppression of cell proliferation within the germinal zones of the fetal cerebrum. This suggestion is in harmony with the finding that cell division *in vitro* can be stimulated and inhibited by these two adrenergic subtypes, respectively. Since both ventricular and subventricular zones produce a variety of neuronal subtypes ( [McConnell and Kasanowski, 1991](#); [Parnavelas et al., 1991](#); [Rakic, 1988a](#)), it is possible that noradrenaline, or other neurotransmitters, can regulate the proliferation of various types of neurons already in the germinative epithelium. Both  $\gamma$ -aminobutyric acid (GABA), as well as GABA<sub>A</sub> receptor subtypes, are expressed on migrating neurons before they reach their destination in the cortex ( [Schwartz and Meinecke, 1992](#)). *In vitro* studies indicate that these signaling molecules may play a role in differential cell death and, therefore, can influence the areal- and layer-specific density of GABA-containing local circuit neurons ( [Vaccarino et al., 1995](#)). Collectively, these findings demonstrate that the range of morphologies and patterns of connections of cortical neurons may be specified, in considerable degree, by various neuroactive molecules before postmitotic cells reach their final positions in the cerebral cortex.

## RADIAL UNIT HYPOTHESIS

The cellular mechanisms underlying expansion of the cerebral cortex during individual development and evolution can be explained in the context of the *radial unit hypothesis* ( [Rakic, 1988a, 1995b](#)). In all species, the neocortex consists of an array of iterative neuronal groups (called interchangeably, radial columns or modules) that share a common intrinsic and extrinsic connectivity and subserve the same function ( [Goldman-Rakic, 1987a](#); [Mountcastle, 1997](#); [Szentagothai, 1978](#)). The larger the cortex in a given species, the larger the number of participating columnar units ( [Rakic, 1978, 1995b](#)). The radial unit hypothesis of cortical development postulates that the embryonic cortical plate forms from vertically oriented cohorts of neurons generated at the same site in the proliferative ventricular zone of the cerebral vesicle ( [Fig. 3.3](#), [Fig. 3.7](#), and [Fig. 3.8](#)) ( [Rakic, 1978](#)). Each radial unit consists of several clones (polyclones) that migrate to the cortex following glial fascicles spanning the cerebral wall ( [Rakic, 1988a](#)). In the cortical plate, later generated cells bypass earlier generated ones and settle in an inside-out gradient of neurogenesis ( [Rakic, 1974](#)). Thus, the two-dimensional positional information of the proliferative units in the ventricular zone is transformed into a three-dimensional cortical architecture: the X- and Y-axis of the cells is provided by their site of origin, whereas the Z-axis is provided by their time of origin ( [Fig. 3.8](#)). The radial unit hypothesis provides the explanation for the large expansion of cortical surface that occurred without a concomitant significant increase in thickness during phylogenetic and ontogenetic development ( [Rakic, 1988a](#)). It also shows how the genes controlling the number of founder cells at the ventricular surface sets a limit



on the size of the cortical surface during individual development, as well as during the evolution of mammalian species ( [Rakic, 1995b](#)). Thus, a relatively small change in the timing of developmental cellular events could have large functional consequences. For example, a minor increase in the length of the cell cycle or the magnitude of cell death in the ventricular zone could result in a large increase in the number of founder cells that form proliferative units ( [Rakic, 1988a](#)). Because proliferation in the ventricular zone initially proceeds exponentially because of the prevalence of symmetric divisions, an additional round of mitotic cycles during this phase doubles the number of founder cells, and consequently, the number of radial columns ( [Fig. 3.9](#)) ( [Rakic, 1995b](#)). According to this model, before the onset of corticogenesis, fewer than four extra rounds of symmetric cell divisions in the ventricular zone can account for a 10-fold difference in the size of the cortical surface. Because the mode of cell division predominantly changes to asymmetric after the onset of corticogenesis, one can predict that by extending cell production in humans by about 2 weeks longer than in macaques should enlarge the cortical thickness by only 10% to 15%, which actually is observed ( [Rakic, 1995b](#)). Thus, as illustrated in [Fig. 3.9](#), even a small delay in the onset of the second phase of corticogenesis results in a proportionately larger cortical surface owing to the increasing number of founder cells at the ventricular zone.

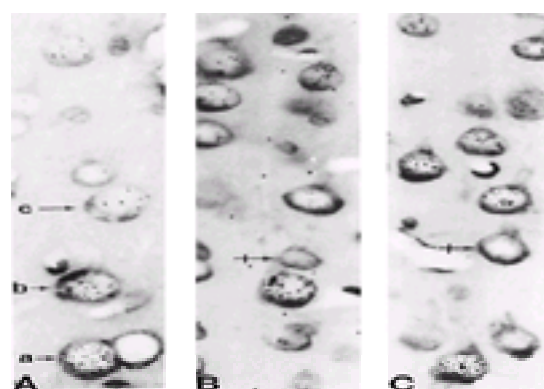


**Figure 3.9. A:** Schematic model of symmetric cell divisions that predominate before the E40. At this early embryonic age, the cerebral wall consists of only the ventricular zone (VZ), where all the cells are proliferating, and the marginal zone (M), into which some cells extend radial processes. Symmetric division produces two progenitors (P) during each cycle and causes rapid horizontal lateral spread. **B:** Model of asymmetric or stem division which becomes predominant in the monkey embryo after E40. During each asymmetric division, a progenitor (P) produces one postmitotic neuron that leaves the ventricular zone and another progenitor that remains within the proliferative zone and continues to divide. Postmitotic neurons migrate rapidly across the intermediate zone (IZ) and become arranged vertically in the cortical plate (CP) in reverse order of their arrival (1,2,3,4). (Modified from Rakic P: A small step for the cell—a giant leap for mankind: A hypothesis of neocortical expansion during evolution. *Trends Neurosci* 18:383–388, [1995b](#)).

The proposal that neurons comprising a given radial column may be clonally related has been tested experimentally using the retroviral gene transfer method that allows *in vivo* analysis of cell lineages in the mammalian brain ( [Sanes, 1989](#)). Use of this approach suggests that most progenitors originating in the same site of the ventricular zone remain radially deployed in the cortex in rodents ( [Luskin et al., 1988](#); [Tan et al., 1998](#); [Ware et al., 1999](#)), as well as in primates ( [Kornack and Rakic, 1995](#)). Furthermore, a number of studies in chimeric and transgenic mice have provided further evidence that a majority of postmitotic, clonally related neurons move and remain radially distributed in the cortex ( [Nakatsuji et al., 1991](#); [Rakic, 1995a](#); [Soriano et al., 1995](#)). Using the retroviral gene transfer method in the embryonic primate brain showed that even in the large and highly convoluted cerebrum, radial deployment of many clones is remarkably well preserved ( [Kornack and Rakic, 1995](#)), and the similarly convoluted human cerebrum develops by using the same cellular mechanism.

## ACQUISITION OF LAMINAR AND RADIAL POSITIONS

Because genesis of cortical neurons in macaque monkeys does not start until E40 ( [Rakic, 1974](#)) and until a few days later in humans ( [Sidman and Rakic, 1982](#)) ( [Fig. 3.3](#)), neuronal progenitors in the ventricular zone prior to that time produce only other progenitors. This so-called symmetric mode of cell division has initially been implied from data on the kinetics of cell proliferation using  $^3\text{H}$ -thymidine labeling of DNA in short-term experiments ( [Rakic, 1975](#)). Thus, radioactivity uniformly became diluted in the ventricular zone 1 or 2 days following injection of the isotope. After E40, however, many progenitor cells begin to produce dissimilar daughters (by asymmetric division), one of which becomes a postmitotic neuron, whereas the other may remain a stem cell or die ( [Fig. 3.9](#)). Initially, evidence that many stem cells in the proliferative units begin to divide asymmetrically also came from  $^3\text{H}$ -thymidine autoradiographic analysis, but only after using longer intervals following injections of the isotope ( [Fig. 3.10](#)). Examination of adult monkeys exposed to  $^3\text{H}$ -thymidine between E40 and E100 revealed that the radioactivity of neurons, progressively situated more superficially in the cortex, diminished stepwise by halves ( [Rakic, 1988a](#)). This finding indicated that, in a majority of mitotic divisions, one daughter cell remains in the proliferative unit and dilutes its radioactivity by subsequent divisions. However, many divisions must continue in a symmetric mode, because a given proliferative unit, which might initially contain a single stem cell, contains several progenitor cells during later stages of corticogenesis. The concept of a gradual shift from a purely symmetric mode of division to a mixture of symmetric and asymmetric modes in the telencephalic ventricular zone is supported by use of double labeling methods, enabling study of proliferation kinetics with two independent S phase markers ( [Takahashi et al., 1992, 1993](#)). Therefore, proliferative units in the ventricular zone should be regarded as polyclones, and each ontogenetic column in the overlying cortical plate should receive neurons from more than one clone. This model is supported by distribution of  $^3\text{H}$ -thymidine-labeled cells in the macaque monkey, which shows that, within the same column, intensely labeled and lightly labeled neurons (presumably from the same progenitor) can be interspersed among unlabeled neurons originating from a different progenitor within the same proliferative unit prior to or after injection of  $^3\text{H}$ -thymidine ( [Fig. 3.10](#)).



**Figure 3.10.** Microphotograph of three autoradiograms, showing neurons in the cortex of an adult monkey that was exposed to  $^3\text{H}$ -thymidine at E70. **A:** The most intensely radioactive neuron (a) lies deeper in the cortex than the two progressively less labeled, more superficially situated neurons (b and c). **B:** and **C:** Unlabeled neurons (crossed arrows) may be interspersed among radioactive neurons within the same ontogenetic column. Further explanation is in the text. (From Rakic P: Specification of cerebral cortical areas. *Science* 241:170–176, [1988a](#).)

Eventually, all postmitotic cells generated in a given proliferative unit of the ventricular zone ( [Fig. 3.5](#)) morphologically form an identifiable stack of neurons in the cortical plate ( [Fig. 3.5](#)). These radially deployed neurons are easily distinguishable and variously termed ontogenetic or embryonic columns ( [Rakic, 1978, 1988b](#)). The number of neurons in an ontogenetic column of the macaque monkey in the second half of gestation ranges between 70 in the anterior cingulate cortex (area 24), where neurogenesis lasts about 1 month, to more than 120 in the primary visual cortex (area 17), where neurogenesis lasts almost 2 months ( [Rakic, 1974, 1988b](#)) ( [Fig. 3.2](#)). However, the number of cells in the column is about 50% larger during midgestation and is eventually reduced by differential cell death, which starts after completion of neurogenesis in primates ( [Williams et al., 1987](#)). Within each column, earlier generated neurons occupy deeper positions, whereas those arriving to the cortex later must pass older neurons to become situated more superficially ( [Fig. 3.2](#)). This relationship among cells with different birthdays, called the “inside-out” gradient of neurogenesis, was suspected by classical anatomists, but has been proven only after use of the  $^3\text{H}$ -thymidine autoradiographic method in a number of mammalian species ( [Sidman and Rakic, 1973](#)). The inside-out gradient of neurogenesis is particularly prominent in primates, in whom each daily injection of  $^3\text{H}$ -thymidine labels a more selective sample of cortical neurons than in rodents ( [Rakic, 1974](#)). It is not clear why newly generated neurons must pass the older ones

instead of piling up below each other, but one of several hypotheses is that this sequence enables feedback signals to pass from the older to the arriving cells.

## PROTOMAP HYPOTHESIS

A major challenge of the cerebral cortex to students is how individual and species-specific cytoarchitectonic areas have emerged from the initially seemingly uniform ventricular zone and cortical plate. Both intrinsic and extrinsic factors have been suggested. One attractive hypothesis is that all cortical neurons are equipotential and that laminar and areal differences are induced by extrinsic influences exerted via thalamic afferents ( [Creutzfeldt, 1977](#)). However, modern neurobiology has provided a considerable amount of new evidence that cells generated within the embryonic cerebral wall contain some intrinsic information about their prospective species-specific cortical organization. To reconcile existing descriptive and new experimental data, we formulated a *protomap hypothesis* ([Rakic, 1988a](#)). This hypothesis suggests that the basic pattern of cytoarchitectonic areas emerges through synergistic, interdependent interactions between developmental programs intrinsic to cortical neurons that can be modified by extrinsic signals supplied by specific inputs from various subcortical structures to a certain extent. According to the protomap hypothesis, neurons in the embryonic cortical plate—indeed even in the proliferative ventricular zone where they originate—set up a primordial map that preferentially attracts appropriate afferents and have the capacity to respond to this input in a specific manner. The prefix *proto* was introduced to emphasize the primordial, provisional, and essentially malleable character of the protomap that is subject to considerable modification by extrinsic influences exerted at later stages ([Rakic, 1988a](#)).

The initial indication that developmental events in the proliferative ventricular zone foreshadow prospective regional differences in the overlying cerebral mantle comes from the observation that neurogenesis of the primary visual cortex, which contains more neurons per radial unit than the adjacent areas, lasts longer ( [Rakic, 1976b](#)). Furthermore, it has also been demonstrated that the mitotic index in the ventricular region subjacent to this area is higher than in adjacent regions ( [Kennedy and DeHay, 1993](#)). Therefore, region-specific differences in production of the ventricular zone can be detected even before neurons arrive at the cortex ( [Algan and Rakic, 1997](#); [Kennedy and DeHay, 1993](#)). Several lines of evidence indicate that, during final cell division, one or both daughter cells start to express a variety of neuron, class-specific signaling molecules ( [Lidow and Rakic, 1994](#); [LoTurco et al., 1995](#)).

Postmitotic cells not only become committed to a neuronal fate but also become restricted in their repertoire of possible fates ( [McConnell, 1988](#)). Numerous studies in which the cytology of postmitotic cells have been examined ( [LoTurco et al., 1995](#); [Schwartz et al., 1991](#)) and/or manipulated by a variety of methods, such as spontaneous mutations ( [Caviness and Rakic, 1978](#); [Rakic, 1995b](#)), ionizing radiation ( [Algan and Rakic, 1997](#)), retroviral gene transfer labeling ( [Parnavelas et al., 1991](#)), transgenic mice ( [Kuida et al., 1996](#)), and heterochronic transplantations ( [McConnell, 1988](#); [McConnell and Kasanowski, 1991](#)) all indicate that certain cell, class-specific attributes are expressed before migrating neurons arrive at the cortical plate and become synaptically connected.

More recently, retroviral tracing experiments and some clonal analyses suggest that the ventricular zone is comprised of a heterogeneous population of cells and that cell lineage contributes substantially to cell fate determination of neurons ( [Acklin and van der Kooy, 1993](#); [Kornack and Rakic, 1995](#); [Kuan et al., 1997](#); [Parnavelas et al., 1991](#); [Williams and Price, 1995](#)). These findings raise the question of whether laminar and areal identities of cortical plate cells provide cues or chemotactic attractants for incoming afferent axons. Data from axonal tracing indicate that afferent connections from subcortical structures and other cortical regions find their way to the specific regions of the cortical plate either directly and/or via the subplate zone ( [Agmon et al., 1995](#); [Catalano et al., 1996](#); [Kostovic and Rakic, 1990](#); [McConnell et al., 1991](#); Richards et al., 1997), suggesting the existence of region-specific attractants for path finding and target recognition. In support of this idea, the development of correct topological connections in anophthalmic mice and in early-enucleated animals indicates that basic connections and chemoarchitectonic characteristics can form in the absence of information from the periphery ( [Kaiserman-Abramof et al., 1980](#); [Kennedy and DeHay, 1993](#); [Kuljis and Rakic, 1990](#); [Olivaria and Van Sluyters, 1984](#); [Rakic, 1988a](#); [Rakic and Lidow, 1995](#)).

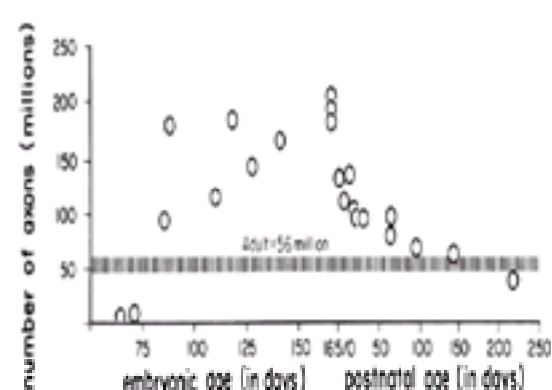
A large number of region-specific and layer-specific morphoregulatory molecules have been observed in the embryonic cerebral wall ( [Arimatsu et al., 1992](#); [Barbe and Levitt, 1992](#); [Buffone et al., 1993](#); [Cohen-Tannoudji et al., 1994](#); [Donoghue and Rakic, 1999a,b](#); Emerling and Lander, 1994; [Ferri and Levitt, 1993](#); [Gitton et al., 1999](#); Grove and Ragsdale, 2000; Levitt et al., 1997; [Porteus et al., 1992](#); [Rubenstein and Rakic, 1999](#)). Function of these molecules is not well understood, but a consensus seems to emerge that many of them contribute to the formation of specified axonal pathways. Thus, tangentially and radially distinct landmarks in the postmitotic cells may facilitate axonal path finding and target recognition that eventually leads to parcellation of the cerebral cortex. However, the initial species-specific protomap seems to be intrinsic to the cortex itself. Recently, it was shown that the cerebrum develops surprisingly well in knockout mice in which synaptic transmission is abolished ( [Verhage et al., 2000](#)) and that disturbance of thalamic input does not prevent formation of the basic cortical protomap ( [Bishop et al., 2000](#); [Miyashita-Lin et al., 1999](#)).

It should be underscored that although the embryonic cerebral wall exhibits gradients of several classes of morphoregulatory molecules, as well as other specific area-specific molecular differences, the protomap within the embryonic cerebrum only provides a set of species-specific genetic instructions and biological constraints. The precise position of interareal borders, the overall size of each cytoarchitectonic area, and the details of their cellular and synaptic characteristics in the adult cerebral cortex are achieved through a cascade of reciprocal interactions between cortical neurons and the cues they receive from afferents arriving from a variety of extracortical sources ( [Rakic, 1988a, 1988b](#)). Such afferents may serve to coordinate and adjust the ratio of various cell classes with the subcortical structures, as has been shown in the primary visual system ( [Kennedy and DeHay, 1993](#); [Meissirel et al., 1997](#); [Rakic et al., 1991](#); [Rakic and Lidow, 1995](#)). In summary, the concept of the cortical protomap includes the role of both intrinsic and extrinsic determinants in shaping the final pattern and relative sizes of the cytoarchitectonic areas.

## OVERPRODUCTION OF NEURONS AND THEIR PROJECTIONS

With few exceptions, most brain structures in vertebrates have a larger number of neurons and axonal connections during development than in adulthood ( [Hamburger and Oppenheim, 1982](#); [Williams and Herrup, 1988](#)). In all parts of the brain so far studied, neurons, as well as their axons and dendrites, were only found to be more numerous during a well-defined phase of development ( [Easter et al., 1985](#); [Purves, 1988](#); [Rakic et al., 1994a](#); [Rakic and Riley, 1983a](#)). The same basic phenomenon of initial exuberance has been observed during development of the cerebral cortex. Judging from the density of pyknotic (degenerating) cells, the visual cortex of fetal monkeys contains about 30% more neurons than that in adult monkeys ( [Williams et al., 1987](#)). The elimination of cells occurs mostly during the second half of gestation, when the superficial cortical layers may lose 35% to 40% of the initial number of neurons that arrived at the cortical plate. In contrast, the deep cortical layers, which are formed earlier, lose less than 25% of the original number. The reason for the lamina-specific difference in the magnitude of neuronal death is not clear, but it may be significant that local circuit cortico-cortical systems that are predominantly situated in the superficial layers (that are generated later) lose more cells than cortico-subcortical efferent systems, which originate from the deep layers (that are generated early).

The phase of massive neuronal elimination from the primate cerebral cortex precedes the phase of axonal elimination in the large cortico-cortical fiber tracts. For example, the newborn monkey has about 190 million callosal axons, compared to less than 50 million in the adult ( [LaMantia and Rakic, 1990](#)). A majority of these axons are eliminated during the first three postnatal weeks at the rate of 8 million per day or 50 per second. Therefore, callosal axons are lost at an estimated average rate of half a million per day or five per second, until the adult value is reached around puberty ( [Fig. 3.11](#)). This enormous loss of callosal axons is not accompanied by a significant change in the number of cortical neurons during the same period. This apparent paradox can be explained, at least in part, by the fact that callosal neurons represent less than 1% of the total cortical cells. However, it should be noted that many collateral branches or secondary axons of callosal cells might be eliminated from neurons that survive to adulthood. Other classes of interhemispheric connections, such as the anterior and hippocampal commissures, are also lost at a similar rate during approximately the same time ( [LaMantia and Rakic, 1994, 1995](#)). As discussed in the following, the number, pattern of distribution, or type of surviving neurons and axons can be modified at critical stages by the deletion of the neurons with which they have a direct synaptic relationship or more distant trophic dependence.





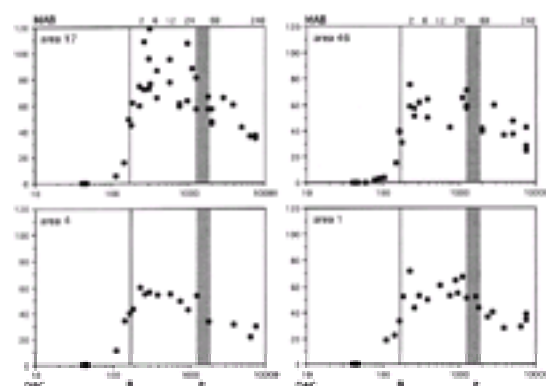
**Figure 3.11.** The estimated total number of axons in corpus callosum as a function of prenatal and postnatal ages in postconceptual days. Each point represents a single animal. Error values (<14%) are smaller than or equal to the size of the dots used to represent each data point. The hatched line indicates the average total number of callosal axons in eight adult monkeys. (From LaMantia AS, Rakic P: Axon overproduction and elimination in the corpus callosum of the developing rhesus monkey. *J Neurosci* 10:2156–2175, 1990.)

The biphasic development of cortical connections, which proceeds from initially diffuse to more sharply defined terminal fields, has been first and, perhaps, best documented in the primate visual area (Hubel et al., 1977; Rakic, 1976a). Subsequently, a similar developmental scenario also has been shown in diverse structures in a variety of species (Easter et al., 1985; Innocenti, 1981; O'Leary, 1989; Purves, 1988). In primates, for example, terminals of the corticostriatal projections in the caudate nucleus are initially distributed in a diffuse manner, across wide territories, before synapses retract from the sites that eventually become occupied by later-arriving afferent systems (Goldman, 1978; Goldman-Rakic, 1981b). These two examples, one afferent (geniculocortical) and the other efferent (corticostriatal), reveal that the formation of both input and output to the cortex may be achieved through dynamic competitive cellular interactions that occur prenatally and involve at least two well-defined steps. In the first step, several input systems project to the target structure in a diffuse and overlapping manner, without regard to its specific target area, the individual nerve cell, or its part. In the second phase, connections sort out and form more selective contacts with specific sets of neurons or their dendrites. However, unlike in cats, in primates, this rearrangement mostly occurs before birth and, thus, does not involve visual experience (Hocking and Horton, 1998; Rakic, 1976a, 1977). The finding that the ocular dominance columns can develop even in the absence of input from both eyes (Crowley and Katz, 1999, 2000) supports the hypothesis that the basic pattern of connectivity is innate, rather than experience dependent.

The functional significance of the large loss of axons in the cerebral cortex is not fully understood. In primates, it occurs after topographic and columnar organization has been achieved (LaMantia and Rakic, 1990; Schwartz and Goldman-Rakic, 1991) and therefore is not involved in establishing a basic pattern of connections. Likewise, in the adult rhesus monkey, area 17 does not have callosal connections, and unlike the developing cat, this region is never interconnected (Kennedy and Dehay, 1993). Callosal axons in the prefrontal cortex are interconnected with appropriate areas already in the middle of gestation, well in advance of the axonal loss phase that in this species occurs mostly postnatally (Schwartz and Goldman-Rakic, 1991). Therefore, axonal elimination in the cerebral cortex is probably involved in synaptic remodeling at the local rather than global level (LaMantia and Rakic, 1990). Indeed, as discussed in the following, loss of interhemispheric axons coincides with the period of rapid synaptic production rather than synaptic elimination.

### OVERPRODUCTION OF CORTICAL SYNAPSES

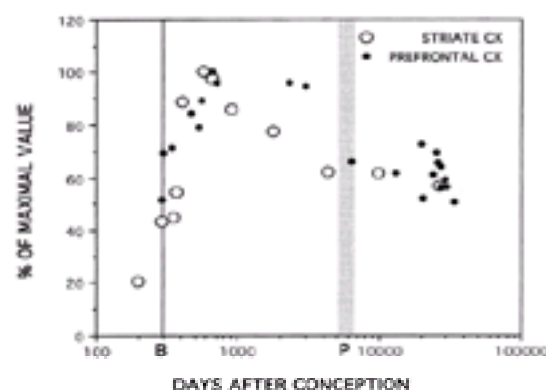
In most structures of the vertebrate brain, where quantitative data has been obtained with reasonable accuracy, both the density of synapses and their total number are higher during one stage of development than in the adult state (Easter et al., 1985; Purves, 1988). The cerebral cortex is not an exception to that rule, and the magnitude of the overproduction in this structure is even more pronounced. A bewildering number of growing axons eventually must establish synaptic junctions with a specific neuron class in each cortical area or, even more precisely, with only a part of their body and dendritic tree. In the primate cerebral cortex this precise pattern of synaptic connectivity in the target structure is achieved through a marked overproduction and subsequent elimination. For example, the cerebral cortex in both human and macaque monkeys contains more synapses during infancy than in adulthood (Bourgeois and Rakic, 1993; Huttenlocher and de Courten, 1987; O'Kusky and Colonnier, 1982; Rakic et al., 1986; Zielinski and Hendrickson, 1992). In the newborn monkey, the density of synapses per unit volume of neuropil and their total number is approximately equal to the adult values. However, during the first 2 to 3 months of life, synaptic density continues to increase until it reaches about 40% more than in an adult (Fig. 3.12). The phase of high synaptic density lasts throughout adolescence and decreases significantly during sexual maturation, which in this species occurs during the third year of life (Rakic et al., 1986, 1994a). The decline in number of synapses owes primarily to the elimination of asymmetric junctions located on dendritic spines, whereas synapses on dendritic shafts and cell bodies remain relatively constant. The course of decline in density and absolute number of synapses is perhaps best documented for the macaque monkey primary visual cortex. The number of synapses lost in area 17 of a single cerebral hemisphere is about  $1.8 \times 10^{11}$  (Bourgeois and Rakic, 1993). The magnitude of this loss is stunning when expressed as a loss of about 2,500 synapses per second in the striate area of each hemisphere during a period of about 2 to 3 years.



**Figure 3.12.** Histograms of the density of synapses per  $100 \mu\text{m}^3$  of neuropil in the primary visual (area 17), prefrontal association (area 46), primary motor (area 4), and primary somatosensory (area 1) at various ages. Each black circle represents the value obtained from a single electron microscopic probe, and data are corrected and transferred into N per volume according to the Anker and Cragg formula. The time is represented on a semilogarithmic scale. B, birth; DAC, days after conception; MAB, months after birth; P, days after puberty. (Data compiled from Bourgeois J-P, Rakic P: Changing of synaptic density in the primary visual cortex of the rhesus monkey from fetal to adult stage. *J Neurosci* 13:2801–2820, 1993; Bourgeois J-P, Goldman-Rakic PS, Rakic P: Synaptogenesis in the prefrontal cortex of rhesus monkey. *Cereb Cortex* 4:78–79, 1994; Zecevic and Rakic, 1991; Zecevic et al., 1989.)

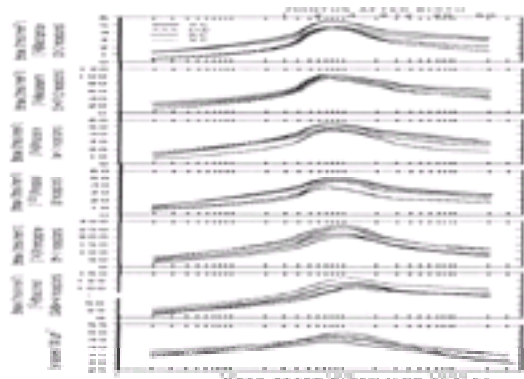
Quantitative analysis of synaptogenesis in the motor (Zecevic et al., 1989), somatosensory (Zecevic and Rakic, 1991), visual (Bourgeois and Rakic, 1993), prefrontal (Bourgeois et al., 1994), and limbic (Granger et al., 1995) cortex in the macaque monkey indicates that a basic course of synaptogenesis, and, in particular, the ascending phase, occurs concomitantly (Fig. 3.12). Thus, contrary to the prevailing views, the entire population of synaptic junctions in the association Brodmann's area 46 matures concurrently with, rather than after, the primary sensory and motor areas. Although during this exponential phase there is a sequential accretion of some classes of synapses from diverse origins, overall the course of synaptogenesis is framed within a "window of time" centered on the exponential phase, which is common for all areas of the neocortex (Bourgeois et al., 1999).

The cortical synapses in humans also seem to form synchronously in diverse cortical areas, although the available data initially suggested a delay in the rapid phase of synaptogenesis in the prefrontal, as compared to that in the visual striate area (Huttenlocher, 1979; Huttenlocher and de Courten, 1987). However, if one eliminates the technical and procedural differences, the course of synaptogenesis in human and monkey is remarkably similar across the entire cortex. For example, when the data on synaptogenesis obtained from the human prefrontal (Huttenlocher, 1979) and human visual cortex (Huttenlocher and de Courten, 1987) were normalized and replotted to the maximum value of the curve on a semilogarithmic scale (Bourgeois et al., 1999; Rakic et al., 1994a), both curves overlap, as they do in the monkey (Fig. 3.13). Moreover, by means of linear regression analysis, the two sets of values were fitted to a straight line, and statistical comparison between the two regression lines failed to reveal a significant difference in their slopes. Therefore, it appears that synaptogenesis both in humans and nonhuman primates proceeds synchronously in the sensory and association cortex (Rakic et al., 1994a).



**Figure 3.13.** Synaptogenesis in human cerebral cortex. Synaptic density in the human striate area (open, large circles) and prefrontal cortex (black, small circles) based on the studies of [Huttenlocher and de Courten \(1987\)](#) and [Huttenlocher \(1979\)](#), respectively. We replotted each point from their published data on a semilogarithmic scale as a function of conceptual age and normalized to the maximum value of the curve. Under these conditions, both curves overlap as they do in monkeys. For further explanation see text. (From Huttenlocher PR, de Courten C: The development of synapses in striate cortex of man. *Hum Neurobiol* 6:1–9, 1987; Huttenlocher PR: Synaptic density in human frontal cortex—developmental changes and effects of aging. *Brain Res* 163:195–205, 1979; Rakic P, Bourgeois J-P, Goldman-Rakic PS: Synaptic development of the cerebral cortex: Implication for learning, memory, and mental illness. *Prog Brain Res* 102:219–235, 1994a.)

The time course of synaptogenesis in different cortical areas corresponds well with changes occurring simultaneously in cerebral metabolism during development of both the human and macaque cerebral cortex ([Chugani et al., 1987](#)). In humans, the use of fluorodeoxyglucose, which indicates the level of metabolic activity in positron emission tomography, reveals that, after birth, metabolic activity also increases concurrently in the prefrontal, motor, somatosensory, and visual cortex ([Chugani et al., 1987](#)). These studies lend support to the idea that maturation of diverse cortical areas in both monkey and human occurs simultaneously rather than in a pronounced sequential order. Further, the synchrony in synaptogenesis observed in the nonhuman primate is in harmony with biochemical and functional data on cortical maturation in the same species. Biochemical studies ([Goldman-Rakic and Brown, 1982](#)) suggest that concentrations of dopamine, noradrenaline, and serotonin increase rapidly in the cortex of the macaque monkey over the first 2 months and approach adult levels by the fifth month after birth. Recent studies of the accumulation of major neurotransmitter receptor sites in different cortical areas show that their maximum density is also reached between 2 and 4 months after birth in this species ([Lidow et al., 1991](#); [Lidow and Rakic, 1992](#)). The curves of the increase in receptor density are very similar to those of synaptogenesis in all areas examined irrespective of level of activity ([Bourgeois et al., 1989](#)) (Fig. 3.14). However, the phase of decline of receptor density appears to slightly precede the phase of synaptic decline. These observations from divergent cortical areas suggest that initial formation and maintenance of synapses, as well as their biochemical maturation, may be determined by intrinsic signals, which are common to the entire cortical mantle.



**Figure 3.14.** Developmental changes in the overall changes (across all layers) in density of the synapses and specific binding of radioligands labeling a representative selection of neurotransmitter receptor subtypes in the prefrontal, primary, motor, somatosensory, and primary visual cortical regions of the developing rhesus monkey. For receptor densities, the lines were obtained by locally weighted least square fit with 50% smoothing (KALEIDA GRAPH, Synergy Software, Reading, PA) based on mean  $B_{max}$  values obtained from the measurements of the entire cortical thickness in at least two animals at birth, 1, 2, 4, 8, 12, 36, and 60 months of age. Age is presented in postnatal days on a logarithmic scale. (The primary data are from Lidow MS, Goldman-Rakic PS, Rakic P: Synchronized overproduction of neurotransmitter receptors in diverse regions of the primate cerebral cortex. *Proc Natl Acad Sci USA* 88:10218–10221, 1991; Lidow MS, Rakic P: Postnatal development of monoaminergic neurotransmitter receptors with primate neocortex. *Cereb Cortex* 2:401–415, 1992; Rakic P, Bourgeois J-P, Eckenhoff ME: Concurrent overproduction of synapses in diverse regions of the primate cerebral cortex. *Science* 232:232–235, 1986.)

The concurrent course of synaptogenesis and formation of neurotransmitter receptors in functionally different areas of the cerebrum is at variance with the tacit assumption, widespread in the literature, that development of the cerebral cortex follows an hierarchical sequence of structural and functional development from sensory to motor and, finally, to the association cortex ([Filimonov, 1949](#); [Flechsig, 1920](#); [Greenfield, 1991](#); [Yakovlev and LeCourse, 1968](#)). We argue that myelination of large axonal tracts is not a very useful criterion of functional maturation of a given cortical area and, furthermore, that hematoxylin staining of sections used to measure the amount of myelin is not a reliable quantitative method. There are clear exceptions to the hierarchical rule of cortical maturation even in these studies. For example, the corticospinal motor system is among the last to myelinate and becomes stained by hematoxylin only during the second year of life, well after association pathways are myelinated ([Yakovlev and LeCours, 1978](#)). On the other hand, concurrent structural (synaptic) and biochemical (neurotransmitters, receptors) maturation of the cortical mantle appears reasonable when one takes into account that integration of sensory, motor, limbic, and associative components are essential for even the simplest cortical functions ([Goldman-Rakic, 1987a, 1988](#)). This concept of concurrency becomes even more compelling when one considers the coupling between early maturation of the neuronal circuits of the cortex and the first expression of cognitive capacity that requires coordination of sensory and associational cortical areas ([Spelke et al., 1994](#)).

Isochronic development of anatomically and functionally diverse regions indicates that establishment of cell-to-cell communication in this structure may be orchestrated by a single genetic or humoral signal. This phase of primate life may provide a long period of unparalleled opportunity for competitive, activity-driven stabilization among various initially overproduced intercortical and intracortical connections, which comprise the largest fraction of cortical synapses ([Goldman-Rakic, 1987a](#)). This period of supernumerary synapses in the cerebral cortex can be considered as a stage in life with maximal opportunity and minimal commitments, thus providing an enormous window of opportunity for the generation of cortical diversity beyond genes.

## SYNAPTIC ELIMINATION AND FUNCTIONAL VALIDATION

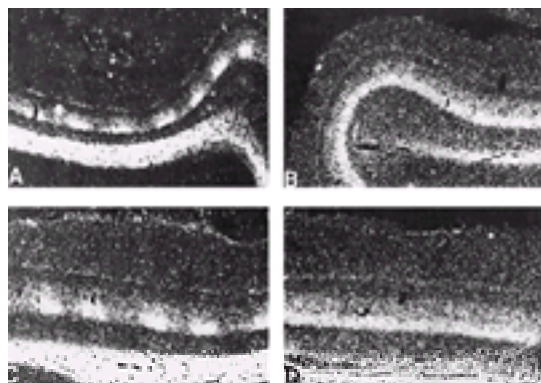
Studies of the cerebral cortex in a variety of mammalian species support the hypothesis of competitive interactions as a mechanism of attaining point-to-point connectivity during segregation of initially more numerous and diffuse projections ([Easter et al., 1985](#); [Edelman, 1987](#)). The balance between overproduction and elimination of neurons and axons and competition between them determines the size and site of territories devoted to a given terminal field. The first, and perhaps the best-documented example of developmental processes, which proceeds from diffuse to sharply defined terminal fields, is the primate binocular visual system ([Hubel et al., 1977](#); [Rakic, 1976a, 1977, 1991b](#)). This basic principle has since been shown in a variety of cortical areas ([Easter et al., 1985](#); [Innocenti, 1981](#); [O'Leary, 1989](#); [Shatz, 1983](#)). Perhaps the most prominent cellular mechanism for selective elimination of inappropriate and less used synapses is the process of sorting out connections through competition. When it occurs during postnatal development and is associated with sensory stimulation, it is also known as the Hebbian mechanism or synaptic stabilization ([Changeux, 1993](#); [Hebb, 1949](#)). However, in either case, this pruning involves elimination of already formed synapses, rather than creation of new connections.

After the number and density of synapses in the cerebral cortex reach adult levels, they remain relatively stable throughout the life span in both humans and nonhuman primates ([Bourgeois et al., 1999](#); [Huttenlocher and de Courten, 1987](#); [Rakic et al., 1986, 1994a](#)). Obviously, even though we learn and acquire an enormous amount of information during adulthood, the number of synapses in the cerebral cortex during this period remains basically the same. From crude



quantitative analysis of the overall density and number of synapses, one cannot exclude the possibility that certain classes of synapses are constantly being produced, whereas others are being eliminated in the same proportion (Rakic et al., 1986). However, the absence of profiles of immature synapses or their early forms, as well as the lack of hard ultrastructural evidence for their degeneration under normal conditions, suggests that the turnover of the synaptic population in primates must be either absent or negligible. Based on these findings, we argue that during infancy, childhood, and adolescence, learning basic skills and formation of intellectual capabilities may be associated with considerable changes in the population of synapses by both formation and elimination of selected synaptic junctions, as well as strengthening of their efficacy. In contrast, after puberty, learning and memory probably depend primarily on changes in the strength of already existing synapses. It should be pointed out that this concept does not negate the capacity for functional plasticity that remains in the adult cerebral cortex (Merzenich, 1988), nor does it contradict the existence of competitive interactions based on strengthening of the selected connections in response to stimulation (Edelman, 1987; Shatz, 1990). However, this hypothesis could explain the substantial qualitative and quantitative differences in learning capability before and after sexual maturity. This mode of learning may be particularly important in primates, who depend on retention of acquired knowledge by a permanent set of neurons and their connections during a prolonged life span (Rakic, 1985).

The theory of competitive elimination of synapses during development of the cerebral cortex was initially obtained from studies of monocular eye deprivation, which resulted in a decrease in the size of ocular dominance columns subserving the occluded eye and enlargement of the functional eye (Hubel et al., 1977). This was the first clear anatomic evidence that the pattern of neuronal connectivity can be visibly changed by a brief deprivation of function during the critical postnatal period of development. Even more dramatic changes are observed when the eye in a monkey fetus is enucleated before geniculocortical fibers have entered the cortical plate (Rakic, 1981a, 1983). In such cases, there is no trace of ocular dominance columns in the visual cortex; therefore, neurons that normally receive input from, and subserve the left eye now receive input from the right eye (Fig. 3.15). Retino-geniculo-cortical connections fail to withdraw to appropriate territories even after the blockade of electrical activity in one eye by tetrodotoxin (Dubin et al., 1986; Stryker and Harris, 1986). Some additional experiments performed on the developing cerebral cortex are reviewed in the following to illustrate the extent of changes in neuronal organization that can be induced by interference with the course of normal development.



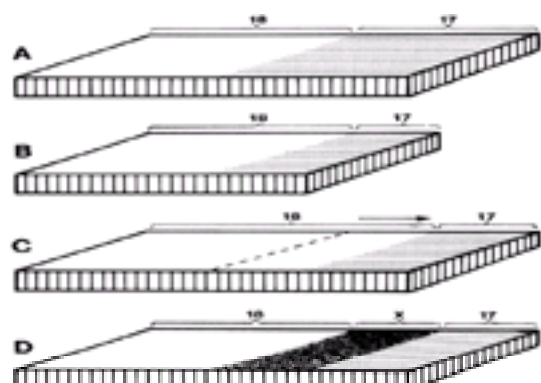
**Figure 3.15.** Dark-field illumination photomicrograph of autoradiograms, showing distribution of transneuronally transported radioactive tracers in the primary visual cortex of normal adult monkeys (A and C) and in adult monkeys in whom one eye was enucleated during the first third of pregnancy (B and D). Note the lack of alternating ocular dominance columns in the cortex of experimental animals. The distribution of silver grains over sublayers of layer IV remains similar to that in the control. (From Rakic P: Genuculo-cortical connections in primates: Normal and experimentally altered development. *Prog Brain Res* 58:393–404, 1983.)

## EXPERIMENTAL MANIPULATION OF THE CORTICAL PROTOMAP

The cerebral cortex is basically an analyzer that associates diverse inputs originating from various sensory receptors communicated to its neurons via precisely and systematically organized thalamocortical connections. Although information essential for developing a basic species-specific pattern of these connections must be imprinted in our genome (see the preceding), interference with normal interaction with information derived from the periphery, where stimulation arises, can have a devastating effect. The role of afferents in the development of cortical parcellation can be examined by altering the number of axons comprising specific thalamocortical systems at early embryonic stages and determining the size and cytoarchitectonic pattern in corresponding target areas of the cortex in adult animals. For example, binocular enucleation performed in the monkey embryo around E60 reduces the number of geniculate neurons to less than one-half the number in age-matched controls without altering any other component in the thalamocortical system (Rakic, 1988a; Rakic et al., 1991). Furthermore, the occipital lobe in early enucleates displays dramatic and remarkably reproducible changes in convolutions of the occipital operculum. These changes are milder in later-operated cases and cannot be induced by downsizing geniculocortical input during the second half of gestation. Use of anterograde and retrograde transport of axonal tracers in early enucleates reveals the presence of topographically well-defined reciprocal connections of the occipital lobe with a vestige of the lateral geniculate nucleus (Rakic, 1988a).

In specimens with an experimentally produced smaller lateral geniculate, area 17 is well differentiated from adjacent area 18, and its thickness and characteristic laminar pattern are surprisingly normal. However, the surface area and the total number of neurons in area 17 in the early-enucleated subjects are less than half those in age-matched controls (Rakic et al., 1991). Despite the drastically reduced number of geniculocortical afferents, area 17 contained the normal number of neurons per unit volume of each layer and per each radial column. The distribution of major neurotransmitter receptors in early enucleates, in spite of some modification, retained the basic laminar pattern characteristic of area 17 (Rakic and Lidow, 1995). Furthermore, cytochrome oxidase blobs in layers II and III, which are thought to subserve color and form vision (Livingstone and Hubel, 1984), were segregated and maintained in the visual cortex of early binocular enucleates (Kuljis and Rakic, 1990). Finally, synaptic density per unit of neuropil, as revealed by quantitative electron microscopy, achieved normal range in all layers (Bourgeois and Rakic, 1996). These results indicate that the basic cytologic, synaptic, and biochemical characteristics of area 17 can develop in the absence of stimulation from the retina. However, the number of ontogenetic columns and therefore the size of the surface of area 17 is reduced to match the number of geniculocortical axons in the enucleated animals (Rakic, 1988a).

The reduction in size of area 17 in animals enucleated at early embryonic ages could result from either creation of fewer radial columns or from an increase in their elimination (Fig. 3.16). However, neither possibility seems likely because enucleation was performed after all proliferative units in the ventricular zone should have been formed, and cell death restricted to entire columns of cells has never been observed. It is possible, therefore, that the total number of ontogenetic columns in the cortices of animals that were operated on remained the same and that the adjacent cytoarchitectonic area, which normally receives input mainly from the adjacent thalamic nucleus (pulvinar), expanded (Fig. 3.16). Such changes would require respecification of neurons genetically programmed to become part of area 17 to accommodate input characteristic to area 18. This mismatch may create a “hybrid” cortex (Fig. 3.16) that retains some characteristics of area 17, but takes on some features of area 18 as a result of receiving a different set of afferents (Rakic, 1988a). These findings may provide a hint about how a new type of cytoarchitectonic area can be introduced by input-target mismatching during evolution (Rakic et al., 1991).

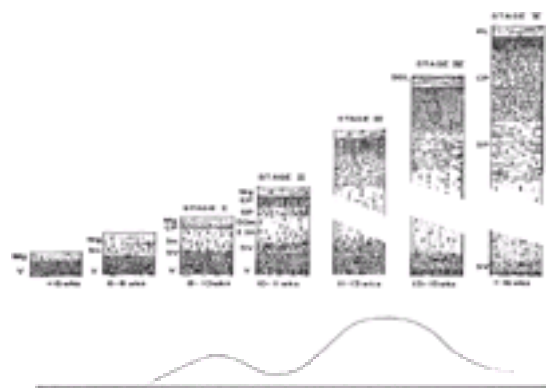


**Figure 3.16.** Schematic representation of the possible modes of decrease in the size of area 17 caused by experimental reduction of thalamic input. A: Relation between areas 17 and 18 in a normal animal. B: Differential cell death. C: Encroachment of adjacent area 18 into the territory of area 17. D: Formation of an abnormal cytoarchitectonic area (X) consisting of neurons genetically destined for area 17 that receive input characteristic for area 18. Further explanation is in the text. (From

Regulation by thalamic input to the developing cortical plate is probably only one part of a complex interactive process that occurs during parcellation of the neocortex. For example, prenatal resection of the fetal cortex, which eliminates or decreases the amount of cortico-cortical input to the subplate zone at early stages, also affects the pattern of cortical convolutions in some unoperated areas on both sides ( [Goldman-Rakic, 1980](#); [Goldman-Rakic and Rakic, 1984](#)).

## TIMING OF CORTICAL HISTOGENESIS IN HUMANS

Because our ultimate goal is to learn more about human neocortical development, it is important to determine whether the development of the human cortex proceeds according to time schedules relative to birth and shares the same cellular and molecular mechanisms uncovered in nonhuman primates. A comparison of some of the morphologic features of the cortex in human fetuses of different ages ( [Kostovic and Rakic, 1980, 1990](#); [Poliakov, 1949, 1959, 1965](#); [Rakic and Sidman, 1968](#); [Sidman and Rakic, 1973](#)) with those of the monkey may help to determine the corresponding time and sequence in these two species. Poliakov's comprehensive histologic studies of cortical development in humans have been reviewed in more detail elsewhere ( [Sidman and Rakic, 1982](#)) and are briefly summarized here and illustrated in [Fig. 3.17](#).



**Figure 3.17.** Semidiagrammatic drawings of the human cerebral wall at various gestational ages, listed in fetal weeks below each column. The stages refer specifically to an arbitrarily chosen area midway along the lateral surface of the hemisphere. (Detailed in Sidman RL, Rakic P: Development of the human central nervous system. In: Haymaker W, Adams RD (eds): *Histology and Histopathology of the Nervous System*. Springfield, IL, Charles C Thomas, 1982, pp. 3–145.) In addition, the subplate zone, situated below the cortical plate, appears in the last three stages. (Kostovic I, Rakic P: Developmental history of transient subplate zone in the visual and somatosensory cortex of the macaque monkey and human brain. *J Comp Neurol* 297:441–470, 1990.) Because there is a gradient of maturation, as many as three of five stages of cortical development may be observed in different regions of the neocortex in the same fetal brain. In the three columns on the right, the intermediate zone is not drawn in full because the thickness of the cerebral wall has increased markedly compared with earlier stages and cannot fit into the drawing. The curve below the drawing schematically indicates waves of cell migration to the neocortex assessed by the density of migrating neurons in the intermediate zone. CP, cortical plate; Im, intermediate zone; I.Im and O.Im, inner and outer intermediate zones, respectively; Mg, marginal zone; PL, plexiform layer; SGL, subpial granular layer; SP, subplate zone; SV, subventricular zone; V, ventricular zone; wks, age in fetal weeks. (From Rakic P: Defects of neuronal migration and pathogenesis of cortical malformations. *Prog Brain Res* 73:15–37, 1988b, with permission.)

### Stage I. Initial Formation of the Cortical Plate (Sixth to Tenth Fetal Week)

During the seventh fetal week, postmitotic cells begin to migrate from the ventricular zone outward to form a new accumulation of cells at the junction of the intermediate and marginal zones. Already, by the middle of this period, synapses of unknown origin are present above and below the cortical plate ( [Molliver et al., 1973](#)). This stage corresponds approximately to the level of cortical development found in the monkey fetus between E40 and E54, depending on the region.

### Stage II. Primary Condensation of the Cortical Plate (Tenth and Eleventh Fetal Weeks)

At this stage, the cortical plate increases in thickness, becomes more compact, and is clearly demarcated from the fiber-rich part of the intermediate zone, which seems to have fewer cells per unit volume, indicating that the first major wave of migration is almost spent ( [Fig. 3.17](#)). The end of this stage corresponds approximately to the E55–E59 period in the monkey when the majority of efferent neurons of layers 6 and 5 are generated in most regions of the cortex.

### Stage III. Bilaminate Cortical Plate (Eleventh to Thirteenth Fetal Week)

The uniform and compact cortical plate of the second stage becomes subdivided into an inner zone occupied mainly by cells with relatively large, somewhat widely spaced oval nuclei and an outer zone of cells with densely packed, darker, bipolar nuclei ( [Fig. 3.17](#)). This heterogeneity results from the more advanced maturation of the deep-lying neurons that arrived at the cortical plate during earlier developmental stages, plus the addition of a new wave of somas of immature neurons that take up more superficial positions. This period is also characterized by the appearance of the cell-sparse, fiber-rich subplate zone situated below the cortical plate. This transient embryonic zone in the human fetus is particularly wide in the regions subjacent to the association areas ( [Kostovic and Rakic, 1990](#)). Stage III roughly corresponds to the level of development achieved in the monkey between E59 and E64.

### Stage IV. Secondary Condensation (Thirteenth to Fifteenth Fetal Week)

During this period of gestation, the ventricular zone becomes progressively thinner, whereas the subventricular zone remains relatively wide ( [Fig. 3.17](#)). The cortical plate again becomes homogeneous in appearance and resembles, in a sense, a thickened version of Stage II. The reason for this change may be that, in Stage IV, most of the young neurons in the cortex become considerably larger as they differentiate, whereas relatively few new immature neurons enter the cortical plate. The result is a more uniform appearance. At the end of this stage, an accumulation of large cells appears below the cortical plate and the subplate zone enlarges further ( [Kostovic and Rakic, 1990](#)). Depending on the cortical region, this stage appears in the monkey between E64 and E75.

### Stage V. Sixteenth Fetal Week into the Postnatal Period

Morphologic data are inadequate in determining the length of time and the quantity of neurons that continue migrating to the human neocortex after 16 weeks; hence, the line at the right side of the curve is dotted in [Fig. 3.17](#). By the fifth lunar month, relatively few neuron precursors seem to be proliferating in the reduced ventricular zone of the human cerebral hemispheres, although many neurons generated prior to the 16th week have yet to reach the cortical plate. Comparison of the <sup>3</sup>H-thymidine autoradiographic results in the monkey ( [Rakic, 1974, 1977](#)) with comparable stages in humans ( [Kostovic and Rakic, 1990](#)) indicate that probably all neurons of the human neocortex are generated before the middle of gestation. Toward term, the subplate zone dissolves, and, as the intermediate zone becomes myelinated and transforms into white matter, only a vestige of the subplate neurons remain as interstitial neurons ( [Kostovic and Rakic, 1980](#)). After all cortical neurons have been generated and attained their final position, their differentiation, including formation of synapses, continues to proceed until reaching a peak during the second postnatal year. The subject of synaptogenesis in the cerebral cortex of both monkey and humans has been described in the preceding sections.

## RELEVANCE TO MENTAL ILLNESS

It is generally agreed that many psychiatric and neurologic disorders that become expressed in childhood and adolescence have a genetic and developmental background, but the exact time and place of the onset of a defective process has been difficult to place. Advances made in understanding normal cortical development provide insight into the pathogenesis of some inherited and acquired cortical malformations observed in animals and humans. However, the biological



bases of many psychiatric disorders, such as schizophrenia, obsessive-compulsive disorders, or various forms of congenital mental retardation have been more elusive. In such cases, methodologic limitations allow even relatively large changes in the relative size of cortical areas or in the synaptic number to remain undetected. In other cases, the defect may be biochemical, but only quantitative analysis of the distribution of the involved molecules could reveal the effects. A few examples are provided here of the visible and relatively well-defined disorders that are explicable in light of the present knowledge of corticogenesis that was reviewed on the preceding pages. These examples are selected because they also elucidate the main principles governing the development of the cerebral cortex.

One rare condition that is highly relevant for understanding development of the cerebral cortex is the formation of the cortex in the absence of sensory input from early stages of development. For example, it is surprising but well established that, when a child is born without both retinas, area 17 has a normal thickness and lamination but a greatly reduced surface ([Bolton, 1900](#); [Brunquell et al., 1984](#)). Although the cause of most cases of congenital anophthalmia is unknown, the result is wholly explicable by findings from bilateral enucleation experiments ([Rakic, 1988b](#); [Rakic et al., 1991](#)), which indicate that this cortical abnormality is caused by a defect in eye formation during the first third of gestation. The resection of retinas during the first third of gestation, provide a useful model system to study the pathogenesis of secondary anophthalmia. So far, it has been learned that the visual cortex in these animals develops a normal density of synapses and normal distribution and binding intensity of the major neurotransmitter receptors ([Bourgeois and Rakic, 1996](#); [Rakic and Lidow, 1995](#)). Other pertinent examples of cortical malformation are focal malformations found in both humans and mice, in which only a segment of the cortex confined to several radial units is highly disturbed and sharply delineated from adjacent areas ([Nowakowski, 1984](#); [Sherman et al., 1985](#)), indicating that only cells originating from a small part of the ventricular zone have been affected.

The classification of most cortical malformations in humans is traditionally based on the appearance of the cerebral cortex at autopsy. However, advances made in developmental neurobiology in the past decade allow some new interpretations. For example, according to the radial unit hypothesis, the pathogenesis of some cortical malformations can be classified into two major categories ([Rakic, 1988a,b](#)). The first category comprises malformations in which the number of radial units in the cortex is reduced, whereas the number of neurons within each ontogenetic column remains relatively normal. It can be expected that defects in this category result from an early-occurring event that alters the number of proliferative units at the time they are being formed in humans within the first 6 weeks of gestation. Once the number of proliferative units in the ventricular zone is established, albeit in fewer numbers, each unit can produce a normal or even greater number of neurons, which become crowded in the diminished cerebral vesicle. It could be expected that the cortex would have a smaller surface area in spite of a normal or enlarged thickness and the presence of massive neuronal ectopias in the white matter. These features are actually observed in some human malformations, such as lissencephaly and pachygyria ([Hong et al., 2000](#); [Volpe, 1987](#)).

The second category consists of malformations in which the initial formation of radial units is not affected, whereas the number of neurons within ontogenetic columns is reduced. The defect in this category should begin after the sixth fetal week, when the normal complement of proliferative units has already been established. Such malformations can be caused by interference with cell proliferation via intrinsic (genetic) or extrinsic (irradiation or viral infection) factors. Diminished production of neurons in the proliferative units results in fewer neurons within ontogenetic columns; therefore, the cortex is thinner. The number of neurons in ontogenetic columns could also be affected by cell death or by a failure of their migration. In the latter case, some neurons may survive in ectopic positions within the white matter. All of this can be observed in the so-called polymicrogyria brain ([Volpe, 1987](#)).

It should be recognized that only few cortical malformations have features of only one or the other general category described in the preceding, but in practice, most show a mixture of both. The proposed classification suggests possible developmental mechanisms by separating defects of unit formation from defects of ontogenetic column formation. In support of the radial unit hypothesis and the concept of a cortical protomap, experimental and neuropathologic data reveal that each step (formation of proliferative units, formation of ontogenetic columns, and formation of cytoarchitectonic areas) can be separately affected by genetic defects or by extrinsic factors ([Haydar et al., 1996](#); [Rakic, 1988b](#); [Sidman and Rakic, 1982](#)). It can, therefore, be predicted that genetic alteration as well as mechanical, chemical, or viral lesions of distant but synaptically related structures that result in reduced input to the cortex would affect subsequent developmental events and provide the setting for new cell relationships, the net outcome of which could be the emergence of a unique cytoarchitectonic map.

Of particular relevance to the psychiatric disorders of adolescence may be abnormalities that are associated with phases of synaptic overproduction and elimination. It is well established that many major psychoses, including schizophrenia, become manifest at the end of adolescence and in early adulthood. In addition, a growing number of studies of postmortem tissue samples from schizophrenics and of magnetic resonance images of the brains of living patients have provided a solid factual basis for morphological signs, such as enlarged ventricles, which predate the illness ([Weinberger, 1987](#)). The evidence for a genetic predisposition to schizophrenia ([Kety et al., 1968](#)) has naturally led investigators to consider the developmental antecedents of this major psychosis, and several hypotheses have invoked events related to synaptogenesis ([Bloom, 1993](#)). For example, it was proposed that schizophrenia might be caused by a fault in programs of synaptic elimination during adolescence ([Feinberg, 1982](#)). More recently, this concept has gained support by the finding that cortical neurons in schizophrenics have fewer synaptic contacts as judged by the reduction in neuropil ([Goldman-Rakic and Selemon, 1997](#); [Selemon et al., 1997](#)) by a computational model, which tests predictions relevant to particular symptoms in schizophrenia ([Hoffman and McGlashan, 1993](#)).

More direct evidence for the possibility of excessive pruning comes from recent studies of noninvasive methods in living patients ([Pettegrew et al., 1993](#); [Williamson et al., 1991](#)). In these studies, the structural integrity of the prefrontal cortex in drug naive schizophrenics shows excessive elimination of dendrites and axons. Complementing the *in vivo* studies ([Selemon et al., 1997](#)) found that there is a higher density of neurons per unit volume in prefrontal areas of schizophrenics compared with that in controls—a finding compatible with the loss of synaptic neuropil in schizophrenia. [Glanz \(1993\)](#) have reported preliminary evidence of reduced synaptophysin labeling in the prefrontal cortex of schizophrenics. The suggestion that Notch signaling pathway may be involved in regulation of synaptic pruning ([Sestan et al., 1999](#)) opens the possibility to test this hypothesis. These studies indicate a pathologic process at work in the various cortical regions of patients—findings that are compatible with a neurodevelopmental perspective. Although these changes occur at inflection points in the synaptogenesis curve, there is no direct evidence that they are causative in nature. It is likely, however, that knowledge of the normal course and mechanisms of synapse formation and the influence of various exogenous and endogenous events on synapse stability and turnover are essential prerequisites to determining the locus and timing of etiologic factors in diseases that affect the cortex and alter cognitive function. This is the ultimate justification for studies in nonhuman primates, where conditions are experimentally controlled and monitored.

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## 4 NEUROCHEMISTRY OF CHILDHOOD PSYCHIATRIC DISORDERS

George M. Anderson, Ph.D. and Donald J. Cohen, M.D.

[Neurochemical Research: Aims, Scope, and Methods](#)

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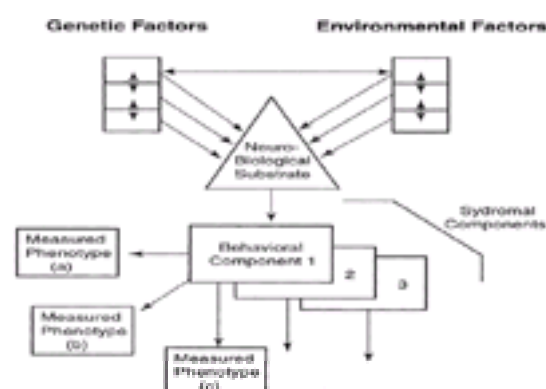
[Chapter References](#)

Accelerating advances in basic neuroscience have led to a much improved understanding of brain development and functioning and hold the tantalizing promise of understanding the alterations that may exist in childhood psychiatric disorders ( [Andreasen, 1997](#); [Kandel and Squire, 2000](#)). Research on the molecular control of developmental neurobiology ( [Chapter 1](#)) is particularly relevant. The recent elucidation of the genetic bases of a number of single-gene childhood psychiatric disorders ( [Chapter 2](#)) provides additional hope, and to some extent paradigms, for tackling the more complex molecular and biological influences in autism, attention deficit/hyperactivity, Tourette syndrome, anxiety, posttraumatic stress disorder (PTSD), depression, and suicide. Identification of causative factors in Parkinson's disease, Huntington's disease, Alzheimer's disease, and other adult neurologic and psychiatric disorders further encourages the notion that the biological bases of childhood psychiatric disorders can be ascertained.

Along with the successes in identifying the genetic and etiologic bases for syndromes like fragile X and Rett, there is accumulating evidence that genetics plays a large role in all of the common childhood psychiatric disorders. Thus, family and twin studies of adults and children have left no doubt that genetics and biology often can be even more important than environmental factors in influencing normal and abnormal behavior; however, the last decade has been one of disappointment in trying to find single gene or loci explanations for the more common syndromes and symptoms of child psychiatry. This research has made it increasingly clear that the genetics and biology of the common disorders are complex, in that multiple genes appear to contribute to the syndromes ( [Benjamin and Gershon, 1996](#); [State et al., 2000](#)). This is not surprising given the complex and diffuse nature of the neural systems that subserve the relevant brain processes ( [Abercrombie and Zigmond, 1995](#); [Heninger, 1999](#)). The genetic complexity is further multiplied by the wealth of variation arising from gene-environment interactions: illuminating the complexity will require the application of new and appropriate genetic methods ( [Burmeister, 1999](#); [Collier et al., 2000](#); [Pauls, 2000](#); [Petronis and Gottesman, 2000](#); [Weiss and Terwilliger, 2000](#)).

Recognition of the importance of genetics and complexity of the disorders is changing in fundamental ways how the disorders or behaviors are viewed; thus, the field of biological psychiatry is changing in its approach. Whether the field is termed "molecular psychiatry," "biological psychiatry," or "clinical neuroscience" is of little consequence; fundamental changes are being prompted by a better appreciation of the scope of the endeavor. An emerging overarching theme is that all biological, neuropharmacologic, and behavioral investigation needs to be performed in a genetic context. There is also a greater focus on components or domains of behavior and a greater interest in quantifying the traits or variables of interest. This move toward examining components is an understandable attempt to simplify the situation, and it has long been offered as an alternative to the more usual approach oriented toward diagnostic categories. The rationale for examining what have been referred to as "elementary units of psychological dysfunction" ( [van Praag, 1997](#)), "core or candidate symptoms" ( [Leboyer et al., 1992](#)), quantitative phenotypes ( [Leckman et al., 2001](#)), endophenotypes ( [Almasy and Blangero, 2001](#)), or "core psychopathological processes" ( [Krueger, 1999](#); [Wittchen et al., 1999](#)) is becoming more and more compelling. Finally, there is a greater appreciation of the advantages of combining methodologies. It is now clear that all sources of potentially relevant phenotypic information need to be considered, and the interrelationships among the different phenotypes can be fruitfully examined.

One formulation of the interrelated aspects of biological psychiatry is presented in [Fig. 4.1](#). The mutual interacting influences of genetic and environmental factors are shown determining the neurobiological systems that, in combination, form the substrate of relevant behaviors. The behaviors can be assessed in a number of ways, providing a range of measured phenotypes (a-c). In most cases, syndromes and disorders consist of multiple behavioral components, depicted by the receding additional behavioral components (1-3).



**Figure 4.1.** Schematic of the interrelated factors underlying biological psychiatry. The mutual interacting influences of genetic and environmental factors are shown determining the neurobiological systems that combine to form the substrate of relevant behaviors. The behaviors can be assessed through a range of measured phenotypes (a-c). The multiple behavioral components of most syndromes and disorders are depicted by the receding additional behavioral components (1-3).

The field of biological psychiatry has traditionally emphasized neurochemical and neuroendocrinologic approaches ( [Chapter 7](#)). Other fields that are now proving critical to advancing an understanding of brain neural transmission in psychiatry include neurophysiology ( [Chapter 5](#)), neuropsychology, neuroimaging ( [Hendren et al., 2000](#)) ( [Chapter 9](#)), psychopharmacology ( [Chapter 76](#) and [Chapter 77](#)), and pharmacogenetics/pharmacogenomics ( [Anderson and Cook, 2000](#)). Elucidation of the bases of the childhood disorders can be expected to proceed through an iterative process involving mutually beneficial relationships among all of the perspectives ( [Bailey et al., 1996](#); [McBride et al., 1996](#)). Although advances in a particular area typically will be incremental, they usually will have multiple applications in other areas. The multiple reciprocal relationships among all methods of phenotypic analysis and the field of genetics will be especially important; it has been pointed out that the circular aspect of this undertaking means that genotypic information will help to clarify phenotypes ( [Hyman, 2000](#)). The evolving approach to studying brain and behavior is discussed at length by Werry and colleagues in [Chapter 8](#). The importance of being ever mindful of the developmental context when studying childhood psychiatric disorder is clear ( [Cohen and Young, 1977](#); [Dawson et al., 2000](#); [Leckman, 1999](#); [Skuse, 2000](#); [Young et al., 1984](#)). In a sense, the diagram of [Fig. 4.1](#) needs to be thought of as moving through the fourth dimension of time, affected by (and affecting) the developmental chronology.

### NEUROCHEMICAL RESEARCH: AIMS, SCOPE, AND METHODS

#### Aims of Clinical Neurochemical Research

Biological or molecular psychiatry has been concerned with investigating possible alterations in neural transmission, with most studies focusing on the monoamine neurotransmitters. The research has had three main aims: the identification of useful markers of disorders, explication and description of pathophysiology, and



determination of etiology. These goals are not mutually exclusive: In most cases, hypotheses concerning etiology serve to direct the search for markers. Markers can be sought that reflect either the state of an illness or the trait of being predisposed toward an illness. State markers are not necessarily genetically influenced and should revert to normal on recovery or remission. They are presumably not related to the most basic etiologic alteration but to some aspect of symptom mediation. Trait markers can be considered to be associated in some way with the genetic vulnerability; they are usually present before, during, and after illness; and they are often present in family members of probands with heritable illness. The association of a trait marker with illness could be coincidental, owing to genetic linkage. Although such a linked marker might be extremely useful for pedigree analysis and gene localization, it would in itself give little or no information as to etiology.

Trait markers may be dichotomous, discrete, or continuous. Much of the work of biological psychiatry has been directed, implicitly or explicitly, toward finding trait markers in continuous variables that have been hypothesized to be related to etiologic alterations. Preferably, a marker is easily and reliably measured, with little overlap existing between normal and affected groups. A trait marker would be expected to be useful in diagnosis, subtyping, family and genetic studies, establishing prognosis, and selecting therapy.

The goals of understanding pathophysiology and etiology hold the promise of cure. Studies of etiology have typically examined several variables related to a neurotransmitter system that has been postulated to be altered or dysfunctional. Increased knowledge of pathophysiology and etiology should lead directly to the development of more sensitive and specific marker and the use of new, rational interventions.

## Neurotransmitter Systems

A number of neurotransmitter systems have been hypothesized to be involved in childhood psychiatric disorders (Cohen and Young, 1977; Rasmussen et al., 1990; Rogness et al., 1992; Young et al., 1983, 1984). Most extensively studied have been the three monoamine transmitters, serotonin (5-hydroxytryptamine, or 5-HT), dopamine (DA), and norepinephrine (NE). Other systems studied include opioid peptides, g-aminobutyric acid (GABA), and acetylcholine (ACh). The emphasis on monoamines has been a result of both the importance of these systems in mediating behavioral phenomena and the marked effects of pharmacologic agents known to affect monoamine functioning.

The metabolism of serotonin (5-HT) is depicted in Fig. 4.2. Serotonin is produced after hydroxylation and decarboxylation of the essential amino acid tryptophan. Cell bodies of 5-HT-containing neurons are located in the raphe nuclei of the hindbrain and project to nearly all areas of the central nervous system (Green, 1988; Iverson and Iverson, 1981; Jacobs and Azmitia, 1992). Interest in central 5-HT functioning derives from 5-HT's important role in processes as diverse and important as sleep, mood, appetite, perception, and hormone secretion (Lucki, 1998), as well as its critical role in neurodevelopment (Lauder et al., 1995; Rubenstein, 1998). A schematic diagram of a serotonergic neuron is shown in Fig. 4.3. Serotonin produced from tryptophan (TRP), can be taken up by the vesicular monoamine transporter (VMAT2) and stored in vesicles for subsequent release or metabolized by the mitochondrial enzyme monoamine oxidase (MAO). Once released, 5-HT can interact with presynaptic or postsynaptic receptors, diffuse to extrasynaptic sites, or be taken up by neuronal or glial membrane 5-HT transporters (5-HTTs). Blockade of the 5-HTT by selective serotonin reuptake inhibitors, such as fluoxetine (Prozac), results in higher levels of synaptic and extracellular fluid 5-HT and leads to greater 5-HT receptor stimulation.

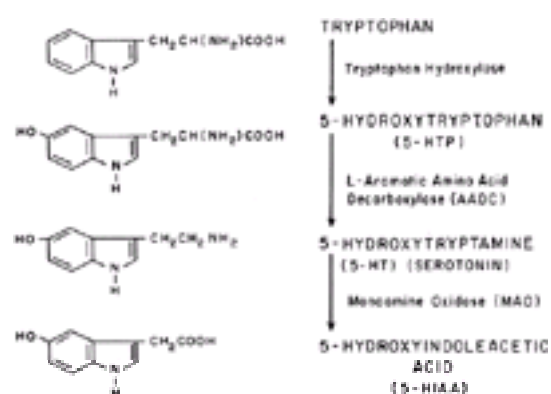


Figure 4.2. Synthesis and catabolism of the indoleamine neurotransmitter serotonin (5-HT).

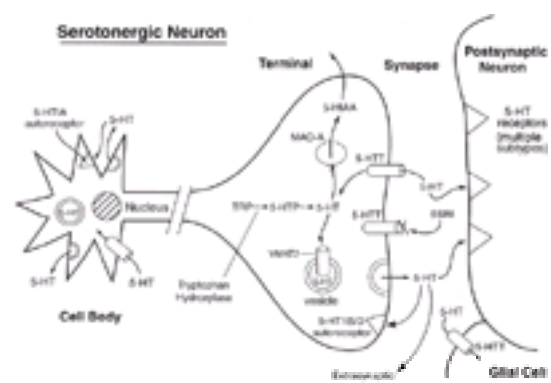
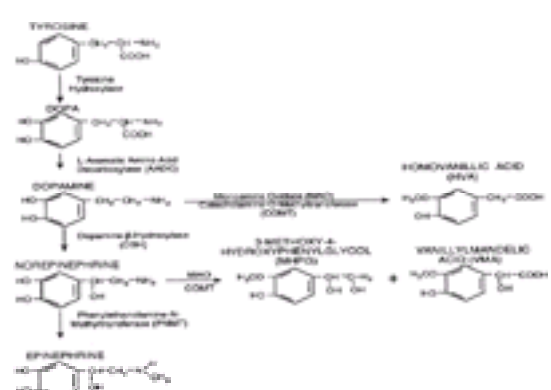


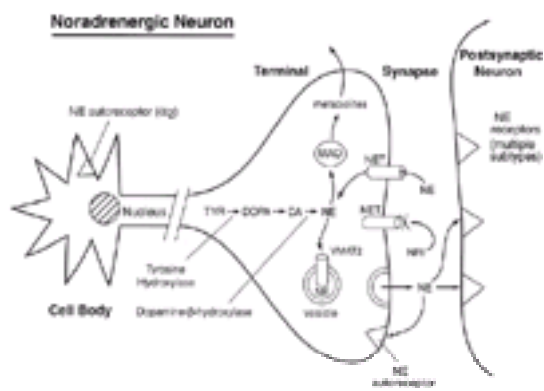
Figure 4.3. Diagrammatic illustration of a serotonergic neuron. Serotonin (5-HT) synthesized from tryptophan (TRP) by the rate-limiting enzyme tryptophan hydroxylase, can be taken up by the vesicular monoamine transporter (VMAT2) and stored in vesicles for subsequent release or metabolized by the mitochondrial enzyme monoamine oxidase. Released 5-HT can interact with presynaptic or postsynaptic receptors, diffuse to extrasynaptic sites, or be taken up by neuronal or glial membrane 5-HT transporters (5-HTTs). Inhibition of the 5-HTT by selective serotonin reuptake inhibitors (SSRIs) results in higher levels of synaptic and extracellular fluid 5-HT, leading to greater 5-HT receptor stimulation.

The metabolism of the catecholamines NE and DA is shown in Fig. 4.4. DA is synthesized from tyrosine, after hydroxylation and decarboxylation, and is found in highest concentration in the midbrain, although extensive cortical projections also occur. DA has been shown to be critical in reward, modulating movement, and cognition (Iverson and Alpert, 1982; Moore and Bloom, 1978; Randrup and Munkvad, 1974). NE is produced from DA by the action of the enzyme dopamine- $\beta$ -hydroxylase (DBH). Nearly all NE-containing neurons project from the locus ceruleus and innervate midbrain and cortical areas. A noradrenergic neuron is depicted in Fig. 4.5. The vesicular and membrane uptake, metabolic, and release processes present in serotonergic neurons have direct counterparts in noradrenergic and dopaminergic neurons. Major differences concern the membrane transporters (norepinephrine transporter, NET; dopamine transporter; or the 5-HTT) and types of presynaptic and postsynaptic receptors present. The three monoamine systems are highly functionally interrelated, making direct connections with one another; often, also, simultaneously modulating other transmitter systems.



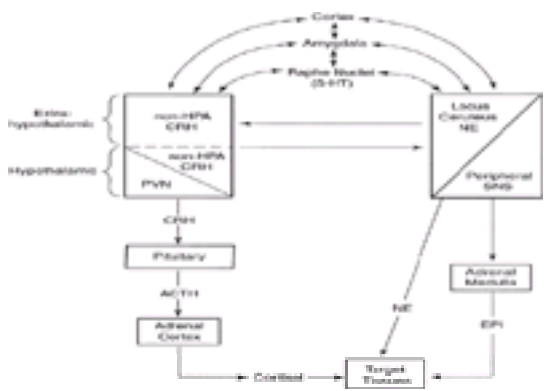


**Figure 4.4.** Synthesis and catabolism of the catecholamine neurotransmitters dopamine, norepinephrine, and epinephrine.



**Figure 4.5.** Diagrammatic illustration of a noradrenergic neuron. Norepinephrine (NE) synthesized from tyrosine by the rate limiting enzyme tyrosine hydroxylase, can be taken up by the vesicular monoamine transporter (VMAT2) and stored in vesicles for subsequent release or metabolized by the mitochondrial enzyme monoamine oxidase. Released NE can interact with presynaptic or postsynaptic receptor. Inhibition of the norepinephrine transporter (NET) by NE reuptake inhibitors (NRI) results in higher levels of synaptic and extracellular fluid of NE and leads to greater NE receptor stimulation.

Stress responses, central and peripheral arousal, and learning and memory are all critically modulated by noradrenergic neurons ( [Amaral and Sinnamon, 1977](#); [Arnsten, 1997](#); [Aston-Jones et al., 1991](#); [Iverson and Iverson, 1981](#)). The critical role of the noradrenergic system in the stress response is shown in [Fig. 4.6](#). The extensive interaction of the central/peripheral NE system with the hypothalamic-pituitary-adrenal (HPA) axis is evident and is summarized in the review of [Chrousos and Gold \(1992\)](#).



**Figure 4.6.** Diagram of the two major components of the stress response system: the central noradrenergic/sympathoadrenomedullary system and the hypothalamic-pituitary-adrenal (HPA) axis. The extensive interaction of the central/peripheral norepinephrine systems with the HPA axis is evident. Not shown are the extensive hormonal and neuronal inputs and feedback occurring from the periphery to the central nervous system.

## Methodologies of Neurochemical Research

The strategies employed in neurochemical research can be grouped into three areas: (a) measurement of neurotransmitters and their metabolites; (b) measurement of neurotransmitter-related enzymes; and (c) measurement of neurotransmitter receptors (number, affinity, and functioning). The neurotransmitters and their metabolites have been measured in postmortem (PM) brain tissue, cerebrospinal fluid (CSF), plasma and blood elements, and urine. Enzymes have been determined in the brain, CSF, and blood, whereas receptor measurements have been carried out with brain and blood specimens. Other biological research has included neuroendocrine studies, typically measuring blood hormone concentrations after administration of challenge agents, as well as brain-imaging studies employing computed tomography (CT), magnetic resonance imaging (MRI), positron emission tomography (PET), and single-photon emission computed tomography (SPECT) to examine neuroanatomic structure, blood flow, and receptor binding. With all approaches, a critical issue is to what extent the measure provides information about the central neurotransmitter system of interest. Even when measurements are made on brain tissue itself, the relationship to neuronal functioning can be tenuous. This issue becomes even more problematic when measurements are made in peripheral fluids and tissue. The degree of correlation between a particular measure and central neurotransmitter functioning is difficult to judge and depends on the specific measure and system under study.

### POSTMORTEM BRAIN

Analysis of postmortem (PM) brain tissue allows the most direct access to the systems of interest. One can measure levels of neurotransmitters, their precursors and metabolites, associated enzymes (both synthetic and catabolic), reuptake sites, and presynaptic and postsynaptic receptor sites. This approach has been extensively employed in the study of neuropsychiatric disorders, including Alzheimer's disease, Parkinson's disease, Huntington's disease, schizophrenia, suicide, and depression ([Arai et al., 1984](#); [Bunney et al., 1997](#); [Gross-Isseroff et al., 1998](#); [Korpi et al., 1986](#); [Lee et al., 1978](#)). The anatomic resolution and the wide range of neurotransmitter-related analytes have made PM studies of paramount importance. Yet there are some disadvantages inherent in this approach, including PM degradation, subject recruitment, control matching, and the fundamental limitation arising from an inability to measure functionality ([Perry and Perry, 1983](#)).

### CEREBROSPINAL FLUID

Measurements of transmitters, metabolites, and enzymes in CSF have been considered to be the next best thing to PM analyses when attempting to assess central systems. Although a number of the compounds measured in CSF are derived predominantly from the brain, other analytes are thought to originate from spinal cord metabolism or to diffuse into the CSF from the blood ([Garelis et al., 1974](#)). Even when compounds are solely of brain origin, lumbar CSF concentrations are affected by clearance rates. The greatest limitation of CSF measures is their failure to provide information regarding anatomic localization. A resultant practical shortcoming is that only global or marked changes in brain neurochemistry are detected.

The DA metabolite homovanillic acid (HVA) has been extensively assayed in CSF and is probably substantially derived from the brain metabolism of DA ([Elchisak et al., 1978](#); [Elsworth et al., 1987](#); [Garelis et al., 1974](#); [Sourkes, 1973](#)). However, CSF levels of NE and the NE metabolite 3-methoxy-4-hydroxyphenylglycol (MHPG) appear to primarily reflect NE metabolism in the spinal cord ([Kopin et al., 1983](#); [Ziegler et al., 1977](#)). Similarly, the 5-HT metabolite 5-hydroxyindoleacetic acid (5-HIAA) is apparently predominantly of spinal cord origin ([Bulat, 1977](#); [Moir et al., 1970](#); [Weir et al., 1973](#)). Recent studies of 5-HT in nonhuman primate cisternal CSF indicate that this measure may provide an alternative, improved route to assessing central 5-HT function. It is often unclear to what extent peptides and amino acids measured in lumbar CSF reflect brain, spinal cord, and peripheral metabolism.

### BLOOD AND URINE

Measurements of monoamine metabolites in blood and urine have been performed widely; however, the issue of peripheral versus central contribution is problematic. It is clear that the vast majority of plasma and urine 5-HIAA is of peripheral origin, arising from the catabolism of 5-HT produced in the enterochromaffin cells of the intestine ([Udenfriend et al., 1956](#)). This has limited the utility of measurements of blood and urine 5-HIAA levels to those situations where the rate of gut 5-HT

synthesis is of interest. Urine 5-HT has been rarely measured because it too probably reflects only gut synthesis of 5-HT. Blood levels of 5-HT have been more frequently studied because of 5-HT's localization in platelets. Similarities between platelet and neuronal uptake, storage, and release of 5-HT have made the platelet of special interest ([DePrada et al., 1988](#); [Pletscher, 1988](#); [Sneddon, 1973](#)). Findings in autism have further stimulated research in the area.

Plasma and urine HVA concentrations have been widely determined in an attempt to assess central DA functioning ([Bowers, 1991](#)). A number of studies have attempted to ascertain the central contribution to plasma (and urine) levels. Known sources of peripheral DA and HVA include the sympathetic nervous system, adrenal glands, and kidney. When peripheral DA synthesis and metabolism are inhibited with debrisoquin, a peripherally acting MAO inhibitor and neuronal blocker, plasma and urine HVA levels decrease to about 50% of baseline, indicating that at least half of the measured HVA originates in the periphery ([Maas et al., 1985](#); [Riddle et al., 1986](#)). Changes in the norepinephrine metabolite MHPG after debrisoquin administration are even more marked, indicating that less than 25% of plasma or urine MHPG is of central origin ([Kopin et al., 1988](#)). Investigators have also measured plasma and urine levels of the catecholamines themselves. It is clear that all plasma and urine NE arises from the adrenal glands and sympathetic nervous system. However, the tight coupling between the central norepinephrine neurons and the sympathetic nervous system suggests that plasma and urine NE might still be useful indices of central noradrenergic functioning. At the least, measurements of NE and related compounds appear to offer a useful index of sympathetic functioning. It is even clearer that measurement of epinephrine (EPI) in blood and urine offers an unambiguous reflection of adrenomedullary activity. Unfortunately, clearance and catabolism affect the measured levels of the monoamines and their metabolites. Urine measurements can actually offer advantages in that differences in clearance are less a problem than with plasma. Urine samples can be more easily obtained, although it is difficult to observe acute (<1- or 2-hour) changes.

Several monoamine-related enzymes have been measured in blood specimens. These include MAO B in platelets, catecholamine-O-methyltransferase in red blood cells, and dopamine-β-hydroxylase (DBH) in serum. Genetic influences on the enzymes have been recently described, making the measures of greater interest. This appears to be especially the case for DBH, with a recent report indicating that more than 50% of the population variance in serum DBH activity can be explained on the basis of a specific promoter polymorphism (-1021C>T). The virtual absence of serum DBH activity in individuals homozygous for the lower frequency -1021T allele is reflective of the highly functional nature of the polymorphism ([Zabetian et al., 2001](#)).

Neurochemical analyses of blood and urine have been made both in unperturbed (baseline) situations and after administration of centrally acting agents; the latter approach often bears a strong resemblance to neuroendocrine challenge studies. These neuroendocrine studies have attempted to assess central 5-HT, DA, NE, and opioid functioning by determining plasma levels of growth hormone, prolactin, cortisol, and other hormones. Hormone levels are determined after administration of challenge agents that are meant to selectively stimulate specific central neurotransmitter receptors. A number of factors tend to confound the neuroendocrine approach, including pulsatile and fluctuating release, poor specificity of the challenge agents, and the difficulty in relating findings in the hypothalamus, pituitary, or adrenal gland (or thyroid gland) to etiology.

The numbers and affinities of a range of neurotransmitter receptors have been measured in blood elements. Platelet serotonergic (5-HT<sub>2</sub>) and adrenergic (α<sub>2</sub>) receptors, platelet 5-HT uptake sites (imipramine-binding sites), and white blood cell adrenergic (β) receptor sites have been measured using radioactively labeled ligands. The measurement of receptor function has been less prevalent, although a host of studies have examined the platelet uptake of tritiated 5-HT and binding of reuptake inhibitors ([Owens and Nemeroff, 1994](#)).

## NEUROCHEMICAL RESEARCH IN CHILD PSYCHIATRY

In the following sections, neurochemical research on neurotransmitter ontogeny and the relatively common childhood psychiatric disorders of autism, Tourette syndrome (TS), and attention deficit hyperactivity disorder (ADHD) are considered in detail. The reader is also referred to the individual chapters on these disorders and to reviews of the substantial neurochemical and neuroendocrine research carried out in other childhood disorders. These latter areas include studies of childhood depression ([Ryan and Dahl, 1993](#)), anxiety ([Foote, 1999](#); [Pine, 1999](#)), and the effects of trauma ([Kaufman and Charney, 1999](#); [Pynoos et al., 1999](#)). Neurochemical research in these areas has been extensive and the recent studies demonstrate a movement toward a more symptom-based, multiperspective, and genetic approach. Although neuroendocrinology has been a large part of child biological psychiatry, this area is not a focus of our chapter.

### Neurochemistry of Normal Development

Many recent exciting advances have led to a better picture of neuronal development, both at the cellular level and in relation to cell-cell interactions and complex circuit assembly. Animal studies have examined the ontogeny of neurotransmitter synthesis, storage, release, and uptake, as well as receptor number, affinity, and coupling. Work on the developing primate brain has been most informative as to what changes may be occurring during different periods of childhood ([Kandel and Schwartz, 1981](#); [Purves and Litchman, 1986](#); [Rakic, 1988](#); [Shepherd, 1983](#)).

Studies of neurotransmitter ontogeny in psychiatrically normal children have predominantly dealt with the measurement of neurotransmitters, metabolites, and associated enzymes in CSF, blood, and urine. These studies have helped to establish normative ranges for contrasting with values seen in neuropsychiatric disorders and have also served to point out periods of change and allow the validity of animal models to be assessed.

A large study of CSF monoamine precursors and metabolites in children treated for leukemia found large decreases in HVA and 5-HIAA over childhood and adolescence. Mean values for HVA in subjects ages 2 to 5, 6 to 11, and 12 to 17 years decreased from 92 to 62 to 44 ng/mL, respectively, whereas 5-HIAA declined from 30 to 21 to 17 ng/mL, respectively. Changes in the precursors were substantially less. The multiple lumbar punctures performed allowed an estimation of mean intraindividual variation of 20% to 25% to be made ([Riddle et al., 1986](#)). The ontogenetic declines in HVA and 5-HIAA levels also have been observed in developing (12- to 42-day-old) rat pups. Changes in rat CSF metabolite levels tended to parallel declining brain metabolite levels, whereas levels of total brain monoamine levels (DA and 5-HT) increased ([Shaywitz et al., 1985](#)). Studies of CSF metabolite ontogeny in humans have been extended to the neonatal period, where even higher levels of 5-HIAA, HVA, tryptophan, and tyrosine are seen ([Anderson et al., 1985](#)). Mean levels of 5-HIAA and HVA in newborns were 143 and 184 ng/mL, respectively, substantially higher than in the 2- to 5-year-old period. Neonatal levels of MHPG were also significantly higher (27 ng/mL) than those seen in 2- to 5-year-olds, although unlike 5-HIAA and HVA, MHPG levels did not decline further after the period of 2 to 5 years of age. Another compound with an early, rapid maturational decline is the other acidic DA metabolite 3,4-dihydrophenylacetic acid (DOPAC) ([Anderson et al., 1988](#)). Although the causes of the maturational declines are not clear, the dissociation between the ontogenies of DOPAC (with its early, rapid decline) and HVA (with its more gradual decline) indicate that acid transport and clearance changes are not major determinants.

These studies of normal neurochemical ontogeny provide a context when examining levels seen in childhood disorders and after neonatal drug exposure. In studies of CSF levels of monoamine and metabolites in neonates exposed prenatally to cocaine, significantly lower levels of the dopamine metabolite HVA were seen in the exposed infants ([Needman et al., 1993](#)). Further studies are needed to see whether this decrease persists over the first months and years of life. A short-lasting decrease simply may be due to an acute pharmacologic effect; longer lasting reductions in HVA probably are a result of reductions in DA innervation.

Blood concentrations and urinary excretion rates of the catecholamines and their metabolites are generally observed to decline over childhood ([Dalmaz and Peyrin, 1982](#); [Gitlow et al., 1968](#)). This apparently is due both to decreasing synthesis and increasing activity of the catabolic enzymes MAO and catechol-O-methyltransferase (COMT) ([Young et al., 1984](#)). It is not clear whether the decreasing synthesis corresponds to decreasing functional release. Definite statements about relative central and peripheral contributions to plasma or urine catecholamine metabolite levels cannot be made, although the situation in childhood does not appear to be more favorable than in adulthood. Blood levels of 5-HT have been observed to decrease with age; however, most of this effect appears to occur at puberty ([McBride et al., 1998](#)). This suggests that platelet uptake and storage of 5-HT are fully developed in early childhood. In recent studies, platelet 5-HT levels in nursing infants have been used to index the extent of transporter blockade occurring after exposure to selective serotonin reuptake inhibitors (SSRIs) through breast milk ([Epperson et al., 1997](#)). The decline in levels seen after SSRI administration reflects the reduction in transporter activity because all 5-HT in the platelet is accumulated by uptake.

### Attention Deficit Hyperactivity Disorder

The symptomatology of attention deficit hyperactivity disorder (ADHD) includes inattention, distractibility, and impulsivity, with or without hyperactivity ([Carey, 1988](#); [Shaywitz and Shaywitz, 1982, 1992](#)) ([Chapter 52](#)). Early work focused on the presence of this behavioral pattern in children who had suffered from encephalitis or brain damage. Although the idea that there existed some sort of minimal brain damage (MBD) led to the use of MBD as a diagnostic category, no consistent or specific abnormality was demonstrated. The behavioral sequelae of encephalitis along with the remarkable effects of stimulants on ADHD children have led to a long-standing and continuing interest in the role of DA. Family and twin studies have strongly supported the idea that there are inherited components to the disorder



([Hewitt et al., 1997](#); [Levy et al., 1997](#); [Pauls et al., 1983](#); [Thapar et al., 1999](#)).

A great number of neurobiological studies have been carried out; the majority involves the measurement of neurotransmitter metabolites in blood and urine, either at baseline or after pharmacological perturbation ([Zametkin and Rapoport, 1987a](#)). Several neuroendocrine challenges have been performed, whereas there are only a limited number of studies involving receptor assessment ([Hunt et al., 1982](#); [Shekim et al., 1994](#)). An increasing number of brain-imaging studies have been reported ([Castellanos, 1996](#); [Hendren et al., 2000](#); [Schweitzer et al., 2000](#)) ([Chapter 9](#)). These studies have tended to focus attention on the frontal cortex and midbrain DA nuclei and their projection areas.

#### DOPAMINE

The simplest of the catecholamines has been extensively studied in ADHD; much of the work has been prompted by neuropharmacologic observations. Neuroimaging studies, the development of animal models ([Lipton et al., 1980](#); [Shaywitz et al., 1976](#)), and the recent replicated genetic investigations indicating an association with ADHD symptomatology and alleles of the dopamine transporter and D4 DA receptor genes ([Anderson and Cook, 2000](#)), have further increased interest in DA. Although the ameliorative effects of the stimulants methylphenidate, amphetamine, and pemoline have strongly implicated DA, the stimulants are known to also affect norepinephrine and epinephrine. The hypodopaminergic hypothesis also has been questioned because of the relatively benign or slightly beneficial effects of DA blockers.

Although the neurochemical evidence is extensive, it fails to conclusively support a role for DA involvement ([Zametkin and Rapoport, 1987a](#)). The limited CSF data suggest a possible lowering of HVA in ADHD. Baseline measurements of HVA in plasma and urine, and DA in urine have not revealed differences between ADHD and control subjects. Platelet levels of MAO activity are unchanged or decreased in ADHD, whereas serum DBH was similar in ADHD and control subjects. Urine or plasma levels of DA and HVA also have been measured after administration of stimulants or tricyclic antidepressants with no clear group differences emerging. Neuroendocrine studies in ADHD are limited and provide no definite information regarding possible group differences.

#### NOREPINEPHRINE AND EPINEPHRINE

Arousal mechanisms almost certainly play an important part in symptoms of hyperactivity, impulsivity, distractibility, and inattention ([Halperin et al., 1997](#); [McMahon, 1984](#); [Oades, 1987](#); [Ornitz et al., 1997](#); [Satterfield et al., 1974](#)). The crucial role of the central noradrenergic system and sympathetic nervous system (SNS) in regulating arousal, together with noradrenergic effects of stimulant medication, has led to hypotheses of noradrenergic involvement in ADHD ([Hunt et al., 1984](#); [Mefford and Potter, 1989](#); [Mikkelsen et al., 1981](#); [Pliszka et al., 1996](#); [Shekim et al., 1979](#); [Snyder and Meyerhoff, 1973](#)). Treatment studies employing the noradrenergic-specific agents clonidine and guanfacine, as well as an increasing appreciation of the role of central NE in attention and cognition ([Arnsten, 1997](#); [Arnsten et al., 1996](#); [Aston-Jones et al., 1991](#); [Williams and McGaugh, 1993](#)), have served to maintain interest in the role of NE in the symptoms of AD/HD.

Baseline measurements of NE in serum and MHPG in plasma have not revealed differences between ADHD and control subjects. The data with respect to MHPG and NE in urine are less consistent, with MHPG excretion, for instance, decreased, unchanged, or increased in ADHD ([Baker et al., 1993](#); [Zametkin and Rapoport, 1987a](#)). Platelet  $\alpha_2$ -adrenergic receptors are reported to be lower in ADHD ([Shekim et al., 1994](#)); as mentioned, serum levels of the NE synthetic enzyme DBH was similar in ADHD and control subjects. Urine or plasma levels of NE and MHPG also have been measured after administration of stimulants or tricyclic antidepressants. Although neurochemical changes are observed after the drug treatment (e.g., lowered MHPG with desipramine, increased NE with yohimbine), no conclusive differences in neurochemical response to the agents have been shown between ADHD and control subjects.

Research establishing a positive association between classroom performance and epinephrine (EPI) excretion ([Frankenhaeuser, 1971](#)), along with reports of stimulant-induced EPI release and long-standing observations of cognitive enhancing effects of systemically administered EPI ([Frankenhaeuser and Jarpe, 1963](#); [Williams and McGaugh, 1993](#)) suggest a possible role for EPI in attention. Several reviews have discussed how altered interaction of the adrenergic and noradrenergic systems might contribute to symptoms of ADHD ([McCracken, 1991](#); [Mefford and Potter, 1989](#); [Pliszka et al., 1996](#)). Although studies of baseline EPI excretion have not found differences between ADHD and control groups, three studies ([Anderson et al., 2000](#); [Hanna et al., 1996](#); [Pliszka et al., 1994](#)) have found substantially lower rates of EPI excretion during cognitive testing in ADHD patients compared to normal controls. In studies examining the effects of amphetamine or methylphenidate in patients with ADHD, both drugs increased EPI excretion, but relatively smaller adrenomedullary responses were seen in patients compared to controls following acute ([Rapoport et al., 1978](#)) or chronic ([Elia et al., 1990](#)) dosing. A similar reduced adrenomedullary response was observed in children with ADHD during hypoglycemic challenge when plasma levels of epinephrine were measured. Thus, the finding of a blunted epinephrine response in ADHD has been seen consistently across a number of situations. In the most recent study ([Anderson et al., 2000](#)), the blunted response appeared to be specific to the inattention component or domain of ADHD. Further work in this area appears warranted: It would not be surprising if genetically determined variations in the functioning of each of the catecholamines contributed some part to one or more of the component behaviors of ADHD.

#### SEROTONIN

Interest in a role for serotonin (5-HT) in ADHD was stimulated by early reports of decreased platelet 5-HT in affected children. Subsequent studies have not replicated this finding and have found normal levels of platelet and urine 5-HIAA ([Zametkin and Rapoport, 1987a](#)), as well as normal numbers and affinities of platelet imipramine-binding sites in ADHD subjects ([Weizman et al., 1988](#)). In addition, studies of CSF 5-HIAA have not found differences between ADHD and control subjects. On the whole, the neurochemical research and the minimal treatment response to serotonergic agents have made it seem less likely that a 5-HT alteration is etiologic. Even so, it appears that the role of 5-HT in disruptive behaviors, particularly with respect to impulse control, deserves further consideration ([Anderson, 1993](#); [Zubieta and Alessi, 1993](#)).

#### Autism

Autism is a pervasive developmental disorder with disturbances in social relations, language, and communication ([Cohen and Volkmar, 1997](#); [Waterhouse et al., 1996](#)). The three monoamine transmitters have been extensively studied in autistic children, and some work has also been carried out in relation to the opioid transmitters ([Anderson and Hoshino, 1997](#); [Cook et al., 1990](#); [Lotspeich and Ciaranello, 1993](#)).

#### NOREPINEPHRINE AND EPINEPHRINE

As one of the two major components of the stress response system ([Chrousos and Gold, 1992](#)), the sympathetic/adrenomedullary system has been of interest in autism owing to the hyperarousal, hyperactivity, and overreaction to novel situations often seen in autism. It should be mentioned that the HPA axis, the other major component of the stress response ([Fig. 4.6](#)), has also been well studied in autism. However, these neuroendocrinologic studies are reviewed elsewhere ([Tordjman et al., 1997](#)). The functioning of the sympathetic/adrenomedullary system has been assessed through measurements of norepinephrine (NE) and epinephrine (EPI) in plasma or urine. In addition, plasma and urine levels of the major NE metabolites, 3-methoxy-4-hydroxyphenylethylglycol (MHPG) and vanillylmandelic acid (VMA) have been determined. Serum levels of dopamine- $\beta$ -hydroxylase—the synthetic enzyme secreted along with NE from sympathetic neurons—also have been studied ([Anderson and Hoshino, 1997](#); [Minderaa et al., 1994](#)).

The results of the studies examining these neurochemical indices of sympathetic/adrenomedullary system functioning are summarized in [Table 4.1](#). The urine measures reflect basal functioning because of the absence of a stressful situation involved in the collection of the sample and owing to the long-term nature of the collection. Plasma levels of MHPG and VMA are basal indices because of their slow response to perturbation, a consequence of their long plasma half-lives. In contrast, plasma NE and the vascular measures respond very quickly to stressful situations. When considered in this fashion, it can be seen that the results are actually fairly consistent. Thus, in nearly all cases, indices that reflect basal functioning of the sympathetic/adrenomedullary system were found to be normal in patients with autism. On the other hand, most of the studies measuring indices of acute stress response have found elevations in patients with autism when they are exposed to the stress of a venipuncture or neuropsychological test. Taken together, the data support the idea that stress response systems are hyperresponsive when individuals with autism are stressed, but that autistic patients are not in a chronic state of hyperarousal. Findings from studies of HPA axis function are consistent with the sympathetic/adrenomedullary results, and support the same conclusions.

Measures of Basal Functioning		
Measure	Finding	Reference
Urine NE	Normal	Loucky et al., 1987; Marinneau et al., 1982; Minderaca et al., 1994
	Decreased	Young et al., 1978
	Increased	Batholomy et al., 1988
Urine EP	Normal	Minderaca et al., 1994
Urine MHPG	Normal	Loucky et al., 1987; Minderaca et al., 1994
	Decreased	Batholomy et al., 1988; Young et al., 1978
Urine VMA	Normal	Minderaca et al., 1994
Plasma MHPG	Normal	Minderaca et al., 1994; Young et al., 1981
Serum DHP	Normal	Lake et al., 1972; Young et al., 1980
CSF AHPA	Normal	Gilberg and Sorenstam, 1987; Young et al., 1981

Measures of Acute Response		
Measure	Finding	Reference
Plasma NE	Increased	Cook et al., 1990; Lake et al., 1977; Loboyer et al., 1992; Loventhal et al., 1990
BP/heart rate	Increased	Koolt and Cohen, 1981; Tordman, 1999

**Table 4.1. Sympathetic/Adrenomedullary Functioning in Autism**

The apparent increased response to stressors could result from a difference in the level of perceived stress, an over elicitation of the physiologic response, or an abnormality in the stress response systems themselves. It will be difficult to determine whether the individual with autism experiences a greater threat, if the response to the threat is less well regulated, or both. Despite the difficulties encountered in stress response research in autism, further research in this area is warranted, given the clinical relevance and possible etiologic nature of alterations in this area.

#### DOPAMINE

A case for altered DA functioning in autism can be made, based on its clear role in mediating motoric disturbances (e.g., stereotypies) and the observation that DA blockers are effective in treating some aspects of autism. The majority of relevant neurochemical studies have examined levels of HVA (Anderson and Hoshino, 1997). The concentration of HVA in CSF has been reported to be slightly decreased, apparently normal, or significantly (approximately 50%) increased in autism. The most recent study, using controls selected from patients undergoing myelography, did not find any difference between autistic and control subjects (Narayan t al., 1993). Measurements of HVA in urine have been in disagreement, with some groups reporting increased excretion and others reporting normal excretion in autism. The only study of plasma HVA reported similar levels in autistic and control subjects (Minderaca et al., 1989). Other relevant measures include urinary DA, reportedly normal in autism, and plasma prolactin, which also has been reported to be normal in autistic subjects (McBride et al., 1989; Minderaca et al., 1989). To summarize, the majority of studies report normal DA metabolism and normal baseline levels of prolactin, a hormone under strong inhibitory control by the tuberoinfundibular DA system. The bulk of the studies suggest that central dopaminergic functioning, to the extent it can be assessed by the measures employed, is normal in autism.

#### SEROTONIN

Initial interest in a role for 5-HT in autism stemmed in part from the powerful effects of serotonergic agents, such as lysergic acid diethylamide, on perception. Research in the area was further stimulated by, and has continued to evolve around, the early studies of Freedman of elevated 5-HT in blood of autistic children (Hanley et al., 1977; Schain and Freedman, 1961). Reports of the presence of 5-HT receptor antibodies in the CSF of autistic patients (Todd and Ciaranello, 1985), of beneficial effects of treatment with serotonin reuptake inhibitors (Cook et al., 1992; Gordon et al., 1993; McDougale et al., 1996a) and with the serotonergic agent fenfluramine (Campbell, 1988; Ritvo et al., 1986), and exacerbation observed after depletion (McDougale et al., 1996b) have also heightened interest in the role of 5-HT in autism. It should be pointed out that careful follow-up studies did not replicate either the autoantibody finding (Yuwiler et al., 1992) or the early reports of fenfluramine's benefits (Leventhal et al., 1993; Stern et al., 1990; Varley and Holm, 1990).

Although most of the 5-HT-related research has focused on the hyperserotonemia of autism, a number of studies of CSF 5-HIAA and several neuroendocrinologic studies of central 5-HT functioning have been reported (Anderson, 1987; Anderson and Hoshino, 1997). CSF studies are in general agreement that few or no differences exist between autistic and control groups' mean levels of 5-HIAA (Anderson, 1994). Neuroendocrine challenge studies have measured growth hormone and prolactin after administration of fenfluramine (McBride et al., 1989), the 5-HT<sub>1B/D</sub> receptor agonist sumatriptan (Novotny et al., 2000) or the 5-HT precursor 5-hydroxytryptophan (5-HTP) (Hoshino, personal communication). The fenfluramine and 5-HTP studies both observed a lowering or blunting of the prolactin response, whereas sumatriptan produced an elevated growth hormone response in autism. The data are not definitive, but they are consistent with decreased central presynaptic 5-HT<sub>2</sub> functioning in autism.

The bulk of the 5-HT-related studies deal with the characterization or elucidation of the hyperserotonemia of autism (Anderson et al., 1990). The basic finding of elevated platelet 5-HT is robust and well replicated. Increased levels are observed regardless of whether concentrations are expressed as amount per volume of blood (e.g., nanograms per milliliter) or as amount per number of platelets (nanograms per 10<sup>9</sup> platelets); in addition, platelet counts and size distribution have been reported to be normal in autism. Although the basic finding of a group mean increase in autistic subjects is well established, the magnitude of the increase is not clear. Recent research has indicated that substantial racial and pubertal effects exist; when these were accounted for a group mean increase of 25% was observed (McBride et al., 1998). The relationship of blood 5-HT to the behavioral aspects of autism and the distribution of the measure in the autistic group are also not clear. Serotonin levels have not been consistently correlated with degree of mental retardation (MR) or other symptomatology, and recent studies have not found an increase in the MR group. In groups of unmedicated autistic individuals, the distribution of blood 5-HT levels appears Gaussian, suggesting that the group mean increase was not caused by a subgroup of hyperserotonemic individuals; however, the possibility of a multimodal distribution cannot be ruled out, given the size of the groups examined.

The typical group mean increase observed in 5-HT is relatively robust (effect size ~1); however, the large degree of overlap with the normal population has prevented the use of blood 5-HT levels in screening or diagnosis of autism. Numerous investigators have been intrigued, nonetheless, by the possibility of discovering the cause(s) of the elevation (Anderson et al., 1990; Cook, 1990; Hanley et al., 1977; Young et al., 1982; Yuwiler et al., 1985). It has been hoped that understanding the mechanism of hyperserotonemia would lead to the development of more specific and useful markers, elucidation of the CNS abnormality, and useful approaches to the treatment of the condition.

Two main possibilities have been considered as potential causes of the hyperserotonemia: (a) increased exposure of the platelets to free 5-HT as a result of either decreased catabolism or increased synthesis of 5-HT and (b) altered platelet handling of 5-HT. The issue of decreased catabolism of 5-HT has been examined by measuring platelet MAO activity and monoamine substrates and metabolites. These studies have strongly suggested that MAO activity, and hence 5-HT catabolism, is not altered in autism (Anderson, 1990). The normal excretion rates observed for 5-HIAA in autistic subjects in most, but not all, studies also strongly indicate that 5-HT synthesis is not increased in autism.

Although these data strongly suggest that 5-HT metabolism is unaltered in autism, they do not definitely indicate whether or not the circulating platelet is exposed to increased levels of 5-HT. This issue can be addressed most directly by determining plasma free levels of 5-HT. When high-performance liquid chromatography (HPLC) was used to determine platelet-poor plasma 5-HT levels in autistic subjects, the levels were similar to those seen in normal controls (Cook et al., 1988). Urinary excretion rates for 5-HT in autistic subjects also have been measured, and the group mean was found to be similar to or slightly lower than that seen for normal subjects (Anderson et al., 1989). These measures of plasma and urine 5-HT levels, as well as the evidence regarding 5-HT metabolism, indicate that the platelet is not exposed to increased concentrations of 5-HT. This, in turn, strongly implies that the platelet's handling of 5-HT is altered in autism.

Accumulated evidence warrants focusing research on platelet uptake, storage, and release of 5-HT. Despite work in a number of laboratories, no consensus has been reached concerning relative rates of 5-HT uptake by platelets from autistic and normal subjects (Anderson et al., 1990). The studies have been more consistent in not finding differences in the affinities for uptake in the two groups. Group differences also were not seen when researchers examined the number ( $B_{max}$ ) and affinity ( $K_d$ ) of 5-HT transporter binding sites in autistic and normal individuals (Anderson et al., 1984; Weizman et al., 1987). The question of altered efflux has been studied in some detail after an initial report indicated that platelets from autistic subjects have a greater efflux of preloaded tritiated 5-HT during *in vitro* incubation. In a multicenter study, the consensus was that autistic and normal groups had similar mean efflux rates (Boullin et al., 1982). McBride and associates (1989) examined the 5-HT<sub>2</sub> receptor-mediated augmentation of adenosine diphosphate (ADP)-induced platelet aggregation and found lower 5-HT augmentation in the autistic group than in the normal subjects. In addition to the blunted augmentation response, the autistic group had lower numbers ( $B_{max}$ ) of 5-HT<sub>2</sub> receptors detected by iodinated-LSD binding. Recent investigations examining the possible role of the 5-HT transporter gene in affecting risk to, or expression of, autism have stimulated interest in the role of the transporter in the hyperserotonemia. It now appears that although the promoter polymorphism in the 5-HT transporter gene probably does not influence risk to



autism, it may affect severity in certain behavioral domains. It also seems unlikely to contribute substantially to the hyperserotonemia of autism given the allele frequencies and functional effects observed ([Cook et al., 1997](#); [Klauck et al., 1997](#); [Tordjman et al., 1998, 2001](#)).

#### OTHER STUDIES

Although there has been considerable speculation regarding a role for opioid peptides in autism and related behaviors ([Chamberlain and Herman, 1990](#); [Panksepp et al., 1980](#)), studies of plasma levels of the opioid peptide b-endorphin (BE) do not bear on the issue of central opioids ([Anderson and Hoshino, 1997](#); [Sandman, 1992](#)) and treatment studies with opioid antagonists are not conclusive. It should be pointed out that plasma BE is not centrally derived and the measure actually appears to be highly reflective of HPA axis activity ([Tordjman et al., 1997](#)). Two studies of CSF endorphins have found increased levels of the compounds in the CSF of autistic individuals ([Gillberg et al., 1985](#); [Ross et al., 1985](#)); however, this research has not been replicated to date.

Imaging studies have reported a number of intriguing findings in at least a portion of autistic subjects ([Chugani, 2000](#); [Chugani et al., 1999](#); [Hendren et al., 2000](#)), and neuropathologic studies have found changes in the limbic system and cerebellum ([Bauman and Kemper, 1984](#); [Ritvo et al., 1986](#)). These brain studies, along with the neurochemical research, will help to direct experiments using PM brain tissue. The recent availability of PM specimens from individuals with autism, made possible through the dedicated efforts of Margaret Bauman and colleagues, provides a tremendous new opportunity to study the neurochemistry of autism. Initial results, using the available brain tissue, have appeared ([Blatt et al., 1999](#); [Colantuoni et al., 1999](#)); many more reports are expected. Although we have discussed some of the difficulties inherent in PM brain research, this area has great promise in identifying central alterations in autism.

#### Tourette Syndrome

Tourette syndrome (TS) is a persistent familial neuropsychiatric disorder characterized by multiple motor and phonic tics that are stress-sensitive and that tend to wax and wane in severity ([Chappell et al., 1990](#); [Leckman et al., 1987](#); [Shapiro et al., 1988](#)) ([Chapter 59](#)). Knowledge of the disorder has grown dramatically in the past 10 to 20 years; a clearer picture of the clinical phenomena and an increased awareness of the disorder have allowed better diagnosis and more systematic neurobiological research. Neuroanatomic and neuropharmacologic considerations have prompted a number of neurochemical studies with a focus on the monoamines DA, 5-HT, and NE ([Anderson et al., 1999](#); [Cohen and Leckman, 1994](#); [Kurlan, 1992](#); [Singer and Wendlandt, 2001](#)).

#### DOPAMINE

A role for DA is suggested by the amelioration of tics by neuroleptics, exacerbation of symptomatology after administration of stimulants, and importance of DA pathways in modulating basal ganglia output. The basal ganglia play critical roles in motoric behavior, attention, and emotion and reward: areas of function that intersect in the symptoms of TS ([Leckman and Riddle, 2000](#)). The measurement of the DA metabolite HVA in CSF has not revealed consistent differences between mean levels in Tourette and control groups ([Leckman et al., 1995](#)). Only limited data are available comparing plasma or urine levels of HVA; however, group differences have not been apparent. Studies of PM brain from a small number of Tourette patients have given conflicting results. Singer and colleagues ([Singer et al., 1991](#); [Singer and Wendlandt, 2001](#)) have found increased densities of the DA transporter in basal ganglia regions and suggested that the increased densities are a reflection of increased DA innervation within the striatum; however, observations of the normal striatal levels of DA, homovanillic acid, and tyrosine hydroxylase do not support this idea ([Anderson et al., 1992a,b](#)). Although an initial imaging study reported higher striatal DA transporter binding in Tourette patients, subsequent studies have not confirmed this elevation ([Singer and Wendlandt, 2001](#)).

The notion that hypersensitive central DA receptors may be etiologic in TS has prompted attempts at receptor modulation by administration of dopaminergic agents ([Friedhoff, 1982](#)). Other than the observation of normal baseline plasma prolactin levels, little neuroendocrinologic assessment of central DA receptors has been performed. Interest in possible alterations in relative densities of brain D2 and D1 DA receptors has increased following a report of a relationship between density and tic severity in twins ([Wolf et al., 1996](#)).

#### NOREPINEPHRINE

The importance of noradrenergic projections from the locus ceruleus in controlling states of arousal, as well as reports of symptom amelioration after treatment with the  $\alpha_2$ -agonist clonidine ([Leckman et al., 1985, 1991](#)), are suggestive evidence for altered noradrenergic functioning in TS. There are several reports of lowered urinary excretion of MHPG in TS; however, this finding has not been consistently replicated. Measurements of plasma MHPG ([Leckman et al., 1986](#)), and platelet  $\alpha_2$ -adrenergic receptors and plasma NE ([Silverstein et al., 1984](#)), after clonidine administration or withdrawal have been relevant to the phenomena of rebound and modulation of receptor sensitivity; however, they do not provide definitive information concerning possible alterations in TS. Assessment of the sympathetic nervous system by measurement of autonomic cardiovascular measures has not revealed any substantial differences in the Tourette group ([van Dijk et al., 1992](#)). However, studies of plasma, urinary, and CSF stress hormones before and after a lumbar puncture clearly suggest that at least some Tourette patients have an increased stress response ([Chappell et al., 1994, 1996](#)). This conclusion is consistent with the results of the largest study of NE and MHPG in CSF, which found that, although MHPG levels were normal, concentrations of NE were elevated nearly twofold ([Leckman et al., 1995](#)). Given the much shorter half-life of NE compared to MHPG, the results are indicative of normal basal stress response functioning and increased acute stress responsivity.

#### SEROTONIN

A role for 5-HT has been hypothesized because of the connection between TS and obsessive-compulsive disorder ([Grad et al., 1987](#); [Montgomery et al., 1982](#); [Nee et al., 1982](#); [Pauls et al., 1986](#)). Effective treatment of obsessive-compulsive symptoms in Tourette patients with the serotonergic uptake inhibitors fluvoxamine and fluoxetine has further stimulated research on the connection between the disorders and symptoms ([Riddle et al., 1992](#)). Earlier studies of CSF 5-HIAA reported normal or slightly lowered levels in TS; however, a larger and more recent study found similar levels of CSF 5-HIAA in patients with TS, OCD, or TS plus OCD, and the normal control group ([Leckman et al., 1995](#)). No studies of 5-HT receptor functioning have been carried out in Tourette patients, and early reports of benefit from the immediate 5-HT precursor, 5-hydroxytryptophan, have not been replicated ([Van Woert et al., 1982](#)).

Research on PM brain tissue has found decreases in 5-HT, 5-HIAA, and tryptophan across nearly all cortical and subcortical areas in Tourette patients ([Anderson et al., 1992a, 1992b](#)). Further PM research is necessary in order to replicate the findings; however, the results tend to increase the possibility that 5-HT may be a factor in the symptomatology of TS. In other indolamine-related research, [Dursun et al. \(1994\)](#) reported increased levels of plasma kynurenine in Tourette patients and suggested that this may reflect a shunting of tryptophan metabolism away from the production of 5-HT.

#### OTHER STUDIES

The importance of ACh and GABA in basal ganglia neural transmission has led to treatment studies using choline and other cholinergic agents ([Dursun et al., 1996](#); [Stahl and Berger, 1981](#)), as well as GABAergic agents ([Mondrup et al., 1985](#)). However, studies of CSF acetylcholinesterase ([Singer et al., 1984](#)) and CSF GABA have not found group differences. Reports of increased red blood cell choline have continued to appear ([Sallee et al., 1992](#)) and muscarinic receptor binding in white blood cells has been reported to be drastically lowered in TS ([Rabey et al., 1992](#)).

Immunohistochemical study of met-enkephalin, substance P, and dynorphin in basal ganglia areas of PM brain has suggested that dynorphin is reduced in the lateral globus pallidus in TS, whereas the other peptides appear normal ([Haber et al., 1986](#); [Haber and Wolfer, 1992](#)). Levels of dynorphin measured in CSF now appear to be unaltered in Tourette subjects ([Leckman et al., 1987](#); [van Watum et al., 1999](#)). In other studies of opioid systems in TS, small treatment effects were seen with naloxone ([Chappell et al., 1992](#)), whereas little effect was seen after the administration of the  $\kappa$  agonist, spiradoline ([Chappell et al., 1993](#)). The behaviorally important and related neuropeptides arginine-vasopressin and oxytocin also have been measured in CSF in Tourette patients; the results are inconclusive at this time ([Altemus et al., 1992](#); [Leckman et al., 1994](#)).

The amino acid neurotransmitters have been little studied. In the one study of central excitatory amino acids, PM brain levels of glutamate were lowered in the three projection areas of the subthalamic nucleus ([Anderson et al., 1992a,b](#)). It was hypothesized that this might lead to disinhibition of the thalamocortical circuit. This would tend to place Tourette in the group of hyperkinetic movement disorders, including Huntington's disorder and hemiballismus. As reviewed by [Swerdlow and Young \(2001\)](#), further research is needed to clarify basal ganglia functioning in TS. Imaging studies ([Peterson, 2001](#); [Peterson et al., 1993](#)) and the PM research have provided interesting leads to consider with respect to the functioning of cortico-striato-pallido-thalamocortical pathways.

## CONCLUSION AND FUTURE RESEARCH

The underlying mechanisms that regulate the unfolding maturation of the CNS are regulated by genetic factors concerned with the timing of expression of particular genes. Findings concerning homeobox genes and other regulators of gene expression are relevant to the investigation of the onset of specific disorders at certain phases of brain maturation (e.g., autism very early on, ADHD in the earliest school-age years, TS somewhat later, and obsessive-compulsive disorder several years later). Many steps from genetic to neuronal functioning have been characterized for model systems, including the localization, cloning, and sequencing of the genes for important enzymes and receptor systems. Gene activity can be assessed through measurement of messenger RNA expression (the “transcriptome”), synthesized proteins (the “proteome”), and the full range of small molecules produced through enzymatic activity. Basic neuroscience research will shed more light on the steps involved in synthesis, storage, release, and reuptake of transmitter compounds; mechanisms of coupling between transmitter and receptors; and the cascade of events that follows on receptor stimulation (alterations in membrane channels and other membrane changes, reconfiguration of receptors, activation of second-messenger systems, and altered gene expression).

The neurochemical approach to brain functioning is increasingly being integrated with other perspectives, including the neuroanatomic, neurophysiologic, and developmental. There is an ongoing reappraisal of the role of traditional neurochemical approaches, including measurements of neurotransmitters, enzyme systems, specific proteins, and metabolites. There are sound reasons for the continued prominence of the neurochemical approach—the role of neurochemical research in understanding drug action, the search for mechanisms underlying the range of expression of specific genes, and the recognition that for many disorders there are likely to be polygenic contributions, as well as gene–environment interactions, that are expressed in neurochemical systems. Neurochemical concepts underlie and are being enriched by the availability of methods of brain imaging such as PET, magnetic resonance spectroscopy (MRS) and SPECT, which provide information about brain functioning. With the discovery of more specific ligands for brain neurotransmitter receptors, it is becoming possible to perform neuropharmacologic studies (e.g., localization of receptors and determination of receptor numbers, affinities, and occupancy) in humans that previously were feasible only with isolated brain slices. The detection of specific sites of neurochemical abnormality in PM brain specimens, in turn, may provide suggestions for where to look in the human brain using brain-imaging techniques.

Determining neurochemical phenotypes may make critical contributions to pulling apart disorders. As has been pointed out with respect to schizophrenia, “Instead of . . . mapping (the disorder) per se, it may be fruitful to map the genetic basis for atypical biochemical or physiological responses found in (the disorder)” ( [Lander, 1988](#)). In fact, there should be occasions where biochemistry or neurochemistry can become the independent variable, creating meaningful subcategories for fruitful exploration in a fashion analogous to that seen in other more biologically based fields of medicine ( [Feinstein, 1985](#); [Heninger, 1999](#)).

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## 5 DEVELOPMENTAL ASPECTS OF NEUROPHYSIOLOGY

Edward M. Ornitz, M.D.

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During the developmental progression from infancy through early and later childhood to adolescence, the striking behavioral, emotional, and cognitive changes with which child psychiatrists are familiar accompany neuroanatomic indices of complex brain growth patterns. These include gross measures of brain growth, such as linked brain weight and head circumference measurements (human brain weight is proportional to the cube of head circumference) ([Epstein, 1979, 1986](#)), thickness of cerebral cortex and size of pyramidal cells ([Rabinowicz et al., 1977](#)), progression of regional myelination of specific brain areas ([Yakovlev and Lecours, 1967](#)), neuronal density in frontal cortex ([Huttenlocher, 1979; Rabinowicz, 1986](#)), synaptic density in frontal ([Huttenlocher, 1979](#)) and visual ([Huttenlocher et al., 1982](#)) cortex, and dendritic and axonal differentiation of prefrontal cortex neurons ([Mrzljak et al., 1990](#)).

Two types of maturational brain changes occur. There are progressive changes across the childhood and adolescent years, and maturational growth spurts at particular ages. From infancy to adulthood, there are progressive increases in brain weight ([Huttenlocher, 1979](#)), head circumference ([Epstein, 1979, 1986](#)), and cerebral cortical thickness ([Rabinowicz, 1986; Rabinowicz et al., 1977](#)), and a progressive decrease in neuronal density in cerebral cortex ([Huttenlocher, 1979; Rabinowicz, 1986](#)) and other cortical and subcortical regions ([Rabinowicz, 1986](#)). The decrease in neuronal density is precipitous before birth, with a slower decrease to about 6 to 15 months, after which time adult values are reached ([Rabinowicz, 1986](#)). Synaptic density increases from before birth to 8 months of age in visual cortex ([Huttenlocher et al., 1982](#)), to about 3 years of age in auditory cortex ([Huttenlocher and Dabholkar, 1997](#)), and to between 1 ([Huttenlocher, 1979](#)) and about 3 ([Huttenlocher and Dabholkar, 1997](#)) years of age in prefrontal cortex and then progressively declines to adult values, which are obtained between 12 years of age and midadolescence. The dendritic and axonal architecture in the different layers of prefrontal cortex appear to mature from this time to late in adolescence ([Mrzljak et al., 1990](#)). Concomitantly, there is continuing synaptic elimination throughout childhood and adolescence ([Huttenlocher and Dabholkar, 1997](#)).

These progressive changes in postnatal brain structure seem to involve two overlapping phases, a more rapid one occurring between birth and 1 and 3 years and a slower one occurring between 1 and 11 years of age with continuing changes during adolescence and young adulthood. The manifestation of these changes in prefrontal cortex are of particular importance because of their association with stages of cognitive development during childhood ([Goldman-Rakic, 1987](#)). The earlier prefrontal cortical phase involves the persistence of granular layer IV (which becomes dysgranular in the adult brain), the gradual disappearance of the subplate zone (a transient fetal-neonatal zone associated, in frontal cortex, with prolonged postnatal growth of cortico-cortical pathways) during the first 6 months of postnatal life, and overproduction of cortico-cortical axons associated with excessive synaptogenesis ([Goldman-Rakic, 1987; Kostovic, 1990](#)). In general, during this early childhood phase of progressive frontal cortical maturation, the transient preterm pattern is transformed into the "structurally adult-like pattern of cortical organization" ([Kostovic, 1990](#)). The early phase is also associated with complex changes in frontal cortical cholinergic receptor function, marked by changing acetylcholine esterase (AChE) activity ([Kostovic, 1990](#)). During the later childhood to adolescent progressive phase, the gradual increase in AChE reactivity in layer III pyramidal neurons has been associated with the innervation of cortical association neurons, synaptic reorganization, and cognitive development ([Kostovic, 1990](#)).

Some of these progressive increments or decrements in brain growth measures are not smooth; rather, they are characterized by spurts and lags in development, resulting in peaks and troughs of brain growth at particular age periods. Detailed analyses of the rate of brain weight and head circumference change have suggested peaks in brain growth rates around ages 3, 7, 11 to 12, and 15 years, with troughs in the intervening years ([Epstein, 1974a, 1979; Thatcher, 1980](#)). In the most recent reconsideration of the brain weight and head circumference data, [Epstein \(1986\)](#) concluded that the peaks in brain growth rates were statistically significant at 7, 11 to 12, and 15 years for male brains. Using a different statistical approach, [McCall and colleagues \(1983\)](#) were able to confirm the significance of only the 15-year-old peak. However, detailed neuroanatomic studies of cortical thickness, pyramidal cell development, neuronal density, and myelination document the validity of the earlier stages. Progressive increases in cortical thickness are interrupted by maturational troughs at 2 and 6 years, followed by accelerated growth between 2 to 4 years and 6 to 10 years ([Rabinowicz, 1986; Rabinowicz et al., 1977](#)). (This is a general statement. Particular cortical regions vary somewhat in the timing of these troughs and peaks in rates of cortical thickening.) Pyramidal cell size and shape also show two periods of accelerated change, between 15 months and 2 years and between 8 and 10 years ([Rabinowicz et al., 1977](#)), and neuronal density, which reaches adult values by 15 months of age, shows a consistent small peak of cell density around 6 years of age in frontal, parietal, temporal, and limbic cortex ([Rabinowicz, 1986](#)). From the cortical thickness, pyramidal cell size and shape, and neuronal density data, [Rabinowicz \(1986\)](#) concluded that: (a) an important period in cortical maturation occurs between 15 and 24 months and (b) a period of cortical "remodeling" takes place between 6 and 8 years. He also referred to unpublished data that suggested additional periods of "cortical remodeling" between 10 and 12 years and around 18 years.

Myelination proceeds at different rates for different brain regions, but, in general, myelination of most tracts is completed at or before 2 to 3 years of age. The reticular formation, the nonspecific thalamic radiations, and the great cerebral commissures are not completely myelinated until between 8 and 10 years of age. Myelination of the intracortical association areas continues into the second and third decades ([Lecours, 1982; Yakovlev and Lecours, 1967](#)). A more recent study suggests earlier maturation of myelination ([Brody et al., 1987](#)), but data are available to only 2 years of age. Myelination of the superior medullary lamina of the parahippocampal gyrus shows a progressive increase throughout childhood. Axons in the superior medullary lamina connect limbic and cortical structures and, hence, "could play some role in the corticolimbic integration of emotional behaviors with cognitive processes" ([Benes et al., 1994](#)).

In general, there appear to be four periods of major structural change in brain development, which punctuate the progressive increases and decreases in the size of the brain and its substructures at both the macroscopic and microscopic levels. The first takes place during early childhood, between about 15 months and 4 years of age. The second occurs during or toward the end of late childhood, some time between 6 and 10 years of age. The third is associated with prepuberty, and the fourth, at least in males, occurs in midadolescence. [Epstein \(1974b\)](#) has suggested correlations between these accelerated periods of brain growth and spurts in mental age occurring during the same time periods. Although some critics have found these correlations to be unconvincing because of a paucity of behavioral data ([Fischer and Silvern, 1985](#)) and an inability to confirm concordance between spurts in brain and mental growth statistically ([McCall et al., 1983](#)), it is noteworthy that the first of these periods of brain growth overlaps the piagetian mental stages of sensorimotor and preoperational behavior, whereas the second coincides with the piagetian stage of concrete operations, and the third and fourth overlap the piagetian stage of formal operations ([Inhelder and Piaget, 1958](#)). Thus, the stages of brain growth seem to occur in association with significant mental developments that involve important advances in both cognitive and emotional functions.

Developmental neurophysiology, the subject of this chapter, focuses on the functional correlates of the structural brain changes that occur during development. These changes in brain function should relate to the behavioral changes occurring during the developmental progression from infancy, through childhood, to adolescence and adulthood. Neurophysiologic studies of development fall into two broad categories: the study of the brain in its resting state and the study of the brain perturbed by external stimuli. The former involves primarily studies of changes in electroencephalogram (EEG) frequencies in the waking state and changes in sleep parameters, notably the tonic and phasic components of rapid eye movement (REM) sleep. The latter includes the startle response and its modulation, nystagmus evoked by vestibular stimulation, and exogenous and endogenous event-related potentials. This chapter is limited for the most part to studies that encompass all or the greater part of childhood, from the preschool period to adolescence, and provide quantitative data permitting the evaluation of maturational changes. Studies from early infancy are included only when comparable data are available later during childhood. The many important studies confined to the newborn and very young infant are not included; an excellent review of these can be found in [Graham and associates \(1983\)](#) in the context of orienting responses.

### DEVELOPMENTAL CHANGES IN THE EEG

[Niedermeyer \(1993\)](#) provides an excellent comprehensive review of *qualitative* descriptions of the maturation of the EEG, including EEG patterns in the premature and full-term newborn. This chapter focuses on *quantitative* studies of changes in EEG frequencies with maturation. Developmental EEG studies have utilized hand



measurement of EEG frequencies ([Eeg-Olofsson, 1980](#); [Lindsley, 1936, 1939](#); [Petersen and Eeg-Olofsson, 1971](#)), analog frequency analysis ([Matousek and Petersen, 1973](#)), spectral analysis ([Matthis et al., 1980](#); [Thatcher et al., 1987](#)), and more recently measures derived from the theory of nonlinear dynamics and deterministic chaos ([Anokhin et al., 1999](#)).

In general, regardless of the method used, EEG maturation between about 5 and 11 years of age has been characterized by a reduction in slower frequencies (those in the delta and theta bands) and an increase in higher frequencies (primarily the alpha band) ([Eeg-Olofsson, 1980](#); [Matousek and Petersen, 1973](#); [Matthis et al., 1980](#)). Some of the slower frequencies tend to increase from 1 to between 5 and 8 years of age and then decrease to 15 years of age ([Petersen and Eeg-Olofsson, 1971](#)). Theta activity shows a developmental peak at 4 years of age ([Matousek and Petersen, 1973](#)). Between 6 and 11 years, the power in the theta band decreases linearly with age. Also, the latency of the theta activity evoked by auditory stimuli decreases with age, and this decrease is highly correlated with the developmental decrease in the latency of the P300 component of the event-related potential (ERP) evoked by the same stimuli ([Yordanova and Kolev, 1997](#)). The frequency of the more extensively studied occipital alpha rhythm increases progressively from 3 months of age to adulthood ([Lindsley, 1936, 1939](#); [Petersen and Eeg-Olofsson, 1971](#)). The relative spectral power of alpha in the 9.5- to 12.5-Hz band increases between 4 and 11 years of age ([Matthis et al., 1980](#)), the amplitude of 9.5- to 12.5-Hz alpha increases from 1 to 21 years of age ([Matousek and Petersen, 1973](#)), and the mean absolute phase of 7- to 13-Hz alpha increases from birth to adulthood ([Thatcher et al., 1987](#)). Most of these progressive increases in alpha activity are exponential, with rapid increases from birth to about 5 or 6 years of age, followed by slower rates of change to adulthood; these changes closely parallel a similar exponential growth curve for brain weight ([Huttenlocher, 1979](#)). A study of nonlinear aspects of EEG organization utilized a measure of EEG complexity to reveal a highly significant effect of age, from infancy through childhood to adulthood. This increasing EEG "complexity" with age is thought to reflect "the evolution of complexity of underlying brain activity" with maturation ([Meyer-Lindenberg, 1996](#)).

The progressive developmental changes in alpha activity are punctuated by spurts and lags. [Epstein \(1979, 1980, 1986\)](#) analyzed the developmental EEG data of [Matousek and Petersen \(1973\)](#) in terms of biennial increments of change and demonstrated EEG developmental stages with peaks of alpha activity around 4, 7 to 8, 11 to 12, and 15 years, and intervening troughs at 6, 9 to 10, and 13 years. These EEG developmental stages coincide quite well with the brain growth stages based on analyses of brain weight-head circumference data, cortical thickness data, pyramidal cell dimensions, and neuronal density changes, as described. The well-defined trough in the development of alpha activity at 6 years, followed by the peak at 7 to 8 years ([Epstein, 1980, 1986](#)), coincides with the beginning of a brain development stage that [Rabinowicz \(1986\)](#) describes as "a remodeling of the cortex." This restructuring of cortical substance involves a relative decrease in cortical thickness, a corresponding decrease in length and increase in width of the Betz cells, and an increased cell density. Thus, functional and structural brain changes coincide at a critical age in child development, the age that for most children is associated with the readiness to learn in a school setting, or, in piagetian terms, to engage in operational thinking.

Using measures of EEG coherence and phase, [Thatcher and colleagues \(1987\)](#) confirmed EEG periods of growth and change around 4 to 6, 8 to 10, 11 to 14, and 15 years and demonstrated hemispheric differences in participation in these developmental stages. The 4 to 6 years period involves spurts in left hemispheric (frontal-occipital and frontal-temporal) coupling and right frontal pole pairing, whereas the 8 to 10 years period is notable for accelerated development of right hemispheric (frontal-temporal) connections. The preadolescent and adolescent periods are bilateral, involving primarily frontal lobe connections. Comparison of the [Thatcher and coworkers \(1987\)](#) findings and further mathematical analysis of the [Matousek and Petersen \(1973\)](#) data by [Hudspeth and Pribram \(1990\)](#) showed "a striking similarity to the timing and sequencing" of the EEG growth spurts ([Thatcher, 1991](#)).

Although relatively little is known about the development of hemispheric differences, it is most interesting to note recent evidence of a developmental shift in dendritic prominence (total dendritic length) from the right to left motor speech areas ([Simonds and Scheibel, 1989](#)). This shift to left-sided primacy of language-related areas occurs during the same 4- to 6-year period during which the spurt in left frontal-temporal EEG coupling occurs ([Thatcher et al., 1987](#)). This ontogenetic shift in hemispheric structure and function parallels an evolutionary trend toward lateralization of auditory cortex ([Thatcher, 1980](#)).

## DEVELOPMENT OF SLEEP PATTERNS

Between birth and late adolescence the total time spent asleep is reduced from about 16 to about 8 hours ([Roffwarg et al., 1966](#)). Of greater interest for the neurophysiology of development is the fact that REM sleep accounts for 50% of total sleep time at birth but only 20% by 3 years, at which time adult values are approached.

The anlage of the REM state makes its appearance in the fetus before birth. By 28 to 30 weeks it is possible to begin to identify REM and non-REM sleep states ([Parmelee and Stern, 1972](#)). At birth REM sleep is relatively "undifferentiated" in comparison with REM sleep in older children ([Emde and Metcalf, 1970](#)). Two major changes mark the differentiation of REM sleep within the first 3 months of life: Starting at approximately 2½ months of age, sleep begins with a non-REM instead of REM period ([Emde and Metcalf, 1970](#)), and the initial high variability in physiologic patterning of neonatal REM sleep becomes organized, so that behavioral and electrophysiologic events uncharacteristic of REM sleep become segregated out of REM sleep and into drowsy and non-REM states. For example, EEG activity during REM sleep in the frequency range of stage 2 sleep spindles in 6- to 8-month-old babies is significantly reduced by 19 to 45 months ([Ornitz et al., 1971](#)). Concomitant with this increasing organization, there is a decrease in REM period length and the percent of total sleep time occupied by REM sleep ([Parmelee and Stern, 1972](#); [Roffwarg et al., 1966](#)) to approximately 40% by 3 to 5 months of age and to 30% by 6 to 23 months ([Roffwarg et al., 1966](#)). By 3 years, although total sleep time has decreased from 16 to 11 hours per day, REM sleep time has decreased from 8 to about 2 hours per day ([Roffwarg et al., 1966](#)). REM sleep is an activated state of consciousness, with a low-amplitude, high-frequency EEG similar to the waking EEG record and a tonic muscular atonia, punctuated by phasic motor and autonomic behavior such as the bursts of rapid eye movements, twitching movements of the extremities, and increased fluctuations of cardiovascular and respiratory activity, and phasic inhibition of responses to sensory input. [Roffwarg and associates \(1966\)](#) suggested that the REM state serves the newborn and young infant as an endogenous source of stimulation when less exogenous stimulation is available. It is notable that the sum of waking time (8 hours) and REM sleep time (8 hours) in the newborn is almost the same as the sum of waking time (13½ hours) and REM sleep time (2 hours) in the 5- to 9-year-old child, supporting the idea that one function of REM sleep during early development is to compensate for reduced time awake. It is most noteworthy, however, that the percent of total sleep time required for REM sleep has decreased to the adult level around 3 years of age, the time of completion of myelination of many subcortical pathways and the first major spurt in brain growth.

The ontogenetic changes in the percent of REM sleep time are accompanied by developmental changes in the phasic motor activity of REM sleep. After adjustment for the differences in the time of night during which REM sleep episodes occur in the course of early childhood development, [Tanguay and associates \(1976\)](#) showed that the rapid eye movements of REM sleep clustered significantly more tightly into bursts with increasing age from 3 months to about 6½ years. This increasing organization of eye movement activity is accompanied by increasing inhibition of the relative amplitude of evoked responses to auditory stimuli during the eye movement bursts, with increasing age from 6 months to about 6½ to 9 years, during which time adult levels of phasic inhibition are reached ([Ornitz, 1972](#); [Ornitz et al., 1969](#)). Hence, neither the phasic motor excitation nor the phasic sensory inhibition of REM sleep are well defined in early infancy; however, these concurrent neurophysiologic processes develop rapidly during early childhood and mature after the preschool period. The phasic activity of REM sleep is generated in the parabrachial reticular formation and mediated by the medial vestibular nucleus (MVN) ([Pompeiano, 1967a,b, 1980](#)). The contribution of the MVN, a central vestibular structure, to the development of phasic motor activity during REM sleep is particularly interesting, as is presented in the next section where we consider the developmental changes in vestibular function in the waking child.

Tonic muscle atonia is an important neurophysiologic component of REM sleep. This atonic state is interrupted by episodes of phasic muscle activity that are most intense in early infancy and show increasing inhibition with maturation, reaching adult levels between 6 and 12 years of age ([Kohyama et al., 1997, 1999](#)). This activity, manifest and measured as chin muscle twitches, occurs simultaneously with the rapid eye movement bursts at a high rate in the newborn. By 4 to 6 months of age, the rate of concurrence of the muscle twitches and rapid eye movement bursts has dropped to adult levels ([Kohyama, 1996](#); [Kohyama et al., 1999](#)); therefore, maturation of the organization of the rapid eye movement bursts (increased clustering) during infancy and childhood is accompanied by both increased sensory inhibition (suppression of auditory evoked responses) and increased motor inhibition (dissociation from the phasic muscle activity).

The functions of the phasic events of REM sleep have rarely been discussed. However, these short, intermittent, recurrent periods of very intense motor activation (e.g., bursts of rapid eye movements, twitching of the muscles of the middle ears, vibrissae, and extremities) are embedded in the longer REM sleep periods, whose function is thought to be the activation of the brain during sleep without arousal of the organism ([Vertes, 1984, 1986](#)). This view is consistent with that of [Roffwarg and coworkers \(1966\)](#), who postulated that REM sleep "serves as an endogenous source of stimulation" in early infancy. Presumably, the need for such endogenous stimulation becomes less with maturation during childhood, as waking time increases and the quality of waking life becomes increasingly active. Hence, as the percent of REM sleep time declines precipitously during the first 3 years of early childhood ([Roffwarg et al., 1966](#)), the phasic motor activity of REM sleep becomes more concentrated in discrete periods of time ([Tanguay et al., 1976](#)), and the phasic sensory inhibition becomes more pronounced ([Ornitz, 1972](#); [Ornitz et al., 1969](#)). The increase in phasic sensory inhibition with maturation may serve to protect the developing child from the waking effect of exogenous stimulation at the time of the most intense motor excitation.



A change in the sleep EEG pattern of non-REM sleep also occurs around 3 years of age. The percent time and number of the 12- to 14-Hz sleep spindles and the duration of the spindles during stage 2 sleep all decrease from 4 months of age, reaching minimal values at 3½ years, and then increase between 4 and 5 years of age ([Tanguay et al., 1975](#)). Spindle rhythms are thought to be generated in reticular thalamic neurons that project to thalamocortical neurons, and brain stem reticular activity blocks the generation of EEG spindling by inhibitory projections to the reticular thalamic neurons ([Steriade, 1988](#)). Perhaps the decrease in EEG spindles around 3½ years of age reflects a peak period of brain stem reticular or reticular thalamic activity associated with the first major spurt in brain maturation.

## DEVELOPMENTAL CHANGES IN VESTIBULAR NEUROPHYSIOLOGY

Vestibular reflexes serve primarily to stabilize gaze during head movements and maintain equilibrium during body movements and are influenced by, and interact with, visual and proprioceptive inputs. The development of these aspects of vestibular reflexes are addressed in this section. Other functional roles for vestibular reflexes, such as compensation for changes in the direction of the force of gravity and maintenance of tonic muscular tone ([Baloh and Honrubia, 1979](#)), have not been systematically studied in the human child and are not considered here.

Vestibular function is most readily studied by quantifying changes in the vestibulo-ocular reflex (VOR). Quantification of the VOR is facilitated by measurement of certain parameters of the ocular nystagmus induced by angular acceleration, and understanding of human vestibular function and its development has depended primarily on this approach. The nystagmus consists of alternating slow and fast conjugate deviations of the eyes. The velocity of the slow deviation in the opposite direction to that of the acceleration reflects vestibular activity. The VOR is the output of a first-order open-loop control system, best characterized by its gain and time constant. In response to constant or impulsive accelerations, the gain is the nystagmus slow-component velocity (SCV) relative to the head acceleration or to the sudden change in velocity. The time constant (the exponential rate of change of the SCV in response to the acceleration) reflects the viscoelastic properties of the cupula (peripheral time constant) and the effect of a central integrator that prolongs the peripheral time constant to that of the nystagmus. In response to sinusoidal accelerations, the gain is the nystagmus SCV relative to the head velocity, and the time constant can be computed from the phase shift between nystagmus and head velocities ([Honrubia et al., 1982](#)).

The vestibular system is both anatomically complete and functionally responsive at or before birth. Histologically, the vestibular apparatus appears complete in the human fetus at 9½ weeks of gestation ([Hooker, 1952](#)). The large neurons of the vestibular nuclei are "unquestionably functional" at 21 weeks of gestation ([Humphrey, 1965](#)). The myelination of the vestibular system is well advanced at 6 months of gestation ([Langworthy, 1933](#)). The endolymphatic and bony labyrinths ([Dayal et al., 1973](#)) and the number of myelinated vestibular nerve fibers ([Bergstrom, 1973](#)) and vestibular hair cells ([Rosenhall, 1972](#)) are mature at birth. Newborn infants respond to acceleration, the slow component of nystagmus deviating in the expected direction (i.e., opposite to that of the acceleration) ([Tibbling, 1969](#)), although the fast component, which is of pontine origin ([Cohen, 1972](#)), may not occur.

There are functional changes in the vestibular system during development from early infancy to adolescence that suggest considerable plasticity of the nervous system during childhood. Vestibular responses to an equivalent stimulus, be it caloric or rotational, differ markedly in preadolescent children from that found in adults ([Conraux and Collard, 1970](#); [Jongkees and Gasthuis, 1973](#)). The responses of children to vestibular stimulation are not only different from the responses of adults but also differ within the childhood age span, with the most marked maturational changes taking place in preschool children ([Ornitz, 1985](#); [Ornitz et al., 1979](#)). During the first year of life, the vestibular system is functioning at its highest level of reactivity, a level that decreases with subsequent development. At the same time, suppression of the VOR by visual fixation is minimal or absent ([Ornitz and Honrubia, 1988](#)). The peaking of vestibular activity during the first year of life is associated with overflow phenomena in the form of behaviors such as rocking and is followed by a subsequent rapid decrement in vestibular reactivity. These features of the maturation of vestibular function suggest that important modifications of vestibular mechanisms take place rapidly during the first few years of life and continue to do so, albeit at a slower pace, throughout childhood ([Ornitz, 1983, 1988](#)).

Those studies that have measured appropriate nystagmus parameters (i.e., the SCV or its expression as VOR gain) have demonstrated the following developmental sequence. Nystagmus is present in the full-term newborn, subject to sufficient arousal, and also in the premature and small-for-dates neonate. Nystagmus is weaker in the latter groups, but after several months of postnatal life, the differences are no longer apparent. The maximum intensity of the vestibular response probably occurs by 6 months, and it is about this time that spontaneous vestibular self-stimulation first occurs, indicating a period of heightened vestibular reactivity. After the first year, the intensity of vestibular nystagmus and spontaneous vestibular behavior decrease rapidly until about 30 months of age, following which there is a gradual decrease toward adult values between 7 and 10 years. This decrease in the intensity of the vestibular response (reduced velocity of nystagmus and reduced VOR gain) is associated with increasing time constants ([Ornitz, 1985](#); [Ornitz et al., 1985](#)). The association of decreasing gain with increasing time constants during development suggests that the immature human vestibular system is characterized by relatively deficient modulation of vestibular reflexes. It can be postulated that the decreasing gain reflects increasing modulation of vestibular function with development and that increasing time constants reflect the development of the modulating mechanisms. Both pharmacologic ([Blair and Gavin, 1979](#)) and neurophysiologic ([Buettner et al., 1978](#)) investigation suggest that the modulating mechanisms reside in reverberating brain stem reticular formation neuronal networks.

Studies of the development of vestibular adaptation to constant acceleration also suggest that the postnatal development of the vestibular system involves central mechanisms. The adaptive process that shapes the time course of the total nystagmus response to constant angular acceleration begins during the decline of the postacceleration primary nystagmus and is amplified during the subsequent secondary nystagmus (nystagmus with slow components in the opposite direction to that of the primary nystagmus) ([Malcolm and Melvill-Jones, 1970](#)). The maximum SCV in all adults occurs at the same time during secondary nystagmus, regardless of stimulus magnitude ([Sills et al., 1978](#)). [Sills and coworkers \(1978\)](#) attribute this consistency to a central, stimulus-independent process. In children, the maximum SCV during secondary nystagmus varies according to age and by 10 years of age is close to the values of Sills and associates for adults ([Ornitz, 1983](#)). These data, together with developmental changes in secondary nystagmus/primary nystagmus peak SCV ratios ([Ornitz et al., 1979](#)), show strong age-dependent effects on adaptation during the childhood years. The maturational changes in VOR gain and time constant ([Ornitz et al., 1985](#)), vestibular adaptation ([Ornitz, 1983](#); [Ornitz et al., 1979](#)), and visual-vestibular interaction ([Ornitz and Honrubia, 1988](#)) all support the notion that the postnatal development of the human vestibular system involves central modulating mechanisms.

The preceding considerations apply to the development of the mechanisms underlying the slow components of nystagmus, which originate in the vestibular nuclei and are modulated by brain stem reticular formation (BSRF) activity. The fast components, on the other hand, are generated in the BSRF, more specifically in the parabrachial reticular formation (PPRF) ([Cohen, 1972](#)). The likelihood of generating fast components in response to near-threshold vestibular stimulation increases progressively from early childhood to young adulthood ([Fuster et al., 1994](#)). Because the same PPRF neurons generate both vestibular fast components and saccades, the maturation of fast component activity may parallel the fine tuning of saccade-mediated vestibulo-ocular mechanisms, which mature throughout childhood. Because the gain of the slow component decreases with development, whereas the fast component and saccadic activity increase, it can be postulated that vestibular responsivity is modified by visuovestibular interaction with maturation ([Ornitz and Honrubia, 1988](#)). This developing interaction, in turn, is mediated by the development of the same BSRF circuits in the PPRF, which lengthen the nystagmus time constant and modulate vestibular output ([Fuster et al., 1994](#)). For more complete reviews of the development of vestibular function during childhood, see [Ornitz \(1983, 1988\)](#).

## DEVELOPMENTAL CHANGES IN STARTLE MODULATION

The magnitude of the startle blink reflex in the human adult can be modulated by nonstartling prestimulation in three ways ([Anthony, 1985](#); [Graham, 1975](#)). Inhibitory modulation of the startle response to an intense sudden stimulus follows brief (20-msec), low-intensity, nonstartling stimuli presented at short intervals (30 to 240 msec) prior to the startling stimulus ([Graham, 1975](#); [Graham and Murray, 1977](#)). At these short prestimulation intervals, the same amplitude inhibition may ([Graham and Murray, 1977](#)) or may not ([Blumenthal and Levey, 1989](#)) occur when the prestimulation is sustained continuously throughout the warning interval, depending on the prestimulation intensity and other experimental parameters. Facilitation of startle magnitude follows longer (>1,400 msec) prestimuli that are sustained continuously throughout the prestimulation interval preceding the startling stimulus ([Graham, 1975](#); [Graham et al., 1975](#)). Facilitation following these long, sustained prestimuli can occur in experimental designs in which the prestimulation interval is constant or is variable in duration ([Anthony, 1985](#); [Graham, 1975](#); [Graham et al., 1975](#)). In contrast, a brief (20-msec) stimulus presented 2,000 msec prior to the startling stimulus induces startle facilitation only in the context of variable prestimulation intervals and not when the prestimulation interval is constant ([Graham, 1975](#); [Graham et al., 1975](#)). Because of the uncertainty associated with the variable prestimulation intervals, the facilitating effect was attributed to an orienting-attentional mechanism. Subsequent research has shown that in the adult human a third type of startle modulation involving nonstartling prestimulation is due to orienting-attentional influences, and the effect can be facilitatory or inhibitory, depending on whether attention is directed toward or away from either the prestimulus or startling stimulus ([Anthony, 1985](#)).

Inhibitory startle modulation is a brain stem function involving the processing of sensory input. Startle inhibition by prestimulation is mediated by an inhibitory pathway in the brain stem tegmentum as demonstrated by lesion ([Leitner and Cohen, 1985](#); [Leitner et al., 1981](#)) and stimulation ([Saitoh et al., 1987](#)) studies in the rat. This



pathway impinges on the primary startle pathway (Davis et al., 1982) at, or prior to, the medial pontomedullary reticular formation (Wu et al., 1988). As with the modulation of vestibular responses by polysynaptic brain stem reticular formation neuronal networks (Buettner et al., 1978) as described, startle modulation also matures between 6 and 10 years of age (Ornitz et al., 1986, 1990, 1991). Thus, a synchronous maturational effect involves two related functions, startle and vestibular modulation, because the motor component of the startle blink reflex, the facial motoneuron, receives vestibular projections (to the facial nucleus) (Shaw and Baker, 1983). Their suggested role is the coordination of facial movements, including blinking, with eye and head movements, all of this being motor activity, modulated in the brain stem, that is involved in the response to sudden sensory input.

Systematically collected data on startle modulation by prestimulation from infancy throughout childhood are available for only experimental paradigms that have not attempted to manipulate the direction of attention (Ornitz et al., 1986, 1990, 1991). Graham and colleagues have carried out such experiments during the first year of life and have reported on differences from adult responses (Anthony and Graham, 1983, 1985; Balaban et al., 1985, 1989; Graham et al., 1981). Anthony and Putnam (1985) compared startle modulation effects in 5-year-olds and adults. Prior to about 4 years of age, inhibitory startle modulation is characterized by fluctuating weak inhibition (25% response inhibition in 2- to 6-month-olds) (Graham et al., 1981), nonsignificant facilitation (17% response increase in 15-month-olds) (Balaban et al., 1989), and again, weak inhibition (23% response inhibition) in 3-year-old boys (Ornitz et al., 1986). In 4-year-old girls and boys, there is nonsignificant startle amplitude facilitation (about 10% and 22%, respectively) 120 msec following prestimulation. In 5-year-old boys, 30% response inhibition has developed. By 8 years, both girls and boys show strong significant response inhibition, 50% and 75%, respectively, approximating adult values. Similar developmental response patterns occur following prestimulation at 250 msec (Ornitz et al., 1986, 1991). Facilitatory startle modulation in response to prolonged sustained prestimulation follows a similar developmental course (Ornitz et al., 1986, 1991).

Both inhibitory and facilitatory startle modulation show, respectively, significant peak losses in startle inhibition and peak gains in startle facilitation at about 4 years of age, followed by a progressive increase in inhibition and decrease in facilitation until 8 years of age, when mature values are obtained (Ornitz et al., 1986, 1990).

Startle can also be modulated by habituation. In response to glabellar taps, the number of reflex blinks to habituation increased from the first year of life until 3 to 5 years of age. Habituation recurred at 6 years of age and became progressively stronger until adulthood (Zametkin et al., 1979). Hence, startle modulation by habituation shows the same loss of inhibitory modulation around 4½ years of age as does startle modulation by prestimulation. In a study of 7- to 11-year-old boys (Ornitz et al., 1996), habituation of acoustic startle occurred at the same rate as in young adults (Ornitz and Guthrie, 1989). Startle and its habituation were found to be independent of association with accompanying autonomic, alpha EEG, and tonic electromyogram (EMG) activities; whereas the startle response habituated, prestartle and poststartle cardiac, alpha, and myogenic levels did not (Ornitz et al., 1996). Tonic heart rate actually increased as startle habituated, suggesting sensitization of the state of these subjects during the experiment, a result consistent with the dual-process theory of habituation (Groves and Thompson, 1970).

Startle is also modulated by the affective state of the subject (Lang et al., 1990). Of the various experimental paradigms used to affect the emotional response to the startling stimulus, the fear-potentiated startle response has been of particular interest because of its application to both animal and human studies (Davis et al., 1993; Grillon et al., 1991). Until recently, however, there has been very little application to children. Infants who had shown strong motor responses and *negative* affect to test stimuli at 4 months showed potentiation of startle in the presence of a stranger at 9 months. In comparison, infants who had shown strong motor responses and *positive* affect at 4 months, showed reduced startle in the presence of a stranger at 9 months (Schmidt and Fox, 1998). Thirteen- to 17-year-old adolescents showed fear-potentiated startle both to threat of an air blast directed to the larynx and by darkness (Grillon et al., 1999). Based on animal studies (Davis, 1992; Gewirtz et al., 1998), these findings indicate that mediation by limbic structures, particularly the amygdala and bed nucleus of the stria terminalis, occurs throughout childhood, from infancy through adolescence.

For further review of the development of startle modulation during childhood, see Ornitz (1999).

As described, of the various types of startle modulation, the developmental sequence throughout infancy and childhood has only been completely described for inhibitory startle modulation. Inhibitory startle modulation matures during a critical period of human neurologic growth and development that is characterized by many important structural brain and functional neurophysiological changes, as described. Myelination of the brain stem reticular formation and the great cerebral commissures is not completed until sometime between 8 and 10 years (Yakovlev and Lecours, 1967). Rabinowicz (1986) describes a period of cortical "remodeling" characterized by changes in cortical thickness, pyramidal cell size and shape, and neuronal density that occurs between 6 and 8 years. Epstein (1986) describes a peak of brain growth (inferred from head circumference data) at 7 years, which he attributes to increased neuronal arborization. Epstein (1979, 1980, 1986) has demonstrated several peaks of alpha activity in the developmental EEG data of Matousek and Petersen (1973), one of which is a well-defined trough of alpha activity at 6 years, followed by a peak at 7 to 8 years. Between 8 and 10 years, EEG coherence and phase studies suggest accelerated development of right hemispheric frontotemporal connections (Thatcher et al., 1987).

All of these structural and functional changes in the human nervous system occur during that period of childhood when children are able to engage meaningfully in learning and, in most cultures, are enrolled in school. This is approximately the piagetian period of concrete operations (Inhelder and Piaget, 1958), during which time the child's concept of reality becomes less egocentric and less anthropomorphic, permitting the initial development of logical thinking. Hence, the maturation of inhibitory mechanisms in the brain stem that damp the response to strong external stimuli and the reduction of excitation following sustained stimulation (sensory modulation) parallel the development of cortical (cognitive) processes that require the capacity of the child to separate affectively and conceptually from external objects and events, a capacity that would seem to require inhibitory functions. In fact, tests of frontal lobe functions indicate an accelerated development of the capacity to inhibit the retroactive interference of a stimulus set on the recall of a prior stimulus set during this period of childhood (Passler et al., 1985).

## DEVELOPMENT OF EVENT-RELATED POTENTIALS

Event-related potentials (ERPs) are responses to stimuli in the EEG that may be exogenous or endogenous. The exogenous ERPs are always elicited by external events; they are obligatory responses whose characteristics (e.g., latency and amplitude) vary with the physical parameters of the stimuli. The endogenous ERPs are nonobligatory responses to stimuli, may occur in the absence of an expected stimulus, and are relatively unaffected by the physical parameters of the stimulus (Donchin et al., 1978). Hence, the exogenous ERPs represent relatively lower levels of sensory processing (stimulus registration), whereas the endogenous ERPs represent relatively higher levels of processing of the information conveyed by sensory stimuli (e.g., expectation, importance, or relevance of the stimulus, recognition of novelty). The endogenous ERPs are evoked by experimental conditions that influence attention and give significance to stimuli.

### Exogenous Event-Related Potentials

The extensive literature prior to 1978 on the maturation of exogenous ERPs during childhood has been thoroughly reviewed by Klorman and coworkers (1978). They found that, in general, the latencies of these responses to auditory, visual, and somatosensory stimuli decreased rapidly during the first year of life and changed more slowly or not at all from early childhood to adolescence. Because the exogenous ERP components are characterized by latencies less than 200 msec, the more marked decreases in latency during the first year were attributed to the early completion of myelination of specific thalamocortical sensory pathways. In contrast, the endogenous ERP components, which have longer latencies, show maturational changes throughout childhood, and these changes were attributed to the much later myelination of nonspecific brain stem and reticular thalamic pathways (Klorman et al., 1978). In the auditory system Eggermont (1992) has shown that the *rate* of maturation (during the first 2 years) of P2 and N2, ERP components occurring around 200 msec that may have either exogenous or endogenous ERP characteristics, depending on the conditions in which they are evoked, is the same as the rate of maturation of wave 5 of the exogenous brain stem auditory evoked response (BSAER) suggesting similar developmental rates for responses mediated by brain stem and more rostral auditory pathways.

The exogenous ERPs occur in all sensory modalities and include the very early brain stem and certain later auditory-evoked responses (AERs), the somatosensory-evoked potentials (SEPs), and the pattern reversal visual-evoked potentials.

### BRAIN STEM AUDITORY-EVOKED RESPONSE

The *brain stem auditory-evoked response* (BSAER) is composed of a series of five waves with peak latencies from about 1 to 6 msec (in adults). Based on experimental animal and clinical patient studies (Fabiani et al., 1979), these peaks represent auditory transmission from the auditory nerve (wave 1), through the cochlear nucleus (wave 2), the superior olivary complex (wave 3), to more rostral regions of the brain stem (waves 4 and 5). Thus, wave 1 represents peripheral auditory transmission, and the remaining components represent progressive central auditory transmission through the brain stem. With maturation during infancy and early childhood, the latencies of the several BSAER components shorten but at different rates. The peripheral transmission (wave 1) matures early, reaching the adult latency between the sixth week (Salamy and Mckean, 1976) and second month (Mochizuki et al., 1983) of postnatal life. The central transmission time, measured as the brain stem transmission time between waves 1 and 5, matures more slowly. Adult values are approached between ages 3 and 5 years (Fabiani et al., 1979);



Mochizuki et al., 1983) and finally reached at 8 years (Fabiani et al., 1984), the same time that another measure of brain stem development, startle modulation, matures (Ornitz et al., 1986, 1990, 1991).

Developmental amplitude changes in BSAER components have received less attention than latency changes (Jiang et al., 1993). Jiang and colleagues (1993) have observed an increase in wave 5 amplitude from birth to 5 years of age, followed by a gradual decrease in amplitude toward adult values. This peak of BSAER activity coincides developmentally both with the peak loss of inhibitory prestimulation modulation of startle (PMS) and the peak increment of facilitatory PMS (see the preceding) and the peak increment of the P300 component of the auditory ERP (see the following). The implications of these several manifestations of heightened responsivity to sensory stimuli are discussed later in this chapter.

#### AUDITORY-EVOKED RESPONSE

*Auditory-evoked response* (AER) peaks occurring between 50 and 200 msec can have both exogenous (i.e., obligatory) and endogenous (i.e., nonobligatory) characteristics, depending on the experimental conditions. These include the positive potentials P1 (at about 50 msec) and P2 (between 140 and 170 msec) and the negative potentials N1 (about 100 msec) and N2 (220 and 270 msec). The latencies refer to established adult values. Ponton and associates (2000) have provided systematic data on the maturational changes that occur between 5 and 20 years of age when these AER peaks are evoked by simple monaural auditory stimuli (a brief train of clicks) at a rapid stimulation rate, under experimental conditions that minimize the influence of attention, and are recorded at multiple electrode sites. It is emphasized that all of these experimental conditions may affect the assessment of maturational sequences and the exogenous or endogenous characteristics of the evoked activity. Under these conditions (Ponton et al., 2000), the P1 latency decreases exponentially from 80 to 110 msec at age 5 years to 30 to 50 msec at age 20 years and does not become adult-like until age 15 years. When recorded at lateral scalp electrodes, the P1 amplitude shows a striking step-like increase and decrease between ages 10 and 11 years. In contrast to the considerable developmental changes in P1, the later positive wave P2 shows no age-related latency changes and only a modest decrease in amplitude. The N1 latency shows an exponential decrease similar to that of P1; the N1 amplitude, recorded frontocentrally, does not develop a consistent negativity until about age 16 years. The negativity at N2 shows an exponential increase in latency and, at lateral electrode sites, an increased amplitude negativity at age 10 years followed by an abrupt decrease in amplitude. Hence, the pattern of developmental change described by Ponton and colleagues (2000) includes a shift from an exponential maturational latency decrease in the earlier peaks (P1 and N1), no maturational latency effect on P2, and a maturational increase in the later N2, and an abrupt change in amplitude at age 10 years for peaks P1 and N2. The maturational latency decrease for waves P1 and N1 has also been found in children in response to simple verbal stimuli (Paetau et al., 1995; Sharma et al., 1997). Other studies of the exogenous auditory-evoked N1 also found latency decrements with increasing age, across the age range 8 to 17 years (Tonquist-Uhlen et al., 1995) and longer latencies in 4- to 8-year-olds than in adults (Bruneau et al., 1997). The latencies of the N1 recorded from temporal scalp regions in response to both tones and speech sounds also decreased across the age range 3 to 16 years (Pang and Taylor, 2000). In contrast to the slow development of N1 negativity recorded at fronto-central sites (Ponton et al., 2000) as described, the amplitude of the auditory N1 was found to be greater over the temporal cortices in 4- to 8-year-old children than in adults (Bruneau et al., 1997; Pang and Taylor, 2000; Tonquist-Uhlen et al., 1995). A shift in N1 amplitude from a parietal to a fronto-central predominance occurred after 8 years of age in two studies (Bruneau et al., 1997; Tonquist-Uhlen et al., 1995) but not until 15 years of age in a recent study using relatively more rapid stimulation (Pang and Taylor, 2000). It should be noted that the maturational increase in latency of N2 reported by Ponton and colleagues (2000) is at variance with the maturational decrease reported in other studies of N2, most of which have used binaural stimulation and task-related conditions that elicit endogenous characteristics of this event-related potential. These studies are reviewed in the following discussion of wave N2 as an endogenous ERP component. Also, in response to verbal stimuli, and longer interstimulus intervals than those used by Ponton and associates (2000), the latency of the exogenous N2 decreased in children between 3 and 15 years of age (Paetau et al., 1995).

#### SOMATOSENSORY-EVOKED POTENTIALS

The *somatosensory-evoked potentials* (SEPs) comprise a series of scalp-recorded negative and positive peaks that vary in peak latency from about 20 to 200 msec following electrical stimulation of the fingers or the median nerve at the wrist (Klorman et al., 1978). Desmedt and his colleagues (Desmedt et al., 1980) have carried out the most extensive studies of the maturation of the SEP, focusing on the negative neonatal component with a peak latency of about 30 msec and a duration of over 15 msec. By adulthood, this broad, prominent peak has evolved into a narrow, smaller negative component with a peak latency of 22 msec and a duration of less than 5 msec (Desmedt et al., 1980). To assess the significance of these changes in terms of the known increase in conduction velocity with maturation, it is necessary to consider the onset latency of this SEP component in relation to body growth. When the onset latency is divided by body length, the resulting negative exponential function shows a rapid decrease in conduction time relative to somatosensory pathway length, from 46 msec/m in the newborn to less than 20 msec/m by 1 year of age to a mature (adult) value of 11 msec/m by 8 years of age (Desmedt et al., 1980). These maturation changes in overall conduction times along the entire somatosensory pathway reflect maturational changes in both peripheral and central conduction velocities. Desmedt and associates (1980) have estimated the peripheral conduction velocity at birth to be about 27 m/sec; adult values around 65 m/sec are obtained by 1 or 2 years of age. In contrast, the central conduction velocity of the SEP is about 10 m/sec at birth and reaches an adult value of 50 m/sec by 8 years of age. Hence, the SEP shows a similar maturational progression to that of the BSAER: rapid maturation of the peripheral system and slower maturation of the central brain stem pathway. For both systems, about the 8th year appears to be a critical period for brain stem maturation.

#### VISUAL-EVOKED POTENTIALS

The *visual-evoked potentials* (VEPs) can be evoked in the EEG by simple flashes or by more complex visual stimuli. Because of the variability of the evoked response to flashes, most modern investigations of the VEP utilize the method of pattern reversal. The pattern reversal VEP is evoked by, for example, the alternating dark and light squares of a checkerboard pattern. Developmental studies carried out prior to 1978 utilized the flash-evoked VEP method, and reviews of these early studies suggested both latency increases (Klorman et al., 1978) and decreases (Moskowitz and Sokol, 1983) with increasing age during childhood. One current study of the flash-evoked VEP found significantly decreased latencies and increased amplitudes of the visual N1 wave (a negative deflection between 80 and 180 msec) across the age range 8 to 15 years (Carillo-de-la-Peña et al., 1999).

Using the pattern reversal VEP and measuring the consistent prominent positive component at 100 to 120 msec in adults, Moskowitz and Sokol (1983) found that the latency in response to a pattern of large checks decreased rapidly during the first year of life, reaching adult values at about 1 year of age. In response to a pattern of small checks, developmental changes were slower, so that by 5 years of age, adult values were still not obtained. Similar developmental differences in response to the two sizes of checks were also observed in earlier studies (Sokol and Jones, 1979), and Spekreijse (1978) found that the latency for VEP responses to small checks matured at about 10 years of age.

The source of the differential developmental response to large and small pattern elements is not well understood. Moskowitz and Sokol (1983) have drawn attention to reviews of the neurophysiologic and neuroanatomic evidence for the presence of two parallel pathways from the retina to the lateral geniculate nucleus to the visual cortex that process different aspects of visual input. One system consists of cells (Y cells) that respond with short latencies to *transient* events and functions, to detect movement and gross patterns; the second system is comprised of cells (X cells) that respond with long latencies to *sustained* input and functions, to discriminate fine patterns (Breitmeyer and Ganz, 1976; Lennie, 1980). Moskowitz and Sokol (1983) suggest that it is the short latency, transient system, which responds to large patterns, that matures rapidly, and the long latency, sustained system, which responds to small patterns, that matures more slowly. However, neuroanatomic studies of the human lateral geniculate nucleus (LGN) (Hickey, 1977) and neurophysiologic studies of the kitten LGN (Daniels et al., 1978) suggest that it is the sustained (X cell) system that matures earlier and the transient (Y cell) system that matures later. These results are not completely comparable to those of Moskowitz and Sokol (1983) because the time course for the increase in size of human LGN cells in both systems (adult size is attained by cells associated with the sustained system by 12 months of age and by cells associated with the transient system by 24 months) is much shorter than the time course for development of VEPs in response to large and small checks (mature responses by 1 year and by 5 to 10 years of age, respectively).

The issue of differential development of transient and sustained response systems is important because the dual functioning of these two systems seems to be a characteristic of a more general principle of the processing of sensory information in the nervous system (Graham et al., 1981). In the auditory modality, Graham and Murray (1977) have proposed that inhibitory and facilitatory startle modulation (see description in the previous section) are associated with two neuronal systems, described by Gersuni (1971), that transmit auditory information about transient and sustained events. Neurons in the auditory system capable of detecting the onset of a signal (transient event) with great precision (short-time constant neurons) and other neurons capable of temporal accumulation of information (sustained event) about the signal (long-time constant neurons) are found in both the cochlear nucleus (Radionova, 1971) and the inferior colliculus (Gersuni et al., 1971). In contrast to the early development during infancy of VEP responses to large checks, which Moskowitz and Sokol (1983) associate with the transient system, inhibitory startle modulation, which Graham and her colleagues (Graham and Murray, 1977; Graham et al., 1981) also associate with the transient system, develops late during childhood (Graham et al., 1981; Ornitz et al., 1986, 1991). For both the visual system (Breitmeyer and Ganz, 1976) and the auditory system (Gersuni, 1971), the transient system is responsive to novel patterns, stimulus onsets, or other aspects of environmental change, while the sustained system is involved with more elaborate processing and analysis of the information in the stimulus. For visual responses to nonstartling stimuli, there is agreement that the transient system (Y cells) also functions to inhibit (or regulate the tonic inhibition of) the activity of the sustained system (X cells) (Breitmeyer and Ganz, 1976; Lennie, 1980) in order to adjust



information processing to the requirements of response to new input. For responses to startling stimuli, it has been suggested that the transient system inhibits responses to new input in order to protect information processing (i.e., processing of sustained stimuli) from distraction by responses to new sensory input ( [Graham and Murray, 1977](#)). These somewhat contradictory hypotheses and the differing developmental sequences should generate fruitful research concerning the neurophysiology underlying the development of attentional mechanisms and cognitive processes in different sensory systems during childhood.

Studies of the pattern onset visual-evoked potential have identified a striate cortex component, which changes as a function of age. A single positive peak at about 130 msec in children under 8 years of age evolves into a negative-positive complex with peak latencies of about 100 and 150 msec between 9 and 16 years of age ( [Ossenblok et al., 1992](#)). This change is attributed by Ossenblok and colleagues, to the changes in synaptic connectivity in striate cortex that occur in comparable age periods ( [Huttenlocher et al., 1982](#)).

A general characteristic of the maturation of the auditory, somatosensory, and visual exogenous event-related potentials is the presence of at least two developmental sequences, a rapid maturational progression that is completed during the first or second year of life and a slower progression that is not completed until after 5 years of age and that may extend more slowly until 8 to 10 years of age or even until adulthood. [Eggermont \(1988\)](#) has provided a mathematical model that represents the developmental latency changes as the sum of a series of decaying exponential functions that apply equally to the three stimulus modalities. The time constants derived from these functions describe these two different rates of development of the exogenous ERPs as well as a third very rapid developmental process during the first few weeks of life.

### Endogenous Event-Related Potentials

Many endogenous ERPs have been described in experiments incorporating tasks designed to require meaningful responses to stimuli. These include negative and positive ERP components of different latencies and durations. The earlier (<300 msec) negative and positive components tend to reflect various aspects of orienting to stimuli, whereas later (>300 msec) components tend to reflect utilization of the information contained in stimuli ( [Hillyard and Picton, 1987](#)).

Several prominent negative endogenous ERP components have been identified. Wave N1 (peak latency around 100 msec) is sensitive to transient aspects of stimuli ( [Näätänen and Picton, 1987](#)) and reflects feature detection ( [Näätänen et al., 1988](#)), increasing in amplitude with increasing confidence in correct detection ( [Hillyard and Kutas, 1983](#)). Developmentally, in 175 normal subjects of both sexes, the latency (but not the amplitude) of wave N1 in response to auditory stimuli decreased linearly from 4 to 16 years of age ( [Fuchigami et al., 1993](#)). In a smaller study (40 subjects between 7 and 20 years of age) limited to girls, [Johnson \(1989\)](#) found neither amplitude nor latency changes in wave N1 with maturation in response to *auditory* stimuli, whereas demonstrating a linear latency decrement (but no amplitude changes) in response to *visual* stimuli, in wave N1 across the same age range. However, [Johnstone and associates \(1996\)](#) reported decreasing amplitude of N1 at frontal sites in response to attended tones across the age range 8 to 17 years. The latter result in an experiment using target stimuli that evoke an endogenous response is contrary to the increasing frontal N1 with maturation found in those experiments structured to evoke exogenous responses, as described earlier in this section.

Direct measurement of wave N1 has been augmented by the study of difference waves wherein the difference between the negative event related potentials in the latency range of N1 (and extending into that of N2, see the following) in two conditions (e.g., while attending or ignoring a stimulus) is measured. This provides neurophysiological correlates of stimulus discrimination and of selective attention. Two types of difference waves have been defined and used to study the development of these two functions throughout childhood and adolescence ( [Oades et al., 1997](#)). Component Nd, the difference negativity, also referred to as the processing negativity, overlaps wave N1 when an attended (e.g., targeted) auditory stimulus is differentiated from an ignored stimulus ( [Näätänen and Picton, 1987](#)). This increased and more prolonged negativity reflects an early stage of stimulus processing that preferentially extracts information from the attended auditory stimulus ( [Hillyard and Hansen, 1986](#); [Hillyard and Kutas, 1983](#); [Hillyard and Picton, 1987](#)). Hence, the processing negativity reflects selective attention. In response to visual stimuli, an analogous ERP component NA has been associated with stimulus recognition and compared to the auditory processing negativity ( [Ritter et al., 1983, 1988](#)). The mismatch negativity (peak latency about 150 to 200 msec) reflects a response to relationships between two stimuli ( [Näätänen and Picton, 1987](#)), evaluating novel or deviant stimuli against the background of standard or expected stimuli ( [Näätänen et al., 1989](#)). Hence, the mismatch negativity reflects automatic detection of stimulus change.

In preadolescent children, compared with adolescents and young adults, the processing negativity in response to targeted longer-duration syllables interspersed among "irrelevant" shorter-duration syllables is significantly smaller, suggesting a neurophysiologic correlate of the child's reduced efficiency in directing attention among competing inputs ( [Berman et al., 1990](#); [Friedman, 1991](#)). Between 7 and 24 years of age, the amplitude of an occipitally derived difference wave for attended vs ignored visual stimuli with a latency between 150 to 300 msec becomes increasingly negative with increasing age, indexing increasing capacity for visual selective attention with greater maturity ( [Van der Stelt et al., 1998](#)). A scalp topography study of the development of auditory event related potentials was consistent with these findings: Processing negativity did not mature until 17 years of age ( [Oades et al., 1997](#)).

In contrast to the late maturing processing negativity, the mismatch negativity (MMN) develops earlier, by 14 years of age in children and adolescents studied between 8 and 22 years old ( [Oades et al., 1997](#)). In other studies, the mismatch negativity evoked by barely perceptible differences in speech sounds had already reached adult levels in school age children ( [Ceponienė et al., 1998](#); [Kraus et al., 1993, 1999](#)) and mismatch negativity has actually been demonstrated in premature infants born 30 to 35 weeks after conception ( [Cheour-Luhtanen et al., 1996](#)). With consideration for state-of-consciousness (i.e., the sleep cycle) and some scalp topographical differences, the MMN in the newborn ( [Alho et al., 1990](#); [Leppänen et al., 1997](#)), at 3 months of age ( [Cheour et al., 1997](#)) and at 8 months of age ( [Pang et al., 1998](#)) is remarkably similar to that of older children and adults. [Cheour and colleagues \(2000\)](#), in a comprehensive review, find "that a prominent, developmentally stable MMN can be obtained from children and even infants," that, as in adults, the MMN is not affected by attention and that it is a response to a change in the stimulus. The developmental differences are limited to a broader and more central scalp distribution and longer latency in infants than adults, and to the fact that the MMN can be obtained in sleep in infants but not in adults ( [Cheour et al., 2000](#)).

The effect of distractibility in children suggests the potential functional utility of the mismatch negativity. In distractible children, compared to nondistractible children, the late component of the mismatch negativity was significantly reduced and current density maps of that component revealed weaker frontal lobe activation ( [Kilpeläinen et al., 1999d](#)). Because the mismatch negativity reflects frontal lobe mediation of auditory sensory memory functioning ( [Näätänen and Winkler, 1999](#)), it is suggested that distractibility in children may reflect dysfunction in frontally mediated aspects of auditory sensory memory ( [Kilpeläinen et al., 1999d](#)). Also, the significantly attenuated MMN in response to frequency differences in young dysphasic children suggests a defect in the central automatic processing of auditory frequencies ( [Holopainen et al., 1997](#)). Although these findings suggest potential clinical utility of the MMN in infants and children with auditory processing impairment ( [Kraus et al., 1999](#)), the relatively low stability of the MMN in individual children and the greater intersubject variability (relative to adults) make diagnostic application in children unlikely at the present time ( [Cheour et al., 2000](#); [Kurtzberg et al., 1995](#); [Uwer and von Suchodoletz, 2000](#)).

Wave N2 (peak latency about 200 msec) also reflects registration of stimulus deviance from a steady-state background ( [Hillyard and Kutas, 1983](#)). It seems to reflect a stage of discriminative stimulus processing, associated with stimulus classification ( [Ritter et al., 1983](#)), that precedes the further processing associated with later ERP components ( [Hillyard and Picton, 1987](#)). A late slow wave with negative polarity that develops around 1,200 msec and persists beyond 2,700 msec has been associated with increasing conceptual difficulty ( [Ruchkin et al., 1988](#)).

Wave N2 has been studied developmentally. It was first described in 5- to 11-year-old children by [Symmes and Eisengart \(1971\)](#) as a large negative wave in response to attention-getting stimuli and later referred to as Nc by [Courchesne \(1977, 1978\)](#), who described it both in response to surprising or novel stimuli and also to target stimuli that must be discriminated from background stimuli ( [Courchesne et al., 1987](#)). This negative ERP component is large and long in latency, between 4 and 7 months of age, after which time there is a progressive reduction in amplitude and latency throughout childhood and adolescence ( [Courchesne, 1978](#); [Courchesne et al., 1987](#); [Enoki et al., 1993](#); [Friedman et al., 1984](#); [Fuchigami et al., 1993](#); [Johnstone et al., 1996](#); [Van der Stelt et al., 1998](#)). [Fuchigami and associates \(1993\)](#) did not find developmental amplitude changes in N2, which, however, was measured peak-to-peak from the preceding wave P2. By late adolescence, this wave has assumed the adult form of wave N2 ( [Friedman et al., 1984](#)). Its latency is about 700 msec in infancy, 400 msec during childhood ( [Courchesne et al., 1987](#)), and 200 to 250 msec in adolescence ( [Enoki et al., 1993](#); [Friedman et al., 1984](#)). Its changing stimulus parameters seem to parallel the changing perceptual styles and interests that occur with maturation. In this regard, it is noteworthy that children under 13 years of age show a similar wave N2 to that of adolescents and adults in response to the requirement to identify a target stimulus, but show a large delayed negative wave in response to surprising novel stimuli. (See Fig. 7 in [Courchesne, 1978](#), where the large N2 in preadolescent children in response to novel stimuli was labeled Nc.) Although described as a special ERP component found in childhood and given a special label ( [Courchesne, 1977](#); [Courchesne et al., 1987](#)), there seems to be little reason not to consider this component as a developmental precursor of wave N2, with a changing configuration throughout its developmental course ( [Friedman et al., 1984](#)). In both children ( [Courchesne, 1978](#)) and adults ( [Knight, 1984](#)), it has a predominantly fronto-central scalp topography and increases in amplitude when elicited in response to novel as compared to target stimuli. Hence, wave N2, which reflects stimulus classification, undergoes striking developmental changes in both latency and amplitude. Its developmental precursor, Nc, seems to reflect the earliest ERP evidence of cognitive processing since as early as 4 to 7 months of age it shows increased amplitude and longer



latency in response to infrequent compared to frequent events ([Courchesne et al., 1981](#); [Karrer and Ackles, 1987](#)). It is during this period, the first 6 months of postnatal life, that major changes in frontal cortical laminar structure and cortico-cortical connectivity occur ([Kostovic, 1990](#)).

Positive endogenous ERP components have also been described. These include the P2, the P3a, the slow wave, fronto-central positivities, and the P300 ([Donchin and Coles, 1988](#); [Fabiani et al., 1987](#); [Ruchkin et al., 1988](#)). The *endogenous P2 increases* in amplitude with increasing age from 8 to 17 years in response to both standard and target stimuli presented in the oddball paradigm ([Johnstone et al., 1996](#)). The oddball paradigm elicits endogenous ERP components, as discussed in respect to P300 elicitation (see the following). This maturational effect is in contrast to the modest *decrease* in amplitude with increasing age that occurs when the experimental conditions elicit an exogenous P2 (see the preceding). The P300, also labeled P3 and P3b, and the related P3a have received the greatest experimental attention and theoretical analysis ([Donchin and Coles, 1988](#); [Donchin et al., 1986a,b](#); [Fabiani et al., 1987](#); [Johnson, 1988](#)) and have received considerable developmental investigation in children and adolescents ([Courchesne, 1978, 1983](#); [Courchesne et al., 1987](#); [Finley et al., 1985](#); [Friedman et al., 1984](#); [Goodin et al., 1978](#); [Martin et al., 1988a,b](#)).

The P300 is a positive peak in the ERP, occurring from somewhat less than 300 msec to nearly 1000 msec after a stimulus ([Hillyard and Kutas, 1983](#)) with maximum amplitude over the parietal scalp. Human neurosurgical/neurophysiologic studies have suggested multiple generators of the P300, particularly the medial temporal lobe ([Halgren, 1988](#)), the temporal-parietal junction ([Knight et al., 1989](#)), the frontal lobe ([Wood and McCarthy, 1985](#)), and the thalamus ([Velasco et al., 1986](#); [Yingling and Hosobuchi, 1984](#)). Lesion studies in the monkey suggest that noradrenergic projections to these structures from the locus ceruleus are important in the generation and modulation of the scalp-recorded P300 ([Pineda et al., 1989](#)).

The amplitude of P300 increases with increasing relevance of a task associated with a stimulus and with decreasing probability that the stimulus will occur ([Donchin et al., 1986a,b](#)). Hence, P300s are usually elicited in experiments in which the subject is asked to identify certain stimuli (e.g., higher-pitched tones) that occur infrequently during a series of frequently occurring and ignored stimuli (e.g., lower-pitched tones). This is usually referred to as the oddball paradigm and the P300 evoked in this paradigm is often referred to as the P3b. The P3b is larger in response to the less frequent target stimulus and shows a parietal predominance. In contrast, the P3a (also called the novelty P3) designates a P300 response to novel or deviant but nontarget stimuli. The P3a shows a frontal predominance in contrast to the parietally predominant P3b, and its latency is considerably shorter than that of the P3b. More generally, the peak amplitude of the P300 appears to be responsive to the distinctiveness of stimuli, reflecting the mental evaluation of surprising events, and hence to reflect mental activity associated with revisions of the subject's current model of the environment ([Donchin and Coles, 1988](#); [Donchin et al., 1986b](#)). The peak latency of P300 is thought to reflect the time required to categorize stimuli ([Hillyard and Kutas, 1983](#)) and thus to update the subject's model of the environment ([Donchin and Coles, 1988](#); [Donchin et al., 1986b](#)). More difficult discriminations result in longer latencies ([Donchin et al., 1986b](#)), suggesting that the level of mental abstraction utilized in performing a task may be reflected in the peak latency of P300 ([Donchin et al., 1986a](#)). [Johnson \(1988\)](#) has discussed the control of P300 amplitude and latency in the context of information theory: The degree to which P300 amplitude and latency are affected by stimulus probability and task relevance (which is affected by task and stimulus complexity as well as stimulus value) is modified by the degree to which the subject is uncertain about having correctly identified the stimulus. Greater uncertainty means that a smaller proportion of information is transmitted; this is reflected in decreased amplitude and increased latency.

Hence, in the human adult, the P300 wave reflects a considerable degree of information processing, becoming larger and occurring earlier as a greater proportion of information is transmitted, that is, as prior uncertainty is resolved by a stimulus or, in information theory terms ([Shannon and Weaver, 1963](#)), as equivocation is reduced ([Hillyard and Kutas, 1983](#); [Johnson, 1988](#)).

Developmentally, the P300, which is not present in infancy ([Courchesne et al., 1981](#)), has been demonstrated in response to infrequently presented target stimuli in 3-year-old children ([Courchesne, 1983](#); [Courchesne et al., 1987](#)). Its small amplitude increases to maximum values between 3.7 and 6 years of age, after which amplitude may decrease to adult values between 10 and 13 years of age ([Courchesne, 1978, 1983](#); [Courchesne et al., 1987](#); [Wijker et al., 1989](#)), show no further change until adulthood ([Fuchigami et al., 1993](#); [Martin et al., 1988b](#); [Mullis et al., 1985](#)), or actually increase ([Polich et al., 1990](#)). Although differential effects of target stimulus probabilities, stimulus modality, or the nature of the required response to the target stimulus have been proposed to explain these differences, perusal of the preceding literature revealed no consistent relationship between these experimental variables and the discrepant developmental results. In one study ([Johnson, 1989](#)), both visual and auditory stimuli and two types of responses to target stimuli were used. Stimulus modality had no effect on P300 amplitude between 7 and 20 years of age, but P300 amplitude decreased with age when the subjects were asked to press a button to the target stimuli and showed no developmental effect when subjects were asked to count the targets. In response to orthographic and phonologic targets, a developmentally maximum amplitude occurred around 11 years of age ([Taylor, 1993](#)), followed by a decrease in P300 amplitude until young adulthood.

Regardless of the developmental course of P300 amplitude changes following the preschool period, the earliest manifestation of increased P300 amplitude in response to target stimuli relative to background stimuli, that is, information processing, occurs some time between 3 and 5 years of age ([Courchesne et al., 1987](#)). This developmental neurophysiologic landmark coincides with the developmental peak loss of inhibitory startle modulation and the associated peak increment of facilitatory startle modulation ([Ornitz et al., 1986, 1990, 1991](#)), the developmental peak dishabituation of the startle blink ([Zametkin et al., 1979](#)), and an early maturational period of EEG changes characterized by developmental peaks of theta ([Matousek and Petersen, 1973](#)) and alpha ([Epstein, 1979, 1980, 1986](#)) activity and coherence and phase changes, suggesting increased left fronto-occipital and frontotemporal coupling and right frontal pole pairing ([Thatcher et al., 1987](#)). Hence, the P300, reflecting responsiveness to the distinctiveness of stimuli and the transmission of information, becomes functional during a major developmental period of functional cortical reorganization (reflected in EEG changes) and reduced brain stem control over response to sensory input (reduced inhibitory and increased facilitatory startle modulation). This constellation of endogenous ERP, EEG, and startle modulation changes, peaking around 4 to 5 years of age, coincides with the piagetian mental stage of preoperational behavior and may reflect or be part of a neurophysiologic developmental stage that underlies the heightened awareness and capacity to absorb new information from the environment and the relative inability to modulate reactions to the environment that is so characteristic of the preschool child. This preschool stage of neurophysiologic development follows the end of the first major period of structural brain maturation, which occurs between 15 months and 4 years and includes accelerated increases or changes in brain weight ([Epstein, 1979](#)), cortical thickness ([Rabinowicz et al., 1977](#)), pyramidal cell size and shape ([Rabinowicz, 1977](#)), neuronal density ([Rabinowicz, 1986](#)), and completion of myelination of the sensory-specific thalamic radiations ([Lecours, 1982](#); [Yakovlev and Lecours, 1967](#)). It precedes the second period of brain growth and restructuring, which begins around 6 years of age ([Epstein, 1986](#); [Rabinowicz, 1986](#)).

The latency of P300 follows a different developmental pattern than that of amplitude. It decreases exponentially with maturation in response to both target and background stimuli, from over 800 msec in 3-year-olds to 300 to 400 msec in adolescents and young adults ([Courchesne, 1978](#); [Courchesne et al., 1987](#); [Finley et al., 1985](#); [Fuchigami et al., 1993](#); [Goodin et al., 1978](#); [Howard and Polich, 1985](#); [Johnson, 1989](#); [Johnstone et al., 1996](#); [Martin et al., 1988a,b](#); [Oades et al., 1997](#); [Taylor, 1993](#); [Van der Stelt et al., 1998](#)). Unlike P300 amplitude, the maturational changes in latency are continuous from the earliest demonstration of the P300 response and seem to be independent of the response to the target ([Johnson, 1989](#)). According to the interpretation of P300 latency as reflecting the time required to categorize stimuli ([Hillyard and Kutas, 1983](#)), the decrease in P300 latency with maturation may index the development of this aspect of information processing. The decrease in P300 latency between 5 and 14 years of age also correlates strongly with increasing scores on tests measuring memory span ([Howard and Polich, 1985](#); [Polich et al., 1990](#)), and in 4- to 7-year-olds, P300 latencies are significantly shorter in children with IQs greater than 140 ([Martin et al., 1993](#)).

It should be noted, however, that factors other than maturation and more efficient information processing can affect P300 latency in children. For example, when the intervals between target stimuli in the oddball task are prolonged, more distractible children actually have shorter latency P300s than less distractible children, possibly because the stimulus processing in the more distractible children shifts from an information processing mode to an orienting mode (orienting to novel events is known to generate shorter latency responses ([Kilpeläinen et al., 1999a](#))). The shorter latency P300 generated in response to the less frequent target stimuli by the more distractible children also showed a frontal rather than the usual parietal predominance, providing further evidence that distractible children processed the target stimuli as if they were novel events, that is, they oriented to rather than cognitively processed the target stimuli ([Kilpeläinen et al., 1999b](#)). When normal 9-year-old children were compared with adults, the children's P300 responses were more affected than the adults' by the intervals between target stimuli. When the intervals between target stimuli were prolonged, there was marked shortening of P300 latency for the children and little effect for the adults, suggesting that children are more likely than adults to process infrequently occurring target stimuli as if they were novel stimuli ([Kilpeläinen et al., 1999c](#)). The greater vulnerability of distractible children than nondistractible children and of children than adults to react to infrequent target stimuli as if they were novel stimuli and hence respond with a P3a, or novelty P3, instead of a P3b suggests that the neurophysiologic representation of the target stimulus during a long intertarget interval decays more rapidly in children. Hence, each successive target is reacted to as if it were novel. The capacity to generate a neurophysiological response to novel stimuli, the P3a or novelty P300, is intact throughout childhood from at least 5 years of age ([Cycowicz et al., 1996](#)). A short latency P3a-like response to very strong sudden stimuli that evoke startle responses occurs in children as well as adults and is modulated by prepulse modulation ([Sugawara et al., 1994](#)) and habituation ([Hirano et al., 1996](#)) as is the startle response itself.

Recent attempts to apply topographical analyses (variations in ERP waveforms across scalp locations) to developmental changes in ERPs during complex cognitive tasks (picture matching and continuous recognition memory) have led to the conclusion that by 7 years of age late ERPs, P300 and a negative deflection at 400 msec (probably a variant of wave N2), are similar in children and young adults, except for the longer latencies in children, reflecting the reduced efficiency in speed of



stimulus evaluation and information processing ( [Friedman, 1991](#); [Friedman et al., 1988](#)). A recent innovation in developmental ERP studies has utilized topographic analysis of long-latency ERP changes in response to tasks which measure the cognitive transitions from the piagetian preoperational stage (2 to 7 years) to the stage of concrete operations (7 to 11 years) and from the latter to the stage of formal operations (11 to 15 years). In this complex study, [Stauder \(1992\)](#) recorded ERPs to pictorial analogs of the piagetian liquid conservation task in 5- to 7-year-olds and to pictorial analogs of the Raven Standard Progressive Matrices in 9- to 11-year-olds. In the younger children, the transition from nonconservation (preoperational level) to conservation (stage of concrete operations) was associated with a reduction in a broad late (up to 1,000 msec) ERP positivity over frontal electrode sites. In the older children, the transition to improved performance on the Standard Progressive Matrices (at the onset of the stage of formal operations) was associated with the reduction of a broad late (700 to 2,000 msec) negativity in the ERP in the more anterior scalp locations. In a related study, 5- to 7-year-old girls were classified as nonconservers or conservers. The ERPs evoked by the choice stimulus in a piagetian liquid conservation task were more positive (in the P300 latency range) frontally in the nonconservers and more positive parietally in the conservers ([Stauder et al., 1993](#)). This demonstration of ERP changes in response to actual performance on tasks indexing developmental transitions is currently the most direct approach to the neurophysiology of cognitive development. These findings of neurophysiologic transitions between developmental stages are congruent with the delineation of the three neurophysiologic stages described in this chapter and with their relationship to the three piagetian stages of preoperational behavior, concrete operations, and formal operations. The ERP topographic findings also point to the predominance of frontal cortical structural and functional change during childhood, a progressive development that is accompanied by the changing response to tests of frontal lobe functioning throughout childhood ( [Levin et al., 1991](#); [Welsh and Pennington, 1988](#)).

Further discussion of the complex effects of scalp topographical changes on the sequences of event related potential development is beyond the scope of this review. For the interested reader, [Oades et al. \(1997\)](#) and [Van der Stelt et al. \(1998\)](#) provide data and detailed reviews of this important topic.

## SUMMARY AND OVERVIEW OF DEVELOPMENTAL NEUROPHYSIOLOGY

Neurophysiologic development during childhood must be considered against the background of what is known about the maturation of the structure of the human brain. Both brain maturation and neurophysiologic development are characterized by progressive changes throughout long periods of childhood and by accelerations, usually followed or preceded by decelerations, in the rate of change, resulting in peaks and troughs of both structural and functional change. Although there is a tendency for the neurophysiologic spurts in development to coincide with structural spurts in maturation, there may be lags, such that important periods of functional change occur during slowdowns in structural growth or between periods of structural reorganization.

Following very rapid developmental and structural changes occurring from the end of gestation through the early weeks of postnatal life (not considered in this chapter), there are two major progressive structural sequences.

The first and more rapid takes place between birth and 1 to 3 years and includes general reductions in neuronal densities, increases in synaptic densities, and completion of myelination of the sensory-specific thalamic radiations. Many important early progressive neurophysiologic changes are associated with these structural developments. These include the marked reduction in REM sleep time and the rapid early reductions in BSAER transmission time, peripheral conduction velocity, and latency of VEPs in response to large pattern changes, and the early development of the mismatch negativity.

The second progressive sequence of neuroanatomic change is slower and includes a gradual increase in brain weight and cerebral cortical thickness from infancy to adulthood, a decrease in synaptic density between 1 and 11 years of age, and continuing myelination of the reticular formation, nonspecific thalamic radiations, and cerebral commissures until 8 to 10 years, and of the intracortical association areas until the second and third decades of life. Associated neurophysiologic events include progressive increases in EEG alpha activity between infancy and adulthood and decreases in slower EEG frequencies between 5 years and adulthood; increased organization of the phasic motor activity and phasic sensory inhibition during REM sleep between infancy and 9 years of age; progressive changes in vestibular function (increased gain, prolongation of time constants, and changes in adaptation) between infancy and 10 years of age; further reduction in BSAER transmission time, SEP central conduction velocity, and VEP latency in response to fine pattern changes between early childhood and 8 to 10 years of age; and reduction of latency of the P1, N1, and P300 components of the event-related potential between infancy and adolescence.

The progressive changes in brain growth are punctuated by four maturational spurts. The first takes place between about 15 months and 4 years of age. It includes an acceleration in cerebral cortical thickening and changes in pyramidal cell size and shape. Associated neurophysiologic changes include a spurt in EEG alpha activity and a developmental peak of theta activity at the end of this period.

The second spurt in brain maturation takes place between 6 and 10 years of age. Brain changes include a second acceleration in the rate of cerebral cortical thickening, a small increment in cortical and subcortical neuronal density, accelerated synaptic elimination, and further changes in pyramidal cell size and shape. Myelination of reticular tissue is also completed during this period. Associated neurophysiologic activity includes a spurt in EEG alpha activity, a peak in EEG coherence and phase, suggesting accelerated development of right hemispheric frontal-temporal connections, and an abrupt increase followed by a steplike decrease in amplitude of the event-related potentials P1 and N2. During this second major spurt in brain growth and change, the developmental progressions of several important neurophysiologic functions are completed. The organization of rapid eye movements into bursts and phasic sensory inhibition during REM sleep, the gain and time constant of vestibular nystagmus, the modulation of startle by inhibitory and facilitatory prestimulation, the central transmission time of the BSAER, the central conduction velocity of the SEP, and the latency of the VEP in response to fine patterns all reach adult values. Hence, by the end of this second spurt in structural and functional brain development, at around 10 years of age, many significant subcortical functions have matured, and accelerated development of some measures of cortical activity have taken place. This period coincides with the piagetian period of concrete operations, that is, the beginnings of logical thinking (Chapter 10). Many of the mature subcortical mechanisms are of an inhibitory nature, suggesting a relationship between the capacity to inhibit and the readiness for higher cognitive processes. During this period, there is an accelerated development of the ability to inhibit the retroactive interference of a second set of stimuli on the recall of a prior set. This is a frontal lobe function. It is suggested that the developing capacity to inhibit, which is requisite for frontal lobe mediated cognition, may be linked to the development of subcortical inhibitory mechanisms.

Prior to the second maturational spurt in brain growth and change, and following the first such spurt (i.e., between 4 and 6 years), there is an important period of neurophysiologic development characterized by a particular constellation of EEG, ERP, and startle physiology changes. Initiated around 4 years of age by developmental peaks in EEG alpha and theta activity and changes in EEG coherence and phase, suggesting advances in frontal cortex connectivity, this period is notable for a peak increment in event-related potential activity ranging from the exogenous wave 5 of the BSAER to the endogenous P300 and a concurrent peak deficit in inhibitory startle modulation. This stage of neurophysiologic development involving increased reactivity to stimuli at both cortical and subcortical levels, occurring between an earlier period of cortical maturation and a later period of cortical "remodeling" (per Rabinowicz), coincides with the piagetian stage of preoperational behavior. The neurophysiologic characteristics suggest the heightened awareness coupled with the inability to inhibit that characterize the preschool child.

Brain weight changes and histologic evidence for cortical remodeling suggest two additional spurts in brain maturation and change around puberty and during midadolescence. These late brain growth periods are also accompanied by neurophysiologic changes manifested by peaks of EEG alpha activity and changes in coherence and phase measures involving frontal cortex connectivity. During adolescence, the N1 component of the ERP develops a consistent negativity, and the progressive changes in the ERP components N2 and P300 terminate in adult values.

An overview of the relationships between neurophysiologic development and brain growth suggests a general but not absolute concordance. There is evidence for considerable plasticity in neurophysiologic development, such that important developmental neurophysiologic changes occur long after structural change seems to be complete (as exemplified by the course of vestibular development) or during lulls in structural change. A major component of plasticity in neurophysiologic development may also be attributed to neuronal plasticity in the case of cerebral cortex. Here there is overproduction and redundancy of synaptic connections early in childhood, with synapse elimination with maturity. [Huttenlocher \(1990\)](#) has suggested that neural plasticity can be understood as the incorporation of some of the excessive synaptic contacts into functioning systems. These synaptic contacts become stabilized, while "labile contacts that fail to be incorporated" are eliminated. Developmental neurohistological studies in primates indicate a similar early synaptic excess followed by elimination of excessive synapses with maturity ([Goldman-Rakic, 1987](#)). Similar cognitive functions of delayed response in the monkey and object permanence in the infant emerge during the period of synaptic excess; [Goldman-Rakic \(1987\)](#) has suggested that whereas a critical mass of synapses is associated with the emergence of this type of cognition, "mature capacity may depend upon the [later] elimination of excess synapses." It is significant that important neurophysiologic development changes, notably those that are related to the ability to inhibit, either emerge or attain adult values during the school-age years (i.e., between 6 and 12 years of age). This is the period when, regardless of cultural differences, children are engaged in some type of formal learning. This is also the time when, following a period of cortical remodeling ( [Rabinowicz, 1986](#)), there is an extended period of synaptic elimination ( [Huttenlocher and Dabholkar, 1997](#)).

An overview of the relationships between neurophysiologic development and behavior suggests three significant stages that, not surprisingly, coincide with the

preschool- and school-age periods of childhood and early adolescence. Between 3 and 5 years, there is a neurophysiologic stage of increased receptivity coupled with disinhibition in response to environmental stimuli. Between 6 and 10 years, there is a neurophysiologic stage of increased inhibition in response to stimuli, coupled with increasing development of neurophysiologic indices of information processing. In early adolescence, there is a third stage of development during which there is neurophysiologic evidence for further development of intracortical, particularly frontal, connectivity and final maturation of event-related potential indices of information processing. These three neurophysiologic stages coincide with the piagetian stages of preoperational behavior, concrete operations, and formal operations. Hence, the developing neurophysiologic capacity to register, process, and extract information from stimuli, coupled with the increasing ability to inhibit responses to stimuli, appears to underlie the changing style of learning that progresses from the egocentric (preschool child) to the more objective but concrete (school child) and finally to abstract thinking (adolescence).

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## 6 DEVELOPMENTAL ASPECTS OF PSYCHONEUROIMMUNOLOGY

Jacqueline A. Bartlett, M.D. and Michael Irwin, M.D.

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Psychoneuroimmunology is the study of interactions among behavior, central- and peripheral nervous systems, neuroendocrine hormones, and the immune system. Although research in this field is relatively new, emergent observations suggest that these interactions constitute one mechanism by which factors such as stress or depression influence health. Substantial data are now available to show that both acute and chronic psychological factors can influence the numbers and functional capacities of multiple immune cells, including lymphocyte and granulocyte function. Less is known about the effects of stress on the immune system of a developing organism, although perturbations occurring in early life, or even before birth, may alter the developmental course of the immune system and result in altered immune responses throughout life.

This chapter reviews experimental evidence concerning the neural and endocrine pathways by which the central nervous system (CNS) may regulate immune function as well as the short- and long-term immunologic effects of fetal or childhood experiences. Preclinical and clinical investigations are presented that document the influence of both stressors and psychosocial processes on immunity. Before beginning the discussion of interactions among the brain, behavior, and immunity, we provide a brief overview of the immune system.

### OVERVIEW OF THE IMMUNE SYSTEM

The immune system functions to discriminate “self” from “non-self” cells, protecting the organism from invasion by pathogens such as viruses and bacteria or from abnormal internal cells such as cancer cells (Cohn, 1985; Hood et al., 1985). The immune response consists of three stages or phases. During the induction phase, foreign or other non-self antigens are detected. The activation phase then involves mobilization and/or proliferation of specific immunocytes. Finally, during the effector phase, these foreign or non-self antigen sources are destroyed, neutralized, and removed (Abbas et al., 1994). These immune functions are closely regulated and usually performed without damage to the host, although an overresponsive immune system is purported to lead to autoimmune disease in which the organism's own tissues are attacked (Cohen, 1988; Morimoto et al., 1987; Paul, 1984; Strakosch et al., 1982; Talal, 1980).

The organs of the mammalian immune system include the thymus, spleen, bone marrow, and lymph nodes (Hood et al., 1985; Paul, 1984). The working cells of the immune system are represented by distinct populations: lymphoid type cells, including lymphocytes comprised of T and B cells and natural killer (NK) or large granular lymphocytes; and myeloid derived cells including granulocytes and monocytes (Hood et al., 1985; Paul, 1984; Ritz, 1989).

Surface markers that are thought to reflect cell functions identify cells of the immune system. These markers are referred to as cluster designation numbers or CD, which are identified by monoclonal antibodies to these cell surface antigens for identification purposes. Immature or stem cells have the CD marker number 34; therefore, they are called CD34-positive cells. Similarly, the CD56 marker often identifies NK cells. Two or more CD markers identify subtypes of particular cell types; for example, T helper/inducer cells are CD3- and CD4-positive, whereas T-suppressor or cytotoxic cells are CD3- and CD8-positive. Conceptualizations of immunocytes have changed over time. Cells of the immune system were first named by their putative function and more recently by CD designations.

All cells of the immune system are derived from the pluripotent stem cell in the bone marrow, which becomes either a myeloid or lymphoid cell precursor. These cells further differentiate within the thymus, bone marrow, or at other sites in the body.

T lymphocytes develop from stem cells in the bone marrow and migrate to the thymus, where they mature into several subsets, including cytotoxic T cells, T-helper cells, and T-suppressor cells (Hood et al., 1985). These T cells circulate into the periphery and are found in the lymph nodes, blood vessels, and spleen. Briefly, the cytotoxic T cell is characterized by its ability to seek out and destroy either cells infected with viruses or tumor cells that have acquired foreign non-self antigens (Henney and Gillis, 1984; Zinkernagel and Doherty, 1979). In the development of the cytotoxic T-cell response, a foreign antigen is first encountered and incorporated onto the surface of an antigen-presenting cell such as a macrophage. After the antigen is presented to the T cell, recognized, and bound by a specific receptor on the T cell, the T cell multiplies and becomes capable of attacking any cell that presents that specific foreign surface-antigen (Henney and Gillis, 1984). Other types of T lymphocytes, such as T helper cells, secrete interleukin-2 (IL-2) and regulate the proliferative response of the T cell to antigenic stimulation (Henney and Gillis, 1984). Reexposure of the cytotoxic T cell to an antigen produces a more rapid and extensive reaction than that found on initial presentation.

The B cell is primarily involved in the humoral response. Like the T cell, the B cell arises from a precursor stem cell in the bone marrow, although in humans its site of maturation remains unknown (Hood et al., 1985), but likely includes the bone marrow and lymph nodes. Following initial antigen processing by the macrophage, the antigen-MHC complex on the accessory cell surface engages the receptors of an appropriate T-helper cell antigen and the macrophage releases interleukin-1 (IL-1). Thus stimulated, the T helper cell proliferates forming a clone, and secretes factors such as IL-2, which stimulate T- and B-cell growth. The activated B cell, in turn, proliferates and differentiates into plasma cells that synthesize and secrete antigen-specific antibodies, of which the five major classes of immunoglobulins (Igs) are

IgM, IgG, IgA, IgE, and IgD (Goodman, 1991). IgM is produced soon after antigenic stimulation. Cytokines liberated by activated T helper cells, such as IL-4 and IL-5, subsequently promote the switch from IgM production by plasma cells to the production of other isotypes such as IgG (Nossal, 1987; Oppenheim et al., 1991). IgA is found primarily in the secretions of the body (i.e., nasal mucus, saliva, etc.). IgE in combination with a specific antigen binds to mast cells and mediates the immediate hypersensitivity response. IgE may also be important in the development of allergic reactions (Oppenheim et al., 1991). The function of IgD is not known, although its prominence on B cell surfaces during certain stages of development suggests that it may be involved in cell differentiation (Goodman, 1991).

A distinct subpopulation of lymphocytes comprised of NK cells has been described in addition to T and B cells. The NK cell is immunologically nonspecific and does not require sensitization to specific antigens to perform its cytotoxic activity (Herberman, 1980; Lotzova and Herberman, 1986; Trinchieri, 1989). Thus, the NK cell responds to a variety of cell surface markers, as long as the markers differ from "self" markers, and lyses a wide variety of cell types. Although the role of the NK cell in tumor surveillance remains controversial (Lotzova and Herberman, 1986; Ritz, 1989), substantial evidence has demonstrated the importance of the NK cell in the control of herpes and cytomegalovirus (CMV) infections in humans (Biron et al., 1989; Padgett et al., 1968; Ritz, 1989; Sullivan et al., 1980) and animals (Bancroft et al., 1981; Bukowski et al., 1985; Habu et al., 1984).

Granulocytes and macrophages are derived from myeloid rather than lymphoid cell precursors. These cells mature in the bone marrow and are sent out into the peripheral circulation from whence they are sent to inflammatory sites throughout the body as needed. These cells engulf or phagocytize bacteria or other materials (e.g., foreign proteins or apoptotic cells) and destroy the bacteria while it is within the cell body likely through the production of superoxides or nitric oxide. Granulocytes then remove the deactivated or dead cells. Myeloid derived cells (monocytes, antigen presenting cells) also are important in lymphoid cell function, as described.

The immune responses of lymphoid cells are typically divided into two important components: cellular immunity and humoral responses (Gilliland, 1983; Nossal, 1987; Paul, 1984). There is evidence that T and B cells interact and cooperate in many cellular immune responses and most humoral immune responses, although cellular immunity is mediated primarily by T lymphocytes and humoral responses by B lymphocytes and their soluble products (Hood et al., 1985).

Regulation of immune responses involves the secretion of cellular factors known as cytokines or lymphokines (Oppenheim et al., 1991). These cytokines together form a network of regulatory signals that show considerable overlap in activity and patterns of synergism, as well as antagonism. For example, the lymphokine 1 IL-1 is produced by nearly all immunologic cell types including NK cells, T and B lymphocytes, brain astrocytes, microglia, and macrophages (Dinarello and Mier, 1987). IL-1 acts mainly as an endogenous adjuvant serving as a cofactor during lymphocyte activation, inducing the synthesis of other lymphokines and activation of resting T cells (Dinarello and Mier, 1987). For example, IL-1 stimulates T lymphocytes to synthesize and release IL-2, and IL-1 further acts on NK cells to induce the expression of the IL-2 receptor. Binding of IL-2 by its receptor on the NK cell is a crucial step in the activation of such cytotoxic cells (predominantly large granular lymphocytes) to form lymphokine activated killers that are able to lyse a wide range of target cells in a non-major-histocompatibility-complex restricted manner (Henney and Gillis, 1984).

## MEASURES OF IMMUNE FUNCTION

The immune system can be evaluated by measures that assess the *number* of different cell types as well as the *function* of various components of cellular and humoral immunity. To quantitate the number of cells in various subpopulations, specific monoclonal antibodies are available that bind to unique surface markers (CD) on cell types such as T helper, T suppressor, and NK cells (Bernard and Boumsell, 1984). Although enumeration of cell types reveals the balance of different cell types needed for the optimal immune response, numbers of different cell types do not necessarily correlate with functional capacity, and changes of cell numbers in the peripheral circulation may merely reflect a redistribution of cell types from various immune compartments (Murray et al., 1992).

Measurement of the *function* of the immune system can involve *in vivo* and *in vitro* techniques. Two examples of *in vivo* assays of immunity are measurement of the delayed type hypersensitivity (DTH) response following administration of skin tests and measurement of antibody responses to a specific antigen. Both techniques provide valuable data about the physiologic response of the organism to an antigenic challenge, and they have been proposed to be more relevant in the clinical assessment of immunocompetence than *in vitro* measures of immune function. However, these assays are expensive to perform, and they cannot be utilized in longitudinal studies because subsequent immunologic evaluations are altered by primary immunization.

An indirect assessment of cellular immune function includes the measurement of antibody titers to latent viruses (Glaser et al., 1985). The cellular immune response is thought to be important in controlling latent viral infections; reactivation of such viruses as herpes viruses can occur during conditions in which cellular immunity is compromised. In turn, synthesis of virus or viral proteins is increased and elevations in antibody titers are detected in serum.

Two *in vitro* correlates of cellular immune function, mitogen-induced lymphocyte proliferation, and NK-cell activity (NKCA), have been widely used to assess cell-mediated immune function. Both of these assays evaluate the function of cells *ex vivo* outside the body. Mitogen-induced lymphocyte stimulation determines the proliferative capacity of lymphocytes following activation *in vitro* either with plant lectins such as concanavalin A (Con A) or phytohemagglutinin (PHA), which predominantly activate the T lymphocyte to divide, or with pokeweed mitogen that induces proliferation of B cells (Keller et al., 1981). The proliferative response is quantitated by the cellular incorporation of radioactively labeled thymidine or idoxuridine into newly synthesized deoxyribonucleic acid (DNA).

The other frequently used *in vitro* assessment of cellular immunity involves measurement of NK activity. Assay of NK lytic activity is carried out by the coincubation of isolated "effector" lymphocytes with radioactively labeled "target" tumor cells. The release of radioactivity by the lysed target cells is proportionate to the activity of the effector NK cells (Herberman and Ortaldo, 1981; Irwin et al., 1987).

Humoral immune function is generally assessed by the measurement of plasma concentrations of specific immunoglobulins, particularly IgM and IgG. Typically, enzyme linked immunosorbent assays (ELISA) are utilized. Immunoglobulin levels in serum provide an index of B-cell responses but do not assess the mechanisms by which this response is regulated (Schleifer et al., 1986).

Assessment of granulocyte function involves measuring both phagocytic and killing ability of these cells, by an assay originally developed by Weir (1967) and subsequently refined by Keller and colleagues (1981). Phagocytes are separated from whole blood until the composition of the cell population is predominantly neutrophils (PMNs), with a small percentage (<5%) of monocytes. Cultures are maintained on blood agar plates and fresh cultures are used for each assay. Then bacteria, *Staphylococcus aureus* (American Type and Culture collection #29213) are first mixed with normal human serum for 25 minutes for opsonization. Phagocyte function is assessed by incubation of the phagocyte cells with the opsonized *S. aureus* for 10 minutes at a 100:1 bacteria to phagocytic cell ratio. Cells are then washed and divided into five aliquots. Aliquot 1 is lysed immediately and the supernatant placed on blood agar to obtain the number of *S. aureus* phagocytized. This time zero plating is a sensitive *in vitro* measure of the adherence of bacteria to the cells and of the cells ability to phagocytize the bacteria. Aliquots 2 and 3 are incubated at 37°C, aliquot 2 for 1 hour and aliquot 3 for 2 hours, prior to lysis and *S. aureus* counts. Aliquots 4 and 5 are kept on ice, aliquot 4 for 1 hour and aliquot 5 for 2 hours as laboratory controls. Results of killing ability are expressed as the number of *S. aureus* colonies that grow after 1 or 2 hours incubation, subtracted from the number phagocytized.

Assay of levels of lymphokines (e.g., IL-1, IL-2, interferon [IFN]) in the plasma or following lymphocyte stimulation recently have been employed to quantitate possible alterations in the regulation of these cellular factors important in humoral and cellular immune responses (Glaser et al., 1990). IL-1 promotes lymphocyte differentiation via increased expression of IL-2 receptors, whereas IL-2 provides a signal for the proliferation and differentiation of immune cells (Oppenheim et al., 1991). Assay of these lymphokines is proposed to provide information about the immunologic mechanisms that may be related to changes in *ex vivo* measures such as lymphocyte proliferation or NK activity (Glaser et al., 1990; Maes et al., 1991).

## REGULATORS OF IMMUNITY

The day-to-day function of the immune system is influenced by many factors (Lawrence and Kim, 2000), including products of the immune system itself (e.g., cytokines), neurotransmitters, hormones, neuropeptides, ambient or environmental antigens or pathogens, and temperature. Immunity and these immunomodulators can vary with age, gender, and race. For example, during puberty the hormonal milieu in which the immune system functions changes drastically with respect to many hormonal immune modulators. In animal studies, altered noradrenergic innervation of spleen, lymph nodes, and the thymus have been reported in association with aging (Madden et al., 1998). Further, replacement of growth hormone in animals and humans has been reported to reverse some of the age-associated changes in the immune system and thymus (Burgess et al., 1999). The following is a review of some of the relevant and important immune regulators.



## Central Nervous System

A multitude of experimental evidence now exists that the brain can influence immune function, in part by coordinating the autonomic nervous system (ANS) and neuroendocrine pathways. Early experiments placed electrolytic or other destructive lesions in the hypothalamus and demonstrated alterations in lymphoid tissue architecture ([Cross et al., 1980](#); [Isakovic and Jankovic, 1973](#)), either impairment or enhancement of lymphoid cell activation ([Brooks et al., 1980](#); [Cross et al., 1988](#); [Keller et al., 1980](#); [Roszman et al., 1982](#)), impairment of delayed type hypersensitivity ([Macris et al., 1970](#); [Stein et al., 1976](#)), and suppression of NKCA ([Cross et al., 1984](#)). Less focal lesions in cortical tissue have revealed not only that cellular and humoral immune responses are modulated by ablation of parieto-occipital neocortex, but also that these effects are observed only following right hemisphere lesions ([Barneoud et al., 1987](#)).

Stroke represents a naturally occurring CNS lesion in humans. In a study of ischemic stroke, [Fiorina and colleagues \(1999\)](#) reported impaired cellular immunity and energy in association with diminished nocturnal melatonin secretion, altered sleep patterns, and mood disturbance. Although this study did not consider location of the lesion, mood disturbances following stroke have been linked to frontal lesions ([Singh et al., 2000](#)).

[Kang and associates \(1991\)](#) found that women with extreme right frontal activation as measured by electroencephalography (EEG) had lower levels of NK activity than did left frontally activated individuals. These observations are intriguing in light of psychophysiologic evidence that emotions are asymmetrically processed in the frontal cortex, with negative emotions (e.g., disgust) producing greater relative right frontal activation, and positive emotions (e.g., amusement) producing greater relative left frontal activation ([Wheeler et al., 1993](#)). Furthermore, clinical depression and a history of clinical depression are both associated with decreased relative left frontal activation ([Henriques and Davidson, 1990, 1991](#)).

## Autonomic Nervous System

The autonomic nervous system (ANS) is a pathway for direct communication from the brain to cells of the immune system. Anatomic studies have revealed an extensive presence of autonomic fibers in both primary and secondary lymphoid organs ([Bulloch and Pomerantz, 1984](#); [Felten et al., 1981, 1984, 1985](#); [Felten and Olschowka, 1987](#); [Kendall and al-Shawaf, 1991](#); [Leposavic et al., 1992](#)), innervating both the vasculature and parenchyma of the tissues. In a pattern of classic anatomic connection, preganglionic cell bodies located in the intermediolateral column of the spinal cord synapse with the ganglionic cells that are found either in the sympathetic chain or collateral ganglia ([Felten and Olschowka, 1987](#)). Immunohistochemical studies have demonstrated that nerve fibers enter the lymphoid organs, such as the spleen, along with the vasculature and branch into the parenchyma ([Felten et al., 1985](#)) and areas in which lymphocytes (primarily T cells) reside ([Ackerman et al., 1987](#); [Felten et al., 1985](#)). These noradrenergic fibers are not only adjacent to sites where T cells congregate but, at the electron microscopic level, end in synapse-like contacts with lymphocytes in the spleen ([Felten and Olschowka, 1987](#)). Together, these observations establish an anatomic link between the brain and immune system.

It has been hypothesized that the development and differentiation of lymphoid tissue are guided by the ANS ([Ackerman et al., 1987](#); [Bulloch, 1985](#); [Bulloch et al., 1987](#)). For example, the thymus was penetrated by vagal fibers before its immune and endocrine functions develop ([Hammar, 1935](#)). Furthermore, in the spleen, development of catecholaminergic nerves in the periarteriolar lymphatic sheaths parallels changes in T-cell compartmentalization ([Ackerman et al., 1987](#)), suggesting that norepinephrine influences lymphocyte movement into the spleen and the development of T- and B-cell compartments shortly after birth. Sympathetic innervation of the spleen also may be essential for the development of splenic immune cell function. Fischer 344 rats that were chemically sympathectomized with the neurotoxin 6-hydroxydopamine (6-OHDA) in the first week of life showed impaired NK cytotoxicity, mitogen-stimulated lymphocyte proliferation, and antibody responses lasting up to 3 months as compared to saline-treated controls ([Ackerman et al., 1991](#); [Madden et al., 1993](#)).

In addition to direct neuronal influence by the ANS, chemicals released during ANS activity influence immunity. In the adult animal, norepinephrine appears to act as a neurotransmitter in the spleen. Norepinephrine is released within the spleen; early studies by [Von Euler \(1946\)](#) found that splenic nerve stimulation yields a release of norepinephrine. Furthermore, *in vivo* dialysis techniques documented a 1- $\mu$ m concentration of norepinephrine in the rat spleen ([Felten et al., 1986](#)), a concentration that is more than 100-fold higher than that in blood, suggesting local release of norepinephrine within the spleen.

Lymphocytes have been found to receive signals from sympathetic neurons by adrenoceptor binding of norepinephrine, epinephrine, and dopamine ([Aarons and Molinoff, 1982](#); [Bidart et al., 1983](#); [Brodde et al., 1981](#); [Miles et al., 1984](#); [Motulsky and Insel, 1982](#); [Williams et al., 1976](#)). These b-receptors are linked to adenylate cyclase ([Katz et al., 1982](#); [Strom et al., 1977](#); [Watson, 1975](#)) and appear to have a functional role in the modulation of cellular immunity. *In vitro* incubation of lymphocytes with varying concentrations of either norepinephrine or epinephrine decreases NK activity ([Hellstrand et al., 1985](#)) and mitogenic responses ([Hadden et al., 1970](#)). As preincubation with a b-antagonist reverses the inhibitory effects of norepinephrine *in vitro* ([Hellstrand et al., 1985](#)), b-adrenoceptor binding is believed to mediate an inhibition of cellular immunity ([Livnat et al., 1985](#)). *In vivo* studies have shown that either surgical denervation of the spleen or chemical sympathectomy with 6-OHDA produces an augmented antibody response to thymus-dependent antigens such as sheep red blood cells ([Besedovsky et al., 1979](#)), an enhanced plaque-forming cell response to thymus-independent antigens ([Miles et al., 1981](#)), and altered T- and B-cell responsiveness to mitogen stimulation (Hall and Goldstein, 1981; [Livnat et al., 1985](#)). Others have found in animals and humans that infusion of adrenergic agonists results in a down-regulation of b-adrenergic receptors in circulating mononuclear cells ([Aarons and Molinoff, 1982](#); [Krall et al., 1980](#)) and acute changes in NK-cell function and lymphocyte responses to mitogens and a specific antigen ([Heilig et al., 1993](#); [Livnat et al., 1985](#); [Pezzone et al., 1992](#); [Schledowski et al., 1993](#); [Tonnesen et al., 1984](#)). Endogenous release of sympathetic catecholamines appears to mediate the immunomodulatory effects of acute stress. Animals exposed to aversive stress or central doses of neuropeptides such as corticotropin-releasing hormone (CRH) that activate sympathetic outflow show suppression of cellular immunity or *in vivo* specific antibody responses that can be reversed by autonomic blockade ([Irwin et al., 1988](#)), or chemical sympathectomy ([Irwin et al., 1990](#)). Pharmacologic blockade of b-adrenergic receptors restores suppressed splenic lymphocyte responses to Con A produced by foot shock stress and conditioned immunosuppression ([Cunnick et al., 1990](#); [Luecken and Lysle, 1992](#)).

In human studies, the importance of the sympathetic nervous system (SNS) in altered immunity is underscored by the rapidity of change following an acute laboratory stressor. Further, the immune changes reported were even greater in subjects who also had elevated cardiovascular responses to the stressors ([Herbert et al., 1994](#)). In a study of immunity in spinal cord injured (SCI) patients, only those patients with high levels of injury (cervical), and loss of SNS integrity were found to have altered lymphocyte function compared to normal healthy matched controls ([Campagnolo et al., 1994](#)). Similarly, tetraplegic but not paraplegic SCI patients were found to have impaired phagocytic ability compared to matched, healthy controls ([Campagnolo et al., 1997](#)).

Chronic elevation of sympathetic tone has also been found to mediate a reduction of immunity ([Irwin et al., 1991](#)). In animals, for example, induction of a chronic hyperadrenergic state (experimental congestive heart failure) or 2-week infusion of the b-agonist isoproterenol reduces NK activity, *in vivo* antibody responses, and lymphocyte proliferation ([Waltman et al., 1992](#)). Similar to findings following acute stress, the immunosuppressive effect of chronic elevated sympathetic tone is completely antagonized by B-receptor blockade. In humans, chronic elevated sympathetic tone may also contribute to the modulation of immune function during severe life stress and depression as reflected by circulating concentrations of neuropeptide Y ([Irwin et al., 1991](#)). Neuropeptide Y is present in peripheral sympathetic nerves and is released following emotional stress potentiating the effects of vasoactive catecholamines and other pressor substances ([Romano et al., 1991](#)). [Irwin and associates \(1991\)](#) have shown that plasma concentrations of neuropeptide Y are elevated in depressed patients, as well as aged individuals and persons undergoing severe Alzheimer caregiver stress. Furthermore, activation of the SNS and release of neuropeptide Y is associated with a reduction of natural cytotoxicity in depression and life stress. Additional findings also support the hypothesis that elevated sympathetic activity in depression is associated with immune alterations. In depressed patients, excretion of 3-methoxy-4-hydroxy-phenylglycol (MHPG) has been used as an index of total body noradrenergic turnover or sympathetic activity and MHPG excretion was inversely related with lymphocyte proliferative responses in depressed patients ([Maes et al., 1989](#)).

Together, these data suggest that the ANS is important in immune function. Further, they suggest that elevated sympathetic tone in patients with major depressive disorder and/or in persons undergoing life stress is inversely correlated with cellular immune function.

## Endogenous Opioid Peptides

Pharmacologic evidence has shown that some cells of the immune system have opioid receptors ([Sibinga and Goldstein, 1988](#)). Opioid binding on immune cells was first suggested by the finding that nanomolar concentrations of opioids affect active rosetting of human T lymphocytes ([Wybran et al., 1979](#)). Morphine inhibits rosetting, whereas met-enkephalin stimulates it, and both effects are blocked by naloxone. Using radiolabeling techniques, specific opioid binding on human phagocytic leukocytes ([Falke et al., 1985](#); [Farrar, 1984](#); [Lopker et al., 1980](#)), platelets ([Mehrishi and Mills, 1983](#)), and lymphocytes ([Mehrishi and Mills, 1983](#); [Ausiello and Roda, 1987](#)) has been demonstrated.



Endogenous opioid peptides are able to influence *in vitro* the function of most cell types of the immune system; both inhibitory and immunoenhancing effects of opioid peptides have been described. [Johnson and coworkers \(1982\)](#) found that  $\alpha$ -endorphin, met-enkephalin, and leu-enkephalin (all with approximately equal potency) decrease the proliferation and antibody production of splenocytes in the plaque-forming assay; an effect that is blocked by naloxone. Confirming these results, [Heijnen and colleagues \(1986\)](#) demonstrated that (des-tyr) b-endorphin is also active in suppressing the plaque-forming cell response. PHA induced proliferation is affected by opioid peptides, although both stimulatory ([Bocchini et al., 1983](#); [Farrar, 1984](#); [Gilman et al., 1982](#)) and inhibitory effects ([McCain et al., 1986](#); [Puppo et al., 1985](#)) have been reported, and these opioid effects on mitogen-induced lymphocyte proliferation, and IL-2 production are not blocked by the specific opiate antagonist naloxone ([Gilmore and Weiner, 1988](#)).

A naloxone reversible enhancement of human natural cytotoxicity by very low concentrations of b-endorphin (10 fM) and met-enkephalin (10 pM) has also been reported. However, some studies have described an inverted U-shaped dose response for B endorphin and a bidirectional effect of the enkephalins and selective opiate agonists on human NK activity; subjects with "low" (below the median) NK activity show stimulation with enkephalin, whereas the cytolytic activity of cells from the "high" group is inhibited by similar doses of enkephalin ([Oleson and Johnson, 1988](#)). Recent evidence suggested that endorphins modulate NK activity through the (6–9) amino acid region (i.e., the  $\alpha$ -helix portion of b-endorphin) ([Kay et al., 1987](#)).

Systemic administration of opioids, as well as the release of opiate peptides following stress, appear to modulate changes of NK cytotoxicity in animals, although the physiologic importance of opioid modulation of other *in vitro* correlates of immune function can not yet be adduced with confidence. In rats repeatedly administered intermittent inescapable foot shock over four consecutive days ([Shavit et al., 1984](#)), a significant suppression of NK activity occurs that can be completely antagonized by the preadministration of naltrexone. Unconditioned (foot shock) and conditioned suppression of lymphocyte proliferation responses can be attenuated by naltrexone ([Lysle et al., 1992](#)). Daily injection of morphine for 4 days at doses of 10, 30, or 50 mg/kg produces a similar suppression of cytotoxicity ([Shavit et al., 1984, 1986](#)). In contrast, inhibition of NK cytotoxicity in a forced-swimming paradigm was unaffected by pretreatment with naltrexone ([Ben-Eliyahu et al., 1990](#)), and naltrexone dose-dependently aggravated reductions in primary and secondary antibody responses to sheep red blood cells induced by restraint stress ([Ray et al., 1992](#)).

Nevertheless, direct peripheral effects of opioid agonists on the immune system are also possible, because opioid agonists bind on specific receptors on cells of the immune system ([Carr et al., 1988](#)). Indeed, experimental and correlative evidence suggest that peripheral increases of endogenous circulating concentrations of b-endorphin stimulate NK activity, similar to the findings generated *in vitro*. In the rat, release of b-endorphin into the plasma following acute exposure to forced immobilization are correlated with immediate, poststress increases of splenic NK activity ([Irwin and Hauger, 1988](#)). Likewise, clinical research has found exercise-induced enhancement of NK cytotoxicity is completely antagonized by the preadministration of the opiate antagonist naloxone ([Fiatarone et al., 1988](#)). Finally, plasma levels of b-endorphin are positively correlated with NK activity in depressed patients ([Darko et al., 1992](#)).

Opiate-induced modulation of NK cytotoxicity is also mediated in part by the CNS. A reduction of splenic NK activity is achieved by the administration of a dose of morphine given into the cerebral ventricle that is a thousand times smaller than that required when given systemically ([Shavit et al., 1986](#)). Peripheral administration of a morphine analog (one that does not cross the blood–brain barrier to enter the CNS) is not effective in altering NK activity ([Shavit et al., 1986](#)). Moreover, intracerebral infusion of morphine into the periaqueductal gray, but not other brain regions, produces a suppression of splenic NK activity ([Weber and Pert, 1989](#)). Finally, peripheral administration of naltrexone has been shown to block conditioned suppression of lymphocyte proliferation responses, but N-methyl naltrexone, which does not cross the blood–brain barrier, is unable to reverse the suppression ([Lysle et al., 1992](#)).

### Serotonin

Serotonin is a neurotransmitter that appears to mediate nervous system and immune system interactions ([Mossner and Lesch, 1998](#)). Substantial evidence has demonstrated that serotonergic systems are altered in depression, and *in vitro* data have shown that serotonin modulates delayed type hypersensitivity responses, T-cell activation, and NK and macrophage activity ([Mossner and Lesch, 1998](#)). Further, serotonin suppresses lymphocyte response to PHA *in vitro* ([Bonnet et al., 1984](#)), whereas the addition of serotonin to enriched mononuclear cells induces a twofold, dose-dependent enhancement of NK activity, similar to the stimulation induced by IL-2 ([Hellstrand and Hermodsson, 1987](#)).

The induction of NK activity is monocyte-dependent and likely mediated by specific binding of serotonin at the 5HT<sub>1</sub> receptor on the monocyte; the enhancing properties of serotonin are mimicked by the 5-HT<sub>1</sub>-specific receptor agonist 8-OH DPAT and completely antagonized by the serotonin receptor antagonist cyproheptadine. Clinical studies have not yet characterized whether expression and regulation of the 5-HT<sub>1A</sub> receptor on monocytes is altered in depressed patients, and it is not known whether monocyte 5-HT<sub>1A</sub> activity has a role in the reduction of NK activity in depression.

## NEUROENDOCRINE INFLUENCES ON IMMUNITY

The neuroendocrine system is an important regulator of immune responses ([Anisman et al., 1996](#); [Marx et al., 1998](#)). The following is a brief review of some of the more important hormonal effects on immunity.

### Corticotropin-Releasing Hormone

Corticotropin-releasing hormone (CRH) has been postulated to be a physiologic CNS regulator that integrates biological responses to stress ([Axelrod and Resine, 1984](#); [Taylor and Fishman, 1988](#)) and that may affect the susceptibility to various illnesses, including autoimmune, infectious, and neoplastic disease ([Elenkov et al., 1999](#)). Its role in modulating immune function through a central site of action has been examined ([Irwin et al., 1987](#)). In addition to its well-established role as a hypothalamic regulator of the pituitary secretion of ACTH and b-endorphin ([Vale et al., 1981](#)), CRH is also found to act in the CNS at extrahypothalamic sites inducing an increase in the firing rate of the locus ceruleus ([Valentino et al., 1983](#)), activating the ANS as reflected by increased plasma concentrations of norepinephrine and epinephrine ([Fisher et al., 1982](#)), and producing a pattern of behavioral responses such as decreased feeding and increased locomotor activity ([Britton et al., 1982](#); [Sherman and Kalin, 1985](#); [Sutton et al., 1982](#)). Moreover, central administration of CRH induces a dose-dependent suppression of splenic NK activity ([Irwin et al., 1987](#)), which is mediated by central activation of the SNS ([Irwin et al., 1988](#)). Finally, recent data suggested a physiologic role for the release of central endogenous CRH in stress-induced suppression of cellular immunity; central administration of an antiserum to CRH completely antagonizes the effects of inescapable shock on splenic NK activity and lymphocyte proliferation ([Irwin et al., 1990](#); [Saperstein et al., 1992](#)). Consistent with the previous findings that show that activation of the pituitary adrenal axis is dissociated from acute stress-induced changes in cellular immunity ([Keller et al., 1983, 1988](#)), peripheral administration of antiserum to CRH antagonizes stress-induced release of ACTH and corticosterone but has no effect on the suppression of NK activity ([Irwin et al., 1990](#)).

### Glucocorticoids

Cortisol has many effects on the immune system ([Byron et al., 1992](#)), and the secretion of corticosteroids has long been considered as the mechanism of stress-induced and/or depression-related suppression of immune function ([Cupps and Fauci, 1982](#); [Munck et al., 1984](#); [Parrillo and Fauci, 1978](#); [Riley, 1981](#); [Selye, 1976](#)). Specific intracytoplasmic corticosteroid receptors have been identified in normal human lymphocytes ([Lippman and Barr, 1977](#)), and these receptors bind corticosteroids and appear to play a role in the regulation of cellular function through modulation of cyclic AMP levels ([Parker et al., 1973](#)). Glucocorticoids have widespread effects on the immune system, including the trafficking of immune cells and the secretion of cytokines ([Chrousos, 1995](#)).

*In vitro* studies have further demonstrated that glucocorticoids act to inhibit IL-2 production *in vitro* with resulting suppression of lymphocyte responses to mitogenic stimulation ([Gillis et al., 1979](#)), natural killer cell activity (antibody-dependent cytotoxicity is relatively refractory to glucocorticoids) ([Parrillo and Fauci, 1978](#)), and the differentiation and function of macrophages ([Baybutt and Holsboer, 1990](#)). In the thymus, glucocorticoids influence apoptosis of thymic lymphocytes and the clonal deletion. This effects removal of self-antigen responsive cells ([Wilder, 1995](#)).

Even though pharmacologic data from *in vitro* and *in vivo* studies demonstrated that corticosteroids suppress a number of parameters of immunity ([Cupps and Fauci, 1982](#)), increased adrenocortical activity is not always correlated with changes in immune function in an organism responding to aversive stressors. For example, acute administration of either forced immobilization ([Irwin and Hauger, 1988](#)) or audiogenic stress ([Irwin et al., 1989](#)) produces an activation of adrenal steroid secretion but does not alter immune function as measured by NKCA. Likewise, with repeated exposure to the stressor, pituitary adrenal activation is dissociated from a reduction in cytotoxicity ([Irwin and Hauger, 1988](#); [Irwin et al., 1989](#)). Finally, stress-induced suppression of lymphocyte function following unpredictable, unavoidable tail shock occurs both in adrenalectomized ([Keller et al., 1983](#)) and in hypophysectomized animals ([Keller et al., 1988](#)). Nevertheless, stress-related reductions of



lymphocyte and antibody responses in which adrenal activation clearly participate have been reported ([Cunnick et al., 1990](#); [Dobbs et al., 1993](#)). A possible resolution of these discrepancies may lie in the site of action for adrenal corticoids. Stress-induced decreases in splenocyte mitogen responses can be abolished by pharmacologic blockade of  $\beta$ -adrenergic receptors, but the responses of peripheral blood lymphocytes are only partially restored ([Cunnick et al., 1990](#); [Pezzone et al., 1992](#)). In contrast, adrenalectomy prevents stress-induced inhibition of mitogenic responses in peripheral blood, but not splenic lymphocytes in rats, and use of the glucocorticoid receptor antagonist RU486 attenuates stress-induced reductions in antibody responses to herpes simplex virus in mice ([Pezzone et al., 1992](#)).

Clinical research has found no relationship between adrenocortical activity and immunity in depressed patients and stressed persons. In depressed patients, decreased lymphocyte responses to mitogens are not associated with dexamethasone nonsuppression ([Kronfol and House, 1985](#)) or with increased excretion rates of urinary free cortisol ([Kronfol et al., 1986](#)). Furthermore, in bereavement in which a reduction of NK activity has been demonstrated, this immunologic change occurs even in subjects who had plasma cortisol levels comparable to control subjects ([Irwin et al., 1988](#)).

## ACTH

A receptor for ACTH has been identified on lymphocytes, also raising the possibility that some aspects of altered lymphocyte function in depression are regulated by this peptide. *In vitro* studies have shown that ACTH inhibits the antibody response at an early state in the antibody response ([Johnson et al., 1992](#)). In regard to cellular immunity, [McGlone and coworkers \(1991\)](#) evaluated the effects of ACTH administered *in vitro* and *in vivo* on NK cell function. Exogenous, peripheral administration of ACTH at physiologic concentrations increases both NK activity and IL-2 stimulated NK cytotoxicity compared to levels in saline controls. Because *in vitro* doses of ACTH have no effect on NK activity ([McGlone et al., 1991](#)), the action of this peptide *in vivo* is likely by an indirect mechanism (i.e., alteration of leukocyte traffic or suppression of central CRH). Finally, one clinical study of 26 depressed patients failed to demonstrate an association between plasma concentrations of ACTH and lymphocyte proliferation ([Darko et al., 1989](#)), although the effects of ACTH on NK activity have not yet been evaluated.

## DHEA

Dehydroepiandrosterone sulfate (DS) and its active metabolite dehydroepiandrosterone (DHEA) are also adrenal hormones, which play an important role in immune regulation. DHEA has been related to greater production of IL-2 and enhanced T-cell cytotoxic function ([Suzuki et al., 1991](#)). In a study of postmenopausal women, treatment with DHEA was associated with a decrease in CD4+ cells and an increase in T-helper cells ([Casson et al., 1993](#)). Several studies of the influence of DHEA on immune response to the influenza vaccine in elderly patients found different results, two suggesting that the response was enhanced ([Araneo et al., 1995](#); [Evans et al., 1996](#)) and one noting no effect ([Dannenbergh et al., 1997](#)).

## Prolactin

Prolactin (PRL) is involved in the regulation of the immune system ([Bernton et al., 1989](#)). Prolactin receptors have been found on T and B lymphocytes ([Russell et al., 1985](#)) and prolactin effects NK activity ([Nagy et al., 1983](#)) and lymphocyte proliferative responses ([Hiestand et al., 1986](#)). However, understanding the effects of PRL on the immune system has been complicated by the discovery that lymphocytes and uterine tissue can synthesize and secrete PRL ([Montgomery et al., 1992](#); [Pellegrini et al., 1992](#)). The question of which PRL, perhaps all, serves as a signaling molecule in the immune system remains to be answered ([Matera, 1996](#)). Basically, PRL levels are thought to correlate positively with immunity, meaning that higher levels are stimulatory, whereas lower levels are inhibitory ([Yu-Lee, 1997](#)). Further, there is speculation concerning how PRL affects immunity. Although there are PRL receptors on immunocytes, the effects are mediated at least in part by influencing interferon regulatory factor-1 (IRF-1) ([Yu-Lee, 1997](#)). However, PRL also influences thymic hormone release, and effects on T cells appear to be dependent in part on their stage of maturity or differentiation ([Murphy et al., 1992](#)).

Impairment of both humoral and cell-mediated immune response has been demonstrated in hypophysectomized rats as well as animals treated with bromocriptine, a dopamine agonist that inhibits prolactin secretion. This impairment can be reversed by treatment with exogenous, physiologic doses of prolactin ([Bernton et al., 1988](#)). In addition, antibodies to prolactin have been reported to inhibit lymphocyte proliferation ([Hartmann et al., 1989](#)).

In humans, preliminary observations by [Darko and colleagues \(1989\)](#) have found that plasma levels of prolactin positively correlated with T-cell mitogen proliferation, although [Clodi and associates \(1992\)](#) failed to show any immune alteration as reflected by serum concentrations of immunoglobulin, IL-1, soluble IL-2 receptor, lymphocyte subsets, and NK activity in patients with prolactinomas. The effects of hyperprolactinemia have been conflicting with respect to NK activity; some studies show decreased NK cell function ([Matera, 1996](#)). Chronic prolactin elevation may lead to adaptive changes that effectively abolish the immunomodulatory effects of prolactin.

## Growth Hormone

Growth hormone (GH) is a polypeptide that plays a pivotal role in growth and development. In addition to its endocrine and metabolic effects, growth hormone is likely to have significant effects on the immune system ([Murphy et al., 1992](#)), particularly T-cell development ([Weigent and Blalock, 1990](#)). In growth hormone-deficient mouse strains, hematopoietic and B-cell progenitor deficiencies, marked atrophy of the cortical region of the thymus, and impairment of cell-mediated immunity have been found and can be reversed by treatment with growth hormone ([Dumont et al., 1979](#); [Murphy et al., 1992](#)). Likewise, thymic size in the aged rat can be increased by GH ([Kelley et al., 1986](#)). In adult rats, GH is reported to stimulate macrophage superoxide release ([Edwards et al., 1992a, 1992b](#)) and confer resistance to bacterial infection ([Edwards et al., 1992](#)). In contrast with these observations, [Cross and colleagues \(1992\)](#) have indicated that hypopituitary mice lag behind heterozygous litter mates with respect to development of immunocompetence but that normal immune responsiveness does fully develop.

Exploratory clinical studies in depression have failed to identify any relationship between growth hormone and immune function ([Darko et al., 1989](#)), although the numbers of depressed subjects studied is small.

Investigation of the effects of GH (and PRL) on the immune system is complicated by the ability of GH to bind to PRL receptors; however, data suggest that GH may have both stimulatory and inhibitory effects on the immune system in humans. Several studies in children have investigated GH and other growth related factors and immunity. In a study by [Yang and coworkers \(1999\)](#), growth hormone and insulin-like growth factor I (as well as insulin-like growth factor binding proteins 1–6) are expressed by immunocytes and in immune organs where they are thought to function in an autocrine or paracrine fashion. In the study of children with GH deficiency, impaired phagocytic ability of neutrophils and monocytes was reversed with long-term GH replacement therapy (RT) ([Manfredi et al., 1994](#)). [Rapaport and colleagues \(1986\)](#) also studied *in vivo* effects of human GH RT on peripheral blood leukocytes. Although no changes in immunoglobulins, polymorphonuclear leukocyte function, or T-cell percents were found, a decreased percentage of B cells were reported. Similar findings were also reported by [Bozzola and coworkers \(1988\)](#); when immunocytes from both HGH deficient patients and normal controls were exposed to HGH *in vitro*, B cells were decreased and lymphocyte proliferative responses were enhanced.

## SUBSTANCES OF ABUSE AND IMMUNITY

Substances of abuse, including alcohol, nicotine, cocaine, and heroin, can influence the immune system. The effects may relate to prenatal exposure, as in cases of fetal alcohol syndrome, or substance use of the individual. The common substances of abuse, including alcohol, nicotine, cocaine, marijuana, and opiates, have been reported to influence immunity. Even single dose or acute exposure to these substances is shown to impact on immune responses. Further, early or *in utero* exposure to these substances may impact immunity and even have prolonged effects, such as those reported in fetal alcohol syndrome. The following is a brief review of human studies concerning these substances of abuse and their effects on immunity.

Chronic alcohol use and alcoholism are associated with immune alterations such as decreased defenses against infection ([MacGregor, 1986](#); [Szabo, 1999](#)) and increased infectious morbidity and mortality ([Adams and Jordan, 1984](#)). More recently, reports of acute exposure, both *in vitro* and *in vivo*, suggest that immune effects are seen in nonalcoholic subjects, especially regarding monocytes. [Szabo \(1998\)](#) reported that as little as 2 mg/kg of vodka consumed over 30 minutes led to transient (at 4 hours postconsumption) increases in IFN- $\gamma$  and IL-12 following monocyte stimulation with bacteria. In addition, alcohol had a biphasic effect on IFN- $\gamma$  production; bacterial stimulation of monocytes at 16 hours postconsumption was associated with decreased IFN- $\gamma$  production. [Girouard and coworkers \(1998\)](#) also investigated alcohol effects on cytokine production of monocytes from nonalcoholic subjects. Monocytes were exposed to alcohol and stimulated with bacteria or cytokines. Production of IL-10 and IL-12 was affected; specifically, IL-10 production was secondarily increased after exposure to alcohol with attendant enhancement of IL-12 production. Consistent with these data, [Irwin and Miller \(2000\)](#) reported that African-American alcoholics showed increased stimulated production of IL-10 and decreased expression of IL-6 as compared to matched comparison controls. However, in another study investigating lymphocyte numbers, type, and function, but not cytokine production, no differences were found between healthy nonalcoholic controls and healthy alcohol-dependent subjects ([Schleifer et al., 1999](#)). In that study,



however, granulocyte phagocytic ability was diminished in the alcohol-dependent subjects. Further, [Singhal and colleagues \(1999\)](#) reported increased apoptosis of macrophages following an alcohol binge in healthy subjects. These data strongly suggest that alcohol influences the immune system and that myeloid derived cells are particularly susceptible.

Cigarette smoking has long been considered a major health risk, with effects on immunity via direct actions or possibly endocrine-mediated mechanisms ([Sopori and Kozak, 1998](#)). Nicotine is reported to affect the HPA axis ([Rosecrans and Karin, 1998](#)) as well as to affect humoral and cellular immunity ([McAllister-Sistilli et al., 1998](#)). Additional research supports the notion that specific immunologic alterations occur in adult smokers, affecting a variety of parameters. Adult male smokers have been found to exhibit higher white blood cell (WBC) counts and lower NK activity than nonsmoking controls. [Tollerud and coworkers \(1989\)](#) reported elevated WBC and lymphocyte numbers but lower NK cell counts in adult smokers compared to nonsmokers. In the only study of adolescent smoking effects on immunity, Keller and colleagues (in review) reported decreased cell numbers but not lower NKCA in adolescents (12 to 18) who reported smoking for 1 year or more.

Smoking also has been associated with elevated levels of IgM and IgA rheumatoid factor (RF), with a more persistent elevation of RF than nonsmokers, indicating that tobacco use increases the production of IgM and IgA RF. These findings are similar to results reported by [Von Herzen and colleagues \(1998\)](#), who found that smoking in adult male twin pairs was significantly associated with elevated IgA antibody levels in smokers with implications for chronic *Chlamydia pneumoniae* infection.

Pulmonary immune function is also a focus of recent studies. IL-8 concentration in bronchoalveolar lavage fluid was significantly higher in smokers than nonsmoking adult subjects. Finally, cigarette smoking also alters nasopharyngeal lymphoid tissue. [Finkelstein and coworkers \(1997\)](#) reported that smokers exhibited deformed and migrating cytotoxic lymphocytes in the nasopharyngeal mucosa, which were attached to epithelial, ciliated, and goblet cells. Cell damage, particularly mucosal invasion and epithelial cell damage, occurred as a result of the uncommon amount of migrating T lymphocytes in the smoking subjects.

Perhaps most notably, depression and smoking status interact to produce greater declines of NK activity than that found in depressed or smoking groups alone ([Jung and Irwin, 1999](#)).

Use of hashish or marijuana, both of which are derived from *Cannabis sativa*, is thought to diminish host resistance to infections ([Cabral and Dove Pettit, 1998](#)). The primary psychoactive component, d-9-tetrahydrocannabinol (THC) and other cannabinoids purportedly are immunosuppressive, affecting lymphocytes, macrophages, and NK cells ([Cabral and Dove Pettit, 1998](#)). THC has been reported to diminish ([Specter et al., 1990](#)) or produce variable change ([Luo et al., 1992](#)) in response to mitogen stimulation and to have indeterminate effects on antibody production ([Nahas and Osserman, 1991](#)) in humans. Although phagocytosis and killing activity by human monocytes are reportedly decreased ([Specter et al., 1991](#)), cannabinoids are reported to both increase and decrease individual immune parameters, depending on dose, chronicity, and timing of exposure ([Klein et al., 1998](#)). Actual health risk of cannabinoid use to humans requires further study.

Opiates such as morphine are thought to affect the immune system via CNS pathways and direct effect on some types of immunocytes ([Eisenstein and Hilburger, 1998](#)). Primarily, the indirect opiate-neural-immune pathway has an immunosuppressive effect, down-regulating all subtypes of immune cells, whereas morphine appears to directly diminish macrophage and granulocyte function but not NK cell or B-cell function ([Eisenstein and Hilburger, 1998](#)). Further, chronic exogenous opioids impact on IL-1 mediated immunity, substantially influencing the body's defense mechanisms ([Chang et al., 1998](#)).

## STRESS AND IMMUNITY: PRECLINICAL STUDIES

### Aversive Stressors and Disease Susceptibility

Many workers have identified links between the stress associated with physical and psychological stimuli and the increased susceptibility of animals and humans to a number of immune-related disorders such as infectious and neoplastic disease ([Griffin, 1989](#); [Rozlog et al., 1999](#)). To evaluate altered resistance to infections, a number of different aversive stressors have been used in rodents. Psychosocial stimulation of animals involving either avoidance conditioning, physical restraint, or auditory stimulation is associated with decreased resistance to herpes simplex, Coxsackie B, poliomyelitis, or vesicular stomatitis virus, and tuberculosis ([Friedman and Glasgow, 1966](#)). High population density affects the susceptibility of mice to the malarial parasite *Plasmodium berghei* ([Plaut et al., 1969](#)) and isolation housing is associated with increased risk of encephalomyocarditis virus infections ([Friedman et al., 1969](#)). Restraint stress was reported to diminish cellular immune and inflammatory responses to viral infection in mice ([Hermann et al., 1994](#)), an effect mediated by corticosterone and catecholamines ([Dobbs et al., 1993](#)). It appears that both the virulence of the organism as well as the psychophysiological state of the animal affects disease risk. If the infectious agent produces a chronic or insidious infection and the animals are stressed at the time of exposure, then symptoms of disease are more likely ([Ader, 1983](#); [Friedman et al., 1965](#); [Griffin, 1989](#)). Preliminary research with macaque monkeys infected with simian immunodeficiency virus (SIV) found that early psychosocial factors, such as rearing condition (i.e., with mother or peers) and the number of changes in housing (i.e., from a familiar social group to an unfamiliar group or to isolation) were significantly correlated with disease-related variables such as latency to leukopenia, weight loss, and survival ([Capitanio and Lerche, 1991](#)).

Findings from studies of the role of stress in susceptibility to neoplastic disease are heterogeneous, depending on the age and species of the animal, the type of tumor (viral or chemically induced), the timing of the stressor in relation to tumor inoculation, and the quality of the stressor (inescapable or escapable) ([Griffin, 1989](#); [Justice, 1985](#); [Riley, 1981](#); [Sklar and Anisman, 1981](#)). In general, it appears that simultaneous exposure of rats or mice to inescapable shock and tumor cells facilitates tumor growth, whereas administration of the stressor before implantation inhibits its growth ([Shavit et al., 1985](#)). Exposure to escapable shock has no impact on tumor development ([Shavit et al., 1985](#)).

### Effects of Stressors on Immune Responses

Behavioral responsiveness to inescapable aversive stimulation has provided an animal model to investigate clinical depression ([Irwin et al., 1989](#)). Aversive stressors such as sound exposure ([Irwin et al., 1989](#); [Monjan and Collector, 1977](#)), rotation ([Kandil and Borysenko, 1987](#); [Riley, 1981](#)), intermittent shock ([Keller et al., 1981, 1983](#); [Shavit et al., 1984](#)), and forced immobilization ([Irwin and Hauger, 1988](#)) have been shown to affect lymphocyte responses to mitogen stimulation and/or NK activity in a manner that depends on their dose- and time-response profiles. For example, using an audiogenic stressor repeated at daily intervals, [Irwin et al. \(1989\)](#) have replicated the findings of [Monjan and Collector \(1977\)](#), who found that the initial stress-induced immune suppression is followed by an increase or enhancement of natural cytotoxicity. [Keller and associates \(1981\)](#) have demonstrated a relationship between the intensity of an acute stressor and the degree of suppression of T-cell function. A progressive decrease of PHA-induced stimulation is found in animals that receive apparatus, low-level tail shock, or high-level electric tail shock, respectively, as compared to home cage controls ([Keller et al., 1981](#)). Further, stress, specifically the stress of social isolation, has been related to decreased proliferative responses, NKCA and macrophage-mediated cytotoxicity, as well as increased tumor metastases and greater tumor burden ([Wu et al., 2000](#)).

Cellular *in vivo* immune responses and antibody responses are also altered by aversive stressors. Delayed hypersensitivity reaction are decreased in mice exposed to heat stress ([Pitkin, 1965](#)) and the graft-versus-host response is suppressed in animals subjected to limited feeding ([Amkraut et al., 1973](#)), an effect that is independent of adrenocortical levels ([Amkraut et al., 1973](#)). Reduced antibody responses are found in monkeys exposed to a number of aversive stimuli ([Felsenfeld et al., 1966](#); [Hill et al., 1967](#)), in mice housed in high- versus low-density grouping ([Solomon, 1969](#)), and in mice subjected to changes in housing condition (either from individual to group housing or from a group to the return to an individual cage) ([Edwards et al., 1980](#)). Similar to the effects of stressor timing on susceptibility to infectious or neoplastic disease, restraint produces a significant reduction in antibody response to sheep red blood cells only if the stress is applied before the time of immunization; there is no change in antibody production when the stress is applied after antigen injection ([Okimura et al., 1986](#)). Further, NKCA was reported to decline during captivity in free-range rhesus monkeys ([Laudenslager et al., 1999](#)), suggesting that continued or prolonged stress can produce continued immune alteration.

### Modulation by Psychological Factors

In addition to the effects of stressor characteristics, the psychological characteristics or response of the animal to the stressor has been related to the immunologic consequences of the stress. [Laudenslager and colleagues \(1983\)](#) found that rats exposed to inescapable uncontrollable electric tail shock have reduced lymphocyte activity, whereas animals that receive the same total amount of shock but are able to terminate it did not show altered immunity. Furthermore, [Mormede and coworkers \(1988\)](#) reported that even stressor predictability, in the form of a warning stimulus preceding inescapable foot shock, completely reverses the shock-induced suppression of lymphocytes. Consistent with the hypothesis that fear ultimately determines the amount of immunosuppression induced by aversive stimuli, a nonaversive stimuli associated with electric shock can be conditioned to impair lymphocyte proliferation independent of the direct effect of the physically aversive stimulation ([Lysle et al., 1988, 1992](#)).

In a study of nonhuman primates, [Cohen and colleagues \(1997\)](#) found that lower social status was associated with increased risk of infection following exposure to an



adenovirus. They also found that lower social status was associated with lower body weight, less aggressive behavior, and greater cortisol responses to social change stress.

### Immunologic Consequences of Early Life Experiences

Infancy has been suggested to be a period of life during which the organism is particularly sensitive to psychological perturbation, and stressful experiences during this period may have long-term physiologic impact. Early life experiences such as premature maternal separation produce alterations in many processes regulated by the hypothalamus, including body temperature, sleep, metabolic rate, and maturation of autonomic processes ( [Ackerman et al., 1979](#); [Hofer, 1984](#)). There is now evidence that the physiologic effects of these experiences also include changes in immune function.

Because of their social nature, nonhuman primates have been the subjects of many studies involving the effects of psychological stress on immune responses in young animals ( [Coe, 1993](#)). One of the earliest studies reported that lymphocyte proliferation responses to stimulation with Con A and PHA were reduced in two pairs of infant pigtail macaques undergoing separation from their peers ( [Reite et al., 1981](#)). This effect was later reproduced in infant bonnet macaques briefly separated from their mothers ( [Laudenslager et al., 1982](#)). In both of these studies, lymphocyte responses were reduced for the 2-week period of separation and returned to their original levels following reunion.

Since that time, numerous experiments involving multiple species of New and Old World nonhuman primates have replicated these early findings and extended them to other measures of immunity, including lymphocyte subset numbers, levels of complement proteins and immunoglobulins, macrophage chemiluminescence, and DTH responses ( [Boccia et al., 1989](#); [Coe et al., 1988a,b](#); [Friedman et al., 1991](#); [Gordon et al., 1992](#); [Gust et al., 1992](#); [Laudenslager et al., 1990](#)). Immune responses and cell numbers have proven to be sensitive to psychosocial stressors ranging from mother–infant separation to the formation of novel social groups.

Some of these studies also have shown, however, that animals' physiologic responses to these stressors are influenced by a number of psychological factors. The largest stress-related differences in immune function are typically found in the monkeys that are observed to have the most severe behavioral responses to both experimental ( [Laudenslager et al., 1990](#)) and natural stressors ( [Laudenslager et al., 1993](#)). The magnitude of change in immune function following psychosocial stress also depends on several factors, including the time at which immunity is measured, the environment in which the animal experiences the stressor, and the age of the animal at the time of the stressor ( [Coe, 1993](#)). Stress-related reductions in antibody responses to a bacteriophage, for example, are attenuated in monkeys housed with familiar, but not unfamiliar, companions or in a familiar location for the duration of the separation ( [Coe, 1993](#)).

Longitudinal assessments of immunity have revealed that early life experiences like those described in the preceding may have long-term immunologic consequences. Pigtail monkeys briefly separated from their mothers during the first year of life showed greater reductions in lymphocyte proliferation responses as adults than control monkeys ( [Laudenslager et al., 1985](#)). In 40-day-old rats separated from their mothers on postnatal day 15, lymphocyte responses are lower than responses in rats that remained with their mothers until day 22 ( [Ackerman et al., 1988](#)). Mice that were maternally deprived and weaned at 15 days postpartum show a depressed antibody response to sheep erythrocyte immunization at 7 and 8 weeks of age, but no differences in body or adrenal weight. Finally, preliminary evidence suggests that rats prematurely separated 100 days after weaning may show greater susceptibility to severe respiratory infection ( [Schleifer et al., 1986](#)).

Several reports have provided additional data that tumor growth in the adult animal is affected by early life experience. Daily handling of mice prior to weaning leads to increased rates of mortality following implantation of a lymphoid leukemia ( [Levine and Cohen, 1959](#)), and handling of rats during the first 3 weeks of life affects the growth of a carcinosarcoma ( [Ader and Friedman, 1965](#)).

More chronic early life experiences, such as rearing environment, have been shown to exert long-term influence on the immune system. Infant rhesus monkeys reared in a nursery by human hands for the first 6 months of life showed greater lymphocyte responses to stimulation with Con A than monkeys reared with their mothers ( [Coe et al., 1989](#)). These differences persisted through 2.5 years of age, despite that fact that both groups of monkeys were housed with peers after their first year ( [Coe et al., 1989](#)). The augmented lymphocyte responses, which contrast with the reduced responses typically reported following acute stress, may be the product of an abnormally high T-helper to T-suppressor cell ratio in nursery-reared monkeys (Lubach et al., 1994).

New evidence suggests that early vulnerability to environmental stressors may begin before birth. Rhesus monkey neonates whose mothers experienced psychosocial stress or ACTH treatment during pregnancy exhibited abnormal postnatal development of neuromotor reflexes ( [Schneider et al., 1992](#); [Schneider and Coe, 1993](#)). Ongoing studies indicate that these monkeys also exhibit reduced lymphocyte proliferation responses in mixed lymphocyte reactions, reduced cytotoxic responses, and altered lymphocyte b-adrenergic receptor expression ( [Coe, 1993](#)).

## STRESS AND IMMUNITY: CLINICAL STUDIES

### Limitations of Current Studies

Research has suggested that stress leads to altered immunity and may lead to increased susceptibility to disease ( [Glaser et al., 1999](#); [Rozlog et al., 1999](#)). No study in humans has yet delineated a causal chain showing that severe life stress or a particular psychological state produced an immunologic response that then resulted in an altered clinical outcome. In one of the few prospective studies in this area, [Cohen and associates \(1991\)](#) recently showed that psychological stress was associated in a dose–response manner with an increased risk of acute infectious respiratory illness. Importantly, this association was owing to increased rates of infection rather than an increased frequency of symptoms after infection; however, measures of cellular immune function were not obtained in this study.

To understand the relationship between stress and disease, all the processes involved in an “X” to “Y” to “Z” model must be studied. Some studies, like that of [Cohen and colleagues \(1997, 1998\)](#) have shown that life events and psychological processes influence the development of disease, whereas other studies have demonstrated that psychological processes result in immune changes. Neither is sufficient because these psychosocial factors could produce independent and unrelated effects both on immune function and disease states ( [Ader, 1987](#)). Prospective studies are needed that examine all these links simultaneously ( [Braveman, 1987](#)). Furthermore, *in vitro* immune measures may not accurately reflect immunologic competence *in vivo* and, thus, may not be useful predictors of increased illness susceptibility ( [Calabrese et al., 1987](#); [Hood et al., 1985](#)). For example, increased numbers might compensate a decrease in one immune parameter such as NK activity or function of other cell types with no resultant increased risk for acute viral infections. Despite these caveats regarding biological relevance, changes in the immune system in persons undergoing stress may have clinical implications and the hypothesis is presented that immunologic alterations mediate the relationship between stressful life events and poor health outcome.

### MORBIDITY AND MORTALITY IN BEREAVEMENT

In the past 20 years, studies of conjugal bereavement have demonstrated an elevated mortality rate among bereaved spouses that varies with age, sex, and time elapsed after the loss ( [Bowling and Benjamin, 1985](#); [Clayton, 1974](#); [Cox and Ford, 1964](#); [Ekblom, 1963](#); [Kaprio et al., 1987](#); [Kraus and Lilienfeld, 1959](#); [Mellstrom et al., 1982](#); [Parkes et al., 1969](#); [Rees and Lutkins, 1967](#); [Ward, 1976](#); [Young et al., 1963](#)). The results are heterogeneous, although there appears to be an increased mortality during the early periods succeeding the loss, particularly among the middle-aged to older widowers ( [Helsing et al., 1981](#)). Besides an increase in mortality, widowhood is associated with the onset of specific immune related medical disorders such as influenza and pneumonia, as well as other diseases including cardiovascular problems ( [Jacobs and Ostfeld, 1977](#); [Klerman and Izen, 1977](#); [Parkes, 1970](#)), possible increases in physician consultations and hospitalizations ( [Maddison and Viola, 1968](#); [Mor et al., 1986](#); [Parkes, 1964](#)), and decreases in general well-being ( [Maddison and Viola, 1968](#); [Parkes, 1970](#)).

### Depression and Illness

#### ADULT STUDIES

Both the onset and course of medical illness may be affected by stress ( [Keller et al., 2000](#)). In a study of 42 patients admitted to a general medical service, 31 of these patients had experienced the onset of their illness within 1 week of a significant loss ( [Schmale and Iker, 1966](#)). In addition, available data suggest an association between depressive profiles and the development of cancer ( [Kielcolt-Glaser and Glaser, 1999](#); [Spiegel, 1996](#)). In 40 women hospitalized for cone biopsies having class III Pap smears, feelings of hopelessness identified at patient interview correctly classified eight of the 14 women with cancer and 23 of the 26 women without cancer ( [Schmale and Iker, 1966](#)). In a 10-year longitudinal study, feelings of hopelessness and depression at baseline are associated with an increased incidence of cancer among 1,353 persons examined in a Yugoslav village ( [Grossarth-Maticcek et al., 1985](#)). Finally, the Western Electric Study has demonstrated, with 20 years of follow-up, an association between depression as measured by the *Minnesota Multiphasic Personality Inventory* and an increased incidence in mortality for cancer

([Persky et al., 1987](#)). Even after adjustment for age, number of cigarettes smoked, alcohol intake, occupational status, and family history of cancer, the relationship between depressive personality and cancer persisted. Conversely, one study found that depressive symptoms are not significant predictors of cancer morbidity with or without adjustment for age, sex, marital status, smoking, family history of cancer, and hypertension ([Zonderman et al., 1989](#)).

In general, the findings of increased cancer incidence in clinically depressed patients are consistent with a "null or weak relationship" ([Fox, 1989](#)). Nevertheless, several follow-up studies have demonstrated a relationship between affective disorders and clinical depression and increased cancer morbidity and mortality, particularly in male patients over age 40 who had primary diagnoses of affective disorders ([Kerr et al., 1969](#); [Varsamis et al., 1972](#); [Whitlock and Sisking, 1979](#)).

PNI investigations have also suggested that psychiatric interventions that reduce psychological distress may have beneficial effects on cancer survival ([Sellick and Crooks, 1999](#)). [Spiegel and coworkers \(1989\)](#) found that group psychotherapy of breast cancer patients improved quality of life and extended life expectancy for patients with metastatic breast cancer. [Fawzy and colleagues \(1990, 1993\)](#) showed that other types of cancer patients improve both psychologically as well as immunologically with psychosocial group therapy. Additionally, in a study of lymphoma, leukemia patients, [Richardson and coworkers \(1990\)](#) found similar quality and quantity of life enhancements.

#### CHILD STUDIES

The relationship between life events and occurrence of medical disease in children has been less extensively studied than that in adults. However, the onset and course of several immune-related diseases have been linked to stressful life experiences. Bronchial asthma and allergic rhinitis, two disorders thought to be mediated by humoral immune responses, occur more frequently in children undergoing life events who have limited abilities to cope psychologically with these changes ([Czubalski and Zawisza, 1976](#); [DeAraujo et al., 1973](#); [Holmes et al., 1951](#); [Knapp, 1969](#)). Streptococcal infections and increased antibodies to streptolysin-O are more likely in members of families in which high levels of stress are noted ([Meyer and Haggerty, 1962](#)). Serious, and often fatal, viral infections have been found in emotionally deprived children ([Spitz, 1965](#)).

One study in children examined prospectively the relationships between stress, immunologic change, and respiratory illness in children. In this study ([Boyce et al., 1993](#)) healthy children were studied immunologically before and after starting kindergarten. During the course of the study, an earthquake struck the area, adding a second stressor to the study. The data showed that children differed in their response to entering kindergarten with respect to changes in immune parameters. However, more importantly, those children who tended to have increases in immune measures (CD4+/CD8+, response to PWM) following entrance into kindergarten also had higher numbers of respiratory infections following the earthquake.

The role that psychological and emotional factors play in increasing the susceptibility of youngsters to medical illnesses including cancers is not yet known, although [Greene and Miller \(1958\)](#) and [Jacobs and Charles \(1980\)](#) have conducted investigations of the relationship of such factors to the onset of leukemia and lymphoma in children and adolescents. Experiences of separation and loss appear to be one of multiple factors that are associated with the development of leukemia in children. For example, of 33 patients with lymphocytic and myelogenous leukemia, 31 are found to have experienced one or more losses or separations in the 2-year period prior to the onset of their illness, with half of these experiences occurring in the 6 months prior to that time ([Greene and Miller, 1958](#)). Further, [Drummond and Hewson-Bower \(1997\)](#) have reported that stress in children was associated with a history of recurrent colds and flu and lower salivary IgA.

#### Life Events and Immune Changes

Although there has often been disagreement about the existence of changes in immunity related to life stressors, a recent metaanalysis of the relationship between stress and immunity concluded that there is substantial evidence for stress-induced modulation of the numbers and functional capacities of multiple immune cell types ([Herbert and Cohen, 1993b](#)). Important variables influencing immunity included stressor duration and the extent to which the stressor involved social (e.g., loss of spouse) rather than nonsocial (e.g., loss of job) events ([Herbert and Cohen, 1993b](#)). A more detailed consideration of the types of stressor that have been linked to altered immunity follows.

#### Clinical Studies of Bereavement

Clinical studies of immune function in bereaved subjects have demonstrated altered immunity, including suppression of lymphocyte responses to mitogenic stimulation, reduced NKCA, and alterations of T-cell subpopulations. The relationship between bereavement and immunologic functioning was first reported by [Bartrop and colleagues \(1977\)](#) who compared measures of lymphocyte responses to mitogen stimulation between 26 men and women whose spouses had died and 26 age- and sex-matched control subjects. At both 3 and 6 weeks after the death of the spouses, during which time active bereavement occurs, T-cell responses to low doses of PHA and Con A are reduced in the bereaved group. T- and B-cell numbers do not differ between groups. Using several specific antigens (streptokinase, mumps antigen, and purified protein derivative of tuberculin), delayed hypersensitivity reactions on routine skin testing are also not different. In a prospective study, [Schleifer and colleagues \(1983\)](#) replicated the earlier findings, demonstrating a significant suppression of lymphocyte responses during the first 2 months following the death of a spouse and an intermediate level of mitogen responsivity during the 4- to 14-month period after bereavement.

Other *in vitro* parameters of cell-mediated immune function are also found to be altered during bereavement. Measures of NK cell cytotoxicity, total lymphocyte counts, and T-helper and T-suppressor cell numbers were compared among three groups of women: those whose husbands were dying of lung cancer, those whose husbands had recently died, and women whose husbands were in good health ([Irwin et al., 1987a,b](#)). In the groups who were undergoing moderate or severe life stress (comprised significantly by women anticipating or bereaving the death of their spouse), a reduction of NKCA is found as compared to the values in the low stress, nonbereaved comparison subjects. Neither the absolute number of lymphocytes nor T-cell subpopulations including number of T-helper, T-suppressor/cytotoxic cells and ratio of T-helper to T-suppressor/cytotoxic cell is different between the groups.

Psychological responses, not merely the event, might contribute to the immune changes in bereavement. Consistent with this hypothesis, severity of depressive symptoms is correlated with reduced NKCA, a loss of T-suppressor/cytotoxic cells, and an increase in the ratio of T-helper to T-suppressor/cytotoxic cells ([Irwin et al., 1987a](#)). Using various measures of humoral and cell-mediated immunity, [Linn and colleagues \(1984\)](#) found reduced lymphocyte responses to the mitogen PHA only in bereaved subjects who had high depression scores but not in those bereaved subjects who had few signs of depression. Other immunologic measures, including lymphocyte responses to Con A and pokeweed mitogen, serum levels of IgG, IgA, and IgM, *in vivo* responses to skin tests, and neutrophilic chemotaxis were comparable to those found in the controls.

Finally, in a study of depression in bereaved women ([Zisook et al., 1994](#)) only that subset of bereaved women who also met criteria for major depressive disorder (DSMIII-R) were found to have altered immunity, specifically lower NKCA and lower responses to mitogen stimulation.

#### Effects of Examination Stress

Relatively minor aversive events such as the stress of academic examinations are also associated with altered immunity. [Dorian and associates \(1982\)](#) reported higher white blood cell counts, an increased absolute lymphocyte count, a reduction in PHA reactivity, and a reduction in plaque forming cell response (an index of B-cell function) in eight psychiatry trainees taking their oral examinations as compared to the values in 16 psychiatrists not taking examinations. In a series of other studies conducted by [Kiecolt-Glaser and colleagues \(1984\)](#), the initial observations of examination-related alterations of cellular immunity have been confirmed and extended. Medical students demonstrate significantly lower levels of NKCA and blastogenesis (in response to both Con A and PHA) during final examinations as compared to baseline samples taken 1 month earlier ([Kiecolt-Glaser et al., 1984](#)). Additionally, the students exhibit stress-related decreases in the percentages of total T lymphocytes, helper T lymphocytes, and suppressor T cells ([Glaser et al., 1985](#)) and significant increases in plasma levels of IgG, IgM, and IgA ([Glaser et al., 1986](#)). Moreover, these researchers have speculated that these immunologic changes may be important for the control of recurrent latent herpes virus infections and found that antibody titers to Epstein-Barr virus (EBV), CMV, and herpes simplex virus (HSV) are significantly increased during the first day of examination as compared to either the month before examination or on return from summer vacation ([Kiecolt-Glaser et al., 1984](#)). The same stressor was also associated with significant decreases in the number and synthesis of IL-2 receptors on peripheral blood leukocytes and with increased release of IL-2 by these cells ([Glaser et al., 1990](#)).

In a more recent study, [Borella and colleagues \(1999\)](#) studied military cadets in Italy both during academic stress (examinations) and in periods of no particular academic stress. Their results suggest that examination stress per se did not result in altered NKCA in the group as a whole. Rather, they found that emotionally stable (not anxious) cadets showed increased NKCA at times of academic stress, whereas cadets who were less emotionally stable (more anxious) had decreased



NKCA compared to baseline.

## Coping and Social Support

Style of coping and quality of social support are postulated as determinants of whether severe adversities such as bereavement or caregiving become translated into psychological and physical morbidity. In this regard, discord or dissatisfaction within the marital relationship are found to have effects on immune function ([Kiecolt-Glaser et al., 1987](#)); poor marital quality is associated with reduced immunologic functioning (lower percentages of NK and helper cells, poor mitogen responsiveness, and higher antibodies to some herpes viruses) among married women ([Kiecolt-Glaser et al., 1987](#)) and married men ([Kiecolt-Glaser et al., 1988](#)).

Despite a demonstrated link between coping and psychological adjustment during stress, few studies have evaluated the role of coping responses as defined by [Lazarus and Folkman \(1984\)](#) in mediating stress-induced alterations in immune function. Instead, most studies have evaluated coping responses indirectly, postulating that a diminished capacity to adapt, as indicated by psychiatric symptoms, is a measure of coping. For example, [Locke and coworkers \(1984\)](#) rated the severity of life events in 114 students and on the basis of these reports and measures of psychiatric symptoms divided the sample into four groups; low stress/low symptoms, high stress/high symptoms, high stress/low symptoms, and low stress/high symptoms. Those subjects with few psychologic symptoms in the face of large amounts of stress ("good copers") have significantly higher NKCA than those experiencing high levels of both symptoms and stress. Temoshok (1986) has also found that one factor influencing survival among AIDS patients is the degree of control they experienced over their lives; those who felt they could exert little control died more quickly. Finally, consistent with the findings generated in preclinical studies, administration of an uncontrollable (but not controllable) stress produces greater elevations in the plasma concentration of ACTH and epinephrine in humans ([Breier et al., 1987](#)).

## Chronic Stress and Immune Dysfunction

In addition to the effects of acutely occurring severe, as well as relatively minor, aversive events, chronic stressors that last over periods of one or more years have also been found to result in alterations in immune function. The stresses of Alzheimer's disease caregiving often place family members at risk for depression ([Eisdorfer et al., 1983](#)) and are associated with decreased life satisfaction ([George and Gwyther, 1986](#)) and increased levels of psychiatric symptoms ([George and Gwyther, 1986](#)). Furthermore, immunologic analyses have revealed significantly higher antibody titers to EBV and reduced percentages of total T lymphocytes and helper T cells in 34 Alzheimer caregivers versus 34 matched controls ([Kiecolt-Glaser et al., 1987](#)). Neither T-suppressor cells nor numbers of NK cells differed between the two groups. In another study, [Mills and colleagues \(1997\)](#) found that chronic stress in Alzheimer caregivers was associated with altered lymphocyte B<sub>2</sub>-adrenergic receptors. These changes in immunity may have health consequences, as Alzheimer caregiving has also been associated with significant increases in episodes of infectious illness and illness-associated physician visits ([Kiecolt-Glaser et al., 1991](#)).

## Stress and Immunity in Children

Two studies have reported an association between stress and immunity in children and adolescents. In a study by [Birmaher and coworkers \(1994\)](#) subjects with MDD or conduct disorder and healthy controls (ages 11 to 18 years, Tanner III to IV) were studied. Both lifetime and past year measures of adverse life events for the entire sample was significantly and inversely correlated with NKCA, meaning greater numbers of adverse events were predictive of lower NK activity for all the adolescents studied. In a study of 11 prepubescent (ages 8 to 12 years, Tanner I) subjects with major depression versus 11 normal matched controls, parental divorce or separation was associated with lower granulocyte killing of *staphylococcus aureus*, whereas major depression was associated with greater NKCA ([Bartlett et al., 1997](#)). No other studies of psychosocial stress and immunity have been reported in children and very few studies of stress in humans address myeloid cell function; however, physiologic stress has been reported to produce altered granulocyte function in children ([Krause et al., 1986](#); [Stroobant et al., 1984](#); [Tolone et al., 1989](#)).

## Depression and Immune Changes

Psychological response to distressing life events is an important correlate of immune dysfunction in persons undergoing severe life stress. Further, stress has long been considered a risk factor for the development of anxiety or depressive disorders with the possibility that stress-related immune changes play a role in the development of these disorders ([Leonard and Song, 1996](#)). This and the clinical similarities of bereavement and depression have led to the hypotheses that depression or anxiety may be associated with immune change. [Herbert and Cohen \(1993a\)](#) conducted a metaanalysis of studies of depression and immunity and concluded that depression is indeed significantly related to impaired cellular immunity with severity of depressive symptoms associated with greater decreases in immune cells numbers and activity ([Herbert and Cohen, 1993a](#)).

To clarify the role of depression in impairing cell-mediated immunity, several studies have compared immune responses between depressed patients and control subjects. [Cappel and associates \(1978\)](#) reported that PHA responses are lower in psychotically depressed patients during the first days of illness than following clinical remission. [Kronfol and colleagues \(1983\)](#) replicated these observations in 26 drug-free depressed patients and found blunted lymphocyte responses to mitogenic stimulation with Con A, PHA, and pokeweed during depression. [Schleifer and associates \(1984\)](#) also found suppressed lymphocyte reactivity in severely depressed patients and further described abnormalities of lymphocyte subpopulations in depression: absolute and T- and B-lymphocyte cell counts are reduced, although relative percentages are unchanged. Because no differences in immune measures have been found in mildly depressed outpatients as compared to controls, Schleifer and colleagues have concluded that the severity of depressive symptoms is an important factor associated with altered lymphocyte responses in depression ([1985](#)).

Extending these observations of altered lymphocyte proliferation responses in depression, [Irwin and colleagues \(1987b\)](#) measured the cytolytic activity of peripheral lymphocytes in two groups of subjects: Medication-free, hospitalized acutely depressed patients and age- and sex-matched control subjects studied on the same day as the patients. NKCA was significantly lower in the depressed patients as compared to the control subjects, a finding that has been replicated by [Urch and colleagues \(1988\)](#) and [Mohr and colleagues \(1987\)](#). In addition, severity of depressive symptoms was correlated with a reduction in NKCA.

In an attempt to understand why some (but apparently not all) patients with major depression show immune changes, the contribution of other factors that might affect immunity in depressed patients has been studied. [Schleifer and colleagues \(1989\)](#) have examined the role of age in the relationship between depression and altered immunity. Employing an extensive assessment of the immune system including enumeration of T-lymphocyte subsets, assay of NKCA, and measurement of mitogen-induced lymphocyte stimulation, significant age-related differences between the depressed patients and control are found for numbers of T-helper lymphocytes and mitogen responses. Age-related increases in T-helper cells and in mitogen responses are found in the controls, whereas advancing age is associated with no changes in T-helper number and decreased lymphocyte responses in depressed patients. In a 1996 study of young adults (ages 18 to 35) with unipolar depression, 21 subjects were compared to 21 healthy matched controls ([Schleifer et al., 1996](#)). In these ambulatory patients, depression was related to increased total WBC and increased circulating granulocytes but lower NK cell counts. When NK numbers were controlled in the analyses, subjects were also found to have lower NKCA; however, no changes in lymphocyte proliferative responses to PHA, Con A, or PWM were found. These findings suggest that age and severity of depression are important correlates of immune changes in depression; immune changes in major depressive disorder are present mainly in elderly, severely depressed patients.

In addition to the independent contribution of age to depression-related changes in immunity, alcohol consumption is also reported to play a role in further reducing cellular immunity in depressed patients. Alcohol use, even in moderate doses, is associated with alterations in cell-mediated immune function such as NKCA. The contribution of alcohol use to produce a greater decrement in immunity in depressives with histories of alcohol abuse as compared to depressed patients without such alcoholism has been compared ([Irwin et al., 1990](#)). Consistent with earlier reports, NKCA is significantly lower both in depressed and alcoholic patients (as compared to controls). Perhaps of more interest, patients with dual diagnoses of either alcohol abuse and secondary depression or depression with a history of alcohol abuse demonstrate a further decrease in NKCA as compared to that found in patients with either depression or alcoholism alone.

Alterations in T-cell subpopulations have also been characterized in depressed patients as compared to control subjects. Consistent with the findings of [Irwin and colleagues \(1987b\)](#), who found a relationship between severity of depressive symptoms and an increase in the ratio of T-helper to T-suppressor/cytotoxic cells in bereaved women, [Syvalahti and associates \(1985\)](#) found that depressed patients have a lower percentage of T-suppressor/cytotoxic cells and a higher ratio of T-helper to T-suppressor/cytotoxic cells than that of the control subjects. However, other studies have found no depression-related differences in quantitative measures of lymphocytes including number of T cells, B cells, T-helper, T-suppressor, and NK cells ([Darko et al., 1989](#); [Wahlin et al., 1984](#)).

NKCA alterations have also been reliably reported in depressed patients ([Irwin et al., 1987, 1990](#); [Stein et al., 1991](#)), as well as alterations in numbers of circulating NK cells ([Andreoli et al., 1993](#)), changes that appear to relate to age and gender, as well as depressive disorder ([Evans et al., 1992](#)). In a study by [Miller and colleagues \(1999\)](#) of 32 women with major depressive disorder, subjects were found to have reduced lymphocyte proliferative responses to both PHA and Con A.



However, NKCA was reduced in the older subjects but elevated in the younger subjects, suggesting age and gender play a role in alterations of both numbers and functional ability of immunocytes.

The apparent inconsistencies in psychoimmunologic investigations in humans have led to increasing sophisticated studies that have documented complexities of psychoimmunologic relationships. Confounding factors have been reported including diurnal variation in NK numbers and function in depressed patients ([Petitto et al., 1992](#)) and effects of sleep on NKCA in depression ([Irwin et al., 1992, 1994](#)). Further, the duration of depression has been found to have an age-independent effect on lymphocyte proliferative responses to PHA, PWM, and Con A and the length of time off antidepressant medication was correlated with numbers of circulating lymphocytes, T cells, and NK cells ([Andreoli et al., 1993](#)).

Few studies have investigated relationships between depression and immunity during the course of the disorder. In a study of 20 male patients with MDD and 20 matched controls, [Irwin and coworkers \(1992\)](#) found that the severity of depressive symptoms and NKCA were positively correlated over time. In a follow-up study of the young adults with unipolar depression ([Schleifer et al., 1999](#)) involving 6 weeks of treatment with nortriptyline and alprazolam, clinical improvements in severity of depressive symptoms was associated with decreased numbers of circulating lymphocytes. In addition, decreases in T cells, CD4+ and CD29+ were found; there were no changes in B cell numbers or CD8+ cells. Finally, patients also had decreased responses to PHA and Con A but not PWM. None of these changes were related to nortriptyline blood levels ([Schleifer et al., 1999](#)).

Beyond the use of more sophisticated measures of immune cell function, future investigations need to test the clinical relevance and biological significance of depression-related changes in the immune system. Furthermore, preclinical and clinical studies of the mechanisms that underlie changes in depression in such biological parameters as measures of immunity will help identify the pathways of communication among the nervous, endocrine, and immune systems.

### Depression and Immunity in Children

Only three studies have been published to date concerning immunity in children and/or adolescents with depression. [Shain and colleagues \(1991\)](#) investigated NKCA in 16 hospitalized adolescents (ages 13 to 18) with major depressive disorder and 16 age- and gender-matched controls. Although they found no between group differences in NKCA, they did find both significant age effects and a significant correlation between severity of depressive symptoms (as measured by the Reynolds Adolescent Depression Scale) and NKCA with greater severity of symptoms associated with lower levels of activity.

In [1994](#), Birmaher and colleagues reported a study comparing 20 subjects with major depressive disorder, 17 subjects with conduct disorder (CD), and 20 normal healthy controls (ages 11 to 18, post thelarche/adrenarche and Tanner stage III to IV). Many of the subjects with psychiatric disorders had comorbid conditions or different subtypes of depression. They found no between group differences in WBC counts or number or percentages of lymphocytes, T cells, or NK cells; however, B cells were increased in CD subjects as compared to controls and MDD subjects. No between-group differences were found in response to PHA stimulation, although CD subjects had lower NK activity (trend) compared to both MDD subjects and controls. In a study of immunity in prepubescent depression, [Bartlett and coworkers \(1995\)](#) investigated 20 children with MDD (Tanner stage I) and 20 healthy matched controls. The depressed children had lower NKCA and elevated response to Con A but not PHA or PWM compared to their healthy controls.

### Anxiety and Immunity

Immune changes associated with anxiety disorders including panic disorder and posttraumatic stress disorder (PTSD) have recently been under investigation. [Koh and colleagues \(1998\)](#) reported decreased lymphocyte proliferative responses to PHA in 31 patients with anxiety disorders compared to 31 matched controls. They also found that patients with anxiety disorders had diminished IL-2 production. In a study of PTSD, following a hurricane, [Ironson and coworkers \(1997\)](#) found that NKCA was related to the symptoms of PTSD with more symptoms predicting lower NKCA. The subjects who had lived through the hurricane, compared to laboratory controls, had lower numbers of circulating CD4+ and CD8+ cells and higher numbers of NK cells, as well as lower NKCA. Further, they found that new sleep disturbances mediated the relationship between NKCA and PTSD. [Boscarino and colleagues \(1999\)](#) also investigated PTSD, comparing Vietnam veterans with PTSD, to veterans with no psychiatric disorder. The veterans with PTSD had elevated leucocyte counts as well as elevated CD4+ counts.

### Behavioral/Psychiatric Symptoms Associated with Immune Phenomena

The communications between the brain and the immune system are now understood to involve bidirectional messaging ([Husband, 1993](#); [Lawrence and Kim, 2000](#); [Maier and Watkins, 1998](#); [Solomon, 1987](#)). Both immune cells and immunocyte products (cytokines) cross- and/or are actively transported across the blood-brain barrier with influences on CNS activity ([Watkins and Maier, 1995](#)). Not only are brain, behavioral, and psychological factors thought to influence immune function, but also immune factors are thought to affect brain, behavior, and psychological status.

Cytokines are reported to have behavioral and neuropsychiatric effects in addition to their role in regulating immune responses as reviewed by [Dantzer and Kelley \(1989\)](#). IL-1, IL-6, and TNF- $\alpha$  are reported to produce a group of symptoms referred to in animal studies as sickness behavior ([Kent et al., 1992a,b](#)) including symptoms of social withdrawal, decreased energy, increased sleep, and poor appetite. IL-6 has also been reported to produce diminished concentration and negatively impact mood as well as other alterations in sleep patterns (e.g., diminished time in rapid eye movement sleep) ([Spath-Schwalbe et al., 1998](#)).

There is a growing literature devoted to the impact of the immune response and soluble immune response modifiers on brain function and behavior. Interleukin-1 is an immune response modifier that has received a large amount of attention because of its pleiotropic physiologic and behavioral effects. This cytokine, when introduced into the brain, induces slow wave sleep ([Krueger and Obal, 1993](#)) increased release of hypothalamic CRH ([Sapolsky et al., 1987](#)), and increased circulating concentrations of ACTH ([Vale et al., 1981](#)). Because IL-1 plays a crucial role in regulating a number of immunologic responses [Dinarello and Mier \(1987\)](#), [Sundar and associates \(1990\)](#), and [Brown and colleagues \(1991\)](#) have investigated whether central administration of IL-1 would affect immunity, and both demonstrated that central doses of IL-1 (3.1 to 12.4 fmol) rapidly reduce *ex vivo* cellular immune responses such as NK cell activity, lymphocyte proliferative response to PHA, IL-2 production, and macrophage IL-1 secretion. Importantly, the effects of central IL-1 on immune function are blocked by the administration of an antibody to CRH, indicating that IL-1b likely mediates immunosuppression via central release of CRH ([Saperstein et al., 1992](#); [Sundar et al., 1989](#)). Consequently, the pathways by which IL-1 regulates peripheral immune function are similar to mechanisms involved in central CRH suppression of immunity. Administration of a ganglionic blocker abrogates the effects of IL-1 ([Sundar et al., 1990](#)), similar to observations that autonomic- and sympathetic nervous mechanisms mediate CRH-induced suppression of NK activity ([Irwin et al., 1988, 1990](#)).

Interferon has been used in treatment of illnesses such as cancer and neurobehavioral consequences of these treatments have been reported. Interferon- $\alpha$  has effects on both mood and cognition ([Valentine et al., 1998](#)), including reduced alertness, increased depression, impaired memory, and lack of initiative.

Less specific immune-neuropsychiatric relationships have also been reported. Perhaps the most notable childhood disorder is that of pediatric autoimmune neuropsychiatric disorders associated with streptococcal infection (PANDAS). Although first reported in 1894 by Osler, PANDAS was not much investigated until the late 1980s. PANDAS consist of obsessive-compulsive and/or tic (motor or vocal) symptoms that have a waxing and waning course related to infection with group B hemolytic streptococcal infection. Abnormal neurologic findings and elevated antistreptolysin titers often accompany exacerbations of symptoms. By definition, it has a prepubescent onset that is often abrupt. Symptoms may remit or disappear entirely between episodes. Antineuronal antibodies may be present. Some symptom exacerbations may follow viral rather than streptococcal infection ([Swedo et al., 1998](#)).

## CONCLUSION

The complexities of interactions among brain, behavior, immunity, and health have been noted for centuries ([Leonard and Song, 1999](#)), but we are just beginning to understand some of the specifics of the interactions involved. Animal and adult human studies have advanced our knowledge but few actual investigations have been undertaken in children for whom psychoneuroimmunologic interactions may potentially affect their well being for their entire lifetime. Investigations of these interactions in children and adolescents are currently underway and will provide much valuable information.

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## 7 DEVELOPMENTAL PSYCHONEUROENDOCRINOLOGY

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The unique biologic and behavioral characteristics of newborns, infants, and children in different stages of development imply important differences between adult neuroendocrine processes and those occurring during development. Not only is the developing neuroendocrine system functioning and capable of dynamic responses to the changing environment, but also in the early stages of development, it can undergo long-lasting and permanent changes as a result of these early influences. Clear understanding of the developmental aspects of psychoneuroendocrinology would require investigations at the embryologic, fetal, neonatal, and later developmental stages and at the molecular, cellular, and architectural levels of organization in the anatomic, neurochemical, neurophysiologic, behavioral, and clinical aspects of the brain and endocrine system. This may seem like a mammoth undertaking but is very likely to be extremely informative.

In this chapter, we review the current literature on neuroendocrinology and childhood behavior and ask the question: How do these neuronal cell populations, pathways, neurotransmitters, neuroregulators, and other physiologic mechanisms develop and interact with the developing endocrine system? This approach carries the basic assumption that the same anatomic and physiologic processes, albeit immature, are used in both the developing brain and endocrine system as those utilized in the adult organism. As a result, our purpose may be reduced to investigating how such systems function in the immature state and during different stages of development; however, this approach is likely fraught with several difficulties.

First, the presently accepted view of neuroendocrine functions may be incorrect and incomplete, and searching for their ontogeny may lead to a compounding of these errors. Second, it is entirely possible, and perhaps likely, that the fetus and neonate use neuroendocrine mechanisms that are vastly different from those in the adult. Their repertoire of behavior and responses to stress are certainly different, as are the characteristics of the stressors that elicit neuroendocrine responses. Third, neuronal cell populations, connecting pathways, neurotransmitters, and other physiologic mechanisms may exist transiently during development and be lost in the adult brain and endocrine system. Some neuroendocrine mechanisms that have limited roles in the adult may indeed have sweeping and pluripotent roles in altering the development of the neuroendocrine system and secondarily altering cognitive processes and behavior during childhood. Last, the concept of “critical windows of development” does not apply to the mature brain, whereby short-lived or slight changes in cellular processing or molecular signaling may have drastic or wide-ranging effects during development by altering critical steps that have a “permissive” effect on subsequent developmental events. Many such examples are described in the ensuing sections whereby an environmental insult during a critical period can have persistent or even permanent effects on the future functioning of a neuroendocrine system, and consequently, on the health and behavior of the affected subject (Anand and Scalzo, 2000). This highly complex and vulnerable system generates enormous possibilities for altered and abnormal development or persistently altered regulation, with important implications for the practice of child and adolescent psychiatry.

### NEUROENDOCRINE REGULATION OF BRAIN DEVELOPMENT

Developmental events in the central nervous system (CNS) are regulated by a variety of genetic mechanisms, ionic gradients, neuroendocrine factors, and paracrine influences. Neuroendocrine factors that control the migration, differentiation, and maturation of neuronal cell populations include thyroid hormones, glucocorticoids, androgens and estrogens, and several neuropeptides. Much of the research on neurobiological development and the basic mechanisms of psychoneuroendocrinology has been performed in rodent species, largely rats and mice. Apart from logistic reasons such as laboratory cost and convenience, short gestation, and simple breeding and rearing conditions, three other reasons contribute to this choice. First, the cellular, molecular, and architectural events in the development of the CNS follow remarkably similar patterns in the human and rodent species. Some of the relevant data illustrating this interspecies correlation are summarized in [Table 7.1](#). The rat brain at birth (P0) has a similar developmental maturity as the brain of a 24-week-gestation human premature infant, and at 10 to 14 days of age (P10 to P14) the rat brain corresponds to the brain of a full-term newborn infant (Bass et al., 1977; Eayrs, 1968). Second, the behavior of infant and adult rats has been investigated in much greater detail than that in any other species. The rich repertoire of cognitive and behavioral tests that have been validated and published allow meaningful correlations between neurobiological events and mechanisms, experimental paradigms, drug therapies, and the resulting (often quantifiable) behavioral changes. Third, the remarkable database on mouse, in contrast to rat, genetics makes the mouse the species of choice for studies of behavior genetics. A powerful combination of these investigative advantages makes rodents the primary choice for animal models of human psychiatric and behavioral disorders. Nevertheless, when extrapolating from experimental studies to clinical situations, we must account for major differences in the complexity and adaptability between human and rodent brains. Therefore, this chapter focuses much of the discussion on developmental aspects of psychoneuroendocrinology in rodent and human species, although studies involving nonhuman primates are reviewed as well.

Developmental feature	Rat	Human	Reference
Myelination of the spinal cord (Dorsal horns, etc.)	E16	8 weeks	Mart et al., 1987
	E17	8 weeks	Mart et al., 1987
Ingrowth of olfactory bulb	E14-E15	9-12 weeks	Chiodo et al., 1982
Substantia nigra neurogenesis	E15	50 weeks	Mart et al., 1987
			Nomura and Co., 1984
Termination of the spinal cord	E17	10 weeks	Roy et al., 1987
			Mart et al., 1987
Ret. movements	E16	7-8 weeks	Nakabayashi et al., 1971
Motoricore death	E15-E17	11-20 weeks	De Vries et al., 1982
			Nomura and Co., 1984
			Casperman, 1987
Neocortical neurogenesis	E18-E19	8-12 weeks	Mart et al., 1987
Increased neurotransmitter and receptor expression	E20-E21	20-30 weeks	Chen et al., 1984
			Levine, 1972
			Chiodo et al., 1984
Delayed autonomic reflexes	Up to P10	Up to 1 year	Rogers and Olson, 1984
			Pendergast and Shultman, 1982
Corticospinal tract maturation	P1-P2	1-2 years	Schwartz and Jones, 1982
			Pendergast and Shultman, 1982

**Table 7.1. Correlation of Rat and Human Brain Development**

## Thyroid Hormones

Thyroid hormones influence brain development in three phases during fetal and neonatal life. Phase I includes the influence of maternal thyroid hormones, which extends up to 17 days of gestation in the rat and 10 to 12 weeks gestation in the human. A significant proportion of brain stem and cerebral neurogenesis occurs during this period, and accumulating evidence has started to define the developmental role of thyroid hormones. Placental transport of thyroid hormones occurs in humans (Vulsma et al., 1989) and rats (Morreale de Escobar et al., 1990), with localization in the fetal brain as early as E12 in the rat (Porterfield and Hendrich, 1992) and 10 weeks gestation in the human fetus (Ferreiro et al., 1988). The fetal brain selectively accumulates thyroid hormones (triiodothyronine [ $T_3$ ] > thyroxine [ $T_4$ ]), and thyroid hormone receptors are present from E14 in the rat (Perez-Castillo et al., 1985) and the first trimester of human gestation (Bernal and Pekonen, 1984). The synthesis of transthyretin in the choroid plexus may regulate the transport of thyroxine across the blood-brain barrier (Schreiber et al., 1990), where it is selectively deiodinated to form  $T_3$ , primarily through the action of Type II iodothyronine 5 $\alpha$ -deiodinase (D2) (Koibuchi and Chin, 2000). D2 is found mainly in astrocytes, suggesting that  $T_4$  is taken up from capillaries by astrocytes, deiodinated to  $T_3$ , and transferred to neurons for direct interaction with the nuclear thyroid hormone receptor (Guadano-Ferrez et al., 1997).

Destruction of the fetal and maternal thyroid gland in midgestation results in decreased messenger ribonucleic acid (mRNA) and protein synthesis in the brain (Holt et al., 1973) with specific reductions in mRNAs for constitutive tubulin isotypes Mb5 and Ma1 (Stein et al., 1989). Treatment of pregnant rats from E7 with propylthiouracil was associated with delayed cell accumulation in mesencephalic and motor nuclei (Narayanan and Narayanan, 1985). These experimental observations were correlated with cognitive deficits and delayed motor skills in children exposed to maternal hypothyroidism in early gestation (Letarte and Garagorri, 1989; Rovet, 1986).

During phase II (E17 to E21 in rats, 12 to 40 weeks gestation in humans), the fetal and maternal thyroid glands are active sources of thyroid hormones. The partial neurogenesis, neuronal migration, neurite formation, and synaptogenesis occurring in this period are exquisitely sensitive to the presence of thyroid hormones. Potter and colleagues (1986) found that reduced brain growth caused by maternal hypothyroidism was restored to normal in sheep fetuses after the onset of fetal thyroid function. Maternal hypothyroidism may alter fetal brain development by nonspecific mechanisms such as altered maternal metabolism and compromised nutrient availability, decreased placental growth, circulation, or function, or indirectly, by altering the regulation of trophic hormones (growth hormone [GH], insulin-like growth factor-1 [IGF-1], IGF-II). These specific effects are mediated directly by fetal or neonatal thyroid hormones on the growth and maturation of neuronal cell populations (Porterfield and Hendrich, 1993).

In phase III, which follows birth, brain development is entirely dependent on the activity of the neonatal thyroid gland. The small amounts of thyroid hormones present in human breast milk are insufficient to support normal brain development (Mallol et al., 1982), although breast milk may serve as a significant source of iodine because of the selective uptake of iodine from maternal plasma (Tazebay et al., 2000). Hypothyroid states in phase III mostly affect neuronal maturation and development in the forebrain and cerebellum, together with proliferation and maturation of glial cells that cause myelination throughout the nervous system. This critical period extends from birth to P21 in the rat and up to 2 years in the human, when thyroid deficiencies can cause serious and permanent brain damage. Several lines of evidence highlight the importance of thyroid hormones in the regulation of protein synthesis in neuronal and glial cells (Dussault and Ruel, 1987). Thyroid hormones function as a time clock in phase III, terminating neuronal proliferation and stimulating differentiation, particularly in the rat cerebellum. Triiodothyronine appears to regulate the expression of the genes that encode growth factors, including the neurotrophins (e.g., NT-3) and brain derived neurotrophic factor (BDNF) (Koibuchi, 1999; Koibuchi and Chin, 1999; Lindholm et al., 1993; Neveu and Arenas, 1996), and other crucial peptides including myelin basic protein, neural cell adhesion molecule and reelin (Koibuchi and Chin, 2000).  $T_3$  may also regulate the expression of genes encoding other transcription factors such as hr, Krox 24, and Rora (Koibuchi and Chin, 2000). Myelination of central tracts by oligodendrocytes is preceded by the transient expression of nuclear  $T_3$ -receptors, which are absent before the critical period of active myelination (Besnard et al., 1994).  $T_4$  is also involved in brain development; it recently has been shown to regulate the extracellular deposition of laminin on astrocytes (Farwell and Dubord-Tomasetti, 1999). Laminin provides guidance signals for migrating neurons in the rat cerebellum (Farwell and Dubord-Tomasetti, 1999). Perinatal hypothyroidism also alters the normal expression and function of G proteins in the rat cerebellum (Leung et al., 1996).

Discovery of these cellular mechanisms in neuronal and glial cells has partially defined the important roles of thyroid hormones in normal brain development. These wide-ranging functions, which are deranged by the absence of these hormones, include neuronal migration and differentiation, synthesis of the cytoskeleton and other structural proteins, regulation of transcription and growth factors, myelination of nerve tracts, and development of the signal transduction machinery for numerous neurotransmitters and neurohormones (Table 7.2). These data, therefore, highlight the robust cognitive and behavioral abnormalities resulting from the absence or relative deficiency of thyroid hormones.

Developmental feature	Thyroid Deficiency	Treatment with Thyroxine	Treatment with Corticosterone
Neuronal cell formation	Normal	Deficit	Deficit
Brain volume	Reduced	No effect (P21)	Reduced (P21)
Rate of cell migration	Reduced	Normal	Normal
Rate of synaptogenesis	Reduced (cerebellum)	Normal	Normal
Neurochemical induction	Reduced	Advanced	Normal
Conversion of glucose to glycogen	Reduced	Advanced	—
Neuronal differentiation	Reduced	Advanced	—
Dendritic arborization	Reduced	Accelerated	Transiently decreased
Dendritic spine formation	Reduced	Accelerated	Transiently decreased
Synaptogenesis	Reduced	Accelerated but transient	—
Myelination	Reduced	Normal	—
Behavioral changes	Reduced	Advanced	Reduced
Development of simple behavioral patterns	Reduced	Advanced	Normal
Retention of sets of associative behavior	Reduced	Advanced	Normal

**Table 7.2. Effects of Thyroxine and Corticosterone on Postnatal Brain Development in Rats**

## Glucocorticoids

Glucocorticoids appear to have dual roles in the developing brain, with inhibitory effects on the proliferation and growth of neuronal and glial cell populations, together with stimulatory effects on the synthesis of certain neurotransmitters and maturation of neuronal function (Table 7.2). Although direct effects of glucocorticoids on early brain development have not been demonstrated, low doses may potentiate the production of nerve growth factor (NGF), as well as the genomic and nongenomic effects of NGF and other trophic factors (Greene et al., 1983; Perez-Polo et al., 1977). Glucocorticoids seem to accelerate neurotransmitter synthesis and release, maturation of synapses, and development of ion channels that regulate membrane potential and electrolyte distribution across neuronal membranes (Baethmann, 1985; Puro, 1983). Direct effects on neuronal maturation in the cerebral cortex (Chubakov et al., 1986), reticular formation (Avanzino et al., 1983), limbic system (Telegdy and Vermes, 1975), and spinal cord (Hall et al., 1978) have been reported. These widespread changes may be related to glucocorticoid effects on the maturation of intracellular signaling, enzyme activity (e.g., Na<sup>+</sup>/K<sup>+</sup>-ATPase, glutamine synthetase, tryptophan synthetase), and neurotransmitter synthesis



(Baethmann, 1985; Leret et al., 1993; Slotkin et al., 1993; Telegdy and Vermes, 1975).

Large doses of glucocorticoids given postnatally suppress growth and cell division, particularly in glial cells and cerebellar neurons. These effects are mediated by inhibition of deoxyribonucleic acid (DNA) synthesis and increased production of myelin and gangliosides, which lead to the developmental delay of several behavioral milestones (Dussault and Ruel, 1987). Further studies on the development and distribution of glucocorticoid receptor subtypes and their genomic effects in neuronal and glial cells (O'Banion et al., 1994) will help to further define the developmental roles of glucocorticoids.

### Gonadal Steroids

Gonadal steroid hormones—including the androgens, progestins, and estrogens—seem to regulate developmental mechanisms that lead to sexual dimorphism in the mammalian CNS. However, there is early evidence suggesting that some sexually dimorphic development in the brain is also triggered by genetic, nonhormonal signals (Arnold, 1996). The regulatory role of gonadal steroids was first suggested by Pfaff (1966) based on observations of morphologic changes in the adult male rat following neonatal castration and were supported by the pioneering studies of Raisman and Field (1971b). There are well-defined sensitive periods for the development of sexually dimorphic behavior in many animal species; these changes are mostly androgen-dependent. For example, for a permanent feminizing effect, the castration of male rat pups must occur within the first few days after birth and not later than P7, whereas androgen therapy in female rat pups has long-term masculinizing effects if given during the same period (Bermant and Davidson, 1974). The changes in brain development relate not only to sexual behavior, but also to other forms of behavior that differ between the sexes. The critical periods for sexually dimorphic brain development vary between species and do not bear a constant relationship to birth (being prenatal in guinea pig and rhesus monkey, perinatal in the dog, and postnatal in the rat, mouse, hamster, and ferret) (Goy and McEwen, 1980). Raisman and Field's studies (1971a,b) described the differential neuronal development in the preoptic area and hypothalamus, with differences in synaptogenesis, dendritic arborization, and cellular features. Gonadal steroids mediate their effects on the developing brain by specific classes of nuclear receptors, presumed to modulate protein synthesis by altering genomic transcription (Beyer and Feder, 1987). The distribution of these receptor classes corresponds with sexually dimorphic areas in the brain, including those implicated in the regulation of sexual behavior, gonadotropin secretion, and other gender-specific social behavior (Baum, 1986; MacLusky et al., 1987). Testosterone implants in the female rat were associated with the marked growth and differentiation of neurons in the sexually dimorphic preoptic nucleus and spinal nucleus supplying the bulbocavernosus muscle, whereas castration of male rats at birth resulted in significant decreases in these areas. Further studies involving exposure of female rats to testosterone have shown that the growth and differentiation of the spinal nucleus supplying the bulbocavernosus is exquisitely sensitive to the timing of this exposure. Female rats exposed to testosterone on the two prenatal days when males are normally exposed to this surge (E18 and E19) showed a marked increase in cell number in this spinal nucleus. This increase was significantly greater than that seen in females exposed to testosterone just prior to or following these two specific days (Ward et al., 1996). Also, transplantation of the sexually dimorphic nucleus to the brains of castrated male rats restored mating behavior and gonadotropin regulation (Arushanyan and Borovkova, 1989). In addition to gender differences in gross volume of the brain, sexual dimorphism has been reported in the planum temporale, corpus callosum, amygdala, and cerebellum (Baum, 1986; Beyer and Feder, 1987; MacLusky et al., 1987). There is also a large amount of converging, although indirect, evidence that gonadal steroids directly influence the systematic sex differences in the degree of lateralization of the cerebral hemispheres (Wisniewski, 1998). The sexually dimorphic features of the human brain are summarized in Table 7.3.

Dimorphic Features	Sex Differences
Global characteristics	
Brain weight	M > F by 10%-15%
Brain volume	M > F by 10%-15%
Dendritic arborization	M > F at birth
Hypothalamus	
Sexually dimorphic nucleus	M > F
Suprachiasmatic nucleus	M > F
Thalamus	
Masa intermedia	Present in F > M
Cerebral cortex	
Heschl's gyrus	Different morphology in M/F
Anterior end of Sylvian fissure	Located more anterior in M
Planum temporale	L > R in M, L = R in F
Posterior frontal opercular sulcus	Different morphology in M/F
Corpus callosum	
Genus splenium	M > F
Isthmus	F > M

**Table 7.3. Sexual Dimorphism of the Human Brain**

Androgens and estrogens affect brain growth globally and neuronal differentiation in sexually dimorphic areas may be mediated either directly or secondary to their effects on glial cells. For example, exposure to estrogens stimulates astrocytes to participate in the remodeling of hypothalamic neurons that control pituitary function. Glial cells may modulate neuroendocrine actions by synthesizing and releasing trophic factors such as IGF-1 or transforming growth factor- $\beta$  (TGF- $\beta$ ), which may regulate the release of hypothalamic releasing factors (e.g., luteinizing hormone-releasing hormone, LHRH) directly, or by actions on other neurons or glial cells (Duenas et al., 1994). In the rat arcuate nucleus, gonadal steroids caused a marked up-regulation of IGF-1-like immunoreactivity in astrocytes and tanocytes, associated with permanent changes in the morphology of the arcuate nucleus (Duenas et al., 1994). Further studies to define the precise cellular changes produced by gonadal steroids in the developing brain, together with the genomic and nongenomic mechanisms of these effects, should be related to the gender-specific differences in male and female behavior. Only then will we understand the biological basis for developmental abnormalities in gender-derived social behavior.

### DEVELOPMENT OF THE PITUITARY GLAND

Anatomic origin of the pituitary occurs from the hypophyseal placode, a thickening of the midline somatic ectoderm, which is rostrally continuous with the neural crest (Schwind, 1928). With the closure of the neural tube and formation of the mouth, this hypophyseal placode comes to lie ventral to the floor of the forebrain vesicle in the region of the infundibulum and medial eminence, just rostral to the anterior end of the notochord (Schwind, 1928). The earliest molecular markers for the hypophyseal placode include the mRNAs for the  $\alpha$ -glycoprotein subunit of the gonadotropins and the  $\beta$ -thyroid-stimulating hormone subunit  $\beta$ -TSH, expressed on E11 when the anterior neuropore is just closing (Simmons et al., 1990). This is followed by evagination of the infundibulum from the floor of the developing hypothalamus and the formation of Rathke's pouch by E13. A day later,  $\beta$ -TSH mRNA are localized to the most anterior part of anterior lobe, with proopiomelanocortin (POMC) mRNA expressed in cells just caudally. By E17, ventrally located cells in the anterior lobe first express the mRNAs for luteinizing hormone (LH) and follicle-stimulating hormone (FSH), followed by the expression of GH and prolactin (PRL) transcripts by E18 (Swanson, 1992).

During the next 2 weeks of development, cells expressing GH mRNA proliferate much faster than those containing PRL, indicating a differential genetic regulation of these cell types. The expression of these hormones is preceded by the synthesis of Pit-1 mRNA and protein on E15, a specific transcription factor that up-regulates the transcription rates of both GH and PRL genes (Ingraham et al., 1988). Pit-1 is expressed in somatotropes, lactotropes, and thyrotropes and also serves as a growth factor for each of these cell types (Simmons et al., 1990). Another nuclear transcription factor belonging to the leucine zipper family, called thyrotroph embryonic factor (TEF) expressed on E13, increases the transcription rate of the  $\beta$ -TSH gene (Drolet et al., 1991). Thus, these transcription factors play an important role in the cell- and tissue-specific expression of the anterior pituitary hormones, and their spatiotemporal patterns correspond with the expression of the hormonal genes that they regulate.

### HYPOTHALAMIC-PITUITARY-ADRENOCORTICAL AXIS

#### Ontogeny of the Hypothalamic-Pituitary-Adrenal Axis

In the rat fetus, corticotropin-releasing factor (CRF)-immunoreactive nerve terminals can be detected in the anterior regions of the median eminence from E18 (Bugnon et al., 1982), followed by proliferation and growth of CRF-immunoreactive fibers and nerve terminals into the developing pituitary. This period is also associated with the proliferation of pituitary cells expressing the POMC gene, although it is unknown whether the maturing CRF-containing median eminence nerve terminals have any effect on the differentiation of these cells. CRF mRNA was detected in the ventral neuroepithelial lobe of the fetal hypothalamus on day 17 of gestation, prior to the formation of a morphologically distinct PVN (Grino et al., 1989b). Hypothalamic CRF mRNA levels increased markedly on E19 and E20, with simultaneous increases in POMC gene transcription and fetal circulating ACTH levels (Grino et al., 1989a,b). Following birth on E21, CRF immunostaining is temporarily absent from the hypothalamic median eminence, probably related to the stress of parturition, because CRF perikarya in the PVN- and CRF-containing fibers in the median eminence can be detected by 12 hours after birth (Bugnon et al., 1982). In newborn rats from P0 to P7, the hypothalamic CRF and pituitary ACTH

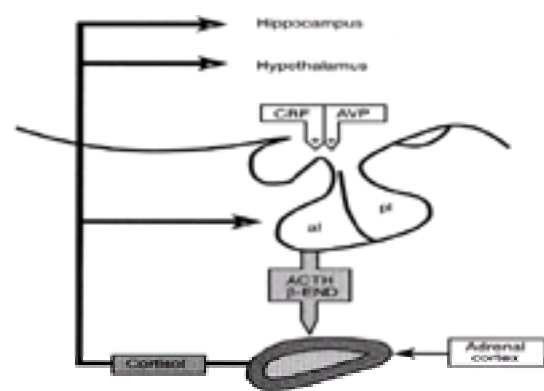


concentrations are approximately 10% of those in adult rats and increase progressively to adult concentrations by 45 days after birth ( Walker et al., 1986a). A targeted mutation in the CRF gene results in CRF-deficient mice, which manifested lung hypoplasia during fetal life. Despite severe glucocorticoid deficiency, these mice exhibited normal growth, fertility, and longevity in postnatal life ( Muglia et al., 1995).

### Corticotropin-Releasing Factor Regulation of the Hypothalamic-Pituitary-Adrenocortical Axis

Within minutes after exposure of pituitary cells to CRF *in vivo* or *in vitro*, a dose-dependent increase in the secretion of POMC-related pituitary hormones occurs, including adrenocorticotrophic hormone (ACTH) and b-endorphin ( Aguilera et al., 1986; Rivier and Plotsky, 1986). ACTH secretion following adrenalectomy or acute stress can be greatly decreased by intravenous injection of a-helical CRF<sub>9-41</sub>, a CRF<sub>1</sub> receptor antagonist, or neutralization with CRF-antiserum, or destruction of the paraventricular nucleus (PVN) of the hypothalamus ( Rivier and Plotsky, 1986; Rivier et al., 1982). These data, taken together with the CRF responses to stress, adrenalectomy, and corticosteroid replacement support the hypothesis that release of endogenous CRF from the paraventricular nucleus regulates the secretory activity of pituitary corticotropes ( Rivier and Plotsky, 1986). CRF may mediate the ACTH response to stressors with a more "psychological" component, whereas both CRF and vasopressin may be involved in the increased ACTH responses to physical stressors ( Mason, 1971; Pich et al., 1993). Both norepinephrine and epinephrine augment cyclic AMP production and the ACTH responses to CRF in pituitary cells ( Abou-Samra et al., 1986; Aguilera et al., 1986). A similar synergism occurs between CRF and vasopressin on ACTH release ( Aguilera et al., 1986). Thus, a variety of neuroendocrine factors have important roles in the regulation of ACTH secretion.

Control of ACTH secretion is mediated by the feedback inhibition of cortisol in primates (corticosterone in rodents). The removal of this feedback by adrenalectomy causes threefold increase in CRF mRNA expression and fourfold to sevenfold increase in arginine vasopressin (AVP) mRNA expression in PVN neurons ( Bradbury et al., 1994; Byer et al., 1988), together with large increases in the synthesis of ACTH ( Fig. 7.1). Feedback inhibition of ACTH release by corticosterone can be divided into rapid and delayed effects, which are partially reversed by exposing pituitary corticotropes to CRF, vasopressin, angiotensin II, and norepinephrine ( Abou-Samra et al., 1986; Levin et al., 1988). The rapid effects on ACTH secretion occur within minutes because of direct steroid effects on ACTH release mechanisms, and the delayed effects occur over hours and days mediated by the inhibition of CRF mRNA expression in the PVN, decreased CRF release from the median eminence, altered CRF receptor-mediated signal transduction, decreased POMC gene transcription, and ACTH synthesis in the pituitary ( Dallman et al., 1994). Chronic *in vivo* or *in vitro* exposure to high CRF concentrations desensitizes the secretory responses of pituitary corticotrophs by down-regulation of the CRF receptor ( Aguilera et al., 1987; Axelrod and Reisine, 1984). In addition to potentiating CRF-stimulated ACTH secretion, vasopressin may also modulate CRF-induced desensitization and down-regulation of CRF receptors in the anterior pituitary ( Aguilera et al., 1987). Occupancy of the high affinity (type I or mineralocorticoid, MR) receptors by low plasma concentrations of corticosterone decreases CRF and AVP expression in PVN cells and normalizes the basal ACTH secretion in the morning. However, occupancy of both MR and low affinity glucocorticoid receptors (GR) is required to reduce the peak ACTH secretion in the evening ( Bradbury et al., 1994; Byer et al., 1988; Kwak et al., 1993). Specific MR antagonists increase basal ACTH secretion, whereas both MR and GR antagonists increase ACTH secretion during the circadian peak or following stress ( Dodt et al., 1993; Ratka et al., 1989). Neurotransmitters other than the glucocorticoids and mineralocorticoids also have been demonstrated to regulate CRF release.



**Figure 7.1.** The hypothalamic-pituitary-adrenocortical axis. ACTH, adrenocorticotrophic hormone; al, anterior lobe of pituitary; AVP, arginine vasopressin; CRF, corticotropin-releasing factor; b-END, b-endorphin; pl, posterior lobe of pituitary. (Negative feedback inhibition is mediated by corticosterone in the rodent species and cortisol in the human species.)

Neurotransmitters that promote CRF release include norepinephrine, acetylcholine, serotonin, neuropeptide Y, interleukin-1B, angiotensin-II and estrogen, whereas inhibitory neurotransmitters include GABA, opioids, atrial natriuretic peptide, substance P, nitric oxide, progesterone, and leptin ( Grossman and Costa, 1993; Heiman et al., 1997; Roy et al., 1999). In addition to feedback inhibition from glucosteroids, CRF is bound by a binding protein that at least theoretically reduces its availability to act on CRF receptors (Reichlin, 1998). CRF binding protein gene transcription is positively regulated *in vivo* by cyclic AMP and CRF ( Cortright et al., 1997), and CRF binding protein mRNA levels are increased by restraint stress in the rat ( McClennen et al., 1998). This suggests that the CRF binding protein may provide an additional feedback mechanism on the HPA axis, although further studies on the ontogeny and precise function of this protein are certainly needed.

Urocortin, a second neuropeptide of the CRF family, also has been discovered recently ( Vaughan et al., 1995). Urocortin shares 45% sequence homology with CRF, but there appears to be limited overlap between urocortin and CRF expression in the rat brain ( Kozicz et al., 1998; Morin et al., 1999; Wong et al., 1998). This suggests that the two neuropeptides may serve different functions. This hypothesis is supported by recent evidence that, in contrast to CRF, urocortin does not significantly affect the release of ACTH from the anterior pituitary under basal conditions, after stress exposure, or adrenalectomy ( Masuzawa et al., 1999; Turnbull et al., 1999).

### Distribution of Corticotropin-Releasing Factor in the Brain

Although the main physiologic function of CRF is to act as the cephalic representative of the HPA axis, the biological role of the peptide involves widespread CNS effects on autonomic, immune, and behavioral functions. CRF immunoreactivity has been localized in hypothalamic and extrahypothalamic structures of the CNS ( Bloom et al., 1982; Swanson et al., 1983, 1986). In addition to CRF-containing cell bodies in the hypothalamic PVN, CRF-containing neurons and nerve terminals are localized in the central nucleus of the amygdala, parabrachial area and substantia innominata, bed nucleus of the stria terminalis, locus ceruleus and the olfactory bulb. Bipolar cerebrocortical cells containing CRF (presumably interneurons) also occur, mainly in layers III and IV of the neocortex ( Swanson et al., 1986). The very high density of CRF binding sites in the primate prefrontal, orbital, and insular cortices differentiates the pattern for CRF receptor distribution in the primate and rodent brain ( DeSouza et al., 1985; Millan et al., 1986; Wynn et al., 1984). These are phylogenetically mature brain regions and control higher cortical functions of primates.

Hypothalamic CRF neurons are present throughout the parvocellular division of the PVN, particularly in a dorsomedial cell condensation just ventral to the anterior commissure, and project to the median eminence. The magnocellular division contains few CRF-positive neurons, and these are functionally associated with vasopressinergic and oxytocinergic neurons ( Swanson et al., 1986). Good correspondence was noted between the distribution of CRF neurons, CRF binding sites, and *in situ* hybridization of CRF receptor mRNA ( DeSouza, 1987; Perrin et al., 1993). The abundance of CRF neurons in extrahypothalamic brain regions, together with its electrophysiologic effects, suggests a neurotransmitter role for CRF, which modulates affective behavior and visceral responses ( Menzaghi et al., 1994) (Table 7.4).

Major Effects of CRF	Major Effects of CRF Receptors in CRF Administration
Increased locomotor activity in familiar environments	Reversed stress and drug-induced anxiolysis
Increased exploration of unfamiliar environments	Attenuated stress-induced fighting
Decreased response to spatial conflict	Reversed defense withdrawal (induced by stress, drug, or genetic) and reduced aggression behavior
Enhanced stress-induced feeding	Attenuated stress-induced feeding
Decreased food intake	Blocked hypothermic state
Disrupted sexual behavior	Attenuated acquisition of conditioned emotional response
Enhanced acoustic startle response	Reduced defense burying
Conditioned fear and aversion	Reversed stress-induced analgesia
Disrupted sleep rhythms	
Increased frequency of grooming and defensive burying	
Reduced values of high doses	

**Table 7.4. Behavioral Effects of Corticotropin-Releasing Factor**

The analgesic properties of CRF have been demonstrated in several studies, and have been posited to be a major mediator for stress-induced analgesia (Chappell et al., 1986; Schafer et al., 1996, 1997). Several studies show that CRF mediates its analgesic actions at all levels of the neuraxis, as well as in the periphery (Lariviere and Melzack, 2000; Schafer et al., 1996, 1997). As previously suggested, the pituitary release of b-endorphin following CRF injection (via various routes: intravenous, intracerebroventricular, intracisternal, and intrathecal) does not explain CRF-induced analgesia, which is a specific CRF effect. From various experimental paradigms it appears that CRF-induced analgesia may be more potent in the setting of inflammatory and prolonged pain, and dysregulation of these mechanisms may play a significant role in the chronic pain syndromes associated with HPA axis abnormalities (Lariviere and Melzack, 2000).

In summary, hypothalamic CRF release triggers a cascade of hormonal events via stimulation of the anterior pituitary and sympathetic outflow, leading to ACTH release and adrenomedullary release of catecholamines. These responses significantly alter cardiovascular, gastrointestinal, metabolic, and immune function, depending on their magnitude and duration. In a complementary manner, the extra-hypothalamic release of CRF seems to mediate the appropriate behavioral and emotional responses to physical and psychological stressors.

### Regulation of Corticotropin-Releasing Factor Receptors

Previous studies using homogenate-binding assays and autoradiography techniques (DeSouza et al., 1985; Hauger et al., 1987, 1988a; Wynn et al., 1984) together with recent molecular approaches (Perrin et al., 1993) have allowed a characterization of the development, anatomic distribution, and regulation of CRF receptors. Activation of CRF receptors leads to the stimulation of adenylate cyclase activity and consequent increases in cyclic AMP levels (Aguilera et al., 1986) in pituitary cells, orbital and frontal cortical neurons, and the amygdala (Millan et al., 1986; Wynn et al., 1984).

Two CRF receptor subtypes, CRF<sub>1</sub> and CRF<sub>2</sub>, have been identified in the rodent (Chalmers et al., 1995; Lovenberg et al., 1995a,b; Potter et al., 1994) and human brains (Chen et al., 1993). These two subtypes have very different patterns of distribution in the brain (Primus et al., 1997). In the rat, the CRF receptor has two different splice variants, CRF<sub>2a</sub> and CRF<sub>2b</sub>. CRF<sub>2a</sub> is the major splice variant located at neuronal membranes, whereas CRF<sub>2b</sub> is found predominantly in nonneuronal elements. The primary endogenous ligand for CRF<sub>2a</sub> appears to be urocortin rather than CRF (Vaughan et al., 1995). Studies using CRF<sub>1</sub>-receptor knockout mice (Smith et al., 1998; Timpl et al., 1998) and studies using antisense oligodeoxynucleotides directed at CRF<sub>1</sub> and CRF<sub>2a</sub> (Heinrichs et al., 1997; Liebsch et al., 1999; Skutella et al., 1998) indicate that the CRF<sub>1</sub>-receptor appears to regulate angiogenesis. CRF<sub>2a</sub> appears to be involved primarily in regulation of feeding and possibly reproductive and defensive behaviors (Steckler and Holsboer, 1999).

The expression of pituitary CRF receptors is altered by physiologic variations in circulating glucocorticoid levels and associated with the delayed corticosteroid inhibition of ACTH transcription and release. Exogenous corticosterone administration produces a dose-dependent reduction in CRF receptor density (B<sub>max</sub>) in the anterior pituitary, thus contributing to the feedback inhibition of ACTH secretion (Hauger et al., 1987). Adrenalectomy causes a decrease in CRF-stimulated adenylate cyclase activity and a down-regulation of pituitary CRF receptors, which is prevented by corticosteroid replacement in adrenalectomized rats (Aguilera et al., 1986; Wynn et al., 1984). The hypersecretion of CRF from hypothalamic neurons results in a homologous, ligand-induced down-regulation of CRF receptors and desensitization of the remaining CRF receptors by uncoupling from adenylate cyclase-linked signal transduction. Nevertheless, CRF receptor regulation is dissociated from ACTH responsiveness following adrenalectomy based on the observation that *in vivo* and *in vitro* ACTH responses to CRF are greater following adrenalectomy, despite the loss of pituitary CRF receptors (Aguilera et al., 1986). In contrast to these changes in the pituitary, CRF receptors in other parts of the brain are unchanged following very high doses of corticosterone or adrenalectomy (Hauger et al., 1987). Release of ACTH and corticosteroids following acute stress does not alter CRF receptor density in the pituitary, although chronic stress causes a down-regulation of pituitary CRF receptors, which is temporally related to the progressive normalization of ACTH secretion during chronic stress (Hauger et al., 1988a). Pituitary ACTH responses to novel stressors or intravenous CRF injection were accentuated in chronically stressed animals, compared with the ACTH responses of nonstressed controls (Hauger et al., 1988a,b). *In vitro* CRF-stimulated cyclic AMP and ACTH release from pituitary cells is reduced following chronic stress, a discrepancy explained by the concomitant effects of vasopressin release *in vivo*, which reverses the desensitization of pituitary cells to resemble those of nonstressed controls (Hauger et al., 1988a). Thus, it appears that vasopressin, norepinephrine, and other neurotransmitters contribute to the *in vivo* sensitization of ACTH responses to novel stressors in chronically stressed animals.

### Hypothalamic-Pituitary-Adrenal Axis Responses to Stress

#### THE STRESS HYPORESPONSIVE PERIOD

ACTH release from fetal or neonatal pituitary glands can be demonstrated *in vitro* following CRF stimulation (Dupouy and Chatelain, 1983; Hary et al., 1984), although the HPA axis *in vivo* remains relatively hyporesponsive to stress for 7 to 10 days after birth in rats (Levine, 1970; Sapolsky and Meaney, 1986; Walker et al., 1986a,b). The threshold for pituitary-adrenal responsiveness is particularly high in the 24 hours after birth, with progressive decreases in the subsequent weeks. Following the increased transcription of hypothalamic CRF mRNA and pituitary POMC mRNA levels in rat fetuses from E17 to E20, CRF and POMC gene transcription decreases markedly during the perinatal period (Grino et al., 1989a,b). A progressive increase in POMC mRNA expression occurs in the rat neurointermediate lobe of the pituitary from P1 to P21, which is corticosteroid insensitive owing to an absence of glucocorticoid receptors. This implies a greater glucocorticoid negative feedback in the perinatal period, leading to secondary inhibition of the expression of CRF mRNA expression in PVN neurons (Grino et al., 1989a,b).

The hypothesis that the stress-hyporesponsive period results from increased corticosteroid inhibition of the HPA axis was supported by the demonstration of enhanced sensitivity to negative feedback, *in vivo* and *in vitro*, to CRF-induced pituitary ACTH release in neonatal rats (Walker et al., 1986a,b). Adrenalectomy of perinatal rat pups was associated with normal ACTH responses to ether stress, although the levels of POMC mRNA in the anterior pituitary after adrenalectomy were lower at P7 than at P14 (Grino et al., 1989a,b). Thus, the relative hyporesponsiveness of the anterior pituitary during the perinatal period may be explained by decreased sensitivity to CRF stimulation and increased sensitivity to corticosteroid negative feedback.

Previous data suggested that b-endorphin responses to CRF stimulation in pituitary cells were similar in 7- and 14-day-old rats (Grino et al., 1989a), although CRF mRNA expression in the PVN increases significantly in 14-day-old rats and remains unchanged in 7-day-old rats following adrenalectomy, an effect reversed by daily CRF injections (Grino et al., 1989a,b). Consequently, the stress hyporesponsive period may result from immature corticosteroid regulation of CRF gene expression in the hypothalamus, perhaps because of the immaturity of afferent inputs to CRF neurons in the neonatal PVN. The ontogeny of CRF receptors in the CNS does not explain the stress hyporesponsive period. Detectable numbers of CRF receptors (40% of receptor density in the adult rat) were measured in the E17 rat CNS (Insel et al., 1988). CRF receptor concentrations increased progressively in the brain until P8, when the receptor density was 312% of adult levels, and then declined to typical adult levels by P21 (Insel et al., 1988). The highest densities of fetal CRF receptors were found in the striatum, whereas after birth CRF binding increased progressively to reach its maximum in the cerebral cortex (Insel et al., 1988). Fetal CRF receptors are coupled to adenylate cyclase-linked signal transduction from E17, and postnatal development is characterized by progressive increases in the percentage of CRF receptors coupled via the stimulatory G-protein to the adenylate cyclase catalytic subunit, with maximum coupling noted in the CNS of adult rats (Insel et al., 1988). Maternal-infant interactions are also involved in the maintenance of the stress hyporesponsive period as outlined in the following.

In contrast, other reports have shown robust adrenocortical responses in neonatal rats soon after birth to physical stressors such as cold-separation stress, which occurred at P0, P9, and P15 (Baram et al., 1997). These studies suggest that careful sampling techniques are required to demonstrate HPA stress responsiveness in



the early postnatal period, which are related to the difficulties in obtaining nonstressed baseline values from neonatal rat pups. Similarly, recent studies showed the rapid transcription of heteronuclear (hnRNA) and messenger RNA (mRNA) for CRF in the hypothalamic paraventricular nucleus of infant rats following even mild aversive stimulation, such as a saline injection ([Dent et al., 2000](#)). Other studies showed the absence of a stress hypo-responsive period in response to an immune challenge (injection of endotoxin), demonstrated by neuronal activation in the hypothalamic PVN accompanied by CRF transcription in neonatal rats ([Dent et al., 1999](#)). Stress hypo-responsiveness of the HPA axis during the perinatal period has not been documented in the human neonate, although neurologic maturity of the newborn rat (at P0) corresponds to that of the premature infant at 24 weeks gestation (data summarized in [Table 7.1](#)).

### STRESS RESPONSES IN HUMAN NEONATES

The hormonal responses of human neonates to surgical operations performed under minimal or no anesthesia are characterized by substantial increases in plasma catecholamines, which return to preoperative values by 6 hours after surgery ([Anand et al., 1985a,b](#)). Responses of the HPA axis to surgical stress are observed with significant perioperative increases in plasma cortisol concentrations in term neonates and significant increases of the precursor steroid hormones (11-deoxycortisol, 11-deoxycorticosterone, 17-hydroxyprogesterone, and progesterone) in preterm neonates ([Anand et al., 1987, 1988](#)). The magnitude and duration of these responses are proportional to the degree of surgical stress, and may be correlated with postoperative clinical outcomes ([Anand, 1993; Anand and Aynsley-Green, 1988](#)). In term neonates subjected to unanesthetized circumcision, plasma cortisol values increase markedly during and after the procedure ([Gunnar et al., 1985](#)).

Fentanyl anesthesia in preterm babies undergoing thoracotomy produces a substantial reduction in the hormonal stress responses to surgery, as indicated by significant decreases in plasma epinephrine, norepinephrine, glucagon, aldosterone, corticosterone, and other steroid hormone responses. The hormonal stress responses of neonates receiving minimal anesthesia lead to significant metabolic derangements (increases in blood glucose, lactate, pyruvate, endogenous protein breakdown, and postoperative complications), which are prevented or diminished in neonates receiving opiates during and after surgery ([Anand et al., 1987, 1988; Anand and Hickey, 1992](#)).

In neonates undergoing cardiac surgery, anesthetic management with halothane and low-dose morphine is associated with substantial catecholamine, corticosteroid, b-endorphin, and glucagon responses during and after cardiac surgery; these responses are blunted in neonates given high-dose sufentanil anesthesia ([Anand and Hickey, 1992](#)). The incidence of metabolic, infectious, and cardiovascular complications postoperatively decreases, together with a significant reduction in postoperative mortality in neonates given sufentanil ([Anand and Hickey, 1992](#)). In addition to the stress of surgery, human neonates who have encountered stressful conditions during birth or unanesthetized circumcision just after birth, maintain an increased responsiveness of the HPA axis with increased salivary cortisol responses and increased pain behaviors when receiving routine vaccinations at 4 and 6 months of age ([Ramsay and Lewis, 1995; Taddio et al., 1997](#)).

### Effects of Early Handling and Maternal Deprivation

Several decades ago, Levine and colleagues noted that repeated handling of rat pups in the first week after birth (or for 21 days, in some studies) results in adult rats with decreased behavioral inhibition in novel environments and decreased HPA responses to a variety of stressors ([Ader and Grotta, 1969; Levine, 1957, 1962; Levine et al., 1967; Meaney et al., 1989](#)). Handled rats (H) had decreased ACTH and corticosterone responses as compared with nonhandled rats (NH) in response to a variety of stressors ([Hess et al., 1969; Meaney et al., 1989, 1991; Viau et al., 1993](#)). Lower peak plasma levels and a faster return to baseline in H rats led to substantial differences between the integrated plasma hormone responses of H and NH rats, and these differences persisted over the entire life of these animals ([Meaney et al., 1988, 1991](#)). These studies showed no differences between the H and NH groups in the adrenal sensitivity to ACTH, the pituitary sensitivity to CRF, the metabolic clearance of ACTH or corticosterone, the plasma levels of corticosterone-binding globulin, or in the basal ACTH or corticosterone levels at any point in the diurnal cycle. These data suggested that long-term effects of neonatal handling were related to neuroendocrine changes above the level of the pituitary. Subsequent studies demonstrated that differences in the HPA axis responses of H and NH rats resulted from increased negative-feedback inhibition in H rats, secondary to an increased expression of glucocorticoid receptors in the hippocampus and median prefrontal cortex of H compared with NH animals ([Meaney et al., 1985, 1987, 1989; O'Donnell et al., 1994; Sarrieu et al., 1988](#)). Chronic corticosteroid treatment (for 5 days) in H and NH rats reversed their differences in both the hippocampal glucocorticoid receptor density and the HPA axis responses to stress ([Meaney et al., 1985](#)). Thus, it appears that up-regulation of glucocorticoid receptors in the hippocampus is a critical determinant of the decreased HPA stress responses in the adult rat subjected to neonatal handling. This receptor up-regulation is dependent on increases in pituitary–thyroid activity and ascending serotonergic systems that act via increased cyclic AMP formation and activation of protein kinase A activity ([Meaney et al., 1987; Mitchell et al., 1990, 1992; Smythe et al., 1994](#)). Inhibitors of thyroid hormone synthesis and serotonin receptor antagonists block this effect ([Meaney et al., 2000](#)). Hippocampal lesions result in increased CRF and AVP mRNA expression in the hypothalamus and increased corticosterone responses to stress ([Herman et al., 1989; Jacobson and Sapolsky, 1991; Sapolsky et al., 1984](#)).

Prolonged separation from the mother during the neonatal period results in a loss of maternal care, which includes the maternal behaviors feeding, grooming, and licking. These maternal behaviors seem to increase cognitive ability during adulthood and maintain the stress hypo-responsiveness of the rat pup HPA axis ([Meaney et al., 2000; Suchecki et al., 1993](#)). An inverse relationship also has been found between time spent in these behaviors as a pup and subsequent adult HPA stress responsiveness ([Liu et al., 1997](#)). Recent studies suggest a vertical transmission of these maternal behaviors and their long-term effects, via either polygenetic determinants or through learned behavioral modes that have major impact on subsequent stress responsiveness as well ([Francis et al., 1999; Liu et al., 2000](#)). Rat pups separated from their mothers in the first 3 weeks after birth exhibit higher basal corticosterone levels and greater corticosterone responses to acute stress than nonseparated controls ([Thomas et al., 1968](#)). Rat pups separated from their mothers for 0, 15, 30, 60, 180, and 360 minutes each day from P2 to P14 showed markedly different HPA responses to stress during adulthood ([Plotsky and Meaney, 1993](#)). Rats separated for 0 and 15 minutes each day behaved like the NH and H rats, respectively, as described; rats separated for 180 to 360 minutes each day showed significantly increased ACTH and corticosterone responses compared with the H and NH groups. These adult rats (following 180 min/day neonatal separation) showed marked increases in CRF mRNA in paraventricular neurons in the hypothalamus and the central nucleus of the amygdala, increases in CRF peptide content in the median eminence, and nonsuppression in a dexamethasone suppression test. These data suggest increases in the hypothalamic mechanisms regulating the ACTH and corticosterone responses to stress ([Hsu et al., 1998; Makino et al., 1994; Plotsky and Meaney, 1993](#)). Following neonatal exposure to stress, glucocorticoid receptor density decreases in the hypothalamus, hippocampus, and frontal cortex and mineralocorticoid receptor density increases in the hippocampus, related to the decreased negative feedback sensitivity of the HPA axis during adulthood ([Ladd et al., 1998; Meaney et al., 1994](#)). The persistence of these effects is determined by the timing of neonatal stress during critical developmental windows. Thus, if prolonged maternal separation (24 hours) occurred on P3, it produced increased ACTH/corticosterone responses to stress on P20; whereas similar maternal separation on P11 produced blunted ACTH/corticosterone responses to stress on P20 ([van Oers et al., 1998; Workel et al., 1997](#)).

The excessive and prolonged HPA axis responses appear to damage hippocampal neurons because of the prolonged exposure to stress-induced elevations of plasma corticosterone concentrations via indirect glucocorticoid effects on NMDA receptor-dependent excitatory pathways ([Cameron et al., 1993, 1995; Gould et al., 1994](#)). In addition to their immunosuppressive and metabolic effects, high circulating levels of glucocorticoids accelerate the loss of hippocampal neurons, leading to cognitive and memory impairments, stress, and perhaps anxiety and mood disorders during adult life ([McEwen, 1994; Sapolsky et al., 1986](#)). Glucocorticoid hypersecretion was associated with the loss of type II glucocorticoid receptors in chronically stressed rats, aged rats, and AVP-deficient Brattleboro rats ([Sapolsky et al., 1986](#)). These long-term effects may result from the impaired feedback inhibition mediated by hippocampal neurons, leading to increased expression and release of CRF and AVP from hypothalamic neurons in response to acute stress, with consequently increased ACTH and corticosterone responses ([Dallman et al., 1993; Ladd et al., 2000; Meaney et al., 1993; Plotsky et al., 1986; Plotsky and Sawchenko, 1987](#)). The transition from low to high serum corticosterone concentrations within the range of physiologic circadian changes or from high basal to stressed values, increases the susceptibility of hippocampal neurons to excitotoxic damage (quantitated by increased neuron loss, spectrin proteolysis, and qualitative changes in neuronal and dendritic morphology) in the CA3 region of the hippocampus ([Meaney et al., 2000](#)). Postnatal handling increases glucocorticoid receptor expression in the rat hippocampus, thus altering the regulation of hypothalamic synthesis of CRF and the HPA response to stress. Recent findings suggest that postnatal handling might alter glucocorticoid receptor gene expression via cyclic AMP–PKA pathways involving the activation of NGFI-A and AP-2 ([Meaney et al., 2000](#)).

As summarized in [Table 7.4](#), central administration of CRF in nonhuman primates causes behavioral changes almost identical to those observed in primate models of depression involving the separation of neonatal monkeys from their mothers ([Kalin and Takahashi, 1988](#)). Several lines of evidence suggest that the hypothalamic hypersecretion of CRF may result from maternal deprivation and predispose to the development of depression in adulthood ([Gold et al., 1988](#)). Evidence for this phenomenon has been found from both rodent and nonhuman primate studies ([Coplan et al., 1996; Owens et al., 1994](#)). Prolonged increases were noted in the cerebrospinal fluid (CSF) concentrations of CRF in young adult monkeys following maternal stress as infants owing to variable foraging conditions ([Coplan et al., 1996](#)). Interestingly, chronic treatment with the antidepressants paroxetine and mirtazapine seem to normalize the behavioral and endocrine stress responses in maternally separated rats ([Ladd et al., 1997; Plotsky et al., 1996](#)). Clinical correlates of these experiments are being obtained from victims of sexual or physical abuse, poor parenting, or exposure to other stressful life events ([Goodyear, 1994; Heim et al., 1998, 2000; Kaufman et al., 1997; Mullen et al., 1993](#)), although the mechanisms mediating such changes in the neonatal brain have remained largely unexplored. One possibility is that lack of  $\text{N-methyl-D-aspartate}$  (NMDA) receptor activity from maternal separation and sensory isolation leads to increased apoptosis in multiple areas of the immature brain ([Ikonomidou et al., 1999](#)). On the other hand, exposure to repetitive pain may cause excessive NMDA/excitatory amino acid activation, resulting in excitotoxic damage to developing neurons. These changes



promote two distinct behavioral phenotypes characterized by increased anxiety, altered pain sensitivity, stress disorders, hyperactivity/attention deficit disorder, leading to impaired social skills and patterns of self-destructive behavior ( [Anand and Scalzo, 2000](#)).

## ALTERATIONS IN CHILDHOOD PSYCHIATRIC AND BEHAVIORAL DISORDERS

### Childhood Shyness

Shyness was defined by [Kagan and coworkers \(1988\)](#) as an initial avoidance of and/or prolonged behavioral restraint to an unfamiliar event. Although some shyness may be expected within the range of normal childhood behaviors, excessive shyness in young children has been correlated with excessive anxiety, social avoidance and isolation, and panic and mood disorders during adulthood, leading to severe psychological impairment. In pathologically shy children, hypothalamic and amygdala-mediated responses to novel situations are hyperreactive. Increased sympathetic nervous system activity was documented in shy and inhibited children exposed to unfamiliar events, by their increased heart rate, pupillary dilation, and peripheral norepinephrine responses, also correlated with high morning levels of salivary cortisol ( [Kagan, 1982](#); [Kagan et al., 1988](#)).

It is likely that shyness results from the diathesis of an abnormally low threshold for limbic–hypothalamic responsivity to novel stimuli or stress. Because CRF is a primary regulator of limbic sites stimulating the sympathoadrenomedullary axis and the HPA axis, CRF may be involved in the pathogenesis of severe childhood shyness. CRF hypersecretion may cause behavioral inhibition and enhanced pituitary–adrenal, catecholamine, and cardiovascular responses in shy children. CRF overdrive in the CNS may be a common pathogenetic mechanism for childhood shyness and major depression in adults ( [Gold et al., 1988](#)).

### Major Depressive Disorder

Chronic hyperactivity of the HPA axis occurs in adults with a major depressive disorder (MDD), which is owing to CRF hypersecretion, at least in part. Pathophysiologic alterations of the HPA axis in depression during the acute phase of illness include hypersecretion of cortisol, increased amplitude of each cortisol secretory episode, and an early onset (i.e., phase-advance) of the nocturnal quiescent period and the subsequent increase toward the morning peak for ACTH/cortisol circadian rhythms. Alterations in corticosteroid feedback, as evidenced by nonsuppression of plasma cortisol levels in response to the synthetic glucocorticoid dexamethasone, decreased ACTH responses to intravenously administered CRF, and increased CSF concentrations of CRF occur, together with down-regulation of the number of CRF receptor sites in the frontal cortex of depressed suicidal patients ( [Francis et al., 1999](#); [Gold et al., 1988](#); [Hauger et al., 1989](#); [Nemeroff et al., 1988](#)). Although some of these HPA axis abnormalities are not unique to depression, their presence in acutely depressed patients suggests that CRF hypersecretion at hypothalamic and extrahypothalamic levels may contribute to the pathogenesis of depression. This overactivity of the HPA axis is a reversible, state-dependent secretory disturbance, which normalizes following recovery from depression. Therefore, these neuroendocrine changes are indicative of a state of stress maladaptation, supporting the hypothesis that pathologic stress responses contribute to the pathogenesis of depression ( [Anisman, 1984](#)).

There is currently considerable debate over the issue of whether the HPA axis of prepubertal children and adolescents with MDD resembles the overactive HPA axis of adult patients with depression. A preliminary study suggested that cortisol hypersecretion could occur in prepubertal children with endogenous depression ( [Puig-Antich et al., 1979](#)), although this was not confirmed in subsequent studies of children and adolescents with MDD ( [Casat and Powell, 1988](#); [Dahl et al., 1989](#); [Kutcher et al., 1991](#); [Puig-Antich et al., 1989](#)). Cortisol hypersecretion occurs in severely depressed and suicidal children and adolescents requiring hospitalization ( [Evans et al., 1987](#); [Dahl et al., 1991](#); [Pfeffer et al., 1991](#)), and during the recurrence of major depressive episodes ( [Rao et al., 1996](#)), although dexamethasone suppression occurs infrequently in childhood and adolescent MDD ( [Dahl et al., 1992](#)). This overall lack of cortisol hyperresponsiveness may be related to a more sensitive plasma cortisol feedback mechanism occurring in all but the most severely depressed children and may represent a developmental stage of the adult syndrome ( [DeBellis et al., 1996](#)). It is possible the progressive changes in the regulation of hypothalamic CRF secretion during childhood may finally manifest in the full-blown adult disorder ( [Liu et al., 1997](#)).

### Posttraumatic Stress Disorder

As seen in major depression, there is some degree of uncertainty as to the continuity of HPA axis findings between children and adolescents and adults in posttraumatic stress disorder (PTSD). Adult PTSD patients demonstrate increased CRF levels in the CSF and blunted ACTH responses, and yet they exhibit a normal cortisol response to exogenously administered CRF ( [Bremner et al., 1993](#); [Smith et al., 1989](#)). Adults demonstrate low urinary cortisol levels, suppression to low doses of dexamethasone, and increased glucocorticoid receptors on lymphocytes ( [Heim et al., 1996](#); [Yehuda et al., 1991, 1995](#)). Adult combat veterans also exhibit elevated urinary levels of catecholamines ( [Yehuda et al., 1992](#)). Although adult PTSD patients exhibit hypocortisolemia, there have been findings of hippocampal atrophy in combat veterans with PTSD ( [Bremner et al., 1995](#); [Gurvits et al., 1996](#)) and adult survivors of childhood abuse ( [Bremner et al., 1997](#); [Stein et al., 1997](#)).

Increased urinary catecholamines were reported in recent studies of children and adolescents with PTSD, similar to the adult studies ( [DeBellis et al., 1994b, 1999](#); [DeBellis and Putnam, 1994](#); [Queiroz et al., 1991](#)). This finding is associated, however, with increased, rather than decreased, urinary cortisol levels ( [DeBellis et al., 1994a, 1999](#)). It has been hypothesized that this difference in cortisol levels may be the result of either a maturational effect or a long-term adaptation of the HPA axis to stress ( [DeBellis et al., 1994a, 1999](#)).

### Childhood Autism

Autism is a developmental neuropsychiatric syndrome characterized by the early childhood onset of severe disturbances in language, cognitive development, absence of object relatedness, social withdrawal, idiosyncratic affective states, motoric stereotypies, and increased locomotor behavior. In an often-cited study, [Schain and Freedman \(1961\)](#) demonstrated that circulating levels of serotonin (5-HT) were markedly elevated in autistic children. Further investigations revealed that blood 5-HT concentrations were elevated in 30% of children with clinically defined (Diagnostic and Statistical Manual-III [DSM-III] criteria) autism. Several mechanisms may account for this finding, ranging from abnormal metabolism of serotonin in the gut, to the properties of platelets that transport serotonin, to CNS mechanisms that control these processes. Systematic studies of peripheral serotonin production, gut motility, and blood flow; platelet numbers, size, storage capacity, and half-life; hormones or other endogenous mediators that may alter the serotonin transporter on platelet membranes; or the central dysregulation of serotonergic pathways are necessary because all could contribute to defining the etiopathogenesis of autism. However, in most studies published subsequently, the patient populations were not defined by hyperserotoninemia but by the clinical features of autism. A great deal of research that focused on defining the abnormal serotonergic mechanisms in autism did not even measure blood serotonin concentrations. This is important because only 30% of clinically defined autistic children have hyperserotoninemia, and the percentage in any subpopulation may range from 0% to 100%. Therefore, separate studies measuring a variable relevant to the mechanism of hyperserotoninemia will differ quantitatively in accordance with the unknown proportion of hyperserotoninemic subjects in the study population, leading to different, or even opposing, conclusions. Two additional potential confounds in this area that have been recently examined are ethnicity and pubertal status. Ethnicity was found to have a greater effect than diagnosis when platelet serotonin levels were compared among prepubertal autistic children, nonautistic mentally retarded prepubertal children, and normal controls of the same age. Ethnicity and diagnosis were both nonsignificant in postpubertal groups and platelet serotonin levels were significantly lowered in postpubertal versus prepubertal subjects ( [McBride et al., 1998](#)). Much of the confusion in this area might be avoided if study populations were defined by their etiologic diagnosis rather than their clinical diagnosis ( [Yuwiler, 1995](#)).

Fenfluramine given in low doses acutely increased central 5-HT neurotransmission via stimulation of 5-HT release, inhibition of presynaptic 5-HT reuptake, and interaction at certain postsynaptic 5-HT receptors as a weak agonist ( [Costa et al., 1971](#)). High-dose or chronic fenfluramine administration exerts neurotoxic effects on presynaptic 5-HT nerve terminals, with a sustained depletion of neuronal 5-HT stores ( [Costa et al., 1971](#); [Garrattini et al., 1975](#)). Fenfluramine therapy in autistic children (administered in studies prior to its withdrawal from the market by the FDA) resulted in significant clinical improvement and was correlated with 50% reduction in blood serotonin levels ( [Ritvo et al., 1984](#)), although these findings were not replicated in subsequent studies. Fenfluramine-induced changes in pituitary prolactin secretion were used to measure serotonergic responsivity in male autistic children ( [McBride et al., 1989](#)). Prolactin responses to fenfluramine were blunted in autistic patients, together with a marked reduction in the magnitude of 5-HT–amplified platelet aggregation and decreased numbers of 5-HT<sub>2</sub> receptors in the platelets of autistic children ( [McBride et al., 1989](#)). It is plausible that hyperserotoninemia and the desensitization of brain serotonergic systems to 5-HT agonists may characterize autism. Decreased activity in serotonergic pathways in autistic patients may explain a number of their clinical findings and suggests that drugs augmenting central serotonergic neurotransmission may have therapeutic effects. Concordance between serotonergic mechanisms in peripheral blood platelets and the CNS has not been proven conclusively, although it is frequently assumed in mechanistic and therapeutic studies.

Stress responses of the HPA axis may be abnormal in autistic children. Cortisol secretion in response to insulin-induced hypoglycemia is elevated and prolonged in autistic children as compared with normal controls ( [Maher et al., 1975](#)). Because the regulation of hypothalamic CRF release involves stimulatory input from serotonergic neurons (via 5-HT<sub>2</sub> receptors), the abnormal cortisol responses to hypoglycemia may reflect abnormalities in central serotonergic neurotransmission



([Calogero et al., 1989](#)). Fenfluramine administration reduces hypothalamic CRF concentrations, presumably by stimulating CRF release, and increases CRF concentrations in the hippocampus, midbrain, and spinal cord ([DeSouza et al., 1989](#)). The clinical improvement noted with fenfluramine therapy in autistic children could be partially related to the normalization of CRF-mediated mechanisms essential for stress adaptation.

Some of the behavioral and motoric symptoms in autism suggest abnormalities in dopamine (DA) neurotransmission. Increased CSF concentrations of the principal DA metabolite homovanillic acid ([Cohen et al., 1977](#)) and decreased GH responses to L-dopa were documented in autistic children ([Deutsch et al., 1985](#)), suggesting that dopaminergic mechanisms influencing GH release may be desensitized in autism. Conversely, the GH responses to insulin-induced hypoglycemia, mediated via the catecholaminergic, serotonergic, and glucoreceptor stimulation of PVN neurons, were prolonged in autism ([Deutsch et al., 1986](#)). There are no data demonstrating alterations in endogenous opioid neuropeptides in autism and no therapeutic effects have been demonstrated with the use of opioid agonists or antagonists in this disorder ([Gillberg and Coleman, 1992](#)).

A number of animal studies show that oxytocin plays a role in social behavior and communication ([Insel et al., 1999](#)). A recent human study showed that plasma oxytocin concentrations in autistic children were half those of normal controls. In these autistic subjects, their age and lack of interpersonal skills were correlated with a failure of the developmental increases in oxytocin levels ([Modahl et al., 1998](#)), although oxytocin levels in the CSF of autistic subjects have not been measured. These disparate neuroendocrine manifestations of childhood autism indicate the need for much greater precision in neurobiologic studies investigating the mechanisms of this neuropsychiatric disorder, with particular focus on regulation of the HPA axis and somatotrophic axis. Certainly brain-imaging studies, both structural and functional, could play a pivotal role in elucidating the pathophysiology of this devastating disorder.

## THE SOMATOTROPHIC AXIS

### Ontogeny of the Somatotrophic Axis

Growth hormone-releasing factor (GHRF) immunoreactivity can be measured in the infundibular nucleus of the hypothalamus from 18-week-old human fetuses ([Bresson et al., 1984](#)); however, GHRF neurons in the arcuate nucleus cannot be detected until 29 weeks of gestation ([Bloch et al., 1984](#)). Consequently, fetal GH regulation may not involve GHRF until mid- to late gestation. In the feline CNS, GHRF terminals are sparse in the median eminence at 15 days postpartum, followed by their rapid proliferation in the first month after birth ([Bugnon et al., 1983](#)). Fetal sheep have higher circulating concentrations of GH as compared with neonatal sheep, associated with the maximum GH responses to GHRF in fetal sheep from 71 to 135 days of gestation ([Gluckman, 1984](#); [Ohmura et al., 1984](#)). In the human species, GH responses to GHRF are fairly constant throughout puberty and adulthood. The sensitivity of the pituitary somatotroph to GHRF does not change after early childhood. The gradual reduction of GH secretion with age is secondary to increasing sensitivity of the pituitary to somatostatin and may be related to the increased production of the somatostatin<sub>2</sub> receptor subtype with age, as recently demonstrated in the rat brain ([Reed et al., 1999](#)).

### Distribution of Growth Hormone-Releasing Factor in the Brain

After [Vale and colleagues \(1981\)](#) reported the isolation, sequencing, and synthesis of a 41-amino acid ovine hypothalamic peptide CRF that stimulated pituitary ACTH and endorphin secretion ([Vale et al., 1981](#)), the same group ([Rivier et al., 1982](#)) and another group ([Guillemin et al., 1982](#)) independently identified and sequenced GHRF from tissue extracts of pancreatic islet cell tumors from two patients with acromegaly ([Guillemin et al., 1982](#); [Rivier et al., 1982](#)). Further research has characterized the distribution of hypothalamic neurons containing these peptides, together with the localization of receptor sites for these regulatory factors in the pituitary gland and other parts of the brain.

Although CRF, somatostatin, and thyrotropin-releasing hormone (TRH) are widely distributed in the mammalian brain, GHRF has a much more limited localization in the human, nonhuman primate, and rodent brains ([Bloch et al., 1983](#); [Merchenthaler et al., 1984](#)). GHRF-immunoreactive cell bodies are primarily located in the arcuate and ventromedial nuclei of the hypothalamus and project GHRF-containing nerve terminals to the median eminence. Other tissues that express GHRF-like peptides include the placenta and parts of the gastrointestinal tract (e.g., in the gastric antrum and upper intestinal tract it is colocalized with the peptide gastrin).

The only nonhypophysial behavioral effect thus far identified for GHRF injected intracerebroventricularly (ICV) is its ability to stimulate food intake ([Vaccarino et al., 1985](#)) and induce sleep ([Frieboes et al., 1995](#)). The intravenous administration of GHRF releases GH without stimulating feeding behavior ([Vaccarino et al., 1985](#)). The regulation of appetite is a central action of GHRF that may facilitate GH-induced somatic growth in children and adolescents.

### Regulation of GHRF Receptors

Specific high-affinity receptor sites for GHRF were demonstrated in the anterior pituitary and other organs, including the exocrine pancreas, where GHRF stimulates pancreatic enzyme secretion ([Seifert et al., 1985](#)). Although GHRF receptors in the pituitary are specific for GHRF, other endogenous mediators such as vasoactive intestinal peptide (VIP) also demonstrate a high affinity binding to pancreatic GHRF receptors ([Pandol et al., 1984](#); [Seifert et al., 1985](#)). The GHRF receptor is a G protein-coupled receptor, which activates cyclic AMP by binding to a stimulatory G protein, which activates adenylate cyclase, increases intracellular free calcium, releases preformed GH, and stimulates GH mRNA transcription and synthesis of new GH ([Barinaga et al., 1983](#); [Cronin and Thorner, 1995](#)).

The HPA axis modulates the somatotrophic axis by multiple mechanisms, largely via the central effects of adrenocortical hormones. *In vitro* experiments have shown that glucocorticoids increase GH gene transcription, increase GH content and release, and enhance GH responses to GHRF from pituitary somatotrophs. *In vivo*, corticosteroids suppress somatic growth and GH responses to various stimuli ([Evans et al., 1982](#); [Gelato and Merriam, 1986](#); [Wehrenberg et al., 1983](#)). The GHRF-binding capacity of the anterior pituitary is decreased by adrenalectomy, and dexamethasone administration substantially increases the number of pituitary GHRF receptors in adrenalectomized rats ([Seifert et al., 1985](#)). The physiologic role of this interaction was substantiated by a similar increase in GHRF-binding capacity in animals with intact adrenal glands, suggesting that the HPA axis is part of a larger hypothalamic neural network that mediates feeding, growth, and energy balance ([Dallman et al., 1994](#); [Seifert et al., 1985](#)). Within this network, corticosteroids may enhance the responsiveness of pituitary somatotrophs by up-regulating GHRF receptors. Stress-mediated glucocorticoid release may exert a negative effect on GH secretion, in order to inhibit feeding and other restorative behaviors. The stress-related or baseline secretion of glucocorticoids may also modulate the central actions of GHRF, such as the stimulation of food intake.

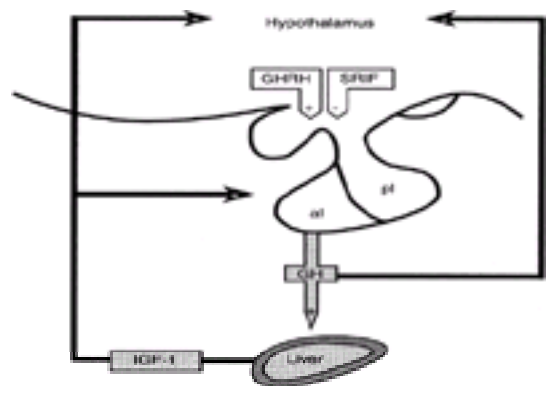
Similar to other transmitter systems, prolonged exposure to hypothalamic releasing factors results in a desensitization of their secretory responses and is associated with a concurrent down-regulation of their specific receptors ([Catt et al., 1979](#)). Thus, prolonged exposure to GHRF stimulation *in vivo* or *in vitro* leads to a significant blunting of the resulting GH responses, associated with GHRF receptor down-regulation ([Bilezikjian et al., 1986](#); [Ceda and Hoffman, 1985](#); [Wehrenberg et al., 1986](#)). The *in vitro* desensitization of GH secretory responses during prolonged exposure to increased GHRF levels does not correspond with the loss of pituitary GHRF receptors ([Bilezikjian et al., 1986](#)). Pituitary somatotrophs become less sensitive to GHRF owing to the depletion of GHRF-sensitive intracellular GH pools. Such desensitized pituitary somatotrophs can respond to very high levels of GHRF, indicating that chronic GHRF infusions may have a therapeutic role in disorders of growth ([Bilezikjian et al., 1986](#)).

### Regulation of Growth Hormone Secretion

#### SOMATOSTATIN

The oscillatory interactions between GHRF and somatostatin are primarily responsible for the regulation of GH release ([Fig. 7.2](#)), although a number of neural factors also influence GH release. GH itself and IGF-1, whose release from the liver is stimulated by GH, both also provide negative feedback inhibition of GH release. The pulsatile secretion of GH results from GHRF pulses released by hypothalamic GHRF neurons, whereas the intermittent secretion of hypothalamic somatostatin causes the variability in the magnitude of GH pulses secreted in response to GHRF. The peaks of GH secretion result from the coincidence of a GHRF peak and a somatostatin nadir from the hypothalamus, and the troughs of serum GH concentrations correlate with the peaks of somatostatin release from the hypothalamus ([Gelato and Merriam, 1986](#); [Hartman et al., 1993](#)). This regulation is illustrated by a sustained increase in GH secretion for several hours in response to a continuous infusion of GHRF during immunoneutralization of somatostatin. In the absence of pretreatment with the somatostatin antiserum, the same GHRF infusion results in pulsatile GH release ([Wehrenberg et al., 1986](#)). Neural influences on GH release include stimulation of GH release by dopamine ([Boyd et al., 1970](#)), norepinephrine, serotonin, acetylcholine,  $\alpha$ -adrenergic receptor stimulation, VIP, nitrous oxide ([Reichlin, 1998](#)), galanin ([Davis et al., 1987](#)), and opioids ([Grossman, 1983](#)). GH secretion is inhibited by TRH under normal physiologic conditions ([Chiara et al., 1977](#)) and  $\beta$ -adrenergic receptor stimulation ([Reichlin, 1998](#)). Somatostatin release is inhibited by GABA and can be stimulated by acetylcholine, substance P, or VIP ([Reichlin, 1998](#)). GH release is also modified by endogenous sleep rhythms, stress, exercise, and the metabolic state ([Reichlin, 1998](#)). The influence of metabolic state may be mediated at least partially by the recently discovered adipose cell

hormone leptin, which stimulates GH secretion ([Tannenbaum et al., 1998](#)).



**Figure 7.2.** The somatotrophic axis. al, anterior lobe of pituitary; GH, growth hormone; GHRH, growth hormone-releasing hormone; IGF-1, insulin-like growth factor-1; pl, posterior lobe of pituitary; SRIF, somatostatin.

Somatostatin acts by the binding of the somatostatin receptor to inhibitory G proteins, which, in turn, lower cyclic AMP and reduce intracellular free calcium through effects on the voltage-sensitive calcium ion channel ([Kleuss, 1995](#); [Schonbrunn, 1990](#)). The effects of lowered cyclic AMP appear to only partially account for the effects of somatostatin, however. Changes in calcium concentration are likely a crucial step in the process, probably acting via increased potassium ion conductance and resultant membrane hyperpolarization ([Patel et al., 1995](#)).

#### EFFECTS OF CORTICOTROPIN-RELEASING FACTOR

Somatostatin can centrally inhibit CRF release and prevent stress-induced ACTH and epinephrine secretion ([Brown et al., 1984](#)). Activation of somatostatin receptors on pituitary cells may decrease ACTH responses to CRF ([Axelrod and Reisine, 1984](#)). Alternatively, high circulating levels of glucocorticoids in depressed patients decrease somatostatin release in the brain, leading to disinhibition of the HPA axis. The nonsuppression of plasma cortisol levels after dexamethasone in depressed patients was correlated with low CSF somatostatin concentrations ([Rubinow, 1986](#)). These data substantiate a pathophysiologic relationship between somatostatin and the HPA axis in depressed patients.

CRF directly stimulates somatostatin secretion from cultured fetal brain cells ([Peterfreund and Vale, 1983](#)). Although CRF has no direct inhibitory effects on pituitary GH secretion, the ICV injection of CRF suppresses basal GH secretion, morphine-stimulated GHRF release, and GH responses to intravenous GHRF ([Rivier and Vale, 1985](#)). Immunoneutralization of endogenous somatostatin abolishes CRF-induced suppression of basal and dynamic GH secretion, whereas the ICV administration of a CRF-receptor antagonist (a-helical CRF<sub>9-41</sub>) abolishes the inhibition of GH secretion after acute stress ([Rivier and Vale, 1985](#)). Therefore, central CRF hypersecretion in pathophysiologic states may alter the regulation of GH secretion by GHRF by stimulating the release of somatostatin. Hyperglycemia in normal human subjects can result in GH hyposecretion and also inhibit pituitary GH responses to GHRF ([Gelato and Merriam, 1986](#)). Hyperglycemia may stimulate hypothalamic somatostatin release, thus altering the responsiveness of pituitary somatotrophs. These interactions further substantiate the view that the HPA axis and somatotrophic axis are embedded within a larger hypothalamic neural network controlling growth, metabolism, and energy balance.

#### Disorders of the Somatotrophic Axis During Childhood

##### GROWTH HORMONE DEFICIENCY

Monosodium glutamate treatment of neonatal rats results in the destruction of GHRF-producing neurons in the arcuate nucleus, leading to GH hyposecretion and growth failure, which are reversible by exogenous GHRF administration ([Millard et al., 1982](#); [Nemeroff et al., 1977](#); [Wehrenberg et al., 1984](#)). Exogenous GHRF does not stimulate cyclic AMP production or GH secretion from pituitary cells of the "lit/lit" dwarf mouse, an animal model of pituitary GH deficiency ([Jansson et al., 1986](#)). The somatotrophs of lit/lit mice were unresponsive to GHRF, although dibutyryl cyclic AMP, forskolin, and cholera toxin stimulated *in vitro* pituitary GH release. These data suggest that the catalytic and regulatory subunits ( $G_s$ ) of adenylate cyclase and the GH release pathway distal to adenylate cyclase can be activated in pituitary somatotrophs from the lit/lit mouse ([Jansson et al., 1986](#)). The GHRF challenge test is being utilized to distinguish GH deficiency states owing to hypothalamic versus pituitary defects. For example, a functional GH response to an injection of GHRF in patients with growth impairment would indicate a hypothalamic defect in the somatotrophic axis ([Gelato and Merriam, 1986](#)). If an intrinsic pituitary defect is excluded, treatment with exogenous GHRF potentially could be used to restore normal growth.

##### PSYCHOSOCIAL DWARFISM

Psychosocial dwarfism is defined as a syndrome of reversible growth failure and developmental delay characterized by reduced growth and hyposecretion of GH ([Powell et al., 1967a,b](#)). The diagnostic criteria proposed for psychosocial dwarfism include: (a) onset between 2 to 3 years of age; (b) delayed bone age and linear growth retardation (<third percentile) without signs of malnutrition; (c) decreased basal secretion of GH and somatomedin; (d) blunted GH responses to secretory stimuli; (e) behavioral changes such as bizarre patterns of food intake, sleep disorders, temper tantrums, withdrawal, delayed cognitive development, and so on; and (f) parental deprivation secondary to psychiatrically disturbed and abusive parents ([Green et al., 1987](#)).

Endocrinologic studies of these patients demonstrated pathologically low fasting GH levels, and reduced GH responses to insulin-induced hypoglycemia, arginine infusion, or exercise, when the patients were in the deprived environment ([Green et al., 1987](#); [Money et al., 1976](#); [Powell et al., 1967a,b](#)). Sleep studies in children suffering from psychosocial dwarfism showed increased stage I sleep, a large reduction in slow-wave sleep (SWS) owing to the absence of stage IV sleep, and a decrease in the total sleep period ([Guilhaume et al., 1982](#)). The SWS phase of sleep is primarily associated with nocturnal GH secretion; therefore, growth failure occurs from the lack of this physiologic stimulus for GH secretion. This SWS-induced GH release is mediated by serotonergic fibers ([Reichlin, 1998](#)). The regulation of GH secretion in psychosocial dwarfism is very sensitive to the child's environment. For example, during a hospital admission or when the child is placed in a beneficial nurturing environment, basal GH secretion and dynamic GH responses normalize rapidly, with initiation of bone growth. If the child returns to the socially deprived environment, GH hyposecretion recurs ([Powell et al., 1967a,b](#)). Normalization of the diminished circulating levels of serum somatomedins occurs in children with psychosocial dwarfism after hospitalization ([D'Ercole et al., 1977](#)).

Maternal deprivation by physical separation or maternal anesthesia was used to develop an animal model of psychosocial dwarfism in preweaning rat pups. The relative lack of active tactile interaction between the rat pups and their mothers decreased the circulating serum GH concentrations, metabolic activity of brain and peripheral tissue, and ornithine decarboxylase (ODC) activity, an index of organ growth and differentiation. These changes were rapidly reversed when the pups were returned to their mother ([Kuhn et al., 1978](#)). Because maternal deprivation did not alter serum concentrations of corticosterone or prolactin, it is unlikely that hyposecretion of GH resulted largely from the effects of stress. Exogenous GH or placental lactogen, combined with normal feeding of these pups, did not reverse the reduced brain and peripheral ODC levels in the absence of the mother. Conversely, vigorous stroking of the separated rat pups or the presence of an awake mother who cannot nourish because of ligated nipples was found to restore growth and maturation ([Kuhn et al., 1979](#)). This maternal tactile stimulation appears to stimulate secretion of GH via serotonergic and noradrenergic effects on GHRF and somatostatin ([Katz et al., 1996](#)). [Kuhn and colleagues \(1990\)](#) have further reported that prolonged periods of maternal separation (>2 hours) result in a suppression of plasma GH, whereas brief periods of separation (i.e., 15 minutes) or handling lead to increases in plasma GH concentrations. Thus, differential environmental conditions during early life may have widely different neuroendocrine effects; there is some evidence that these neuroendocrine changes persist for the entire life of the individual ([Meaney et al., 1994](#)).

GH hyposecretion and subsequent tissue insensitivity to GH in human psychosocial dwarfism may result from the psychogenic suppression of GHRF secretion in the CNS and/or the excessive release of brain somatostatin in the absence of active mothering behavior. This hypothesis was originally proposed by Powell and associates when they stated, "the emotional disturbance in these children may have had an adverse effect upon release of pituitary trophic hormone via the central nervous system." A state-dependent decrease in the basal secretion of corticosteroids is present in children with psychosocial dwarfism, which can be reversed by exogenous ACTH administration ([Powell et al., 1967a,b](#)). Although ACTH secretion may be deficient, adrenocortical sensitivity to ACTH is intact in psychosocial



dwarfism. Early maternal separation leads to marked and long-term increases in the sensitivity of the HPA axis to mild or moderate stressors, secondary to decreased feedback inhibition of PVN neurons expressing CRF and AVP ([Meaney et al., 1994](#)) and to CNS CRF neurons as well ([Coplan et al., 1996](#)), as described.

## MAJOR DEPRESSIVE DISORDER

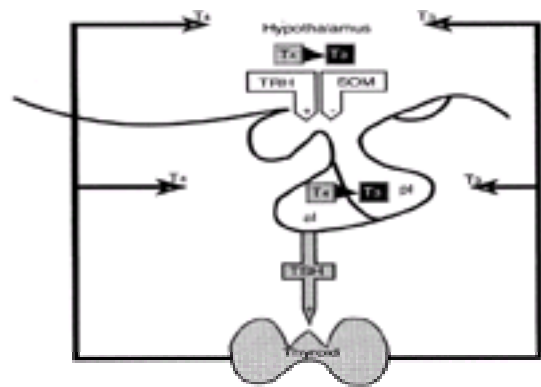
Diurnal hypersecretion of GH occurs in adult patients with endogenous depression, with a phase-advanced circadian timing of GH secretion ([Linkowski et al., 1987](#); [Mendelwicz et al., 1985](#)). This abnormality persists despite a therapeutic response to antidepressants or following complete recovery in remitted patients who are off antidepressants ([Jarrett et al., 1990](#)). The GH responses to insulin-induced hypoglycemia, clonidine, desmethylimipramine (DMI), and zimelidine (but not to the dopamine agonists amphetamine, L-Dopa, or apomorphine) have been consistently shown to reduce during depression. In adult patients with MDD, the blunting of GH responsiveness to these stimuli persists despite clinical remission and response to therapy ([Amsterdam et al., 1987](#); [Charney et al., 1982](#); [Siever and Uhde, 1984](#)).

Further, some (but not all) investigators have reported that GH responses to GHRF are significantly reduced in adult patients with MDD as compared with those in healthy controls ([Krishnan et al., 1987](#); [Lesh et al., 1987](#); [Risch et al., 1988](#)); the blunting of GH responses to GHRF may persist for years after recovery from depression ([Risch and Hauger, 1988](#)). Decreased GH secretion in major depression may either result from desensitization of pituitary somatotrophs by excessive release of GHRF, or from somatostatin hypersecretion in the depressive state. Based on preclinical studies, CRF hypersecretion in depression could suppress basal GH secretion and dynamic GH responses by stimulating the excessive release of somatostatin from extrahypothalamic and hypothalamic sites. However, other studies indicated that GH responses to GHRF are not decreased and may actually be increased in depression ([Krishnan et al., 1987](#)). This conflicting finding and the observation that major depression is associated with an episodic daytime hypersecretion of GH would suggest that hypothalamic somatostatin secretion is reduced, a hypothesis consistent with the measurement of low CSF somatostatin concentrations in depressed patients ([Rubinow et al., 1983](#)).

Prepubertal children and adolescents with major depression also demonstrate abnormalities in GH secretion. In contrast to depressed adults, prepubertal children secrete excessive GH during the total sleep period in the acute episode of endogenous depression and during remission ([Puig-Antich, 1987](#); [Puig-Antich et al., 1984a,b,c,d](#)). Similar excessive GH secretion during sleep was noted in two studies comparing adolescents to matched controls ([Kutcher et al., 1988, 1991](#)), but these findings were not replicated in two similar more recent studies of depressed adolescents ([Dahl et al., 1992](#); [DeBellis et al., 1996](#)). In one of these more recent studies, dividing the MDD group into those patients with and those without suicidality revealed significant blunting of nocturnal GH in the suicidal group, similar to findings in adults ([Dahl et al., 1992](#)). However, this finding in the suicidal MDD subgroup was not replicated in a second recent study ([DeBellis et al., 1996](#)). Abnormal regulation of GH secretion in MDD is supported by the reduced GH responses to insulin-induced hypoglycemia and DMI in depressed prepubertal children and adolescents; these changes persist during remission ([Puig-Antich, 1987](#); [Puig-Antich et al., 1984a,b,c,d](#); [Ryan et al., 1988, 1994](#)). Clonidine challenge did not produce statistically significant changes in GH response in one study ([Ryan et al., 1994](#)). Deficient GH responses were not correlated with the severity or other features of MDD, although the maximum reduction of GH responses to DMI were measured in adolescents who had made a suicide attempt or had prominent suicidal ideation ([Ryan et al., 1988](#)). Clinical application of these data suggests that trait dysregulation of GH secretion may identify depressed adolescents at risk for suicide. State-dependent abnormalities in the HPA axis of adult patients with depression were also associated with increased suicidality ([Arato et al., 1988](#); [Nemeroff et al., 1988](#)). Despite numerous studies in this area, it remains unclear whether alterations in the somatotrophic axis play any precise role in the etiology or pathophysiology of MDD.

## HYPOTHALAMIC-PITUITARY-THYROID AXIS

The effects of thyroid hormones on the development of the brain were described in the first part of this chapter and are summarized in [Table 7.2](#). This section examines the ontogeny of the hypothalamic-pituitary-thyroid (HPT) axis and describes the neuroendocrine regulation of thyroid function under basal conditions and during acute stress, as well as discusses some of the psychiatric correlates of abnormal thyroid function ([Fig. 7.3](#)).



**Figure 7.3.** The hypothalamic-pituitary-thyroid axis. al, anterior lobe of pituitary; pl, posterior lobe of pituitary; SOM, somatostatin; T<sub>3</sub>, triiodothyronine; T<sub>4</sub>, thyroxine; TRH, thyrotropin-releasing hormone; TSH, thyroid-stimulating hormone.

### Regulation of the Hypothalamic-Pituitary-Thyroid Axis

The major thyroid gland hormones are T<sub>3</sub> and T<sub>4</sub>, both of which are released from the thyroid gland. However, about 90% of plasma T<sub>3</sub> is derived from the deiodination of T<sub>4</sub> in the CNS and peripheral tissues. The biological activity of T<sub>3</sub> is much greater than T<sub>4</sub>, and more than 99% of both hormones are bound in serum to proteins such as thyroxine-binding globulin (TBG), albumin, and prealbumin; thus, less than 1% of the circulating concentrations of these hormones are unbound and biologically active. The biological activity of these hormones in the brain is closely regulated by uptake across the blood-brain barrier ([Schreiber et al., 1990](#)), and the activity of the enzyme 5 $\alpha$ -deiodinase-II, which converts T<sub>4</sub> to T<sub>3</sub> in neuronal and glial cells ([Leonard, 1990](#)).

Biosynthesis and release of T<sub>3</sub> and T<sub>4</sub> are controlled by thyroid-stimulating hormone (TSH) secreted from the anterior pituitary, and the biosynthesis and release of TSH is regulated by TRH secreted from nerve terminals in the median eminence into the hypothalamo-hypophysial portal venous system. Dopamine ([Kaptein et al., 1980](#)) and somatostatin ([Ferland et al., 1976](#)) are potent inhibitors of TSH synthesis and release and blunt the pituitary responses to TRH with consequent decreases in serum T<sub>3</sub> and T<sub>4</sub>. Thyroid hormone in turn inhibits the release of somatostatin ([Berelowitz et al., 1980](#)). Homeostatic control within the HPT axis is maintained by the feedback inhibition of TSH biosynthesis and release, mediated mostly by the biologically active circulating concentrations of T<sub>3</sub> and to a lesser extent T<sub>4</sub>. T<sub>4</sub> in the blood gains access to TRH-secreting neurons in the hypothalamus by way of the CSF. The hormone is taken up by epithelial cells of the choroid plexus of the lateral ventricle, bound within the cell to locally produced transthyretin, and then secreted across the blood-brain barrier ([Schreiber et al., 1995](#)). Within the brain, T<sub>4</sub> is converted to T<sub>3</sub>, which selectively reduces TRH biosynthesis in the PVN ([Segerson et al., 1987a,b](#)) by interacting with thyroid hormone receptor subtypes TR-a-1, TR-b-1, and TR-b-2 ([Lechan et al., 1994](#)). This provides another means for homeostatic control of the HPT axis. The secretion of TSH and TRH is under complex control and can be modified by alterations in the activity of several neurotransmitter systems ([Jacobowitz, 1988](#); [Morley, 1981](#)).

Effects of T<sub>3</sub> and T<sub>4</sub> in the brain are controlled by 5 $\alpha$ -deiodinase-II activity, which has been localized only in the brain, pituitary, brown adipose tissue, and placenta. 5 $\alpha$ -Deiodinase-II activity in the brain increases threefold to fivefold within 24 hours of thyroidectomy and decreases by 80% to 90% within a few hours after injection of a saturating dose of T<sub>3</sub>, suggesting that the enzyme is responsible for a homeostatic mechanism by which intracellular T<sub>3</sub> levels are maintained within narrow limits ([Leonard, 1990](#)). Thyroid hormone-related changes in enzyme activity are owing to changes in the half-life of the enzyme and do not depend on changes in the rates of transcription or translation ([Leonard, 1990](#)). Thus, it is reasonable to expect that thyroid hormones play important roles in the normal development and functioning of the intact brain. The impact of thyroid function on brain development has been described in the preceding, and support for the effects of T<sub>3</sub> and T<sub>4</sub> on brain function is obtained from significant changes in the electroencephalogram (EEG) and evoked potentials ([Pohunkova et al., 1989](#); [Sulc et al., 1990](#)), the observation of specific behavioral changes during thyroid disease, and reports of alterations in the HPT axis in certain psychiatric disorders.

### Ontogeny of the Pituitary-Thyroid Axis

With its expression on E11, the mRNA for b-TSH forms one of the earliest molecular markers for the developing hypophyseal placode ( [Simmons et al., 1990](#)). By E14, the mRNA for b-TSH is localized in the anterior-most cells of the developing anterior pituitary. The TEF, expressed on E13, specifically increases the transcription rate of the b-TSH gene in these cells ( [Drolet et al., 1991](#)). The mRNA for TRH is first detected in the hypothalamus on E14 and is localized in parvocellular neurons of the PVN and other hypothalamic neurons by E16 ( [Burgunder and Taylor, 1989](#); [Segerson et al., 1987a](#)). The expression of TRH mRNA reaches an adult pattern of distribution in hypothalamic and extrahypothalamic sites by P21 in the rat ( [Burgunder and Taylor, 1989](#)). Exposure of immature and adult rats to hypothermia up-regulates TRH mRNA expression in the PVN; however, this effect is not seen at the circadian peak of TRH mRNA levels at the onset of darkness ( [Zoeller et al., 1990](#)).

### Effects and Regulation of Thyrotropin-Releasing Hormone Secretion

Regulation of TRH secretion by parvocellular neurons in the PVN occurs by negative feedback inhibition. The mechanisms for feedback inhibition mediated by  $T_3$  and  $T_4$  develop and mature between E20 and P7 in the neonatal rat ( [Taylor et al., 1990](#)). High levels of TRH mRNA in the PVN were noted in animals that were chemically thyroidectomized and reverted to baseline after treatment with  $T_3$  ( [Koller et al., 1987](#); [Segerson et al., 1987b](#)). Regulation of TRH expression is primarily controlled by the direct action of  $T_3$  in PVN neurons and is independent of TSH, other pituitary hormones, and catecholamine input to the hypothalamus ( [Dyess et al., 1988](#); [Zoeller et al., 1988](#)). Support for the primary role of  $T_3$  in feedback inhibition of TRH is provided by transcripts encoding for the  $\alpha_1$ - and  $\beta_1$ -thyroid hormone receptor genes in parvocellular neurons of the PVN ( [Bradley et al., 1989](#)).

TRH is the primary regulator of TSH secretion from the anterior pituitary, and it also stimulates prolactin release. The concentrations of TRH are highest in the median eminence, although substantial concentrations of TRH are localized in extrahypothalamic brain areas including the brain stem, midbrain, preoptic area, septum, basal ganglia and the cerebral cortex, where TRH apparently functions as a neurotransmitter and neuromodulator ( [Timiras and Nzekwe, 1989](#)). TRH effects on anterior pituitary cells are mediated via the phosphoinositol second messenger system; other forms of signal transduction may be involved in the nonpituitary effects of TRH. The neurotransmitter role for TRH is thought to explain widespread changes in behavior and brain functions, such as the antagonism of hibernation and the sedation and hypothermia induced by centrally acting depressants (e.g., barbiturates, ethanol, and anesthetics). In addition, TRH increases body temperature, reduces food intake, stimulates locomotor activity, increases blood pressure and respiratory rate, causes arousal and EEG activation, increases gastric motility, and has antinociceptive effects. These effects may be produced directly or by interaction with a variety of monoamine and peptide neurotransmitters ( [Griffith, 1985](#); [Nemeroff et al., 1984](#); [Timiras and Nzekwe, 1989](#); [Vaccari, 1988](#)). In human subjects, TRH appears to increase the sense of well-being, motivation, relaxation, and coping capacity. These effects were noted in normal subjects and patients with neurologic (Parkinson's disease) or psychiatric disorders (depression, schizophrenia, autism) ( [Loosen, 1988a,b](#); [Loosen and Prange, 1984](#); [Prange et al., 1979](#)), although it is often difficult to separate the direct behavioral effects of TRH from its endocrine effects, leading to secondary changes in behavior, as well as its interactions with various other neurotransmitter systems ( [Griffith, 1985](#); [Nemeroff et al., 1984](#)).

### Thyroid Hormone Receptors

A specific class of intracellular nuclear receptors, found on both neuronal and glial cells, mediates the effects of thyroid hormones ( $T_3$  and  $T_4$ ) on the developing and adult brain ( [Evans, 1988](#)). Although both  $T_3$  and  $T_4$  bind to the nuclear thyroid receptor, most of the biological activity results from  $T_3$  and 80% of the  $T_3$  is derived from the b-deiodination of  $T_4$  in the neuronal cell ( [Dussault and Ruel, 1987](#)). The homology between steroid and thyroid receptors suggests that these belong to a superfamily of nuclear receptors containing three functional domains that mediate hormone binding, DNA binding, and transcription ( [Evans, 1988](#); [Evans and Arriza, 1989](#); [Samuels et al., 1989](#)). The binding affinity of these receptors for  $T_3$  is about 10 times their affinity for  $T_4$ , a factor that roughly parallels the *in vivo* potency ratio for these two hormones ( [Komisaruk et al., 1986](#)).

The expression of thyroid hormone receptors in the human brain precedes the prenatal increase in serum  $T_3$  levels and the peak growth spurt in neuronal differentiation and glial replication ( [Komisaruk et al., 1986](#); [Kuhn and Schanberg, 1984](#)). High densities of thyroid receptors are expressed in the developing brain from early gestation, with preferential localization on neurons rather than glial cells. Specific  $T_3$ -binding sites were barely detectable at 10 weeks gestation and increased tenfold by 16 weeks gestation in the human fetus. Similar increases occurred around birth in rat pups and reach peak levels at P9 ( [Dussault and Ruel, 1987](#); [Komisaruk et al., 1986](#); [Kuhn and Schanberg, 1984](#)). The abundant expression of thyroid hormone receptors in early gestation underlines the important role of  $T_3$  and  $T_4$  in controlling the normal development of the immature mammalian brain.

### Hypothalamic-Pituitary-Thyroid Axis Responses to Stress

Acute stress in the form of critical illness, surgical operation, starvation, burns, or severe systemic diseases produces the "euthyroid sick syndrome" in all age groups. This includes decreased plasma concentrations of  $T_3$ , low or normal total and free  $T_4$ , normal TSH, and significantly increased plasma reverse  $T_3$  concentrations ( [Brandt et al., 1976](#); [Fisher, 1990](#); [Weissman, 1990](#)). Serum TSH responses to exogenous TRH appeared normal after surgical stress, but the maximal TRH-induced increase in TSH and the integrated TSH responses were reduced in critically ill postoperative patients ( [Zaloga et al., 1985](#)). Pituitary hyporesponsiveness to TRH stimulation was directly correlated with serum dopamine concentrations, and with clinical outcome following critical illness ( [McLarty et al., 1975](#); [Philips et al., 1984](#); [Silberman et al., 1988](#)). These findings were confirmed in a recent study, in which the plasma concentrations of cortisol,  $T_3$  and TSH were found to be the most accurate predictors of mortality following critical illness ( [Rothwell and Lawler, 1995](#)). These findings indicate that pathologic alterations in the HPT axis and thyroid function in response to acute stress are associated with marked increases in illness severity and poor outcome.

### Behavioral Manifestations of Thyroid Dysfunction

#### HYPOTHYROIDISM

Infants with untreated congenital hypothyroidism show profound mental retardation with specific motor and sensory abnormalities, in addition to the physical stigmata and metabolic characteristics of cretinism ( [Fisher, 1986](#)). Severe deficits in IQ occur in more than 80% of hypothyroid infants who received delayed treatment, and only 15% of infants who received early treatment with thyroxine ( [Klein et al., 1972](#)). The degree of thyroid deficiency and the age of the patient at the time treatment is begun determine the intellectual prognosis and effectiveness of therapy ( [Dussault, 1986](#); [New England Congenital Hypothyroidism Collaborative Study, 1981](#)). Even in patients with normal or near normal intellectual development, impaired brain development may be manifested in the form of clumsiness (33%), behavior disorders (23%), speech disorders (20%), learning deficits (26%), and poor motor coordination in the majority of children ( [MacFaul et al., 1978](#)). Older children and adolescents may show a variable deterioration in school performance ( [Fisher, 1986](#)).

Acquired hypothyroidism in adults is manifested primarily by impaired cognition, often associated with depression, fatigue, and anxiety, and occasionally the development of psychosis ( [Reitan, 1963](#); [Schon et al., 1961](#); [Whybrow et al., 1969](#)). In some patients, the overt psychiatric disorders may precede the onset of hypothyroid signs and symptoms and may go unrecognized until the failure of therapy with psychopharmacologic intervention ( [Reed and Bland, 1977](#)).

An association has been reported between attention deficit hyperactivity disorder (ADHD) and the rare thyroid disease called generalized resistance to thyroid hormone (GRTH). The occurrence of symptoms consistent with a diagnosis of ADHD was noted in a significantly higher number of adults and children with GRTH than unaffected family members ( [Hauser et al., 1993](#)). These symptoms were consistent with the Hyperactive/Impulsive but not Inattentive DSM-IV subtypes of ADHD ( [Hauser et al., 1997](#)). A subsequent study of 132 children and adolescents with ADHD, however, failed to find evidence of GRTH in any subjects and also found only minor abnormalities in thyroid function tests that occurred at the same rate reported for populations of normal children in the literature ( [Spencer et al., 1995](#)).

#### HYPERTHYROIDISM

The behavioral manifestations of hyperthyroidism may include cognitive changes ranging from subtle defects in attention and concentration (potentially mistaken for ADHD) to overt delirium. Depression may also be seen in hyperthyroidism and is its most common psychiatric manifestation ( [Anfinson et al., 1998](#)). Anxiety also may be seen and manifestations of mania, hypomania, and psychosis may occur rarely ( [Anfinson et al., 1998](#)).

### Thyroid Function in Major Depressive Disorder

The HPT axis is as important for the maintenance of normal affective state and behavior as it is for cognition. As noted, depression occurs commonly in patients with



both hypothyroidism and not uncommonly in hyperthyroidism. Conversely, approximately 30% of euthyroid-depressed patients show a decreased TSH secretory response to TRH administration (Gold et al., 1981; Loosen, 1992). Antithyroid antibodies were found in 20% of psychiatric inpatients with prominent depressive symptoms (Nemeroff et al., 1985) and 9% of patients with unipolar depression (Joffe et al., 1987), evidence of incipient hypothyroidism. Additionally, an inverse relationship between peripheral TSH and regional cerebral blood flow and cerebral glucose metabolism was shown in a PET study of adults with major depression and bipolar disorder (Marangell et al., 1997). Although the endocrine effects of treatment with antidepressants cannot be separated, most studies have shown a significant improvement in thyroid function with remission in depressed patients (Loosen, 1992). This was first suggested by Whybrow and associates (1972), who found that improved thyroid function before treatment in depressed patients was positively correlated with a rapid response to treatment with imipramine. Decreased thyroid function noted in patients with rapid cycling bipolar disorder has been associated with a clinical response to treatment with high-dose T<sub>4</sub> in a pilot study (Bauer and Whybrow, 1990). The few studies evaluating thyroid function in adolescent major depression, however, have produced conflicting results. This is exemplified by three different studies comparing free T<sub>4</sub> levels in depressed adolescents and normal controls. One study found reduced free T<sub>4</sub> (Dorn et al., 1996), one elevated free T<sub>4</sub> (Carstens et al., 1990), and one no change (Sokolov et al., 1994). It should be noted that there were significant methodologic differences among the studies.

Decreased TSH responses to TRH in depressed patients were not correlated with their age or somatic measures, the severity of depression or treatment history, or levels of thyroid hormones, corticosteroids, somatostatin, or dopamine. Decreased TSH responses were not correlated with clinical factors such as primary or secondary depression, unipolar or bipolar subgroups, and patients who were acutely ill or in remission. However, the primary clinical importance of decreased TRH-induced TSH responses derives from their association with a history of violent suicidal behavior and an increased risk of suicide (Loosen, 1988a,b). Repeated TRH tests may be useful for prognostic purposes, although these data have not been validated and require cautious interpretation (Loosen, 1992). The data begin to suggest some similarities between the endocrine correlates of critical physical illness (see the preceding) and severe psychiatric disorders, a concept that may generate a host of interesting hypotheses for future research.

## HYPOTHALAMIC-PITUITARY-GONADAL AXIS

### Ontogeny of the Pituitary-Gonadal Axis

Neurons expressing gonadotropin-releasing hormone (GnRH) first develop anteriorly in the olfactory placode and migrate posteriorly to reach the median basal hypothalamus by 9 to 11 weeks of gestation in the human fetus. The development and migration of these neurons are altered in patients with Kallman's syndrome (hypogonadotropic hypogonadism and hyposmia) associated with a Xp22.3 deletion of the X chromosome (Handelin et al., 1993; Schwanzel-Fukuda et al., 1989). In the normal human fetus, GnRH mRNA is expressed in the fetal hypothalamus from about 10 weeks gestation, and the developing pituitary gland contains b-FSH from 10 to 15 weeks of gestation. The mRNA for the  $\alpha$ -glycoprotein subunit of gonadotropins forms one of the earliest molecular markers for the hypophyseal placode, expressed on E11 in the rat fetus, when the anterior neuropore is just closing (Simmons et al., 1990). By E17 in rats, ventrally located cells in the anterior lobe first express the mRNAs for luteinizing hormone (LH) and follicle-stimulating hormone (FSH) (Swanson, 1992). The hypothalamic-hypophyseal portal circulation develops at about midgestation in most species, leading to a marked increase in gonadotropin secretion (Gluckman et al., 1981). The increased concentration of gonadotropins in peripheral plasma is responsible for the testicular and ovarian maturation that occurs after midgestation. Sheep fetuses exposed to a long-acting GnRH agonist in the latter half of gestation showed the absence of pituitary gonadotrophs and reduced testicular growth at birth (Thomas et al., 1994). Steroidogenic factor 1 (SF-1), an orphan nuclear receptor, regulates the development of gonadotrophs in the pituitary, as well as the enzymes that synthesize sex steroids in the adrenal gland and gonads. In SF-1 knockout mice, pituitary cells lacked the expression of gonadotroph-specific factors, including LH-b, FSH-b, and the GnRH receptor (Ingraham et al., 1994).

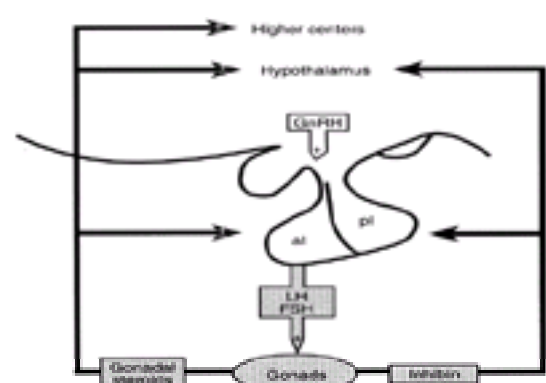
During the third trimester, the expression of steroid receptors in the hypothalamus is associated with the onset of feedback inhibition and a resulting decrease in the secretion of gonadotropins. At birth, the fetal hypothalamus is released from the control of high maternal plasma estrogen concentrations, and gonadotropin secretion increases substantially within 2 to 4 days after birth. Episodic peaks of gonadotropin secretion can be detected for 2 to 4 years after birth, often associated with the secretion of gonadal steroid hormones in the pubertal range (Stein, 1992).

### Neuroendocrine Regulation of Puberty

Puberty is a sequence of events characterized by the secretion of pituitary and gonadal hormones leading to the development of secondary sexual characteristics, gametogenesis, reproductive function, and transformation of the child's appearance to the dimorphic adult state. Regulation of the onset of this epochal life event involves the complex interaction of several neuroendocrine mechanisms, many of which are susceptible to modulation by genetic, psychological, behavioral, dietary, and environmental factors. Some of these regulatory neuroendocrine mechanisms include: (a) changes in the sensitivity of the hypothalamus to inhibitory steroid feedback, (b) synchronization of the GnRH secreting hypothalamic neurons leading to a summation of the stimuli producing the secretion of gonadotropins (FSH, LH) from the pituitary, (c) development of the secretory capacity of pituitary gonadotrophs, (d) removal of inhibitory influences of nongonadal origin (e.g., melatonin), and (e) proposed decreases of the inhibitory influences from higher brain centers. Other somatic and endocrine factors include the amount and distribution of body fat mass (de Ridder et al., 1992), the levels of sex-hormone binding globulin (SHBG) (Mendel, 1989; Sitteri et al., 1972), ovarian estrogen secretion in response to LH (Garibaldi et al., 1993), and the effects of other pituitary hormones and cytokines (Chrandrashekar et al., 1988; Hall et al., 1992).

### CONTROL OF GONADOTROPIN SECRETION

Stimulation of the pituitary gonadotrophs by the hypothalamic decapeptide GnRH released into the hypophyseal portal system leads to the characteristic secretion of FSH and LH pulses as measured in peripheral plasma (Apter et al., 1993) (Fig. 7.4). The pulsatile secretion of GnRH most likely results from intrinsic mechanisms within the network of GnRH-secreting neurons (Mellon et al., 1990), although the frequency of GnRH pulses and synchronization of individual GnRH neurons may depend on other hypothalamic and extrahypothalamic factors (Styne, 1994). In normal children, increased gonadotropin secretion occurs for a period of 2 to 4 years after birth followed by a period of quiescence, which is known as the juvenile phase. At the onset of puberty, the GnRH secretion increases again with marked accentuation of the magnitude and duration of gonadotropic hormone pulses (Grumbach and Styne, 1998). Other pituitary hormones may also alter the secretion of gonadotropins. Studies in transgenic mice overexpressing the human GH gene suggested that GH altered the pituitary sensitivity to GnRH and/or increased pituitary gonadotropin synthesis and secretion (Chrandrashekar et al., 1988, 1989). Decreased body weight, testicular weight, and development following thymectomy in animals were related to the stimulation of hypothalamic GnRH secretion and pituitary LH release by thymosin-b4 (Comsa, 1973; Hall et al., 1992). Conversely, the blockade of GnRH receptors in neonatal rats (P1 to P5) permanently impaired the development of the thymus and maturation of T lymphocytes and inhibited normal sexual maturation (Morale et al., 1991).



**Figure 7.4.** The hypothalamic-pituitary-gonadal axis. al, anterior lobe of pituitary; FSH, follicle-stimulating hormone; GnRH, gonadotropin-releasing hormone; LH, luteinizing hormone; pl, posterior lobe of pituitary.

Inhibition of gonadotropin secretion results from feedback inhibition of sex steroid hormones and inhibin (which suppresses FSH secretion only), and control from higher brain centers. In patients without gonads (e.g., patients with Turner's syndrome, agenesis of the ovary) or in castrated animals, the pattern of gonadotropin secretion during infancy and childhood is qualitatively similar to that of normal children, but the baseline and peak serum levels of gonadotropin pulses are increased significantly (Conte et al., 1975). The absence of gonads does not alter the qualitative pattern of increased gonadotropin secretion during infancy or puberty and the



quiescent phase during childhood, but the quantitative increases in the magnitude of gonadotropin secretion implicate the inhibitory feedback of gonadal steroid hormones ([Grumbach and Styne, 1998](#)). Increased plasma cortisol also decreases the pulsatile secretion of LH and GnRH, implying its role in stress-associated menstrual disturbances ([Saketos et al., 1993](#)).

Inhibitory control from higher brain centers is thought to be responsible for mediating the juvenile phase, which occurs in a steroid-independent manner. Although the exact mechanisms and precise loci mediating this control of gonadotropin secretion are presently unknown, a significant amount of evidence primarily from primate studies indicates a significant role for GABA in the inhibition of the GnRH pulse generator by higher brain centers ([Grumbach and Styne, 1998](#)). Loss of this control is demonstrated in patients who develop chronic increases in intracranial pressure (e.g., secondary to hydrocephalus or subarachnoid cysts) with the onset of central precocious puberty (CPP), which is reversed by surgical procedures (e.g., ventriculoperitoneal shunt placement) that normalize the intracranial pressure. Rare patients with hamartomas of the tuber cinereum, which often contain ectopic GnRH-secreting neurons, also present with central precocious puberty because these ectopic neurons do not receive the descending inhibitory control from higher brain centers ([Grumbach and Styne, 1992](#)). Thus, the onset of CPP may be related to the disinhibition of hypothalamic GnRH neurons resulting from the absence of control from higher brain centers.

Both of the gonadotropins, LH and FSH, can be measured by specific radioimmunoassays, although recent interest in measuring the biologically active component of LH (by testosterone production in rat Leydig cells) and FSH (by aromatase activity in rat Sertoli cells) has demonstrated different patterns of changes from those of the immunoreactive hormones ([Beitins and Padmanabhan, 1991](#); [Kletter et al., 1993](#)). These data revealed that plasma concentrations of biologically active LH may increase manyfold and were not associated with corresponding changes in immunoreactive LH concentrations. Comparison of biologically active and immunoreactive FSH concentrations in serum did not show such striking differences ([Kletter et al., 1993](#)). The role of bioactive gonadotropins in the regulation of the normal and abnormal HPG axis remains speculative.

#### ONSET OF PUBERTY

The amplitude and frequency of nocturnal GnRH pulses from the hypothalamus increase gradually and progressively well before the onset of pubertal development ([Apter et al., 1993](#)). These changes are associated with increased production of pituitary gonadotropins and episodic increases in estrogen and testosterone secretion from the immature gonads. In prepubertal boys, early morning testosterone levels of >0.7 nmol/L were accurate predictors of the onset of puberty within 12 to 15 months ([Wu et al., 1993](#)). Another predictor of pubertal onset was the magnitude of LH secretion in response to exogenous GnRH (100 mg intravenously). Although significant increases in serum LH concentrations were predictive of pubertal onset within 6 to 12 months, the wide variability of individual responses was associated with a number of false-positive and -negative results ([Crowley and Jameson, 1992](#)). Longer-acting and more potent analogs of GnRH activity may be more accurate in determining the onset of puberty ([Cuttler et al., 1993](#)).

Normal pubertal development is characterized by a complex interaction of gonadal steroids, gonadotropins, SHBG, and body mass composition ([de Ridder et al., 1990](#); [Frisancho and Flegel, 1982](#)). The amplitude of nocturnal GnRH pulses is markedly accentuated at the onset of puberty, leading to significant increases in the magnitude and duration of gonadotropic hormone pulses, developmental changes in the gonads, and the production of gonadal steroids. Initially, the GnRH and gonadotropic pulses occur at the onset of sleep and progressively increase to cover the entire night ([Grumbach and Styne, 1992](#); [Morales et al., 1992](#)). Developmental changes in monopolar and bipolar GnRH-immunoreactive neurons located in the medial septal-preoptic area and the diagonal band of Broca in male syrian hamsters were related to the onset of puberty ([Urbanski et al., 1992](#)), although the mechanisms mediating these morphologic changes remain unknown. In normal girls, control of the onset of puberty and the maturation of negative feedback control of the HPG axis are partially independent, thus allowing "catch up" pubertal maturation in girls with a late onset of puberty ([de Ridder et al., 1992](#)).

The change in GH secretion during the pubertal growth spurt is closely associated with the stages of puberty. Plasma concentrations of both LH and GH measured in normal boys and girls (7.2 to 14.6 years age) showed increases in the amplitude and frequency of nocturnal pulses with the progression of puberty ([Bourouignon, 1991](#)). The coordination of the pubertal growth spurt and pubertal development may occur via neuroendocrine mechanisms at the hypothalamus and/or pituitary. Little is known of these mechanisms apart from GH effects on the pituitary sensitivity to GnRH and/or increased pituitary gonadotropin secretion (Chandrashekar et al., 1988, 1989). The role of endocrine and paracrine factors *other than gonadotropins* in regulating the production of gonadal estrogens was suggested in a recent study of early central precocious puberty in girls ([Garibaldi et al., 1993](#)). For example, interleukin-1 (IL-1) altered the expression of LH receptors in the rat ovary, and interferon- $\alpha$  was capable of suppressing estrogen and progesterone release ([Hall et al., 1992](#)). These data indicate that a complete understanding of the mechanisms controlling the onset of puberty is not presently available.

After the onset of puberty, gonadotropin secretion during the daytime increases associated with progressive decreases in the nocturnal gonadotropin secretion that is characteristic of early puberty. The later stages of puberty are identified by the lack of diurnal variation in gonadotropin secretion and development of positive feedback between ovarian estrogen secretion and pituitary LH release to stimulate normal ovulation at the peak of the LH surge ([Morales et al., 1992](#)).

#### Disorders of Hypothalamic-Pituitary-Gonadal Function

##### ANOREXIA NERVOSA AND BULIMIA

Anorexia nervosa is behaviorally defined as weight loss of more than 15% below expected body weight, associated with an intense fear of weight gain, an abnormal body image, and amenorrhea. Bulimia is characterized by recurrent episodes of binge eating followed by purging behavior such as self-induced vomiting, use of laxatives, diuretics, strict dieting, or vigorous exercise ([Kennedy and Garfinkel, 1987](#)). These two disorders have considerable overlap: They first appear during adolescence and young adulthood, are often accompanied by major depression, and have characteristic abnormalities in neuroendocrine function ([Bruch, 1973](#); [Kennedy and Garfinkel, 1987](#)). The etiology of anorexia nervosa and bulimia most likely involves complex interactions between the endocrine system, CNS, and psychological and social factors. A great deal of recent research has examined the hypothesis that primary hypothalamic abnormalities may lead or predispose to the combination of behavioral disorders and neuroendocrine pathophysiology that characterize anorexia nervosa and bulimia. Their neuroendocrinology may be comparable because both cause similar behavioral and psychological disturbances (such as a morbid fear of obesity, distorted body image, poor impulse control, anxiety, and depression).

The weight loss in anorexia nervosa is preceded by amenorrhea, a clinical finding also noted in underweight bulimic patients, but not in those of normal weight ([Boyar et al., 1974](#); [Halmi and Sherman, 1975](#); [Marshall and Kelch, 1979](#)). The amenorrhea in both disorders is attributed to hypothalamic hypogonadism and it may or may not be reversed by the restoration of weight gain. Basal circadian levels of LH and FSH are decreased in pubertal women with anorexia nervosa to plasma levels typical of the prepubertal period ([Boyar et al., 1974](#); [Halmi and Sherman, 1975](#); [Marshall and Kelch, 1979](#)). Exogenous administration of GnRH results in the normal release of LH and FSH, suggesting that the pituitary is intact and that the abnormality exists at the level of the hypothalamus. The hypothesis that the site of the neuroendocrine dysfunction is at or above the hypothalamus, however, does not confirm whether weight loss, protein-calorie malnutrition, or a primary CNS defect results in amenorrhea. CRF hypersecretion may contribute to the hyposecretion of gonadotropins ([Olster and Ferin, 1987](#); [Rivier and Vale, 1984](#)). For example, intracerebroventricular (ICV) administration of CRF produces a dose-dependent decrease in pituitary LH secretion (without any effects on FSH secretion) ([Rivier and Vale, 1984](#)). The stress-induced suppression of LH secretion can be abolished by the central administration of CRF antagonists ([Rivier et al., 1986](#)). Therefore, intrahypothalamic CRF hypersecretion may inhibit GnRH neurons in the arcuate nucleus, via mechanisms that remain obscure.

The neuropeptides neuropeptide Y (NPY) and peptide YY (PYY) may also play a role in the neuroendocrine pathophysiology of eating disorders. NPY is secreted by the hypothalamic arcuate nucleus and then acts on the hypothalamic paraventricular nucleus to mediate increased eating and decreased energy expenditure ([Kaye et al., 1998](#)). ICV administration of NPY to experimental animals produces several physiologic and behavioral changes similar to anorexia nervosa. These changes include gonadal steroid mediated effects on LH secretion ([Kalra et al., 1986](#)), suppression of sexual activity ([Clark et al., 1985](#)), increased CRF release in the hypothalamus ([Haas and George, 1987](#)), and hypotension ([Fuxe et al., 1983](#)). Underweight anorexics have been found to have significantly elevated concentrations of CSF NPY compared to normal volunteers that normalized with recovery (although amenorrheic anorexics continued to show some degree of CSF NPY elevation) ([Kaye et al., 1990](#)). This elevated NPY is clearly not effective in stimulating eating in underweight anorexics but may possibly play a role in the obsessive and paradoxical food interests shown by many anorexics ([Kaye et al., 1998](#)).

CSF PYY concentrations in anorexics were not different from controls ([Kaye et al., 1990](#)), but PYY may play a role in bulimia nervosa. ICV injection of PYY into rats caused massive food ingestion to which tolerance did not develop ([Morley et al., 1985](#)). CSF PYY in normal weight bulimics who were actively bingeing and vomiting were similar to controls, but CSF levels after 1 month of abstinence from bingeing and purging were significantly elevated in bulimics ([Kaye et al., 1990](#)). It is possible that increased CSF PYY early in recovery from bulimia nervosa may contribute to the drive to binge and contribute to the risk for relapse in this disorder.



The recently discovered adipose hormone leptin has also been studied in eating disorders. Leptin changes in anorexia nervosa thus far appear to be consistent with normal physiologic reactions to weight loss ([Kaye et al., 1998](#)). Leptin signaling may play a role in the suppression of reproductive function seen in anorexia nervosa ([Kopp et al., 1997](#)). Leptin also does inhibit the starvation related elevation of NPY in the hypothalamus ([Stephens et al., 1995](#)). The role of leptin in eating disorders continues to be the subject of investigation.

The HPA axis is hyperactive in anorexia nervosa, based on the observations of elevated basal cortisol secretion, increased cortisol responses to stress, an increased number of cortisol secretory episodes per day, and decreased feedback inhibition (based on abnormal dexamethasone suppression test). As patients gain weight, the rate of cortisol secretion normalizes and feedback inhibition is restored, suggesting this defect is state-dependent ([Kennedy and Garfinkel, 1987](#)). Abnormal dexamethasone suppression tests are also characteristic of depression, bulimia without weight loss, late-stage Alzheimer's disease, and protein-calorie malnutrition. Increased plasma cortisol concentrations may reflect both a decreased metabolism and increased HPA axis activity, the latter further suggested by increased CSF levels of CRF in underweight patients ([Kaye et al., 1987](#)). Increased plasma cortisol levels also occur in starved human subjects who do not have anorexia nervosa or depression ([Fichter and Pirke, 1986](#); [Pahl et al., 1985](#)). A generalized reduction in metabolic rate occurs as a physiologic adaptation to protein-calorie malnutrition and cachexia. Anorexia nervosa, bulimia, and MDD share neuroendocrine abnormalities, anorexia, amenorrhea, decreased libido, hyperactivity, agitation, and depression. Thus, it is likely that hypercortisolism, an abnormal dexamethasone suppression test, and hypersecretion of CRF in various neuropsychiatric syndromes reflect a common CRF-mediated stress pathophysiology rather than being etiologic factors unique to any particular disorder.

Patients with anorexia nervosa have increased GH levels; these are restored to normal with increased caloric intake ([Kennedy and Garfinkel, 1987](#)). Increased secretion of GH occurs in protein-calorie malnutrition with chronic hypoglycemia, thereby further supporting the hypothesis that neuroendocrine abnormalities in these two conditions are related to reduced caloric intake. Diminished GH responses to insulin-induced hypoglycemia and L-Dopa, and increased GH responses to GHRF (but not clonidine), occur in anorexic patients ([Brambilla et al., 1989](#)).

Other neuroendocrine disturbances in eating disorders do not suggest that their etiology is related to central neuroendocrine dysfunction ([Kennedy and Garfinkel, 1987](#)). Thyroid function is characterized by a reduction in peripheral T<sub>3</sub> concentrations and increased concentrations of the inactive reverse T<sub>3</sub> in the face of normal circulating levels of TSH and T<sub>4</sub>. These changes in thyroid hormones result from the decreased hepatic deiodination of T<sub>4</sub> to T<sub>3</sub>, typical of catabolic states. The low T<sub>3</sub> decreases the metabolic degradation of cortisol because T<sub>3</sub> replacement in anorexic patients results in a shorter half-life for cortisol. The TSH response to TRH is also blunted in bulimia, similar to the responses seen in MDD, while anorexic patients exhibit a delayed but not blunted TSH response.

Patients with anorexia nervosa exhibit defects in urinary dilution and concentration that result from abnormal secretion of AVP ([Gold et al., 1983](#); [Nishita et al., 1989](#)). Cold intolerance is characteristic of anorexia nervosa and may be related to a central defect in temperature regulation in the hypothalamus. Anorexic patients also manifest various abnormalities of monoamine secretion ([Kennedy and Garfinkel, 1987](#)). Decreased CNS noradrenergic activity and turnover may be a trait marker for anorexia nervosa, as suggested by decreased urinary, plasma, and CSF concentrations of 3-methoxy-4-hydroxy-phenylglycol (MHPG), the norepinephrine metabolite; low CSF levels of norepinephrine; decreased CSF and urinary homovanillic acid (HVA)—which remain unchanged with long-term recovery. Decreased CSF and urinary concentrations of 5-hydroxyindoleacetic acid (5-HIAA), the serotonin metabolite, which normalize with weight gain, suggest state-associated disturbances of serotonin metabolism, in contrast to the state-independent changes in norepinephrine metabolism. Bulimic patients also have decreased central norepinephrine and serotonin function, which can precede, accompany, or follow behavioral changes. Their role in the etiology of eating disorders remains uncertain because similar changes in neurotransmitter metabolism may result from caloric deprivation or weight loss per se. However, it is of interest that several of the selective serotonin reuptake inhibitor antidepressants such as fluoxetine have been demonstrated, in double blind, placebo-controlled clinical trials, to be efficacious in the treatment of bulimia.

#### *HYPOTHALAMIC OBESITY*

A number of endocrine and genetic causes can lead to hypothalamic obesity, including Cushing's disease, polycystic ovary syndrome, castration, and Prader-Willi syndrome, to name a few. Although hypothalamic obesity was described over 100 years ago ([Bray and Gallagher, 1975](#)), studies using stereotaxic lesions have shown that small lesions in the paraventricular nucleus ([Fukushima et al., 1987](#)) or the ventromedial nucleus (VMN) of the hypothalamus ([Parkinson and Weingarten, 1990](#)) can produce hypothalamic obesity. Lesions in the PVN produce hyperphagia leading to obesity (prevented by limiting caloric intake), whereas VMN lesions cause obesity by complex alterations in autonomic function (increased parasympathetic activity and decreased sympathetic activity) ([Bray, 1992](#)). Following a VMN lesion, vagal efferent activity increases acutely (with increased insulin secretion), the thermogenic component of sympathetic activity decreases ([Bray et al., 1990](#); [Sakaguchi et al., 1988](#)), and GH secretion decreases because of injury to GHRH-producing cells ([Bernardis and Frohman, 1970](#)). These neuroendocrine changes lead to decreased energy expenditure for growth or temperature regulation and increased retention of ingested calories as fat. In humans, hypothalamic obesity is often associated with impaired diurnal rhythms, somnolence, and abnormalities in temperature control ([Bray, 1992](#)). Prader-Willi syndrome, characterized by hypogonadism, central obesity, delayed puberty, hypotonia, and mild mental retardation, is associated with deletions or translocations on chromosome 15 or with a normal karyotype. Obesity in these patients is related to a remarkable hyperphagia and hypoactivity. Behavioral difficulties also include cognitive and language problems, temper tantrums in infancy, and poor relationships with peers ([Butler, 1990](#); [Cassidy and Ledbetter, 1989](#)). Thus, central or peripheral abnormalities in the HPG axis may lead to altered caloric intake and metabolism and should be considered in obese patients with behavioral and psychiatric illnesses.

#### *PRECOCIOUS PUBERTY*

Pubertal development before the age of 8 years is defined as precocious puberty. The etiology of precocious puberty can be classified into peripheral, central, and combined causes. Central precocious puberty results from the premature activation of the HPG axis by GnRH or gonadotropins. Peripheral precocious puberty is a gonadotropin-independent process that results from the excessive production of adrenal or gonadal steroids or from ingestion of exogenous sex steroids. Combined precocious puberty involves activation of the HPG axis following abnormal production of sex steroids in the adrenal glands or gonads. The diagnosis and treatment of precocious puberty is not discussed any further here; the interested reader may refer to several recent reviews ([Breyer et al., 1993](#); [Pescovitz, 1990](#); [Reichlin, 1998](#); [Stein, 1992](#)).

Behavioral disturbances, development of breasts and pubic hair, and accelerated growth with premature epiphyseal closure are common early manifestations of precocious puberty. Children with precocious puberty often have poor self-esteem and difficulty with peer and parental relationships ([Jackson and Ott, 1990](#)) and may be at high risk for sexual abuse because of their cognitive and emotional immaturity.

#### *TRANSSEXUALITY AND HOMOSEXUALITY*

The neurobiologic basis for sexual and behavioral orientations has been investigated for several decades. Sex hormones are thought to be the major biological determinants of sexual orientation because of their well-known effects on somatic sexual differences and the development of sexual dimorphism in the brain ([Witelson, 1991](#)). Despite recent advances in neurobiology and behavior, the relative importance of biological and psychosocial factors, and their complex interactions in the development of sexual orientation are poorly understood. Studies associating sexual orientation with systemic sex hormone levels during adolescence and adulthood yielded largely negative results, especially in men (Meyer-Bahlberg, 1984, 1991). Psychoneuroendocrine research on transsexuality and homosexuality is therefore focused on the role of prenatal or perinatal sex hormones on the development of structural sex differences in the brain and on the gender-specific repertoires of reproductive and nonreproductive behaviors ([Gerall et al., 1992](#); [MacLusky and Naftolin, 1981](#); [Tallal and McEwen, 1991](#)).

In many species, the hypothalamus and adjacent areas of the brain undergo sexual differentiation following exposure to sex steroids at a species-specific sensitive interval during the perinatal period ([MacLusky and Naftolin, 1981](#)). Sex differences noted in the human brain are listed in [Table 7.3](#). The sexually dimorphic nucleus (SDN), located in the preoptic area of the hypothalamus, has been posited to be involved in sexual orientation and gender identity; its size is reportedly 2.5 times greater in men and contains twice as many neurons as compared to the number in women ([Gorski, 1985](#)). The size, number, and morphology of neurons in the SDN are determined by exposure to sex hormones in the perinatal period ([Gorski et al., 1978](#)). Small-sized SDNs were found in the brains of transsexuals as compared to heterosexual men ([Swaab and Hofman, 1988](#)), suggesting a role for this nucleus in male homosexuality.

Some of these structural sexual differences in the brain can be produced by exposure of the female fetus or newborn to androgenic hormones. Postnatal treatment of female gerbils with testosterone (or diethylstilbestrol, DES) increased the relative sizes of the SDN and suprachiasmatic nucleus (SCN), whereas similar treatment during adulthood had no effect. Increase in the size of the left SDN was associated with male-type courtship vocalizations ([Holman and Hutchison, 1991](#)). The effects of testosterone are located in sexually dimorphic areas of the hypothalamus and limbic areas associated with reproductive functions ([Wozniak et al., 1992](#)). Administration of testosterone with an aromatase cytochrome P-450 inhibitor or an estrogen antagonist abolishes the effects of testosterone on sexual behavior. The

mechanism of action of testosterone and other androgens therefore appears to depend on their *in situ* aromatization in these areas (Hutchison and Beyer, 1994). Further, the expression of aromatase mRNA in the mouse diencephalon was significantly increased by testosterone via transcriptional control (Yamada et al., 1993). This mechanism may also explain why prenatal exposure to DES, an estrogen, has behavioral and morphologic consequences similar to those of androgen exposure (Herbst, 1981). The control of reproductive behaviors by neuroendocrine mechanisms remains under active investigation.

The importance of these findings results from the exposure of human fetuses to DES, which is thought to be related to altered sexual behavior and the development of homosexual orientation (Meyer-Bahlburg et al., 1995). Human females exposed to prenatal androgens because of congenital adrenal hyperplasia or DES were more masculine in their childhood behavior than their own sisters or unrelated control subjects. These females also showed increased physical activity; increased preference for rough-and-tumble and pursuit play and male-typical toys and activities; and a decreased preference for parental doll play, and female-typical toys and activities. These girls were also more likely to emphasize careers over childbearing in their fantasies about adult life (Berenbaum and Snyder, 1995; Ehrhart and Baker, 1974; Money and Ehrhardt, 1972). Many of these changes in behavior and interest have been shown to persist into adolescence in patients with congenital adrenal hyperplasia (Berenbaum, 1999). However, DES exposure was not shown to produce significant changes in neuropsychological tasks that commonly show gender differences in performance, when DES-exposed women were compared to their nonexposed sisters (Hines and Sandberg, 1996).

Atypical gender role behavior during childhood was documented by history in about two-thirds of homosexual men and women, interpreted as having crossed the line into the other gender's role-play and identity during childhood (Bell et al., 1981; Whitman and Mathy, 1986). To some extent, the neuropsychological differences between homosexual and heterosexual men resemble those found between heterosexual men and women (McCormick and Witelson, 1991). Hormonal studies in transsexuals have been inconsistent thus far (Bosinski et al., 1997).

We must emphasize that, even if a neuroendocrine basis for sexual orientation is confirmed, this may apply to only a percentage, currently unknown, of homosexual men and women, and that the importance of learning, psychosocial role models, and other cultural factors is not diminished by these hypotheses or supporting data.

## FUTURE DIRECTIONS AND DEVELOPMENTAL PSYCHONEUROENDOCRINOLOGY

The past decade has witnessed remarkable advances in neuroscience in general, and in molecular neurobiology and structural and functional brain imaging in particular. All of these approaches can be applied to developmental psychoneuroendocrinology, both basic and clinical, with the resultant incremental advance in our knowledge of developmental neuroscience, a discipline that has always lagged behind the rest of neuroscience because essentially the CNS, particularly the brain, develops differently virtually on a daily basis. As such, neuroanatomists, neurochemists, and behaviorists have to some extent steered clear from this complicated and difficult to untangle area because the CNS substrate changes so rapidly. Indeed, we still do not have at our disposal a high-resolution developmental brain atlas for most mammals, including humans.

Further measurement of the expression of various genes encoding for neurotransmitters, neurotransmitter-linked enzymes, and neurotransmitter receptors and their regulation in development is needed. This will undoubtedly help understanding of complex phenomena such as the timing of puberty, maturation of the stress response, and the pathophysiology of a variety of important clinical disorders, such as anorexia nervosa, precocious puberty, and even complex behavioral alterations such as sexual preference.

Perhaps the greatest promise for clinical studies to help us elucidate the pathophysiology of childhood disorders, such as autism, affective disorders, anxiety disorders, eating disorders, and ADHD, lies with functional brain imaging. Techniques that do not involve radiation exposure show promise for safely studying brain activity in children and adolescents. These methods, combined with structural brain imaging, will undoubtedly point to particular brain regions that need to be further scrutinized in these devastating disorders. In addition, reproducible abnormalities established using functional brain imaging in symptomatic patients then could be followed after treatments for these disorders to determine whether markers for such disorders can be identified. There is great promise that the next decade will bring continued advancements in this area, far beyond what we might have hoped for in the past.

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## 8 BRAIN AND BEHAVIOR

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The boundary between biology and behavior is arbitrary and changing. It has been imposed not by the natural contours of the disciplines but by lack of knowledge ([Kandel, 1991a](#)).

It is not the purpose of this chapter to review in detail the nature of brain structure and function, nor behavior minutely dissected, both of which are covered in other sections of this volume and in specialized reviews and texts such as [Kandel and colleagues \(2000a\)](#). Rather, discussion is restricted to a broad-brushed canvas of the relationship between brain and behavior (used in its widest meaning), concentrating on the principles that are germane to child and adolescent psychiatry. In so doing, certain points must be borne in mind:

First, child and adolescent psychiatry is, above all, concerned with developing behavior and biology, which although continuing throughout the life span, will not again do so at such a fast pace until the last of the Seven Ages of Man (this time negatively). Put another way, it is not that development is irrelevant to mature life; it is just that the pace is slow enough for it to be conveniently ignored, whereas with children and youth, the fact is obtrusive and inescapable.

Second, scientific knowledge applicable to brain and behavior is now so vast and developing so rapidly that no one person can hope to grasp more than an inkling of their interrelationship. Nevertheless, although knowledge (especially in molecular biology and neuroimaging) is expanding rapidly, there is still a remarkable continuity of knowledge over time in neuroscience, as revealed by scholarly texts that have a sense of history such as [Kandel and colleagues \(2000a\)](#). Thus, what was learned in medical school or residency training is far from totally obsolete or useless in understanding brain and behavior today and puts the fundamental principles within the grasp of any child psychiatrist or mental health professional with training in human biology. Particularly compelling is a slow but significant and escalating change in child psychiatry over time. There has been a rediscovery of the idea that many psychiatric disorders once considered psychogenic have primarily biological causes (for example, autism); genetic and bioenvironmental contributions to the development or expression of psychiatric disorders is becoming clearer; and the concept of neurodevelopment in the etiology of some psychiatric disorders such as schizophrenia ( [Weinberger 1987](#), [Weinberger 1995](#) ) has increasingly shown that child psychiatry will assume a key role in the definitive prevention of these adult disorders (see the following).

Third, bear in mind that, despite all the advances, there is still a yawning gap between the laboratory and clinical practice. Most clinical decisions must be made with an imperfectly garnered, largely pragmatic data set that at best touches only lightly on brain–behavior functions. This knowledge gap often produces two extreme positions—those who crudely crash it and see a disordered brain behind everything clinical, and those who, apart from an occasional perfunctory genuflection to the brain, act as if all behavior is determined solely by intrapsychic, interpersonal, or (nowadays) intrafamilial processes. In fact, a good grasp of brain–behavior relationships is one of the unique things that psychiatrists bring to the multidisciplinary team and is a way to command respect for this complex area of science without overvaluing its role in clinical decisions.

However, looking at the development of biological psychiatry since 1950, it is not hard to see that in some areas etiologic breakthroughs that will radically affect clinical practice are imminent. Most of these will affect the major psychiatric disorders (such as attention deficit hyperactivity disorder, obsessive-compulsive disorder (OCD), schizophrenia, and Tourette's disorder), which form an increasing part of child and adolescent psychiatry. Perhaps more important in terms of its larger clinical applicability, better understanding will come too of the genetic and molecular biological basis of human variability ( [Chapter 1](#), [Chapter 2](#), [Chapter 3](#), [Chapter 4](#), [Chapter 5](#), [Chapter 6](#) and [Chapter 7](#) ) and how its derived macrofunctions, such as temperament, cognition, and other developmental abilities, interact with experience to create psychopathology ([Lewis, 1992](#); [Shaw and Bell, 1993](#); [Taylor, 1991](#)). From this knowledge, it is but one step to biologically based modifications, some therapeutic, but most excitingly, some preventive, in a true pediatric psychopharmacology. For this, if no other reason, the child and adolescent psychiatrist must try to stay abreast of developments in brain–behavior relationships.

### GENERAL PRINCIPLES

#### Concepts of Brain–Behavior Relationships

The best way to introduce fundamental concepts is by way of a historical review. The notion that the brain influences behavior is fairly recent, appearing significantly only with 19th century phrenology ([Kandel, 2000a](#)). Before that, humoral (i.e., endocrinologic) theories, derived from Greek medicine, seem to have predominated and gave such words as sanguine, melancholy, and choleric. Hippocrates (460 to 377 BC) summarized the humoral theory of “diseases,” which were conceptualized as imbalance between fluids (“humors”), and there was no dichotomy between disorders of the body and disorders of the “soul.” Descartes (1596 to 1650) established the landmark for the “Cartesian dualism” of body and mind, with the belief that it was impossible that mental and physical interact.

Some of the basic ideas about how the brain influences behavior (and vice versa) arose over 150 years ago.

#### Localization Versus Equivalence

The phrenologists who mapped 35 higher brain functions, such as benevolence, hope, conjugality, and secretiveness, to specific bumps on the skull were the first exponents of one of the two recurring themes: neurobehavioral theory and localization of function. According to [Kandel \(2000a\)](#), the phrenologists were debunked by Flourens, who removed parts of the brains of animals and, finding no site-specific change in behavior, simultaneously created the other evergreen theme, equivalence, which holds all parts of the brain to be capable of performing any function. The reader should note these two themes of localization and equivalence well because they still continue to leapfrog each other in popularity, especially in the area of higher functions such as cognition and emotion. It appears that we are well into another localization era, derived from developments in pharmacology, neurotransmitter physiology, and brain imaging, but it was not always so.

The prime example of a persistent equivalence view in child psychopathology is that of minimal brain damage/dysfunction, in which it is the amount of brain damage, not its site, that is believed to determine the syndrome. As positron emission tomography (PET) scans have shown, both views are correct and complementary—many functions are localized but their learning, execution, or integration with others requires most of the brain to be involved.

The newest themes popularized by neuroimaging researchers are functional anatomy (structures with specific functions) and connectivity (patterns of flow of information between brain areas forming a functional “network” or “circuit”).

### Hard and Soft Effects

Many years ago [Teuber \(1960\)](#) drew attention to another important concept associated particularly with abnormal brain function, that of the hard, or deterministic, view in which the brain is seen as totally determining observed behavior. This view was popular in the eugenics era around the turn of the 19th century and, in child psychiatry, after the encephalitis pandemic of the 1920s in what we now call conduct disorder. A more modern example is seen among those who explain behavior in children, especially younger ones, entirely on the basis of temperament via such concepts as “the difficult child syndrome” without reference to the flow-on effects of interactions between parent and difficult infant, infant and difficult parent, or both ( [Shaw and Bell, 1993](#)) ([Chapter 14](#) and [Chapter 15](#)).

The opposite of the hard is the soft view, in which the brain is seen as irrelevant before the much more powerful influence of experience, a view that seems to have dominated much of child psychopathology research between 1940 and 1970 and is today seen in the thinking of far too many child mental health professionals in (unwitting) parent-blaming.

In fact hard and soft views are less categorical than extreme poles of a single axis, so that intermediate views ( [Eisenberg, 1957](#); [Lewis, 1992](#); [Lewis et al., 1989](#); [Shaw and Bell, 1993](#)) emphasize that behavior is a downstream endpoint in the interaction between brain function and experience. The explosion of molecular genetics and functional neuroimaging research endorses this longer-held view. Concepts of genetic determination soften as the role of experience on genetic coding becomes more apparent, and the plasticity of the neural machinery is revealed in an ebullient new field of neuroscience. This interactive view between biology and experience then seems both the most sensible and valid one. It might suggest that, as indeed it seems at times to do, in view of the enormous variation in human experience and the transactional effect of development, detection of any meaningful relationships between brain function and behavior in children will be impossible—that is, each child is unique. Science does not accept this pessimistic view and seeks to reduce experience to a manageable number of types, as indeed do most developmental theories, whatever their theoretical orientation, for example, attachment or social learning ( [Shaw and Bell, 1993](#)).

## THE BRAIN

### How the Brain Works

As detailed in the previous chapters of this section, the brain consists of billions of cells and their processes—neuronal, which do the work of the brain, and glial, which support, nourish, and clean up the neurons. The principles of neuronal function and synaptic transmission have already been described in previous chapters in this section and/or are too well known to need detailing. More important for the purposes of this chapter is how these individual cells are organized to do the work of the brain. There are two main ways of operating: point-to-point or one-way traffic, rather like telegraph or fax systems. The other is more like the telephone, in which communication goes back and forth between sender and receiver, although in the brain, two electrically separate cables are used. The back-and-forth telephone model is much more characteristic of most brain function than the telegraph model. What were formerly thought of as unidirectional way stations like the thalamus or basal ganglia are now known to transmit, receive, and modulate in a perpetual dialog. Because axonal and synaptic transmission is so ultrarapid, it is clear that some kind of way of keeping things alive and active is needed for slower processes such as memory, thinking, worrying, and the like. This is achieved by reverberating circuits, some of which (e.g., the Papez or emotional/memory circuit) are very extended. Individual neurons and their processes become organized partly by genetic programming and partly by experience, into units called cell assemblies ( [Hebb, 1949](#)), which are very like integrated circuits or chips. These become the functional units in much of the brain's actions, although they do have connections in parallel and series outside these assemblies, very importantly, allowing for cross talk.

### Development of the Brain

This is discussed in detail in this book ( [Chapter 3](#)) and elsewhere ( [Casaer, 1993](#); [Ebels, 1980](#); [Jacobson, 1991](#); [Kandel et al., 2000](#)). Life begins as a single cell, yet the billions of neurons are created, basically differentiated, and crudely in place by birth and by and large are nonreplaceable. However, from later fetal life on, the process shifts from skeletal construction to one of embellishment or differentiation and fine-tuning. Modern developmental neurobiology holds that most of the fundamental organization and architecture of the brain, differentiation of neurons, and axonal development is preprogrammed and uninfluenced by anything but adverse and physical environmental experiences, yet it is also estimated that there are sufficient genes to program only a fifth of the brain's neurons, leaving huge areas of flexibility on which experience can work and that form the basis of learning.

Postnatally, the most significant developments in the brain are: (a) the development of interconnections shown microscopically by an enormous proliferation of dendritic and axonal processes and (b) myelination of axons, which greatly enhances the speed and efficiency of nerve conduction ( [Ebels, 1980](#)). Although approximately 75% of brain growth (as shown by weight) occurs by the age of 2 years ( [Carmichael, 1990](#)), these two processes of differentiation continue ( [Casaer, 1993](#)), although at a decelerating rate, until adulthood. Even thereafter, increase in myelin density ( [Kolb and Whishaw, 1980](#)) and axonal and dendritic growth continue throughout life until final senescence. This means that at the level of cell assembly organization or complex circuitry the brain is actually in a constant state of change, and this is probably one mechanism by which learning occurs.

### Disruptions in Brain Development

As can be seen from previous chapters, the initial process of neurogenesis and development of brain architecture is a highly complex and surprisingly rapid process. It is obvious that brain development should be most susceptible to disruption during the most rapid phase of neurogenesis and organization, when any disruptions may have gross and catastrophic results, such as congenital malformations and severe mental retardation. As pointed out in [Chapter 3](#), there are two major types of disruptions of normal development—pathogenic “software” owing to abnormal genes/chromosomes and errors in execution of the genetic programming induced by subtle changes in the intrauterine environment, such as those induced by viral infections or biochemical abnormalities, for example, from alcohol, tobacco, and drug abuse, maternal malnutrition, placental dysfunction, maternal vomiting, and even stress ( [Leckman et al., 1990](#); [Taylor, 1991](#)) ([Chapter 7](#)). Being highly specialized in function, neurons are exquisitely sensitive to such changes in the biochemical environment—its acidity, ionic composition, and supply of essential nutrients, particularly oxygen and glucose. Both of the latter are vulnerable to marked change in blood levels (e.g., during abnormalities of pregnancy and delivery) and in demand according to the brain activity, which is never zero, because a mature neuron maintains a basal tonic level of readiness for action that is essential to normal function.

As knowledge grows about the surprisingly early and critical role in initial brain organization of neurotropic messengers, most of which are normal biochemical substances that will later fulfill endocrine, such as androgens ( [Peterson et al., 1992](#)) ([Chapter 6](#) and [Chapter 34](#)), and neurotransmitter ( [Casaer, 1993](#)) function, concern must mount about the possibilities of subtle brain disruptions. For example, [Casaer \(1993\)](#) points out that receptors in the cerebellum become sensitive to g-aminobutyric acid (GABA) before its actual productive and, more importantly, inactivation mechanisms develop. He argues that benzodiazepines given to the mother, therefore, might have powerful effects in fetal life. The same may be true for the effect of nicotine *in utero*, because the cholinergic receptors and enzymes are present in the brain very early in life ( [Ernst et al., in press](#)).

Injury and local brain disease can also result in direct loss of nervous tissue, so that after these prenatal pathogens, the most dangerous point is often assumed to be birth. However, the most destructive force at this time is not direct injury to the brain, but that induced by anoxia and hypoglycemia, especially when combined with prematurity ( [Werry, 1986](#)). Modern advances in obstetric and neonatal care have greatly reduced the frequency of this kind of brain disruption, and it is possible that



postnatal head injury is now more of a threat to brain development in children.

Much of the controversy about brain and behavior ([Kreusi, 1990](#); [Rutter, 1982](#); [Taylor, 1991](#); [Werry, 1986](#)) does not involve gross and obvious abnormalities of development usually resulting in one or more of the neurologic “big three” (mental retardation, cerebral palsy, and epilepsy), but rather focuses on the role, compared with that of the social environment, of minor congenital abnormalities or prenatal, perinatal, and postnatal insults to the brain ([Taylor, 1991](#)) in the production of extreme temperamental types such as the “difficult child” or psychiatric disorders such as attention deficit hyperactive disorder (ADHD), developmental learning, tic and OCDs ([Leckman et al., 1990](#)), and schizophrenia ([Werry and Taylor, 1994](#)). For example, there is a close association between social advantage and school performance, but how much actual physical brain development directly contributes to this is unclear ([Madge and Tizard, 1980](#)). One study of children who had marasmus showed that malnutrition and, presumptively, the resultant brain maldevelopment have some effect independent of social environment. The effect on IQ was small, but there were much larger effects on more subtle cognitive functions such as attention and also on emotional stability ([Galler et al., 1983a](#), [Galler et al., 1983b](#), [1985](#)). This along with other evidence suggests that effects may be there but less obvious ([Taylor, 1991](#)). Increasing overpopulation and putative climatic change leading to famines in the horn of Africa and other Third World areas stress the potential devastating importance of this pathogenic process.

## Recovery from Brain Disruptions

### GENERAL PRINCIPLES

Despite the preceding litany of risk factors, one thing is clear about most brain disruptions—there is no simple relationship between brain insult and brain development ([Taylor, 1991](#)). For one thing, there is both a high threshold to injury ([Taylor, 1991](#)) and a strong tendency to recovery or increasing attenuation ([Kreusi, 1990](#)). The basis of this is disputed and probably multifactorial. One factor could be surplus neurons because, as noted, there is a large redundancy of neurons (as high as 50%) gradually lost by adulthood through neuronal death owing to disuse ([Casaer, 1993](#)). Some is owing to cleanup of the products of inflammation, necrosis, trauma, bleeding, and so on. Loss of neurons may be compensated to some degree by development of more dendritic processes, thus getting a bigger bang for the neuronal buck, as it were. Such development, however, is an active process that requires stimulation and so lends plausibility to the hypothesis that active rehabilitation and learning programs should have a role to play in repairing brain insults. But probably the most important factor is that of parallel functions: When one way is obstructed, there is always another. In the brain, this parallelism is derived in part from the fact that chains of neurons are probably connected in series and in parallel. But it is not only neurons that exhibit parallelism; so too do the elementary subfunctions of higher information processing, so that “neural processing for a given function is distributed within the brain and handled at several sites” ([Kandel, 2000a](#)). However, this principle is limited and least true of, for instance, motor or sensory ones, functions that are simple or highly specific and sharply topographically localized with point-to-point representation.

### AGE AND RECOVERY

It is often held that brain injury occurring early in life has a better prospect of recovery because of the putative increased neuronal plasticity, belief that stemmed from the Kennard principle that posits that the earlier the lesion, the better the outcome. This principle is based on the work of Margaret Kennard on the effects of age a lesion onset in primates where cortical lesions made on young animals had less effect on behavior than have similar lesions in adults ([Kennard, 1938](#), [Kennard, 1940](#)). Certainly, all the prospective studies of putatively brain-damaging prenatal and perinatal situations have shown that many initial effects are often largely undetectable by the start of school ([Kreusi, 1990](#); [Rutter, 1981](#); [Werry, 1986](#)). The counter view is that of critical periods, well demonstrated in the area of the development of vision, where early disruption (e.g., strabismus) can lead to functional blindness (amblyopia, [Chapter 3](#)). In a review of the empirical evidence, [Fletcher and Satz \(1983\)](#) concluded that the only thing that makes early damage look less disabling is the increased length of time between assessment of function and damage, or, in short, that time after injury, not age, may be the critical determinant of recovery. In fact, they concluded that there is some evidence that early damage may be more serious than later damage. [Taylor \(1991\)](#) also points out that regrowth and reconnection may not always be beneficial, but may produce new anomalies and that the early consequences (notably motor problems) may be different from the late one (cognitive) and require more subtle detection methods.

Nevertheless, some degree of recovery continuing for a long time is almost inevitable and may give intensive rehabilitative treatments a credibility that they do not deserve.

## Experience and the Brain

So far the emphasis has been on disruptions in the brain, but what evidence is there that experience may influence brain development? Animal experiments have shown that the weight of the brain, complexity of dendrites, and synaptic size are all significantly greater in animals exposed to an enriched environment and that these morphologic changes are matched by improved performance. The increases are greatest if stimulation is given early in life but are still apparent even if applied later ([Kolb and Whishaw, 1980](#), [1995](#)). [Kandel \(2000a\)](#) summarizes the process of brain development: Synapse formation is largely under genetic control, but when in place, synapses require appropriate environmental stimulation for their activation and fine-tuning. Some of this activation may have a critical time frame (e.g., the development of binocular vision, affection bonds, or fluency in a language). After the laying down of synapses, experience works throughout the life span primarily by influencing the effectiveness of these synapses—downgrading some (unlearning) and upgrading others (learning). This view of synaptic strength, which is attributable to [Hebb \(1949\)](#), has much experimental support but does not take account of the fact that the neuronal processes are actually in a constant state of growth, with synapses disappearing and reorganizing. It seems likely that the concept of synaptic strength is conceptually correct but anatomically incorrect. However, it matters little for our purposes; the important and obvious thing is that experience alters the brain.

## Methods of Studying Brain Function

No attempt will be made to cover these in detail. For more detail see other sections and chapters of this book ([Chapter 9](#)), and [Ernst and Rumsey \(2000\)](#), [Rutter \(1981, 1982\)](#), [Taylor \(1991\)](#), and [Werry \(1986\)](#). In humans, because of obvious ethical constraints, natural clinical states such as disease, injury, malformation, and so on must be utilized. Unless the patient dies, the full extent of the abnormality is rarely known, and the child psychiatry literature is full of methodologically pitiful studies that have aggregated “brain-injured” children when there is good reason to suspect that the site, extent, and nature of the brain damage differed widely. Even in disorders of similar etiology such as head injury ([Chapter 33](#)), the variation in brain damage can be enormous, not only because of differences in the site and force of the impact, but also because of variable disseminated or far-distant effects such as deceleration, contrecoup, shearing, and torsion effects. Add to all this, the variation in postinjury experience, and the situation can be seen to be most complex. Only large single effects are likely to be detectable in the usual-size sample of considerably fewer than 100 subjects.

### CLINICAL METHODS (HISTORY AND EXAMINATION)

These are the most popular, yet among the poorest because they are often unreliable, of unknown validity, only lightly correlated with brain damage, and subject to errors of detection, especially for areas of the brain that are of most interest in psychopathology. Much of the study of brain function in child psychiatry before 1980 falls in this category and is quite error-prone ([Rutter, 1981](#); [Werry, 1986](#)).

### PSYCHOPHYSIOLOGY

In more recent years, the crude and largely noncontributory technique of electroencephalography (EEG) has been replaced by more functionally oriented methods such as evoked potentials and other techniques ([Chapter 9](#)), many of which have attained great sophistication through computer averaging, mathematical analysis like power spectral analysis, and a general growth in knowledge about each of the components of the characteristic waveform. In general, evoked potentials ([Chapter 9](#)) are most suited to studies of general brain states such as arousal and vigilance or gross intactness of the sensory apparatus and brain as a whole. However, they have also offered some useful if very technical information on the development of certain brain processes such as attention ([Lincoln et al., 1990](#)) and for the functional analysis of pharmacological effects such as that of the stimulants ([Coons et al., 1987](#)). The combination of EEG and magnetic field techniques has given rise to the new if still complex and costly magnetoencephalography imaging (MEG) based on surface electrical recording ([Rojas et al., 2000](#)). MEG signal represents a direct index of neural activity, which is not the case for most other functional neuroimaging methods such as functional magnetic resonance imaging (fMRI), which measures changes in cerebral blood oxygenation and cerebral blood flow (i.e., indirect indices of neuronal activity).

### ANATOMIC AND FUNCTIONAL PATHOLOGY

These methods are largely limited to postmortem studies, and death is a rare event in most children with psychiatric disorders. It is of interest that good autopsy reports of disorders such as autism are only just emerging. The increasing problem of teenage suicide offers an unfortunate window of opportunity by making

postmortem studies of this population more common. Two recent contributions bear mention: Pandey (1997, 1999) measured two rather novel enzymes involved in signal transduction linked to receptors such as 5-HT 2a. This shows that even anatomic pathology is moving away from the relatively simple measurement of structure and quantification of receptors toward more refined functions such as second messengers.

## BRAIN IMAGING

Brain imaging methods are discussed in more detail in [Chapter 9](#).

Traditional methods, such as ventriculography, of very limited (gross anatomic pathology) and somewhat dangerous nature, have now given way to exciting developments even beyond computed tomography (CT) and magnetic resonance imaging (MRI). New tools that test brain function rather than inform about anatomic structures are revolutionizing the research on brain and behavior ([Chapter 9](#)). These new tools include functional MRI (fMRI) ([Eden and Zeffiro, 2000](#)), MRI ([Yurgelun-Todd and Renshaw, 2000](#)), MEG ([Rojas et al., 2000](#)), and the much more cumbersome PET ([Kuperman et al., 1990](#); [Herscovitch and Ernst, 2000](#); [Martin and Brust, 1991](#)) and single photon emission computed tomography (SPECT) ([Herscovitch and Ernst, 2000](#)). More emergent techniques include optical imaging ([Frostig et al., 1990](#)) and transcranial magnetic stimulation (TMS) ([Pascual-Leone et al., 1999](#)).

All these new methods are beginning to be used in an organized and logical fashion that offers promise in unveiling neural mechanisms mediating psychopathology. However, many challenges still need to be overcome, encompassing technical limitations, adequate theoretical models, and most important in research in children, ethical issues ([Arnold et al., 2000](#); [Ernst, 1999](#)). Despite all these new methods of studying the human brain, so far the clinical yield in child psychiatry has not been too impressive, except: (a) what it has ruled out as causes, (b) in demonstrating the role of neurodevelopment in the expression and changes over time of psychiatric symptoms, and (c) for clear indications that the role of brain factors in etiology of psychopathology is probably much more extensive than formerly believed ([Berthier et al., 1993](#); [Ernst and Rumsey 2000a](#); [Hendren et al., 1991](#); [Kuperman et al., 1990](#); [Leckman et al., 1992](#)).

## NEUROPSYCHOLOGY

Child psychiatry has had a long association with neuropsychology, since at least the days of [Strauss and Lehtinen \(1947\)](#). There is good reason for this, namely, the close relationship between school and clinic and the common association of behavioral and learning problems. Neuropsychology is now an advanced science typified in texts such as [Hynd and Obrzut \(1981\)](#), [Kolb and Whishaw \(1980\)](#), and [Lezak \(1995\)](#), to name but a few. Although such tests are extremely helpful in the assessment of major neurologic brain disorder, their role in most of the problems seen in child and adolescent psychopathology have been controversial at best ([Feagans, 1983](#); [Taylor, 1991](#); [Werry, 1986](#)). Much of the problem lay in the unknown reliability and validity of the tests in the detection and localization of brain disorders. Often they seem to have acquired a quasineurologic status that they may not deserve, especially in the area of learning disabilities ([Feagans, 1983](#); [Werry, 1986](#)).

However, with the advent of functional neuroimaging and techniques of cognitive activation, the neural substrates of many of the classical neuropsychological tests are now being better delineated. Impaired performance on these tests can now more clearly suggest dysfunction in specific brain regions or neural networks. In fact, neuropsychological testing paired with functional neuroimaging is becoming one of the more powerful strategies to study and understand the neural substrates of cognitive and emotional processes in health and disease.

This is not the way they are used in most clinical practices—yet. A recent review ([Halperin and McKay, 1998](#)), for example, points out that there is a common misconception, that large differences in verbal and performance IQ are pathological, whereas more than half the children in the standardization of the sample for the Wechsler Intelligence Scale for Children III (WISC III) had discrepancies greater than 9 points and about 25% greater than 14 points ([Wechsler, 1991](#)).

Projective testing (e.g., Rorschach) has undergone a resurgence of interest too because of supportive reliability and validity studies and the development of systematic ways of administration and scoring the Exner Comprehensive System.

In summary, neuropsychological testing remains an important adjunctive source of information for clinicians and researchers, but caution is needed in using these as indicators of brain function, especially localized brain function.

## BIOCHEMICAL AND PHARMACOLOGIC STUDIES

Again, this is a rapidly developing area. The past tendency to rely primarily on the passive analysis of blood, cerebrospinal fluid, and other body fluids has been improved by the use of active chemical probes of function analogous to the increasing use of evoked potentials in the electrophysiology field. The most useful instruments for this probe are pharmacologic substances, ever-increasing in number and specificity. This is well illustrated in modern theories of schizophrenia and major depression and in child psychiatry, of autism, Tourette's disorder, and ADHD ([Zametkin and Rapoport, 1987](#)) ([Chapter 4](#)).

However, there are limitations to this approach: Few drugs or their target receptors are specific to one functional area or system. The complex interactive nature of brain function, with the convergence of many different pathways at the effector level, may result in confusion of downstream or compensatory effects for the primary disturbance. Some of these may be responsible for the fact that the biochemical theories of schizophrenia and depression do not seem to have advanced much because the cellular actions of the drugs used in their management became known more than 30 years ago. Currently, the use of pharmacologic challenges has fallen somewhat out of favor as the newer neuroimaging techniques (e.g., MRS, PET, SPECT) permit direct probing of biochemical processes in the brain.

## GENETIC STUDIES

Good summaries of this area can be found in reviews by [Kreusi \(1990\)](#), [Propping and Hebebrand \(1990\)](#), [Shields \(1980\)](#), and [Chapter 1](#). The most important methods are family pedigree studies, twin studies, and naturally occurring experiments such as chromosomal abnormalities. The finding of some genetic role in the etiology of behavior cognitive disorders such as autism ([Propping and Hebebrand, 1990](#)), ADHD ([Goodman and Stevenson, 1989b](#)), or Tourette's disorder ([Leckman et al., 1992](#)) permits an inference that the brain is affected either directly or indirectly.

In the last few years tremendous strides in molecular biology have opened up the possibility of converting these assumptions into direct causative relationships. The best example of this is Huntington's disease, where the genetic location has been established, although not the precise biochemical mechanism. However, the past 10 years have been most notable for conceptual advances as opposed to such actual breakthroughs in the localization of genes that specifically account for syndromes or behavior.

The initial enthusiasm for molecular genetic approaches has now been tempered by the growing realization that most of the neuropsychiatric syndromes in child and adolescent psychiatry will be the result of multiple genetic effects as opposed to single genes, by risk factors rather than direct effects and nonshared environmental influences. Fortunately, exciting, complex new methodologies will facilitate such study.

## Summary

Methods of studying brain function and critical influences on it are developing rapidly and to most of us, dauntingly and bewilderingly. They show great promises but, although individually often alluring and sophisticated, still have a long way to go before they can help in pinpointing any role brain function might have in understanding and treating the complex dysfunctions seen clinically. Additionally, there are specific issues relating to children. Many of the indicators of brain dysfunction so useful in adults represent loss of function, whereas in children, the problem is one of failure to develop a function (e.g., enuresis) or, more commonly, slower or imperfect development (e.g., motor incoordination or dysphasia). Put another way, abnormality is often developmental or chronological and quantitative rather than qualitative. In this context it is hard to know whether the "abnormality" is merely a lag in development, a developmental variation (that can have experiential as well as physical causes), or a true abnormality. For example, it is said that both Einstein and Churchill began to talk very late, yet clearly, they had very good brain function. Or again, many of the so-called soft neurologic signs, such as sensorimotor incoordination, are, in fact, merely the kinds of function seen in somewhat younger children (or immaturities), and their status as true neurologic indicators is unclear ([Tupper, 1987](#)) ([Chapter 44](#)).

## BEHAVIOR



## Classification of Behavior

So far the talk has been largely of the brain. Now discussion moves to behavior, defined in the broad way of child psychiatry to encompass all activity of the organism beyond simple physiologic functions necessary to sustain life. There are certain conventions in classifying brain functions that offer a point of convergence with the behavior side of the brain–behavior equation. The neurologic view, derived largely from the study of disease and the ability to detect these diseases with simple clinical examinations, has typically been input (sensation/perception), output (motor), and in between (association). The latter, being much more complex and less reachable, has remained more elusive to study, yet this is the area of greatest interest to psychiatry. Neuropsychiatry has therefore developed somewhat different classifications, which are more dissected, more functional, less topographic, and more oriented to psychological theory, although compatible with the neurologic view. One of these taxonomies of behavior is as follows ([Lezak, 1995](#)).

1. Information processing or intellectual function, further subdivided into receptive function (acquisition, processing, classification, and integration of information); memory and learning (storing and recall of information); thinking (organization and reorganization of information); and expressive functions (communication and enacting of information). Mental activity, although closely allied to intellectual function, is not considered a basic function because it has no clear behavioral end product but is concerned with the efficiency of intellectual function, including attention, level of consciousness, and activity rate or speed.
2. Emotionality (feelings, motivation). This is one of the traditional domains of psychiatry, although some of the previously popular theories, such as Freud's, still require scientific validation.
3. Executive and control functions. These are capacities that enable a person to engage in independent, purposive, self-serving behavior successfully ([Lezak, 1995](#)). A survey of these three areas shows how germane they all are to child and adolescent psychiatry and how artificial are divisions among child psychiatry, pediatric neurology, and neuropsychology. If psychiatry can claim any particular specialization within this area, it must be to whole organism function, but even that is an artifact of poor medical practice, which has aimed to separate patient from illness, mind from body. However, it would be idle to claim that this classification of behavior is complete or satisfactory for much that concerns child psychiatry because it is essentially intraorganismic and pays little credence to the context in which behavior occurs, the stimuli that elicit it, or its social interactive, systemic, or developmental properties ([Shaw and Bell, 1993](#)).

## Development of Behavior

In view of extensive coverage in Section C of this volume, it is unnecessary to do other than summarize the main principles of child development here. If one tried to isolate the main themes in development, they would include the following:

### *LOSS OF STEREOTYPED REFLEXIVE BEHAVIOR*

The neonate and young infant have a series of preprogrammed behaviors, many necessary for survival, such as the rooting and sucking reflexes, which are lost in a few months. Other studies, particularly by ethologists (e.g., Bowlby), have identified other reflexes, such as stranger and separation anxiety, that prevent the newly mobile immature organism from straying from protecting caretakers, or the smiling reflex, and have shown how these initially automatic reflexes become the basis of socialization as they lose their automaticity. Compared with most other species, the human baby has relatively few of these automatic behaviors, and it is thought that this is because this places fewer obstacles in the way of the rapid development of complex and flexible behaviors, such as most intellectual and social function.

However, there is still considerable debate as to precisely how much of behavior, especially in infants and younger children, is deterministic; for example, the exact role of temperament and gender differences ([Chapter 14](#), [Chapter 15](#)).

### *DIFFERENTIATION OF BEHAVIOR*

Throughout development, there is a movement away from gross, poorly targeted behavior involving most of the organism toward highly specialized, focused activities. Examples of this progression can be seen in motor behavior, from the whole body, flailing behavior of the infant to the finely coordinated activities of the watchmaker or musician. But this is equally true of every subcategory of intellectual, executive, and emotional behavior, although perhaps least striking in the latter.

### *INTERNALIZATION OF BEHAVIOR*

Impulsive gross motor or emotional reactions to stimuli decrease in frequency because more and more behavior occurs unseen, as incoming and self-generated information is processed internally by thinking, as developmental epistemologists such as Piaget have described. But this is also seen in the gradual reduction in motor activity level (and hence of hyperactivity) throughout childhood.

### *HIGHER-ORDER CONTINGENCIES*

More and more, behavior becomes governed by a set of complex rewards and punishments—problem solving, intellectual/aesthetic satisfaction, and, above all, social responses—and less and less by primary self-survival needs. Both behaviorists and psychoanalysts, among others, have described this process in their developmental theories. The pace of this development varies with the function. For example, the acquisition of language and vocabulary between the ages of 1 and 3 years is truly astounding ([Howlin, 1980](#)), as is the acquisition of reading skills at the start of school, whereas the process of intellectual development outlined by Piaget occurs more gradually. As a general rule, the more complex the function (e.g., social behavior; very finely coordinated motor skills, as in violin playing), the longer the process as such is likely to be. Of course, too, the pace begins to decelerate and ultimately even reverse as adult life proceeds to its inevitable conclusion.

It is also apparent that many primitive or immature levels of behavior do not disappear completely. It is not only the infant who has temper tantrums or who will ignore any rights of others in egocentric pursuit, but also these would be expected to emerge in most adults only in disaster situations or when the power of the brain is diminished because of such influences as alcohol intoxication or dementia.

## BRAIN AND BEHAVIOR

Having described both brain and behavior, now it is time to try to relate the two to each other and look at their interaction.

### **Brain Anatomy and Behavior**

One approach to the question, “How do we know the brain affects behavior and in what way?” is to look at the relationship between brain anatomy and behavior. Much of the knowledge applicable to humans in this area is understandably derived from disease or other abnormal states, for the very good reasons that animal work has limited extrapolation, and studies of normal function are more difficult to relate precisely to brain function. The problem is even more difficult with children and adolescents whose brains are still immature, and the ways of relating any deviations in development to brain states are handicapped by the lack of good information about exactly what is truly abnormal and what is simply within normal variation.

### *GENERAL PRINCIPLES*

Comparative anatomic studies show that the amount and complexity of brain development, especially in the cerebral cortex, is closely related to the complexity of behavior, both reaching their highest development in the human species. There are strong parallels between the developmental state of the brain and the development of behavior in humans, especially in early childhood. At birth, the cerebral cortex consists of small, underdeveloped neurons, with few dendrites, especially in the newer parts of the brain ([Casaer, 1993](#)). The process of dendrite differentiation and axonal myelination occurs rapidly during the first few years, although it continues to a diminishing degree into adult life. The brain and certain specific behaviors can be seen to be topographically linked in animal experiments, and in humans, not only in disease states but also in normal development. For example, the smiling response in infants coincides with the myelination of the visual cortex ([Casaer, 1993](#)). The linkage is closest for simple input and output functions. Complex functions such as thinking involve many parts of the brain and, further, are subject to the principle of parallelism of functions discussed in the preceding. This means that topography is less well defined in these behaviors in development and disease states, although still discernible at times. For example, recent brain imaging studies in children have shown that anatomic development generally matches what would be expected from known child development. For example, there is a rapid growth up to puberty in areas subserving highest functions: frontal networks (executive planning), corpus callosum (communication between hemispheres), and association and language areas with loss of some tissue in more



primitive areas such as subcortical gray matter. This rapid development, nonetheless, is not even throughout the brain and continues later in some areas (temporal lobes subserving memory and emotion, and occipital cortex concerned with vision). Much function is asymmetrical, and this asymmetry is reflected in anatomic asymmetry ([Davidson and Hugdahl, 1994](#)). For example, the left hemisphere, which subserves much language function, is heavier than the right in most individuals, especially males, with those areas concerned specifically with language being larger in the dominant hemisphere than in the nondominant hemisphere. On the other hand, areas concerned with visuospatial function, subserved largely in the nondominant hemisphere, are larger there than in the dominant hemisphere ([Lezak, 1995](#)).

### **Brain Topography and Behavioral Function**

The precise relationship between specific brain areas and behavior is now reviewed. Much of what follows is set out in [Lezak \(1995\)](#), Lishman ([1987](#), 2000). In child psychopathology, the reviews by Lewis ([1992](#)) and Taylor ([1991](#)) are among the most germane in trying to relate topography to behavior. Only those areas concerned with higher functions are discussed. A word of warning is needed though. Most of the data come either from animal studies or, in humans, brain disorders in adults when differentiation of function is long established. It is not clear how true the findings are of children, especially when the damage occurs very early in life when there is greater opportunity for utilization of alternative pathways and also development of alternative adaptational behaviors ([Taylor, 1991](#)). However, data from neuroimaging research is rapidly updating the knowledge gained through animal and human studies. Although research in children using these new techniques is still lagging behind research in adults, it is increasing and will give a much better understanding of brain and behavior in child development and psychopathology.

#### *DIENCEPHALON*

This is the only midline structure to be discussed and includes the thalamus and hypothalamus. The thalamus is the Grand Central Station of sensation, through which sensory information passes on its way to the cortex and allied nuclei, but there are also important return circuits. It is involved in sensory processing (crossed), local sensorimotor integration, memory, emotion, attention, and alerting. Deficits associated with thalamic lesions are less absolute than are subtle but important impairments, such as a decrease in verbal fluency (left-sided deficits) and difficulty with pattern recognition (right-sided deficits). As is well known, the hypothalamus is concerned with autonomic and endocrine functions and thus is intimately involved in the expression of emotion (including stereotyped responses such as rage) and motivational states such as sexual drive and aggression ([Lewis, 1992](#)).

#### *CEREBELLUM*

In the past 5 years, not only has much been uncovered about the function of this large area of the brain by technical advances in neuroscience, but also there has been a conceptual shift away from earlier more narrowly focused views of the cerebellum as an organ primarily involved with posture and voluntary movement ([Middleton and Strick, 1998](#)). The cerebellum is intimately involved in such diverse activities as motor learning, adaptive plasticity, central timing (the ability of an organism to measure time, for example, in delay responding) motor and nonmotor aspects of behavior, especially context-dependent and adaptive responses and even cognitive processes such as working memory, learning, and language now appear to be involved ([Middleton and Strick 1998](#); Thach, 1996; Thompson, 1986).

#### *BASAL GANGLIA*

These modulate motor and some autonomic functions by varying tone and producing synchrony of movement but are also involved in intellectual functions too, especially the maintenance of attention. Their function is probably better described as one of generally maintaining and regulating motor and information flow and cerebral activity through reverberating circuits between cortex (especially the frontal region) and basal ganglia. Indeed, the basal ganglia has been shown to play a role in a variety of neuropsychiatric conditions involving motor and attentional dysfunctions ([Mega and Cummings, 1994](#); [Peterson et al., 1998](#); [Rauch and Savage, 1997](#)). These diverse functions are proposed to be subserved by five functionally interconnected subcortical regions: caudate nucleus, putamen, globus pallidus, subthalamic nucleus, and the ventral mesencephalic dopamine system. In addition, as many as five parallel basal ganglia thalamocortical circuits have been proposed ([Alexander et al., 1986](#), [Alexander et al., 1990](#)) to convey the output of these subregions through specific thalamic zones to different parts of the frontal cortex. Impairments here are most easily demonstrated in the classical movement disorders of parkinsonism, athetosis, chorea, and dystonias, but disorders of intellectual function are also seen in basal ganglion disorders. In parkinsonism, there is rigidity of attention and inflexibility of cognition, qualitatively rather similar to the motor disorder ([Lezak, 1995](#)). Perhaps of more interest to child and adolescent psychiatrists (apart from drug-induced parkinsonism) is OCD. Although ordinarily thought of as an anxiety disorder, OCD has strong cognitive components that exhibit some of the same kind of rigidity seen in parkinsonism, except that in this case the rigidity is not one of tone but of a circularity or positive feedback loop of thinking. But just as parkinsonism has both motor and cognitive components, so has OCD at times, in the well-known concurrence of obsessive thinking in Tourette's disorder and postencephalitic parkinsonism ([Leckman et al., 1993](#); [Rapoport, 1988](#)). It is therefore not surprising that recent research suggests that in OCD there may be an abnormality of the circuit between the frontal lobes, basal ganglia, and thalamus ([Cohen and Leckman, 1994](#); [Rapoport, 1988](#)). Recent findings suggest that even ADHD, which equally is characterized by both motor and cognitive impairments, may involve primary dysfunction of the basal ganglia ([Ernst et al., 1998](#)).

#### *THE AMYGDALA*

These are bilaterally paired nuclei deep within the cerebral hemispheres and in close relationship with the older olfactory brain. Not surprisingly, their strongest topographical link is to fear-provoked aggression ([Lewis, 1992](#)). But, as with the basal ganglia, lesions of the amygdala have more widespread effects—disturbances of control, spontaneity, and flexibility of affect and cognition. Its importance in child psychiatric disorders is not well recognized, despite its role in disorders of reward mechanisms, such as substance abuse and eating disorders, and probably in anxiety disorders.

#### *WHITE CEREBRAL MATTER (LARGELY FIBERS)*

This consists primarily of three types of fibers: association fibers, linking parts of the cortex together; commissure fibers, connecting the hemispheres; and projection fibers, linking cortex to distant parts. The effects of disorders of association fibers depend on the tightness of the fiber organization, the specificity of the function, and the extent of the lesion. For example, lesions of the tightly bound left arcuate fasciculus linking Broca's area (frontal) and Wernicke's area (parietal) produce construction aphasia in which the patient understands and expresses language well, but cannot repeat phrases accurately and tends to use inappropriate words ([Kandel, 2000a](#)). In most instances, however, damage to the association areas results in subtle defects of integration of higher function between the areas affected and, as a consequence, a more general, less specific type of impairment ([Lezak, 1995](#)). Abnormalities and lesions of the corpus callosum are often surprisingly silent ([Taylor, 1991](#)), perhaps because of alternative and possibly more usual vertical interconnections through the thalamus. However, neuropsychological tests show that slowing and some impairment of tasks that rely on integration of functions between hemispheres is often present. On the other hand, projection fiber lesions such as those of the internal capsule are likely to have predictable and obvious results, such as hemiplegia, because most are involved in highly specific point-to-point representation.

#### *Cerebral Cortex*

Lezak ([1995](#)) points out that cortical functions can be thought of as roughly organized along two dimensions: lateral (left/right) and frontal/posterior. However, it is important to state early on that this oversimplified topography becomes inaccurate when the function concerned involves much of the brain as PET scans have shown is often the case, especially in any complex or novel task ([Martin and Brust, 1991](#)). Currently, the most frequently used taxonomy of brain function is that of Brodmann, which identifies regions by their functional organization rather than their anatomic localization ([Talairach and Tournoux, 1988](#)).

### **Asymmetric Functions**

In addition to similar but crossed primary sensory and motor functions, both hemispheres have specialization of function. In general, the left or dominant hemisphere is concerned with verbal and language functions, and this includes the storage of such information in the memory systems of the temporal lobes. The right hemisphere is concerned with nonverbal, including spatial, information. But these distinctions are not absolute, and the idea of dominance itself is misleading because specialization of function in each hemisphere is more accurate ([Taylor, 1991](#)). For example, the "poor relation" right hemisphere may actually be the one that enriches brain function and experience in general. For example, much of the melody of speech, humor, lateral or intuitive thinking, imagination, artistry, and music, and the visuospatial aspects of mathematics may be quite dependent on the nondominant or right hemisphere, as may attention itself. There are gender differences in right and left hemispheric organization, primarily, in females, later developing and less marked laterality (e.g., of language functions) that is accompanied by less marked morphologic differences in the two hemispheres ([Kelly, 1991](#); [Lezak, 1995](#)). At a functional level these differences are also matched by differences in the cognitive styles of men and women—men are better at advanced mathematics, less verbally fluent, and better visuospatially, although these anatomic, organizational, and functional differences are relative, and there are large areas of overlap between the two sexes ([Lezak, 1995](#)). How these gender differences arise is still a mystery.

The politically fashionable view is that these are the result of socialization; but there is now clear evidence that there are gender differences in endocrine secretion beginning early in fetal life ([Peterson et al., 1992](#)) when the brain is first developing and that these are related to sexual dimorphism in the brain ([Lewis, 1992](#)) although what relationship these have to nonsexual behavior is unclear. In child psychiatry, one of the more durable areas involving asymmetry of function is that of learning disorders, where failure to develop asymmetry is posited to lie behind some cases of dyslexia ([Duane, 1989](#)), although considerable skepticism about this rather durable theory is needed because reading is a most complex process ([Snowling, 1991](#)). Proof of this lies in recent studies examining morphology in this disorder with MRI and autopsy that suggest unilateral changes in the insula and surrounding areas with less interest in the asymmetry. (The insular cortex is lateral to the globus pallidus but deep to the temporal cortex [[Kandel, 2000b](#)].)

#### *Frontal/Posterior Dimension*

The frontal lobes are most concerned with motor and executive functions such as speaking, organizing, and planning complex activities. Defects lead to aphasias, certain kinds of apraxias (inability to plan and execute sequences of movements such as dressing), deficits of attention, and the planning and execution of complex behavior ([Lezak, 1995](#); [Rothenberger, 1990](#)). The parietal, occipital, and parts of the temporal lobes have largely sensory functions, including complex interpretations of sensory data in light of experience and integration of these data across modalities. Deficits here lead to agnosias and receptive aphasias.

#### *Temporal Lobes and the Limbic System*

These are of special interest in psychiatry because of their well-demonstrated emotional and memory function, but they also have sensory functions associated with visceral sensations, olfaction, hearing, and speech. Organizationally, they have close links to midline structures concerned with emotional expression (e.g., the hypothalamus) and memory (e.g., the nucleus basalis of Meynert) ([Cote, 1991](#)), as well as to the cortex as a whole. Stimulation of this region during surgery or by an irritable focus can produce schizophrenic-like positive symptoms such as déjà vu and visceral or other hallucinations, giving strength to the postulate that dysfunction of the left temporal lobe is involved in schizophrenia ([Crowe, 1990](#)). Destructive lesions can result in negative symptoms or deficits (e.g., amnesias) and agnosias (e.g., impaired sound discrimination, amusia, and receptive dysphasias). Emotional disturbances of blandness similar to the negative states of schizophrenia or of poor regulation (e.g., irritability, panics, anxiety, obsessions) are also characteristic.

#### *LONGITUDINAL SYSTEMS*

The most important one for psychiatry is the reticular system of the more rostral brain stem, which has projections throughout the cortex. This system is much more complicated than originally thought, containing homeostatic, postural, activating (noradrenergic), and incoming information-modulating (serotonergic and dopaminergic) sections ([Kelly, 1991](#)).

#### **Factors Affecting Brain and Behavior**

More detailed discussions appropriate to each disorder and brain damage syndromes in general can be found elsewhere in this book and in other reviews ([Gudex and Werry, 1990](#); [Kreusi, 1990](#); [Rutter, 1981](#), [Rutter, 1982](#); [Taylor, 1991](#); [Werry, 1986](#)). Here, discussion is restricted to factors that have general effects on brain-behavior relationships or that illustrate particular points. Much of what follows relates to major brain abnormalities and hence should be applied with caution to lesser or more uncertain lesions or normal states. The caveat about the validity of these largely adult-derived data in children also applies.

#### **Localized Versus Generalized Damage**

Lipowski ([Gudex and Werry, 1990](#)) has classified brain-behavior syndromes as partial (or localized) and generalized, and this typology has been applied to organic brain disorders in children ([Gudex and Werry, 1990](#)). The amnesic disorders and some types of personality change owing to a general medical condition (e.g., frontal or temporal lobe syndromes) are examples of partial disorders, whereas dementia and delirium are examples of generalized syndromes.

#### **Partial Syndromes**

In child psychiatry those of particular interest are the so-called “temporal lobe” syndromes of explosive, aggressive behavior said to be associated with temporal lobe epilepsy or inferred to represent temporal lobe dysfunction from the behavior itself or its putative response to anticonvulsants ([Kandel, 2000a](#)). The validity of this syndrome is still disputed, especially in children ([Hermann and Whitman, 1984](#); [Kreusi, 1990](#); [Lewis et al., 1989](#); [Rutter, 1981](#)), and although there were allusions to it in Diagnostic and Statistical Manual (DSM)-III in the text, it does not appear in DSM-IV, where classifications of personality change owing to medical conditions are defined strictly behaviorally rather than etiologically. However, the idea is not such as likely to die so easily. (See [Taylor, 1991](#).) A number of psychiatric disorders, such as developmental language (right temporal lobe), learning and motor skills disorders ([Duane, 1989](#); [Feagans, 1983](#); [Silver, 1989](#); [Taylor, 1991](#); [Werry et al., 1990](#)), and ADHD ([Rothenberger, 1990](#); [Taylor, 1991](#); [Zametkin and Rapoport, 1987](#)) can be considered localized or partial syndromes. For example, brain imaging techniques suggest a dysfunction of frontobasal ganglia circuits in ADHD ([Castellanos, 1997](#); [Schweitzer et al., 2000](#)), OCD and Tourette’s disorder ([Cohen and Leckman, 1994](#); [Peterson et al., 1998](#); [Rapoport, 1988](#)), and of the temporal lobe in panic disorder ([Kandel, 2000a](#)), whereas schizophrenia seems to have both temporal and frontal lobe deficits ([Weinberger, 1995](#); [Werry and Taylor, 1994](#)). So far, these are only isolated findings, and conclusive evidence of localized or partial syndromes in child psychiatry remains elusive, although newer neuroimaging research in general is supportive of defined functional neural networks and areas.

#### **Generalized Syndromes**

In contrast to syndromes in adults, generalized syndromes such as dementia and delirium, so topical in adult psychiatry, are little studied in children ([Gudex and Werry, 1990](#); [Volkmar, 1992](#)). In adults, generalized brain disorders are marked by decline in all higher functions, although the precise pattern depends on how much and which parts of the brain and which localized functions are affected as well ([Albert, 1981](#)). In child and adolescent psychiatry, degenerative disorders are rare, and greater interest centers on mental retardation, the effects of anoxia at birth, and postnatal head injury. It seems logical to expect that, as in dementia, there should be a general reduction in the overall number of neurons. In Down syndrome, brain size is reduced by about 25% (although interestingly this is not true until ages 3 to 5, suggesting a failure of the brain to develop), and there is also a reduction in the complexity of convolutions so essential to accommodate the increased number of neurons and processes in the human cerebral cortex. At a microscopic level, there appears to be little localization of pathology ([Propping and Hebebrand, 1990](#)); all this suggests that severe mental retardation can be viewed as a generalized syndrome. Whereas gross brain damage/disorder occurring prenatally or perinatally can result in mental retardation, long-term follow-up studies suggest that the effects of the much commoner milder degrees of anoxia are difficult to detect after the first few years of life, when the compensatory effect of a felicitous environment seems to override the more obvious effects of generalized brain damage ([Rutter, 1981](#); [Werry, 1986](#)). More subtle, difficult to detect effects such as attentional and learning disorders remain a strong possibility ([Feagans, 1983](#); [Taylor, 1991](#)); most such learning disorders would be localized, not generalized, syndromes ([Duane, 1989](#)). Head injuries ([Chapter 33](#)) ([Guthrie et al., 1999](#); [Homer and Kleinman, 1999](#); [Lam and MacKersie, 1999](#)) in children and adolescents are frequent—40,000 in the United Kingdom in 1985 ([Middleton, 1989](#)), and approximately 2 million in the United States as of the early 1990s, with 56,000 deaths at all ages ([Kraus and McArthur, 1996](#)). Individuals with head injury provide opportunities to study brain-behavior relationships in a generalized context because, in addition to local and contrecoup lesions, head injury often produces widespread permanent microscopic effects ([Lezak, 1995](#); [Taylor, 1991](#)) as a result of acceleration and torsion effects. Such generalized effects in adults are well described and can be summarized as a decline in mental efficiency affecting attention, speed of mental processes, cognitive efficiency, and, when severe, high-level intellectual functions such as abstract reasoning. In turn, this often is accompanied by irritability, fatigability, and loss of initiative ([Lezak, 1995](#)). In children, in addition, there is evidence that the risk of development of any psychiatric disorder is increased, suggestive of some general effect ([Taylor, 1991](#)) ([Chapter 33](#)). Data on children are less extensive, although Mittenberg and associates (1997) showed that when assessed in a similar manner to adults, postconcussion syndrome is equally similar. Fortunately, severe injury is infrequent in children and adolescents, most of the injuries are mild, and better treatment of the behavioral and neuropsychological problems is appearing. Although [Middleton \(1989\)](#) criticized the widespread belief that mild injuries are without long-term sequelae, recent reviews ([Lord-Maes and Obrzut, 1996](#); [Satz et al., 1997](#)) are more optimistic in that most better studies showing no abnormal findings. However, it is best to keep an open mind for the moment. Much of the more recent work in adults has shown that the postconcussional changes are detectable for several months after even a mild concussion ([Middleton, 1989](#)), suggesting a significant immediate impact. In children the effects are similar but development of the brain continues into adolescence, and there is a change in the relative importance of areas of the brain toward those concerned with higher levels of cognitive function. Thus there may well be “sleeper,” or delayed, effects that become apparent only later. In summary, then, generalized brain disorder probably results in generalized cognitive and behavioral changes, of a degree and permanence depending on the amount involved. However, the brain seems to have some capacity for recovery, especially in the long term. Much more controversial are other possible generalized (or localized) brain-behavior syndromes that are not owing to frank brain pathology but to normal variations such as temperament and certain behavioral traits such as high activity or sociability. Much of the research in this area depends for its biological underpinnings on genetic studies, especially of twins ([Garrison and Earls, 1987](#)). For example, the studies by [Goodman and Stevenson \(1989b\)](#) suggest that activity level has a strong genetic base (50% of the variance) and that even



anxiety/withdrawal and somatizing traits may also have some hereditary base ([Goodman and Stevenson, 1989b](#)). In the end, genes operate through specific cellular effects, and the brain seems the most logical vehicle if temperamental and personality trait variations are biologically influenced.

## Age

Age is of critical interest in child and adolescent psychiatry. One of the more durable beliefs about brain–behavior relationships is that of plasticity; the effects of any abnormalities of brain structure or function are said to be less serious the younger they occur. But there is an equally durable but opposite view that there are critical periods for the development of normal function, which, if missed or impaired, result in permanent disabilities. This is best illustrated by strabismus, which, if not corrected in the first few years of life, results in amblyopia. There is evidence to suggest that the popular idea that younger age reduces the risk of severity or durability of disability may well be false ([Fletcher and Satz, 1983](#); [Middleton, 1989](#)). Further, the notion of sleeper, or delayed, effects emerging only later as a function of development of more complex cognitive and other brain functions must be borne in mind. In his review, [Taylor \(1991\)](#) expresses concern about the failure of neurologists, child psychiatrists, developmental psychologists, and others to get together on this issue, pointing out, for example, that most of the best studies of head injury were done more than 20 years ago and before modern brain imaging techniques could pinpoint the amount and location of damage so critical to meaningful study.

## Gender

Nearly all the putative brain–behavior syndromes in children, such as ADHD, learning and developmental language disorders, autism, schizophrenia, and Tourette's disorder, have appeared markedly in excess in boys. This may well reflect the sexual dimorphism of brain anatomy and development ([Peterson et al., 1992](#)) discussed in the preceding under Asymmetrical Functions, such as, in females, later and less complete development of dominance, lesser differences in size between right and left hemispheres, and/or in males, greater vulnerability to damage ([Kelly, 1991](#)). Also interesting is animal and clinical research on disorders such as Turner's syndrome and on androgen sensitivity in the development of sexual preference and gender identity ([Zucker et al., 1999](#) and [Chapter 58](#)), and developmental research on other kinds of behavior, such as aggressiveness or activity level, that are observed to be different in the two sexes ([Goodman and Stevenson, 1989a](#); [Lewis, 1992](#)), even early in life ([Garrison and Earls, 1987](#)). Research into gender identity, sex-typed behavior, and sexual preference in animals and humans with chromosomal or endocrine anomalies such as Turner's syndrome or androgen insensitivity suggests that brain factors are influential, especially in the prenatal period ([Garrison and Earls, 1987](#); [Kelly, 1991](#); [Rutter, 1980](#)), but so are environmental factors, as shown in the controversial studies of “intersex” children (those with indeterminate anatomic gender at birth) ([Kelly, 1991](#); [Rutter, 1980](#); [Zucker et al., 1999](#) and [Chapter 58](#)). Nevertheless, the very early demonstration of sexual dimorphism (e.g., in androgen levels) in the fetus when the brain is first developing ([Peterson et al., 1992](#)) and clear differences in behavior (e.g., in activity level) in male and female infants suggest that brain–behavior relationships get off to a somewhat different start in the two sexes. How much they then diverge further or converge on the basis of experience is a most important and quite complex question that unfortunately seems to attract oversimplified polarization ([Zucker et al., 1999](#) and [Chapter 58](#)).

## Etiology

Although different etiologies produce different brain–behavior effects—for example, those of trisomy 21, head injury, and disorders of the temporal lobe all differ—there is good reason to posit that these differences will in the end be better explained by precise knowledge of the regions and functions of the brain affected and the pace and durability of any pathologic changes ([Rutter, 1980](#), [Rutter, 1981](#); [Werry, 1986](#)). Quite apart from the needs of treatment, it thus makes good sense to identify etiology as an important defining variable in brain–behavior relationships and to utilize what is known about the general and anatomic pathology (e.g., amount of extravasation, toxicity, and regional vulnerability of the brain) to generate hypotheses about brain–behavior relationships, and then to relate these to clinical facts. This is one of the main approaches in current neuropsychology, which increasingly eschews such global concepts as “brain damage,” dementia, intelligence, and so on ([Albert, 1981](#); [Lezak, 1995](#)); instead, etiology is seen as often having strong functional implications. However, [Taylor's \(1991\)](#) extensive critical review of etiology shows that in children this is full of methodologic complexities, few of which have characterized past studies, such as accurate delineation of areas of dysfunction (many of which are biochemical, not anatomic), precise and comprehensive measures of psychological function, and controlling for environmental and developmental effects

## Experience

It is probably unnecessary in child psychiatry to highlight the role of experience in shaping and modifying brain–behavior relationships. This review and others have given instances in which the outcome of any brain state or dysfunction has been demonstrated to be influenced by experience; for example, limbic dysfunction and violent behavior ([Lewis, 1992](#); [Lewis et al., 1989](#)), sexual preference and the reassigning of gender ([Kelly, 1991](#)) ([Zucker et al., 1999](#)), and the attenuation of long-term effects of perinatal anoxia ([Rutter, 1980](#), [Rutter, 1981](#); [Werry, 1986](#)). There is also no doubt that experience can produce permanent change in normal brain structure and function; that is what learning and long-term memory must be. For example, although the postcentral gyrus sensory map of the brain is basically similar in all humans, animal experiments have shown that this map can be changed quite significantly by immobilizing some fingers and exercising the others, or by sectioning a nerve ([Kandel, 2000b](#)). This process of “reassigning” brain areas seems to be a general property of the nervous system. At a cellular level, reassignment, learning, and memory are thought to be achieved to a significant degree by alteration of synaptic transmission—increasing or decreasing the sensitivity or effective number of active presynaptic release sites or postsynaptic receptors ([Kandel, 2000b](#)). The way that this is brought about is probably complex and not necessarily homogeneous. For example, the elaboration of dendritic processes would increase the number of receptors, whereas biochemical changes at any one synapse could result in changes in the ratio of active to inactive receptors. Considerable progress is being made in moving these conjectures toward fact, particularly in learning associated with emotion. For example, increased presynaptic neurotransmitter release following long-term potentiation (LTP) in the amygdala has been demonstrated. Even more intriguing, the basis for this LTP is owing to a gene and transgenic mice that overexpress the gene exhibit neither LTP nor fear conditioning. This has important implications for brain–behavior relationships, understanding why some children are temperamentally more timid and anxious than others, and for the interaction between brain and environment in the genesis of anxiety disorders. At a macroanatomic level, new brain imaging techniques are identifying those parts of the brain involved in memory and learning. Although some of this has confirmed the role of traditional structures such as the hippocampus and temporal lobes, they have also emphasized the role of other structures such as the prefrontal cortex and amygdala. They are also showing that there are many different kinds of learning and memory depending on the type, cues, and context and that different parts of the brain are associated with each.

As more is learned about the biological basis of learning and memory, especially emotionally laden memory, this may lead to innovative approaches to the core deficits seen in mental retardation as well as the milder more circumscribed learning disabilities. However, it would be naive to go as far as Flourens or Lashley (see “Concepts of Brain–Behavior Relationships” and [Kandel, 2000a](#)), or too many professionals working with children, to think that experience or retraining programs can overcome any or all brain dysfunctions ([Taylor, 1991](#)). It seems likely that certain regions of the brain where localization is well developed and not replicated and/or the amount of residual brain left after damage will limit the extent and scope of experience ([Taylor, 1991](#)).

## A Clinical Example

The clinical cogency of brain–behavior relationships and the principles outlined in the preceding are illustrated now with one brief example, Tourette's disorder, largely because it has some of the most elegant hypotheses of brain–behavior interactions among child and adolescent disorders. What follows is described in more detail later in this volume ([Chapter 59](#)) and elsewhere ([Cohen and Leckman, 1994](#); [Leckman et al., 1990](#), [Leckman et al., 1992](#), [Leckman et al., 1993](#); [Peterson et al., 1992](#)). Here the emphasis is on illustrating clinical relevance of principles, rather than the disorder itself.

The behavior that brings the patient with Tourette's disorder to the clinician is unwanted, involuntary, repetitive, purposeless, stereotyped motor and vocal acts (tics), which may take quite complex forms in severe cases and can be socially disabling. These acts are often preceded by premonitory urges ([Leckman et al., 1993](#)). The disorder is often accompanied by other behavioral difficulties of a disinhibited, impulsive, or obsessive-compulsive kind ([Chapter 59](#)).

Pedigree and twin studies have suggested that Tourette's disorder is the product of a single major autosomal gene, but that the frequency in identical twins is only 50% ([Hyde et al., 1992](#); [Leckman et al., 1990](#)). Also, the disorder is more common in boys, its severity varies across and within individuals, and there is an increased frequency of other disorders, notably OCD and ADHD in relatives.

These mostly clinical observations raise a number of questions that epitomize the nature of brain–behavior relationships in child psychopathology: What is the action of Tourette's disorder genes on the developing brain? Where do they act? What makes males more susceptible? What are the risk and protective factors determining whether or not, how severely, and which of the interrelated disorders appear clinically? How do these protective/risk factors operate on the brain? What is it that influences the day-to-day and minute-to-minute frequency of the symptoms in any one individual? Why does it take several years before the disorder emerges?



Although no definitive answers are yet available to any of these questions, there are some promising hypotheses and leads.

Simplistically, tic disorders (and OCD) seem to reflect neuronal circuits that reverberate instead of switching off—a failure to self-inhibit ( [Leckman et al., 1993](#)). Genes lay down the fundamental anatomic organization of the brain, and minor variations in this could lead to the propensity of some microcircuits to be driven by some ectopic or rogue pacemaker and/or to be over-readily excited by incidental brain activity. Research suggests that the anatomic substrate may lie in the cortico-striato-cortico-thalamic system that regulates the planning and performance of motor behavior ( [Leckman et al., 1993](#)). This could explain why the disorder manifests both cognitive (premonitory urges) and complex motor (tics) symptoms and also why there may be a particular somatic distribution to both ( [Leckman et al., 1993](#)).

The increased frequency in males is of interest because it is characteristic of a number of psychiatric (and physical) disorders and is intimately related to the controversial question of sexual dimorphism of the brain discussed in the preceding. There is now evidence that testicular hormones begin to be secreted as early as the eighth week of fetal life, at a time when the brain is undergoing its major initial development, and are thought to play a major part in sensitizing certain regions to sex hormones ( [Peterson et al., 1992](#)). The way in which this could predispose males to Tourette's disorder and induce it in childhood and adolescence as androgen secretion increases is outlined with supporting evidence in [Peterson and colleagues \(1992\)](#). The delays in onset of the disorder can be explained, in part, by this rise in secretion, but also by maturation and/or experiential realigning of brain architecture, which as already noted is most active during early life but continues at a negatively decelerating rate until death.

The development of the brain is also dependent on a continued optimum biochemical environment and this is vulnerable to disruption. Two such disruptive factors, maternal vomiting during the first trimester and maternal stress, were shown in one study to influence the severity of tics in those with established Tourette's disorder ( [Leckman et al., 1990](#)). Another risk factor, observed in identical twins, is lower birth weight ( [Hyde et al., 1992](#)), suggesting that the power of one twin to get a better share of placental nutrients is a factor in the impact of genes on brain development. Maternal drug, tobacco, and alcohol use are universal ambients also known to alter the fetal environment and fetal development, although their role in Tourette's disorder awaits clarification ( [Leckman et al., 1992](#)).

The influence of psychosocial variables in Tourette's disorder in postnatal life is widely assumed, but as in many other disorders, study of these variables would benefit from the same degree of rigor that now typifies biological studies—*anxiety and/or stress may precipitate or make tics worse and they may be suppressed for variable periods with effort and are more prominent in relaxed or drowsy states (Chapter 59)*. How these might work at a psychophysiological level needs clarification, although one obvious mechanism is through concomitant variations in neurotransmitters such as noradrenalin, dopamine, and serotonin (5-hydroxytryptamine [5-HT]) and neuropeptides, all of which have modulating actions on brain activity additional to any specific transmitter actions ( [Chapter 4](#)). In summary, Tourette's disorder illustrates how genes, gender, maternal stress, and uterine environment might alter brain microstructure and microorganization, making some individuals vulnerable to develop the disorder. The trigger for the emergence of the disorder might be the maturation of “capable” pathways, the rise of sex hormones, and as posited by Weinberger ( [1987, 1995](#)) for schizophrenia, development-specific stresses that activate at risk pathways. Temperamental variables that increase anxiety-proneness may also make individuals more vulnerable. Once the disorder is established, its severity is influenced by variations in levels in arousal, which in turn are influenced by what is happening to the child or adolescent on a moment-to-moment basis. The content of vocal tics reflects the child's learned vocabulary, and educational, supportive, and behavioral programs, suggesting that even presumptively biogenic mechanisms can be influenced to some degree by purely psychosocial variables. In the end, all factors—biogenic or psychogenic—operate through a final common brain pathway to express the disorder as clinicians recognize it.

Tourette's disorder illustrates the power of the biological substrate in this equation, but other disorders could have been chosen equally to illustrate the power of the environment. It is, however, not either/or, but a question of variation in the mix of these two factors and where current knowledge indicates therapeutic intervention may best be made. There is, however, a need in research on psychosocial variables to move beyond correlational studies, be cast more precisely, and be tied functionally to variations in brain function; otherwise, their credibility in this equation may be diminished, which would be most unfortunate.

## CONCLUSIONS

This chapter shows that the brain is an inordinately complex and wondrous mechanism. Its functions are both sharply localized and generalized, depending on the complexity of the function and the number of different regions of the brain necessary to that function. In a way, however, even the generalized functions seem composed of parallel or interacting localized functions so complex that they only seem generalized. Sharply localized functions such as speech or fine motor movements are vulnerable to disruption in disease states, but parallel function protects a lot of higher functions from greater devastation from disease or injury.

Development is a major variable, of relevance in terms of both brain morphology and the unveiling of complex functions. It also protects the brain before and during birth in that the undeveloped state of the cortex particularly makes it more resistant to pressure and anoxia ( [Werry, 1986](#)). Although much of the process of brain development is preprogrammed, inexorable, and with clear limits, experience plays a vital role, especially in more subtle aspects of morphologic and biochemical differentiation, shaping, and reshaping. The behavioral analog of this we call learning, which is a lifelong process, not confined to childhood or adolescence. Although there is now a large body of knowledge linking brain function and topography to behavior, much of this is derived from adults, animals, and disease states of little relevance to child and adolescent psychiatry. It does, however, enable hypotheses to be made about possible relationships and indicate meaningful lines of research.

As [Kandel \(2000b\)](#) points out, the separation of brain and behavior reflected in the divorce of neurology and psychiatry in the mid-20th century was owing largely to the limitations of our knowledge and becomes increasingly obsolete. Rapid developments in molecular biology, neuropsychology, and other neurologic and behavioral sciences are closing this gap, although this may be more apparent at a research and theoretic than at a clinical level. However, such is the pace of development of new knowledge that it is not too fanciful to predict that presumptively major brain disorders such as autism, OCD and Tourette's disorder, and schizophrenia will yield their secrets and cause a major shift in the focus and thinking in much of child and adolescent psychiatry because many of these disorders are neurodevelopmental, with origins in childhood or even fetal life. But this will be but part of the shift. Soon to be revealed too will be the biological (especially the polygenetic) basis of a major preoccupation of child psychiatry, variations of normal behavior/personality such as temperament, hyperactivity, anxiety threshold, and so on. Then doctors will surely be asked to change this or that characteristic in children, probably through pharmacologic or molecular biological means. Trained as they are in human biology in health and disease and steeped in the ethical traditions of medicine, child psychiatrists must accept the responsibility to serve as a bridge to knowledge about brain and behavior so that the clinical picture of the child may be fuller and richer, yet keeping the treatment always in the child's best interests as a unique human being.

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## 9 PEDIATRIC NEUROIMAGING

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Bradley Peterson organized the study group and edited this review.*

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The close of the last millennium witnessed the development and extraordinary growth of brain imaging studies in childhood neuropsychiatric disorders. Although initially lagging behind studies of adult disorders because of caution in applying new methodologies to the study of children, imaging studies of childhood disorders now have made tremendous strides in defining the neurobiological basis of these childhood illnesses. Imaging studies, in fact, arguably offer the greatest promise of improving our understanding of normal brain development and the neural bases of these complicated syndromes *in vivo*. This chapter first presents an introduction to contemporary imaging modalities and their contribution to our understanding of brain development in normal children. It then presents summaries of findings across a broad range of conditions, including preterm birth, autism, fragile X, velocardiofacial syndrome (VCFS), childhood-onset schizophrenia, attention deficit/hyperactivity disorder, obsessive-compulsive disorder (OCD), Tourette's syndrome, pediatric autoimmune neuropsychiatric disorders (PANDAS), mood and anxiety disorders, posttraumatic stress disorder (PTSD), and dyslexia.

## IMAGING MODALITIES

### Computed Tomography

The development and widespread application in the mid-1970s of axial computed tomography (CT) first heralded the advent of neuroimaging in neuropsychiatric disorders. It permitted noninvasive images of the brain and cerebrospinal fluid (CSF) tissues *in vivo* for the first time. Computed tomography scanning utilizes x-radiation, however, which has limited the use of CT in the research of childhood disorders.

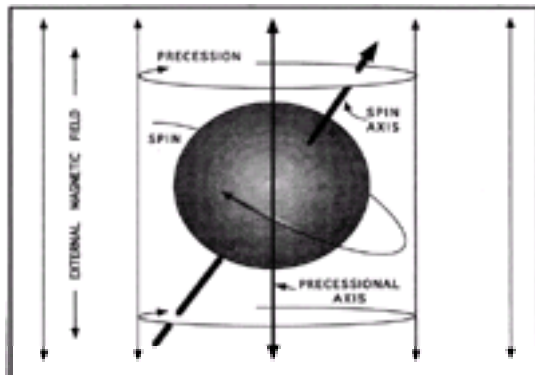
In the acquisition of a CT image, an x-radiation source is physically rotated many times around the subject's head. The varying strength of the x-radiation that passes through the head, which is determined by characteristics of the tissue through which it passes, is then measured multiple times and at multiple angles through the cranium. Computer processing methods then reconstruct the image through calculation of attenuation coefficients for each portion of the brain, and those coefficients are then assigned intensity values, which produces light and dark areas (contrast) in the brain image. Contrast is the main determinant of our ability to discriminate different tissue types in an image. Tissue contrast is vastly inferior to that of magnetic resonance imaging (MRI), which accounts for the favored use of MRI in

neuroimaging studies. Computed tomography scan resolution is approximately 1 mm, although the imaging plane is limited to axial sections.

## Magnetic Resonance Imaging

Structural (or anatomic) magnetic resonance imaging (MRI) allows visualization of the size and shape of various brain regions, in fine detail and with exceptional contrast. Structural MRI scanning has been widely available clinically since the early 1980s, although the remarkable “resonance” property of certain nuclei when placed in magnetic fields had been known for decades prior to the first acquisition of MR images ( [Smith, 1998](#)). Hydrogen is among the nuclei that share this remarkable property, and the fact that it is the most common nucleus in biological tissues is what makes the imaging of brain tissue possible.

This is what is meant by the “resonance” of the hydrogen nucleus. Normally, the hydrogen nucleus spins at a particular frequency with its axis of spin pointed randomly in any direction. When placed in a strong magnetic field, however, this nucleus orients its axis parallel to the external magnetic field. In addition to its usual nuclear spin, the nucleus now also has another motion, that of precession around an axis parallel to the magnetic field, similar to a spinning top or gyroscope ( [Fig. 9.1](#)). While the strong magnetic field is maintained, a very brief radiofrequency magnetic pulse is then emitted from a coil apparatus around the subject's head. The frequency of the applied pulse is carefully chosen to be precisely the frequency that is most efficiently absorbed by the nucleus, its “resonance” frequency. This happens to match the frequency of precession of the nucleus; therefore, “resonance” refers to the absorption of radiofrequency energy by the nucleus at its particular precessional frequency.



**Figure 9.1.** Precession of nuclei occurs when certain spinning nuclei are placed in an external magnetic field. The precession occurs around an axis parallel to the lines of force of the magnetic field. When an appropriate external radiofrequency pulse is applied to the precessing nuclei, the magnitude of the precession increases, effectively tipping the nuclear spin axis toward the transverse plane perpendicular to the precessional axis and the lines of force of the external magnetic field. (Figure courtesy of Brad Peterson.)

Absorption of the resonant energy tips the spin axis toward a plane perpendicular to the precessional axis, in effect increasing the amplitude of the precessing motion. This “excited” nucleus then begins to tip its spin axis back toward the precessional axis, while continuing to precess because of the continued presence of the external magnetic field. The rate at which the nucleus realigns itself with the precessional axis is determined in part by its immediate physical environment, the nuclei surrounding it. The nuclear spin creates a small magnetic field in the nucleus that generates a small radiofrequency signal as the nucleus precesses. Thus, the scanner's radiofrequency pulse excites and tips a precessing nucleus, which produces a radiofrequency signal and which carries information about its unique physical environment in its rate of return toward its precessional axis. In effect, each precessing nucleus becomes a radio transmitter whose broadcasted signal specifies the location of the nucleus and nature of its immediate physical environment.

Highly specialized signal processing techniques are needed to discern this newly emitted signal and decode the information it contains about the excited, resonating nuclei. It is the development and application of these image reconstruction and processing techniques that made MR imaging possible and useful for quantitative studies. Image reconstruction techniques rely on Fourier analysis of the signal to transform into spatial coordinates—the many complex radiofrequency waveforms emitted by the excited nuclei. In addition to providing spatial coordinates of nuclei, these reconstruction methods must also provide signal intensity information that differentiates one nucleus from another. Without this information, spatial localization would simply provide a visually uniform map of hydrogen nucleus localization. Magnetic resonance images also provide intensity information, and hence intensity contrast between different portions of the image, based on the differing tissue environments experienced by the nuclei.

Magnetic resonance imaging techniques provide this information of tissue environment by measuring separately the spin component that is parallel to the precessional axis of the nucleus (and the external magnetic field), and another component that lies in the transverse plane perpendicular to that axis. These components vary in time as the excited, precessing nucleus returns toward its precessional axis, a process called “relaxation.” The time constant for relaxation along the axis parallel to the magnetic field is called “ $T_1$ ” and that within the transverse plane perpendicular to this axis is called “ $T_2$ .” These relaxation times vary according to the tissue characteristics in which the nucleus resides. The intensity value of each picture element (pixel) in an image depends on the timing of the MR measurement relative to the relaxation time constants. Hence,  $T_1$  and  $T_2$  are the major determinants of tissue contrast in MR images.

Because the tissue characteristics determine  $T_1$  and  $T_2$  relaxation times, different tissue characteristics can be emphasized on an MR image simply by varying the intensity values assigned to each combination of  $T_1$  and  $T_2$  times. Thus, the water content of CSF or the fat content of white matter myelin can be varyingly emphasized or deemphasized, depending on the clinical or investigative need. The excellent tissue contrast that MRI produces, its excellent in-plane and between-plane resolution (now approximately 1 mm), and its safety make MRI a useful clinical and research tool.

## Positron Emission Tomography

Positron emission tomography (PET) involves the imaging of radiolabeled tracers that emit positrons. When the tracers are injected or inhaled, the positrons emitted from the tracers soon (within a 3-mm distance from the tracer molecule) collide with electrons. The colliding positrons and electrons annihilate one another, producing two gamma rays that are emitted at 180-degree trajectories from one another. These two gamma rays then pass out of the tissue and are recorded by detectors in the axial plane surrounding the subject. The precise localization of the origination of the gamma rays can be determined by noting which detections occur simultaneously at 180-degree orientations ( [Rebe, 1993](#)).

This site of gamma ray origination varies depending on the pharmacologic properties of the particular radiotracer used. Some tracers, for instance, are primarily confined to intravascular space, and the localization of gamma ray origination can be assumed to reflect the distribution of blood vessels. Other tracers, such as 2-deoxyglucose, are taken up by neurons and phosphorylated. Because the phosphorylated deoxyglucose cannot be metabolized further, the accumulating tracer reflects the degree of activity of the neurons containing them, which is then measured in the amount of radioactivity detected in that particular brain region. Additional tracer types include ligands for specific neurotransmitter receptors. Raclopride, for instance, is a  $D_2$  receptor ligand. The isotopes that are incorporated into the various tracers have short half-lives: that for  $^{15}\text{O}$  (incorporated in water for blood flow studies) is 2 minutes,  $^{11}\text{C}$  (incorporated, for instance, into raclopride for dopamine  $D_2$  receptor studies) is 20 minutes, and  $^{18}\text{F}$  (incorporated into deoxyglucose for metabolic studies) is 110 minutes. In-plane resolution of the newer PET scanners is approximately 3 mm for  $^{18}\text{F}$ -labeled tracers, and 8 mm for  $^{15}\text{O}$ -labeled ones, the difference being caused by differences in the distance between origination and annihilation of the positrons from each of the isotopes in their differing tissue compartments.

## Single Photon Emission Computed Tomography

Single photon emission computed tomography (SPECT) imaging is functionally similar to PET, the primary differences between the two being spatial resolutions and kinds of tracers employed. Whereas PET tracers emit positrons that then produce gamma rays, SPECT tracers emit photons of characteristic energies. Each photon is emitted in a manner that is directionally random and temporally independent of any other photon release, thus accounting for the “single photon emission” portion of the SPECT acronym. Specifics of the photon detection system are the primary determinants of resolution of any particular SPECT scanner. Currently, the spatial resolution of SPECT, although rapidly improving, is still (at approximately 6 mm) inferior to that of PET. Single photon emission computed tomography ligands,

however, typically incorporate long-lived isotopes of technetium and iodine, which, unlike PET ligands, do not need to be synthesized on-site in a large and expensive cyclotron. This is a major cost-saving advantage of SPECT. In addition, the development of new, pharmacologically specific SPECT tracers makes the future of SPECT imaging quite promising.

Although new radiotracers may be developed whose pharmacologic properties and half-life offer the potential of reducing the radiation exposure for children who might undergo PET or SPECT imaging in the future, the need for radiation exposure in these imaging modalities will never be eliminated entirely. Reducing total radiation exposure sufficiently reduces the risks associated with PET and SPECT, so that the techniques might be justifiable for the clinical investigation of children who have severe neuropsychiatric illnesses. Nevertheless, the persistence of radiation exposure, no matter how minimal, is also likely to make the recruitment of normal control children for research protocols prohibitively difficult. The development of novel experimental designs that would contrast various disease groups could make the combination of clinical and research protocols possible in the future, however.

### Functional Magnetic Resonance Imaging

Functional magnetic resonance imaging (fMRI) studies permit the identification of the brain regions that are engaged in processing various types of information or in solving various types of problems. The first reports characterizing changes on MR images during specific cognitive tasks appeared in 1991 ( [Belliveau et al., 1991](#)). The changes on the MRI scans, which consist of subtle regional intensity changes during task activation, are now believed to track blood flow-related phenomena. The regional image intensity changes appear to derive from changes in the contents of oxyhemoglobin and deoxyhemoglobin present during the task, compared with their contents during the control state. Decreases in deoxyhemoglobin concentration during task performance increases the strength of the radiofrequency signal emitted by the underlying tissue, producing a locally increased intensity in the image. Images acquired during task activation can be compared with appropriate control images to determine which pixel intensities (and where in the brain local oxyhemoglobin levels) have changed during the task.

It also appears that the spatial and temporal resolutions of fMRI surpass conventional PET and SPECT imaging capabilities. fMRI can provide in-plane resolution of 1 mm or less, which compares favorably with the current in-plane resolution of the best PET cameras, which is 2 to 3 mm. Functional magnetic resonance imaging's temporal resolution of approximately 1 second is comparable to that of PET but inferior to that of the electroencephalogram (EEG), which has a temporal resolution in the tens of milliseconds—similar to the time scale of neural events. It is doubtful whether fMRI will ever be able to achieve the temporal resolution necessary to resolve discrete neural processes. Nevertheless, its relative safety (especially in children), good spatial and fair temporal resolution, the ability to scan subjects on multiple occasions, and low cost compared with PET or SPECT make fMRI an attractive functional imaging modality and accounts for the vast number of fMRI studies that are now appearing in the literature.

### Diffusion Tensor Imaging

Diffusion tensor imaging (DTI) is a unique and relatively recent application of MRI technology that provides, among other things, detailed information about the orientation of fiber tracts within the brain, especially information about the orientation and integrity of white matter tracts. It provides this information primarily by tracking the nonrandom diffusion of water through those fiber tracts, as follows.

Magnetic resonance images based on T1 and T2 relaxation times provide information on the molecular environment of water molecules. Extravascular water diffuses more or less randomly in tissues, but its movement is influenced by cell membranes (and cytoskeletal structures), which act as barriers to free motion. The first major clinical application of diffusion imaging was in stroke diagnosis. It was shown that the technique demonstrated contrast changes minutes after an insult, whereas conventional T<sub>2</sub>-weighted imaging showed changes only after about 24 hours ( [Moseley et al., 1990](#)). It was later found that diffusion is not isotropic (i.e., the same in all directions) in white matter, which is interpreted as a reflection of the coherent orientation of axons in white matter fibers. According to this model, diffusion is relatively rapid parallel to axons because fewer membranes are encountered moving in this direction than in the perpendicular direction. Therefore, finding the direction of rapid diffusion determines the orientation of axons represented in a pixel or volume element (voxel) of an image. Fibers can be tracked from one voxel to the next by combining this orientation information from neighboring voxels. Continuing this procedure, the paths of fibers can be reconstructed from one region of the brain to another ( [Fig. 9.2](#)). Magnetic resonance fiber tracking can be used to visualize individual axon fascicles in the brain, and provide information on neuronal connectivity. It is thought, although not yet proven, that measurements of fascicle diameter reflect the number of constituent axons and that the rate of water diffusion perpendicular to a fiber depends on axon density.



**Figure 9.2.** Fibers tracked through the internal capsule project to different areas of the cortex. Fibers are shown (in a three-dimensional view) in relation to one of the axial diffusion weighted images from which they are calculated. The head is viewed from the front (along the midline) and somewhat lower than the image slice. (Figure courtesy of Adam Anderson.) See [color plate](#).

Clinical applications of DTI have focused on the integrity of white matter tracts, as measured by the local anisotropy of diffusion (the more highly organized an axon bundle, the less isotropic will be the diffusion). For example, DTI has been used to show loss of white matter structure in multiple sclerosis ( [Werring et al., 1999](#)), schizophrenia ( [Buchsbaum et al., 1998](#); [Foong et al., 2000](#); [Lim et al., 1999](#)), dyslexia ( [Klingberg et al., 2000](#)), and preterm birth ( [Hüppi et al., 1998](#)). It is likely that this imaging method will find many other applications in neuroimaging because it opens an entirely new window on brain structure.

### Magnetic Resonance Spectroscopy

Magnetic resonance spectroscopy (MRS) directly measures the concentrations of certain chemical compounds in the brain. It can be used to measure, for instance, compounds such as *N*-acetyl aspartate, which is thought to be an index of neuron number or integrity ( [Birken and Oldendorf, 1989](#)). It can also measure compounds involved in membrane phospholipid metabolism, pH, lactate, and concentrations of certain neurotransmitters, such as glutamate and  $\gamma$ -amino butyric acid (GABA).

The principles of MRS are similar to those of MRI. Instead of imaging primarily hydrogen nuclei, however, MRS can also image other nuclei that exhibit MR. Magnetic resonance depends on having a net nuclear spin, which entails that only nuclei having odd-numbered protons or neutrons will demonstrate MR. These nuclei include <sup>1</sup>H, <sup>13</sup>C, <sup>19</sup>F, <sup>23</sup>Na, and <sup>31</sup>P. Each of these nuclei has unique resonant frequencies in any given magnetic field because they vary in atomic weight; thus, the MR scanner's excitatory radiofrequency pulse can selectively excite any one of these nuclei simply by applying the radiofrequency pulse of the appropriate frequency. As the excited nuclei relax, the radiofrequency signal that they, in turn, emit is, like that emitted in MR imaging, in part determined by the physical environments immediately surrounding those nuclei. The frequency of the signal is altered slightly, producing a "chemical shift" fingerprint. This fingerprint is characteristic of the specific molecules in which the resonant nuclei are incorporated. The areas under specific spectral peaks then reflect the concentrations of the specific molecules generating the peaks.

In addition to the nuclei that are selectively excited, another difference of MRS from MRI is the importance of using stronger and more homogenous magnetic fields in MRS studies because they allow the preservation and discrimination of the subtle radiofrequency chemical shifts. Imaging in MRS also typically requires the use of surface coils, which localizes the region of interest to a volume of tissue near the cortical surface, although newer image processing methods make the imaging of larger regions of interest possible. Obtaining spectra from structures deep within the brain is very difficult with MRS because of their distance from the radiofrequency



coils and the iron deposits in basal ganglia and brain stem nuclei that disturb the magnetic field locally within those tissues.

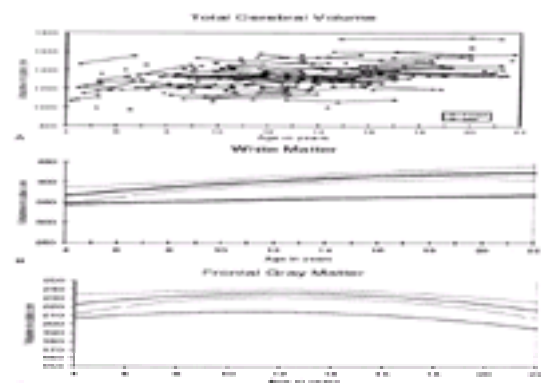
Although all potential uses of MRS are still unknown, the technique promises to provide detailed information on regional brain metabolism of high-energy phosphates such as adenosine triphosphate (ATP), regional membrane phospholipid metabolism, electrolyte balance, and the metabolism of carbohydrates, amino acids, and lipids. The numerous current limitations of MRS include poor spatial and temporal resolution (although this is rapidly improving with the development of scanners with stronger magnetic fields), poor chemical detection sensitivity, and the limited number of biologically relevant nuclei that can be imaged.

## NORMAL DEVELOPMENT

We must first identify the normal patterns of brain development if we are to test the widely held belief that many childhood neuropsychiatric disorders are manifestations of subtle deviations in normal brain development. Recent findings indicate that although total brain size attains 95% of its adult size already by age 5, various brain subcomponents continue to change considerably throughout childhood and adolescence.

### Cortical Gray

Frontal gray matter increases during childhood, reaching a peak at about 11 years in girls and 12 years in boys. Frontal gray matter then decreases during adolescence. Gray matter in the parietal lobe follows a similar developmental trajectory, although temporal lobe gray matter does not reach its maximum thickness until age 16. Occipital gray matter increases mildly throughout the childhood and adolescent years ( [Fig. 9.3](#)). These differing rates of gray matter development seem to be uniquely human ( [Giedd et al., 1999a](#)).



**Figure 9.3.** Age-related changes in selected brain regions. Total cerebral volume for males (blue) and females (red) are shown in ( A). Each line represents one subject. ( B) and ( C) figures show predicted size of total white matter and frontal gray matter with 95% confidence intervals for 243 scans from 89 unrelated males and 56 unrelated females, ages 4 to 22 years. (Figure courtesy of Jay Giedd.) See [color plate](#).

The extent to which the buildup and pruning down phase are influenced by psychiatric illness, genetics, hormones, diet, activity, or other environmental factors is an active area of research. Studies of identical and nonidentical twins, children with neuropsychiatric disorders, and people with anomalous sex chromosomes (e.g., XXY, XYY, XXYY) or hormonal abnormalities (e.g., congenital adrenal hyperplasia) will help to sort out some of the relative influences of these determinants on brain development.

### White Matter

As opposed to the regionally specific, curvilinear developmental trajectories of gray matter volume, white matter volumes tend to increase linearly and at roughly similar rates in the frontal, parietal, temporal, and occipital lobes; however, the corpus callosum (the massive white matter pathway connecting the cerebral hemispheres) increases in size more posteriorly than anteriorly during childhood and adolescence ( [Giedd et al., 1999b](#)). Newer methods to track white matter changes precisely in an individual child over time reveal robust changes in corpus callosum morphology over time intervals as short as 1 year ( [Thompson et al., 2000](#)). White matter density also has been shown to increase with age in speech-related, corticospinal, and frontotemporal pathways (Paul et al., 1999).

### Basal Ganglia

The basal ganglia are subcortical gray matter structures comprised of the caudate nucleus, putamen, globus pallidus, subthalamic nucleus, and substantia nigra. These structures not only are well known to influence movement and muscle tone, but they are also integral components of circuits mediating higher cognitive functions, attention, and affective states. Of the basal ganglia components, only the caudate, putamen, and globus pallidus currently are large enough to be readily quantifiable by MRI. The growth of these structures follows a developmental course similar to the frontal cortical gray matter ( [Giedd et al., 1996b](#)).

### Sex Differences

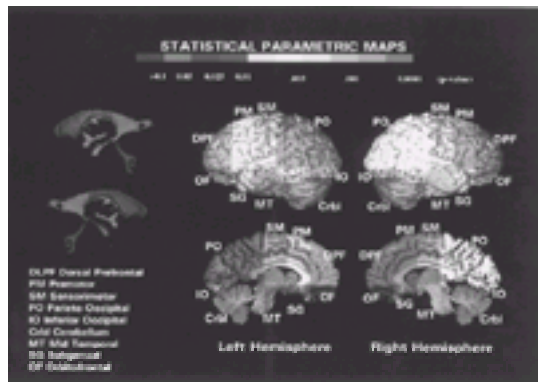
Many brain regions develop differently between boys and girls ( [Giedd et al., 1997](#)). These sex differences may be particularly relevant for child psychiatry because nearly all childhood-onset neuropsychiatric disorders have different ages of onset, prevalence, and symptomatology between the sexes. One example of sexually dimorphic brain development is the amygdala, which increases more robustly in volume in adolescent boys than girls ( [Giedd et al., 1996c](#)). Another is the hippocampus, which increases significantly more in volume in adolescent girls. These sex differences in the amygdala and hippocampus are consistent with the amygdala having a predominance of androgen receptors and the hippocampus having a predominance of estrogen receptors. The temporal lobes, amygdala, and hippocampus subserve functions of language, emotion, and memory, which are known to change dramatically from ages 4 to 18 years. The sizes of these brain structures are highly variable, even in well-screened healthy control groups ( [Lange et al., 1997](#)), thus emphasizing the need in imaging studies for large sample sizes and longitudinal studies to accurately characterize developmental curves.

## PRETERM BIRTH

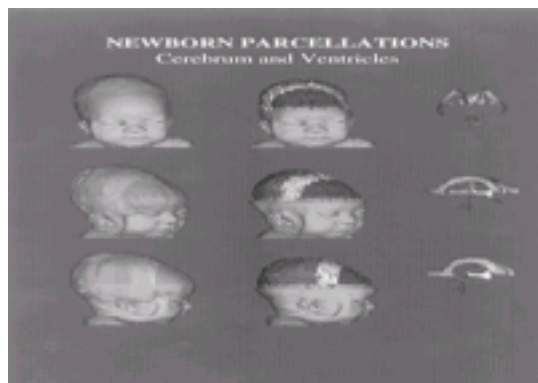
Infants weighing less than 1,500 g (3.3 lbs) at birth account for 1.5% of all live births in the United States ( [Guyer et al., 1999](#)). Survival rates of these infants currently approach 85%. The prevalence of major neurodevelopmental disturbances in these children range from 12% to 32% and are a growing public health concern ( [Hack et al., 1996](#); [Whitaker et al., 1997](#)). Preterm infants, even those who have uncomplicated neonatal courses, frequently experience serious cognitive and educational difficulties ( [Sykes et al., 1997](#)). IQ scores are nearly one standard deviation (15 points) below the population mean ( [Wolke, 1991](#)), more than half of these children require special assistance in school, and nearly 20% repeat a grade in school by the age of 8 years ( [McCormick et al., 1996](#)). The prevalence of mood and anxiety disorders, as well as attention deficit/hyperactivity disorder, is markedly elevated over the rates reported in community-based samples ( [Whitaker et al., 1997](#)).

Prior quantitative MRI studies of preterm infants have suggested the presence of reduced cortical gray and white matter volumes, as well as increased CSF in preterm infants studied at term ( [Hüppi et al., 1996](#), 1998; [Inder et al., 1999](#)). Anatomic abnormalities seem to persist through later childhood and have been noted in qualitative studies of school-aged preterm children, including enlargement of the occipital horns ( [Olsén et al., 1998](#)), white matter damage ( [Olsén et al., 1998](#); [Stewart et al., 1999](#)), and thinning of the posterior corpus callosum ( [Cooke and Abernethy, 1999](#); [Stewart et al., 1999](#)). A recent quantitative study of 25 preterm and 39 term control children studied at 8 years of age supported the findings from these qualitative studies and further documented large volume reductions in sensorimotor, premotor, midtemporal, parieto-occipital, and subgenual cortices bilaterally, as well in the basal ganglia, amygdala, hippocampus, and corpus callosum ( [Peterson et al., 2000](#)). Volumes of the cerebral ventricles were two to four times larger in the occipital and temporal horns of the preterm children. Volumes of sensorimotor and temporal cortices were associated positively with full scale, verbal, and performance IQ scores (the smaller and more abnormal the volume, the lower the IQ). Gestational age at birth was associated positively with volumes of premotor, sensorimotor, and temporal cortices. Frontal volumes were relatively preserved in the preterm children ( [Fig. 9.4](#)). These findings suggest that preterm birth is associated with regionally specific, long-term reductions in brain volume and that those morphologic abnormalities are in turn associated with poorer cognitive outcome. They dramatically emphasize the importance of perinatal and developmental

influences on long-term brain development ( [Fig. 9.5](#)).



**Figure 9.4.** Differences between preterm and term children in regional brain volumes: statistical parametric map. Differences in brain volume between preterm and term children are color-coded and displayed on a subdivided brain and ventricular system. The medial views are located slightly lateral to the interhemispheric fissure to allow visualization of hemispheric tissue. (Figure courtesy of Brad Peterson.) See [color plate](#).



**Figure 9.5.** Imaging of infant brains. The scalp, brain, and ventricular system of an infant is volume-rendered to demonstrate the ability of imaging technologies to measure these small structures in living neonates. The subdivision of these structures is the same as in [Fig. 9.4](#). (Figure courtesy of Brad Peterson.) See [color plate](#).

## AUTISM

Autism is a severe developmental disorder of communication, reciprocal social interaction, and repetitive, rigid, and odd behaviors ( [DSM-IV, 1994](#)). Evidence for a biological basis for autism is substantial ( [Schultz et al., 2000](#)), but there are as yet only a few well-replicated findings from which to begin building a model of pathophysiology. Progress has been slowed by changing diagnostic standards during the past two decades and by a clinical heterogeneity that may well be inherent to the disorder. Current conceptualizations of the pathophysiology of autism emphasize the likely existence of a broader autism phenotype that includes Asperger's syndrome and other syndromic variants. The highly overlapping symptoms of these autism spectrum conditions suggest that they may share common biological foundations, perhaps some kind of “final common pathway” to illness. Converging findings from recent postmortem studies, *in vivo* neuroimaging investigations, and human and animal lesion data suggest that the common biological foundations of autism spectrum disorders may involve abnormalities in overall brain growth, as well as more regionally specific abnormalities in structure and function of the temporal lobe, amygdala, and medial and orbital subdivisions of the frontal lobe.

### Brain Size

One of the more intriguing recent findings is that overall brain size may be increased in autism. Evidence comes from measurement of exterior head circumference ( [Lainhart et al., 1997](#)) and from direct measurement of brain volume in postmortem examinations and *in vivo* neuroimaging studies ( [Piven et al., 1996](#)). It is not yet clear whether all systems are equally affected by the volume expansion, nor is it clear what the functional significance of the expansion may be. One study has reported a selective enlargement of occipital, parietal, and temporal lobes, but not frontal cortex ( [Piven et al., 1996](#)). Increased brain size could come at the expense of the integrity of the interconnections among specialized neural systems, giving rise to a more fragmented processing structure ( [Schultz et al., 2000](#)). This disrupted integration of neural systems could contribute to a lack of “central coherence,” a processing style that makes integration of parts into wholes problematic and that may contribute to the quality of autistic symptoms ( [Frith, 1989](#)).

The timing of the origin of the abnormalities in brain growth is unknown. Some studies suggest that the growth abnormality may be postnatal ( [Lainhart et al., 1997](#)), and others suggest that it may be prenatal ( [Woodhouse et al., 1996](#)). Specifying the point in brain development when the rate of growth is most abnormal in autism would help to identify the underlying cellular mechanisms that are producing the abnormality ( [Schultz et al., 2000](#)). An origin at particular times of fetal brain development could suggest disturbances in the regulation of neuronal or glial cell proliferation, neuronal migration, or apoptosis, for instance.

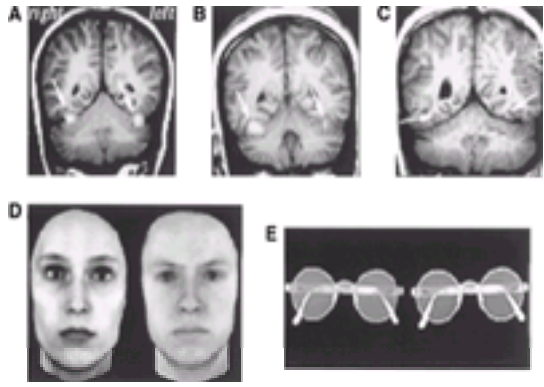
Prenatal origins of disturbed brain development have been suggested by reports of increased frequencies of abnormalities in morphology of the cerebral cortex in autistic individuals ( [Bailey et al., 1998](#); [Berthier et al., 1992](#); [Piven et al., 1990](#); [Schifter et al., 1994](#)). One study found, for example, that seven of 13 subjects with autism had cortical abnormalities, including polymicrogyria in five, schizencephaly and macrogyria in one, and macrogyria in another subject, compared with no abnormalities in 13 control subjects ( [Piven et al., 1990](#)). The cortical abnormalities are thought to derive from disturbances in neuronal migration during fetal brain development.

### Temporal Lobe

The limbic-temporal cortex forms a functional system ( [Amaral and Price, 1984](#)) that mediates social–emotional processes ( [Adolphs et al., 1994](#); [Kling and Brothers, 1992](#)). The amygdala, in particular, plays a critical role in emotional arousal, assigning significance to environmental stimuli and mediating emotional learning ( [Gaffan et al., 1988](#); [Geschwind, 1965](#); [LeDoux, 1996](#)). Persons with autism perform poorly on measures of face recognition ( [Boucher and Lewis, 1992](#); [Klin et al., 1999](#); [Langdell, 1978](#)), emotional perception and comprehension ( [Fein et al., 1992](#); [Hobson, 1986](#)), and imitating emotional expressions ( [Macdonald et al., 1989](#)). The social–emotional dysfunction found in autism may arise from functional disturbances in this temporo-limbic system ( [Baron-Cohen et al., 2000](#); [Schultz et al., 2000](#)).

Regions of the temporal lobe specialized in normal individuals for interpreting facial expressions and identifying facial identity have been mapped with fMRI, PET, and lesion data ( [Fig. 9.6](#)). Functional magnetic resonance imaging studies in autism have reported that a specific region on the ventral surface of the temporal cortex, the fusiform gyrus, fails to respond fully to the human face during perceptual tasks ( [Critchley et al., 2000](#); [Schultz et al., 2000](#)). Thus, deficits in face recognition have a clear functional correlate in higher-order perceptual systems of the ventral temporal lobe in autistic subjects. These functional disturbances could be a primary deficit in autistic individuals, an acquired (secondary) deficit caused by failed emotional learning during critical periods of early social development ( [Schultz et al., 2000](#)), or a nonspecific finding that reflects generalized disturbances in cognitive functioning in autistic individuals. Postmortem findings of abnormalities in size, density, and dendritic arborization of neurons in the amygdala, hippocampus, and entorhinal cortex are consistent with these findings of abnormal temporo-limbic functioning ( [Bauman and Kemper, 1985, 1994](#)).





**Figure 9.6.** Functional magnetic resonance image (fMRI) t-maps of the brain during face perception. The fusiform face area is shown in red or yellow (arrows) in: (A) a young adult with Williams syndrome (WS) and (B) a matched normal control. In both persons, there is a clear focus of face-related activation bilaterally in the fusiform gyrus along the ventral surface of the temporal lobe. Images are in a coronal orientation, with right and left hemisphere reversed by convention. Functional data are superimposed on anatomic images for localization. Note the normal intensity and size of the fusiform face area in WS referenced to the control subject. For contrast, (C) note the lack of activation in the fusiform gyrus in a young adult with autism. fMRI data are from a blocked experiment comparing (D) face perception to (E) nonface object perception during a “same/different” discrimination task on a 1.5 Tesla system, the threshold for displaying activations is set at  $T = 1.5$ . Regions of the brain that activate specifically to objects are shown in blue on the fMRI maps. (Figure courtesy of Robert Schultz.) See [color plate](#).

Other evidence implicating the temporal system in autism include case studies of patients with temporal lobe lesions and autisticlike behaviors ( [Gillberg, 1991](#); [Hoon and Reiss, 1992](#)) and reduced functional activity in the temporal lobes on SPECT scans in autistic subjects ( [Mountz et al., 1995](#)). Furthermore, an association between tuberous sclerosis and autism has long been recognized, and a specific association of the location of the tubers in the temporal lobe with autism has been reported ( [Bolton and Griffiths, 1997](#)).

### Amygdala

Animal models suggest that developmental abnormalities in the amygdala may play a particularly important role in the development of autistic symptoms ( [Bachevalier, 1994](#)). Bilateral damage of the amygdala shortly after birth in monkeys can produce patterns of behavior similar to those of autism, such as social isolation, lack of eye contact, impaired facial expression, and motor stereotypies. Similar lesions in adulthood fail to produce these behaviors. The early postnatal lesions do not immediately produce the signs characteristic of autism spectrum disorders ( [Bachevalier, 1994](#); [Thompson, 1981](#)). Rather, the deficits emerge during early development. These findings are consistent with the hypothesis that autistic symptoms are, in part, a function of faulty early emotional learning mediated by limbic system pathology. Furthermore, monkeys with early lesions to the amygdala and surrounding entorhinal cortex later in adulthood exhibit abnormalities in the frontal cortex on MRS, and the regulation of dopaminergic activity in frontal cortex and the basal ganglia is abnormal ( [Bertolino et al., 1997](#); [Saunders et al., 1998](#)).

Accurate measurement of the amygdala with MRI is difficult. Findings from two studies of amygdala volume have produced opposite findings. Amygdala volumes were significantly smaller in 14 autistic adolescents and adults without mental retardation when compared with 14 age- and IQ-matched male controls ( [Aylward et al., 1999](#)). The size of the hippocampus was smaller only when considered relative to volume of the entire brain, which was enlarged, as expected. Larger volumes of the amygdala were reported, however, in a second study using a very different measurement approach in 15 persons with autism compared with matched controls ( [Abell et al., 1999](#)).

### Frontal Lobe

A  $P^{31}$  MRS study found alterations in phospholipid metabolism and evidence of enhanced membrane degradation in the dorsal prefrontal cortex (PFC) of autistic individuals ( [Minschew et al., 1993](#)). This early study suggested that the dorsal PFC of high-functioning autistic subjects is hypermetabolic. More widespread decreases in frontal lobe perfusion subsequently have been suggested by two SPECT studies ( [George et al., 1992](#); [Zilbovicius et al., 1995](#)).

Functional imaging studies suggest that dorsomedial prefrontal cortex (primarily Brodmann's area 9) is the critical substrate for “social cognition,” that is, for thinking about other's thoughts, feelings, and intentions ( [Baron-Cohen et al., 1994](#); [Goel et al., 1995](#)), and that the ventromedial PFC is involved in processing normal affects ( [Lane et al., 1997a,b](#); [Reiman et al., 1997](#)). Function of these prefrontal cortices seems to be disturbed in persons with autism spectrum conditions. A PET study, for instance, reported reduced dopaminergic activity in the medial prefrontal cortex of autistic subjects ( [Ernst et al., 1997](#)). Reduced glucose metabolism also has been reported in a subdivision of the anterior cingulate gyrus (right Brodmann's area 24) in persons with autism engaged in a verbal memory task ( [Haznedar et al., 1997](#)). A pilot PET study of Asperger's syndrome using a theory of mind task showed specific engagement of the medial PFC, except that the center of activation was displaced below and anterior in patients compared with controls ( [Happé et al., 1996](#)).

### Posterior Fossa

Considerable excitement in the study of autism followed initial MRI reports of reduced volumes of portions of the cerebellum (vermian lobules VI and VII). The initial study reported a 19% reduction in size of these regions in 18 autistic and 12 normal control subjects ( [Courchesne et al., 1988](#)). These initial MRI findings were viewed as consistent with small postmortem studies of persons with autism that revealed a variety of abnormalities, including a significant decrease in the number of Purkinje cells and a variable decrease in granule cells throughout the cerebellar hemispheres ( [Arin et al., 1991](#); [Bauman and Kemper, 1985, 1994](#)). The postmortem comparisons, however, showed that the reduction in Purkinje cell numbers was confined to the cerebellar hemispheres and not observed in the vermis ( [Bauman and Kemper, 1996](#)).

Subsequent studies, some with sizable samples, had trouble replicating the initial findings of cerebellar hypoplasia, particularly when controlling for subject characteristics such as age, sex, and IQ ( [Hashimoto et al., 1992](#); [Kleiman et al., 1992](#); [Piven et al., 1992](#); [Ritvo and Garber, 1988](#)). Subsequently, a reanalysis of data from previous work ( [Courchesne et al., 1988, 1994](#); [Kleiman et al., 1992](#); [Piven et al., 1992](#)) suggested the presence of a bimodal distribution in the sizes of vermian lobules VI and VII in autistic subjects compared with a unimodal distribution in the control subjects ( [Courchesne et al., 1994](#)). This bimodal distribution argued for the existence of hypoplastic and hyperplastic subtypes of vermian size in autistic subjects. Significant IQ differences between autistic and control groups call into question the validity of this finding, however, because IQ seems to be associated with hypoplasia of the cerebellar vermis ( [Piven et al., 1992](#)). These difficulties underscore the importance of rigorously controlled studies in the study of autism.

## FRAGILE X SYNDROME

Fragile X syndrome, one of the leading causes of inherited mental retardation, is caused by a single gene mutation, altering brain development and producing a distinctive physical, cognitive, and neuropsychiatric phenotype. Occurring in one out of every 2,000 to 4,000 live births, the fragile X mutation affects males and females differently, as would be expected in an X-linked disorder. In boys, fragile X is associated with mental retardation, difficulties in visuospatial and memory function, hyperactivity, gaze avoidance, autisticlike features, and poor social communication. In girls who are heterozygous for the fragile X full mutation, the syndrome is associated with variable intellectual functioning (ranging from mild mental retardation to normal cognition), math difficulties, attentional deficits, anxiety, and poor social communication ( [Reiss et al., 2000](#)).

Disruption of the expression of the fragile X gene (FMR1) produces lower levels of the fragile X mental retardation protein (FMRP) and abnormal dendritic spine morphology, which has been observed in human neuropathologic studies of individuals with fragile X ( [Hinton et al., 1991](#)) and studies of fragile X knockout mice ( [Comery et al., 1997](#)). Early studies reported that FMRP normally is most abundantly expressed in neurons of the hippocampus, nucleus basalis, and cerebellum ( [Devys et al., 1993](#); [Hinds et al., 1993](#)). This prompted initial neuroimaging studies to focus on those (and other subcortical) regions in fragile X ( [Reiss et al., 1991a,b, 1994, 1995](#)).

### The Posterior Fossa



When compared to age- and IQ-matched individuals with developmental disabilities and to age-matched controls with normal IQs, 14 males with fragile X were noted to have reductions in the areas of lobules VI and VII of the cerebellar vermis, as well as increases in size of the adjacent fourth ventricle ( [Reiss et al., 1991a](#)). In a complementary study ([Reiss et al., 1991b](#)), females with fragile X also had a reduced size of vermal lobules VI and VII and increases in volume of the fourth ventricles, although these abnormalities in females were not as pronounced as those in males with fragile X. This “intermediate gene dosage effect” of the mutation in heterozygote females is consistent with the sex differences expected in an X-linked disorder.

Hypoplasia of the cerebellar vermis was associated with behavioral and cognitive dysfunction in fragile X syndrome in two studies of school-age girls. In one of those studies, a significant negative correlation of the area of lobules VI and VII with parental ratings of autisticlike behaviors was observed, particularly with measures of social communication and stereotypic behaviors ( [Mazzocco et al., 1997](#)). In the second study, posterior vermis size correlated significantly with Full-Scale, Verbal, and Performance IQ scores, and with measures of executive functioning ( [Mostofsky et al., 1998](#)). The cerebellar vermis is thought to play a role in modulating sensory stimulation and mediating higher-order cognitive functions. This supports the hypothesis that the vermis may be a component of a circuit underlying the behavioral difficulties and cognitive dysfunction observed in individuals who have fragile X syndrome.

That the size of the vermis is reduced in individuals who have fragile X but not those who have nonspecific developmental delays suggests that these findings have some degree of specificity for the fragile X syndrome. The presence of cerebellar hypoplasia in children with autism and the association of cerebellar hypoplasia with autisticlike symptoms in children with fragile X has led some researchers to suggest that fragile X can be viewed as an etiologically homogeneous model for understanding the pathophysiology of autism ( [Reiss et al., 2000](#)).

### **Temporal Lobe**

The presence of deficits in learning and memory in individuals with fragile X syndrome has motivated study of the temporal lobe in this disorder. Although two studies reported a 10% to 20% enlargement of hippocampal volumes in individuals with fragile X ( [Kates et al., 1997](#); [Reiss et al., 1994](#)), a third study was unable to confirm these findings ( [Jakala et al., 1997](#)). Larger sample sizes are needed to settle these discrepancies.

One cross-sectional study also reported that hippocampal volumes were positively associated with age and that superior temporal gyrus (STG) volumes were negatively associated with age in subjects with fragile X ( [Reiss et al., 1994](#)). These associations of age with volume in the hippocampus and STG, combined with the known age-related decline in intellectual functioning in fragile X boys, suggests that the fragile X mutation may continue to exert a detrimental effect on brain development even into later childhood. Longitudinal, prospective studies are needed to test this hypothesis directly ( [Kraemer et al., 2000](#)).

### **Caudate and Thalamus**

One study compared 31 females and 18 males with fragile X to samples of IQ-matched controls and normal controls ( [Reiss et al., 1995](#)). The volumes of the caudate nucleus were increased in both genders with fragile X, lateral ventricle volumes were increased in fragile X males, and thalamic volumes were increased in fragile X females.

### **Frontal and Parietal Lobes**

Although deficits in higher-order cognitive functions (e.g., executive function and working memory) that are putatively mediated by frontal and parietal cortices have been reported in patients with fragile X, morphologic imaging studies of the frontal and parietal lobes have not yet been reported. However, recent functional studies have advanced our understanding of the relation among gene dosage, cortical activation, and behavior in this disorder. An fMRI study, for example, reported reduced activity in frontal-subcortical circuits in individuals with fragile X ( [Hjalgrim et al., 1999](#)). The prefrontal-caudate circuits are associated with the regulation of impulses and attention, and lesions of these circuits have previously been associated with hyperactivity, inattention, and perseveration—all features of the fragile X behavioral phenotype. In addition, a significant association has been reported between FMRP levels and brain activation in the right inferior frontal, left and right middle frontal, and supramarginal gyri during a working memory task in 10 females with fragile X ( [Menon et al., 2000](#)).

### **Conclusions**

Taken together, these findings suggest that the disruption of FMR1 gene expression affects FMRP levels across various cortical and subcortical regions of the brain in fragile X patients, consistent with the recent finding that FMRP is widely distributed throughout the mammalian brain ( [Feng et al., 1997](#)). Nevertheless, we do not fully understand the neural circuits through which FMR1 gene expression disrupts cognitive and emotional functions throughout childhood. Longitudinal studies of brain morphology and function therefore are needed in patients with fragile X. Of particular importance is defining the relation of FMRP protein levels with brain structure, function, and behavior.

## **VELOCARDIOFACIAL SYNDROME**

Velocardiofacial syndrome (VCFS) (also known as the 22q11 deletion syndrome) is caused by a microdeletion on chromosome 22q11.2. It has a variable phenotype that includes congenital anomalies, language delays, learning disabilities, and disorders of attention, affect, and anxiety. Approximately 30% of individuals with VCFS develop schizophrenia in late adolescence or young adulthood. This has stimulated interest in defining the anatomic and functional anomalies of the central nervous system (CNS) that are associated with this specific genetic mutation and that may be associated with the development of severe psychiatric illness.

### **Qualitative Studies**

Initial MRI studies were qualitative and based on clinically acquired scans. White matter hyperintensities, small cerebellar vermis, and cysts adjacent to the anterior horns of the lateral ventricles were reported in a sample of 11 children with VCFS ( [Mitnick et al., 1994](#)). In a study of 11 adults who had both VCFS and schizophrenia ( [Chow et al., 1999](#)), 90% had white matter hyperintensities, 45% midline anomalies, 54% cerebral atrophy or enlargement of the lateral ventricles, and 36% cerebellar hypoplasia.

### **Cerebral Cortex**

Initial quantitative MRI studies of children with VCFS have focused on abnormalities of the cerebral cortex. Two separate studies of the cerebral cortex that used the same measurement methodologies have been reported. The first compared 15 children with VCFS ( [Eliez et al., 2000](#)), and the second compared 10 children with VCFS ( [Kates et al., 2001](#)) to age- and sex-matched control samples. Both studies found total brain volume to be reduced by nearly 11% in children with VCFS. White matter was reduced (most notably in nonfrontal regions of the cortex) more than gray matter in subjects with VCFS. Both studies also found disproportionate reductions in the left parietal lobe of children with VCFS. One study found the reductions limited to parietal gray matter ( [Eliez et al., 2000](#)), whereas the other study found the reductions limited to parietal white matter ( [Kates et al., 2001](#)). Reductions in parietal tissue are consistent with the VCFS neuropsychological phenotype, which includes deficits in visual perception and spatial working memory.

### **Posterior Fossa**

Preliminary studies suggest that regions of the posterior fossa are aberrant in children and adolescents with VCFS ( [Eliez et al., 2000](#)). Both the pons and lobules VI to VII of the cerebellar vermis appear to be significantly reduced in children with VCFS.

### **Temporal Lobe**

Initial quantitative studies suggest the presence of temporal lobe abnormalities in VCFS patients. Although brain-corrected volumetric differences in temporal lobe regions have not been reported between VCFS patients and controls, a cross-sectional study suggested possible age-related reductions in volumes of the temporal lobe and left hippocampus. If this latter finding is replicated, it would provide an additional link between the neuroanatomic anomalies found VCFS and those found in schizophrenia because temporal lobe abnormalities are probably the most robust abnormalities noted in adults who have schizophrenia.

## Conclusions

Neuroanatomic studies of individuals with VCFS are just beginning. Replication of the initial findings is needed. Imaging modalities such as DTI and MRS may prove useful for investigating and understanding the pathophysiology of the white matter reductions that have been reported in the cerebral cortex. Finally, longitudinal studies are needed to help identify the neuroanatomic abnormalities (as well as neurocognitive and behavioral features) that place children with VCFS at the greatest risk for developing severe psychiatric illness in adulthood.

## CHILDHOOD ONSET SCHIZOPHRENIA

Childhood onset schizophrenia is a rare, clinically severe form of schizophrenia associated with the premorbid disruption of cognitive, linguistic, and social development ([Jacobsen and Rapoport, 1998](#)). Although the disorder is rare, understanding the brain changes that mediate the emergence of schizophrenic symptoms at an unusually early age may improve our understanding of the developmental abnormalities that trigger the later onset of schizophrenia and that determine the severity and course of symptoms.

Imaging studies thus far have focused on brain morphology using MRI. Fewer studies have examined indices of brain neuronal integrity using MRS or brain function using PET or SPECT. Studies of brain function in this population have been limited by the severe positive and negative symptoms that these patients experience and their consequent inability during scanning to be still and perform cognitive tasks.

### Brain Volumes

Perhaps the most consistent finding across studies of brain morphology in childhood onset schizophrenia is that these children have smaller brains and enlarged ventricles ([Hendren et al., 2000](#); [Sowell et al., 2000](#)). Longitudinal studies have shown that children with schizophrenia undergo a fourfold greater decrease in cortical gray matter than do controls during adolescence, and that this decrease is most prominent in frontal and temporal regions ([Rapoport et al., 1999](#)). This results in a more rapid decrease in total brain and hippocampal volume and a more marked increase in ventricular volume during this period in patients than controls ([Giedd et al., 1999](#); [Jacobsen et al., 1998](#)). Decline in volume of the right posterior superior temporal gyrus during this adolescent period has been found to predict the severity of positive symptoms of psychosis at follow-up ([Jacobsen et al., 1998](#)). The rate of decline in gray matter volume attenuates by early adulthood, possibly explaining why longitudinal studies of adults with schizophrenia have generally not found progressive age-related changes in brain volume ([Giedd et al., 1999](#)).

Cross-sectional and longitudinal studies of children and adolescents with childhood onset schizophrenia have also documented the presence of a reduced size of the thalamus in the midsagittal plane as well as enlarged basal ganglia structures in patients receiving typical neuroleptic medication ([Hendren et al., 2000](#)). Basal ganglia volumes were found to decrease to normal levels after 2 years of therapy with the atypical antipsychotic medication, clozapine ([Frazier et al., 1996](#)). This reversible enlargement of basal ganglia structures associated with typical neuroleptic therapy has also been observed in adults with schizophrenia. Although the mechanism of this enlargement is unknown, it may stem from depletion of striatal dopamine ([Jacobsen et al., 2001](#)).

### Neurochemical Studies

Magnetic resonance spectroscopy was used to measure the ratio of  $N$ -acetyl-aspartate to creatine (NAA:Cr), an index of neuronal number and integrity, in two samples of patients with childhood onset schizophrenia and in one sample of children with schizophrenia spectrum disorders. All three studies found lower NAA:Cr ratios in the frontal lobes of children with schizophreniform disorders ([Bertolino et al., 1998](#); [Brooks et al., 1998](#); [Sowell et al., 2000](#)). One study in addition observed decreased NAA:Cr ratios in the hippocampus bilaterally ([Bertolino et al., 1998](#)). These findings are similar to the findings of altered brain NAA:Cr ratios in adult onset schizophrenia.

### Functional Imaging Studies

One small study of blood flow using xenon-133 inhalation showed significant hypofrontality in a sample of 10 neuroleptic-naïve adolescents with schizophrenia compared with age- and sex-matched controls ([Chabrol et al., 1986](#)). A second study examined cerebral glucose metabolism using PET in adolescent subjects with childhood onset schizophrenia and healthy adolescent controls ([Jacobsen et al., 1997](#)) during an auditory continuous performance task. Global cerebral glucose metabolism did not differ between groups. A significantly increased metabolic rate was observed, however, in the supramarginal gyrus, inferior frontal gyrus, and insular cortex of the schizophrenia group, and a decreased metabolic rate was observed in the middle and superior frontal gyri.

## Conclusions

Brain imaging studies of patients with childhood onset schizophrenia have demonstrated a consistent pattern of abnormalities similar to those observed in adult onset schizophrenia. The accelerated loss of cortical and subcortical gray matter tissue in these patients, however, may represent a distinct pathologic process that is confined to adolescence. The rate of this accelerated loss of gray matter in certain regions may influence clinical outcome. A critical question to address in future research is whether this accelerated loss of gray matter is unique to childhood onset forms of schizophrenia or whether it is also present premorbidly in individuals who develop schizophrenia as adults. Addressing this question will require longitudinal brain imaging studies of high-risk or prodromal patients.

## ATTENTION DEFICIT/HYPERACTIVITY DISORDER

Early imaging studies of attention deficit/hyperactivity disorder (ADHD) were hampered by the absence of explicitly formulated diagnostic criteria and by the poor ability of CT scans to differentiate between gray and white matter. For the past decade, an increasing number of investigators have applied anatomic and functional MRI, as well as PET or SPECT, to the study of individuals with ADHD. Although many unsettled questions remain, a relatively coherent picture of the neuroanatomic basis of ADHD is beginning to emerge.

### Fronto-Striatal Circuits

A well-known PET study performed in adults with ADHD ([Zametkin et al., 1990](#)) reported decreased cerebral metabolism throughout the brain, particularly in frontal regions. Unfortunately, attempts to replicate and extend this approach to adolescents yielded mostly conflicting and unconvincing results. A number of groups began concurrently to explore anatomic MRI in children with ADHD. Initial studies used small samples and relatively primitive measurement techniques, but they provided further evidence for abnormalities in frontal, as well as in striatal brain regions ([Giedd et al., 1994](#); [Hynd et al., 1990, 1991, 1993](#)). The pattern of fronto-striatal abnormalities, particularly volume reductions in the right hemisphere, was confirmed in the largest anatomic MRI study of boys with ADHD ([Castellanos et al., 1996](#)). Other groups have generally supported those findings, although not always with regard to laterality ([Aylward et al., 1996](#); [Filipek et al., 1997](#)).

### Cerebellum

Numerous functional imaging studies and basic science research have suggested the importance of the interplay between prefronto-striatal circuits and the cerebellum, particularly in the regulation of attention ([Middleton and Strick, 1994](#)). These considerations motivated an examination of the cerebellum in ADHD. One small and well-delineated region, the posterior-inferior vermis, subsequently, was found to be smaller in three independent samples of children with ADHD ([Berquin et al., 1998](#); [Castellanos et al., 2001](#); [Mostofsky et al., 1998](#)). Although the functional significance of a smaller volume in this small region is unclear, it may be relevant that this particular region is unique in that it is one of the very few cerebellar tissues to receive dopaminergic innervation ([Melchitzky and Lewis, 2000](#)).

### Dopaminergic Systems

Although nature is rarely so simple, the longstanding theme of dopamine-linked abnormalities in ADHD continues to accumulate supporting evidence from neuroimaging investigations. Besides the modest but replicated associations between ADHD and a pair of dopaminergic genes ([Swanson et al., 2000](#)), fMRI studies have found abnormalities in dopamine-influenced regions of the brain, such as the striatum ([Rubia et al., 1999](#); [Vaidya et al., 1998](#)), prefrontal cortex ([Rubia et al., 1999](#)) and the anterior cingulate cortex ([Bush et al., 1999](#)). Two ligand-based SPECT studies of the dopamine transporter system in adults with ADHD demonstrated a significant and robust elevation of dopamine transporter levels in the ADHD subjects ([Dougherty et al., 1999](#); [Krause et al., 2000](#)). In one of these studies ([Krause et](#)



al., 2000), all 10 patients were medication-naïve at the time of their initial scan. In addition, all underwent a second scan after 4 weeks of treatment with 15 mg methylphenidate daily. This low-dose treatment resulted in a 30% decrease in dopamine transporter binding to levels somewhat below those of normal controls. If confirmed, these results suggest a relatively straightforward model of adult ADHD pathophysiology in which an excess of dopamine transporters produces a deficit in striatal dopamine functioning owing to excessive reuptake. Whether this abnormality represents the basic genetic “lesion” or whether it reflects a compensatory overproduction of dopamine transporters, however, is unclear. This question will not be answered easily because of the practical impossibility of studying normal control children with even small doses of radiation (required for SPECT or PET) and because even the best PET cameras and ligands are currently unable to quantify dopamine transporter levels *in vivo* outside of the striatum, such as in frontal cortex or cerebellum.

Ongoing anatomic MRI studies in ADHD subjects are examining the role of stimulant treatments in producing deleterious anatomic brain changes, as has been charged by some critics. These studies are also examining the possible abnormalities in brain development associated with ADHD.

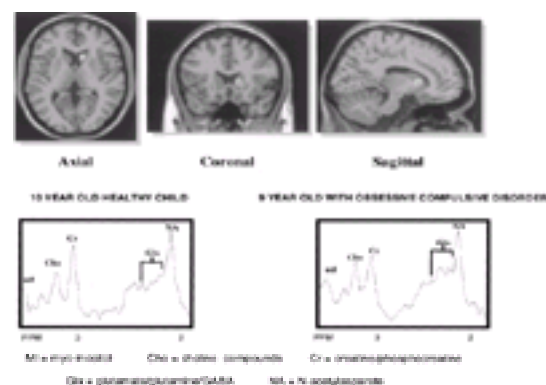
## OBSESSIVE-COMPULSIVE DISORDER

Investigations of childhood OCD have tended to focus on patients near the onset of their illness. This approach minimizes potentially confounding factors that include illness duration and exposure to CNS-active medications.

### Striatum

The striatum is believed to be a primary locus of abnormality in OCD (Rauch et al., 1998). MRI and CT studies of striatal volumes in OCD adults, however, have been highly variable in their findings. Volumetric studies in children with OCD have been more consistent in identifying abnormalities of the striatum. One early CT study reported reduced caudate volumes in adolescent and young adult male OCD patients (Luxenberg et al., 1988). A later MRI study of younger OCD patients, 7 to 17 years of age, also reported smaller striatal volumes (Rosenberg et al., 1997a). Striatal volumes correlated inversely with OCD symptom severity, but they did not correlate with illness duration, suggesting that decreased striatal volumes may be a central abnormality in pediatric-onset OCD.

Elevated glutamate concentrations have also been observed in MRS studies of the caudate nucleus but not in the occipital lobe of treatment-naïve pediatric OCD patients (Rosenberg et al., 2000a) (Fig. 9.7), suggesting that the glutamate abnormalities may be localized to specific striatum-based brain circuits in children with OCD. Glutamate concentrations in the caudate decreased significantly to normal levels after 12 weeks of treatment with paroxetine. Changes in caudate glutamate concentrations correlated positively with changes in OCD symptom severity during treatment, indicating that elevated pretreatment glutamate concentrations in the caudate predicted a better symptomatic response to paroxetine. In contrast, caudate glutamate concentrations did not decrease in pediatric OCD patients after cognitive behavioral therapy (Benazon et al., 2000). Thus, normalization of caudate glutamate concentrations during SSRI treatment in pediatric OCD demonstrates some specificity to treatment modality—it is not simply a generalized treatment effect.



**Figure 9.7.** Illustration of voxel placement in the left caudate nucleus. Proton magnetic resonance spectroscopy of an 0.7-mL volume of interest centered in the left caudate in a 10-year-old healthy control and a 9-year-old treatment-naïve patient with obsessive-compulsive disorder as shown on the T1-weighted magnetic resonance images. (From Rosenberg DR, Keshavan MS, Dick EL, et al.: Corpus callosal morphology in treatment-naïve pediatric obsessive compulsive disorder. *Progr Neuro-Psychopharmacol Biol Psychiatry* 21:1269–1283, 1997a.) (Figure courtesy of David Rosenberg.)

### Prefrontal Cortex

The prefrontal cortex is the major “decision-making” center of the brain. It subserves executive-type cognitive and behavioral processes that include abstraction, inhibiting inappropriate impulses and desires, and delaying responses (Fuster, 1997). An initial study of prefrontal cortical volume in pediatric OCD patients did not reveal significant differences from control patients (Rosenberg et al., 1997a).

A subsequent investigation demonstrated the presence of a significantly larger genu of the corpus callosum in 21 psychotropic-naïve children with OCD compared with healthy controls (Rosenberg et al., 1997b). Because the genu is the region of the corpus callosum that interconnects the ventral portions of the prefrontal cortex in each hemisphere (de Lacoste-Utamsing et al., 1985; Seltzer and Pandya, 1986), the finding of a larger genu in OCD children suggests the involvement of ventral prefrontal cortex in the pathophysiology of OCD. Other investigators have recently reported increased corpus callosal signal intensity localized to the region of the genu and have hypothesized that this may be owing to increased myelin sheath thickness (MacMaster et al., 1999).

These findings prompted examination of subregions of the prefrontal cortex in OCD children (Rosenberg and Keshavan, 1998). Increased anterior cingulate volumes but not in dorsolateral prefrontal or posterior cingulate cortices were observed in OCD children. Increased anterior cingulate volumes in turn correlated positively with severity of OCD symptoms but not illness duration.

### Thalamus

The thalamus is a major motor and sensory relay station, and it is the final subcortical input to the frontal lobe. It stimulates cortical output when released from the inhibitory tonic influence of the striatum (Baxter et al., 1996). Increased thalamic volumes have been reported in OCD children (Gilbert et al., 2000). After 12 weeks of therapy with paroxetine, thalamic volume decreased significantly in these same OCD children to control levels. The change in volume correlated positively with the reduction in OCD symptom severity after the paroxetine treatment. This finding may be specific to SSRI therapy, as no significant change in thalamic volume was observed in treatment-naïve pediatric OCD patients treated for 12 weeks with cognitive behavioral therapy (Rosenberg et al., 2000b). Furthermore, paroxetine-induced reduction in thalamic volumes seems to be localized to medial thalamic regions (Nolan et al., personal communication, January 2001).

These findings are intriguing in view of recent findings in which MRS localized neurochemical marker abnormalities in pediatric OCD patients to the medial but not the lateral thalamus (Fitzgerald et al., 2000). This marker (*N*-acetyl-aspartate) is believed to be a reliable indicator of neuronal viability (Birkin and Oldendorf, 1989). After 12 weeks of effective paroxetine therapy, concentrations of this marker increased in the OCD patients (Amponsah et al., 2000). Longitudinal studies are needed to measure changes in the concentrations of this marker during the course of illness.

## Conclusions

These studies suggest that pediatric OCD may be associated with a reversible glutamate-mediated dysfunction of a ventral prefrontal-striatal-thalamic system. Glutamate concentration may be a marker of illness and a predictor of treatment response. Various brain patterns detected by brain imaging studies, for example, may predict response (or lack, thereof) to a particular treatment (e.g., medication versus cognitive behavioral therapy).

## TOURETTE'S SYNDROME



Similar to theories of the pathogenesis of OCD and ADHD, the etiology of Tourette's syndrome (TS) is thought to involve disturbances in cortical-subcortical circuitry involved in the regulation of cognition, affect, and movement. The common involvement of these circuits across these disorders may account in part for their common co-occurrence in clinical populations ( [Peterson et al., 1998](#)).

## Basal Ganglia

Reduced volumes of the basal ganglia have been reported in both adults and children with TS, particularly in the putamen and globus pallidus ( [Peterson et al., 1993](#); [Singer et al., 1993](#)) (Fig. 9.8). These studies also found some evidence for the presence of reduced asymmetry in these regional volumes and for abnormalities in basal ganglia tissue characteristics ( [Peterson et al., 1994](#)). A subsequent study of monozygotic (MZ) twin pairs with TS reported reduced volumes of the caudate nucleus in members of the twin pairs who had more severe tics ( [Hyde et al., 1995](#)). Because the twins were genetically identical, reduced volumes of the right caudate nucleus were presumably caused by nonshared environmental determinants and not the effects of TS vulnerability genes. These anatomic findings are consistent with reports of natural lesions in the basal ganglia that seem to have produced or worsened tic symptoms ( [Liu et al., 1999](#); [Peterson et al., 1996](#)).



**Figure 9.8.** Basal ganglia definitions. The basal ganglia are important in the pathophysiology of several childhood disorders, including attention deficit/hyperactivity disorder, obsessive-compulsive disorder, and Tourette's syndrome. In the top row are presented varying views of the basal ganglia in a high-resolution MRI scan: axial (*left*), coronal (*middle*), and sagittal (*right*). The caudates are red or light blue, the putamen are green or purple, and the globus pallidus nuclei are blue or yellow. In the bottom row are oblique frontal views of three-dimensional volume rendered basal ganglia nuclei, rotated around the anterior-posterior axis successively by 180 degrees from left to right in the row. (Figure courtesy of Brad Peterson.) See [color plate](#).

A host of functional imaging studies have suggested that basal ganglia are dysfunctional in TS subjects. Reduced resting metabolism and blood flow to these regions has been consistently reported ( [Braun et al., 1993](#); [Chase et al., 1986](#); [Hall et al., 1990](#); [Moriarty et al., 1995](#); [Riddle et al., 1992](#)). Many of the early PET and SPECT studies, however, actually did not have adequate resolution to distinguish clearly metabolism and blood flow abnormalities within the basal ganglia regions or between the basal ganglia and nearby cortical regions. Because of ethical concerns about the use of PET and SPECT in children, metabolism and blood flow studies were of symptomatic TS adults, so that generalizing their findings to children is problematic ( [Peterson, 2000](#); [Peterson and Thomas, 2000](#)).

One fMRI study examined the changes in brain activity associated with the voluntary suppression of tics in TS adults ( [Peterson et al., 1998](#)). These subjects alternated between allowing themselves to tic freely and suppressing their tics completely. Tic suppression was associated with increased activity of the ventral portion of the right caudate nucleus and in numerous cortical regions, especially prefrontal and temporal cortices. Tic suppression was also associated with decreased activity of the ventral globus pallidus, putamen, and thalamus bilaterally. The severity of tic symptoms correlated with the change in activity of the basal ganglia and thalamus regions and suggested that as symptom severity increased, the changes in subcortical activity during tic suppression decreased. These findings suggest that the increases in neural activity of subcortical regions—increases in the right caudate and decreases in the rest of the subcortex—participate in the suppression of tics, and that when these cortical-subcortical braking mechanisms fail, tics may be progressively more likely to escape the inhibitory influences of these circuits on motor behavior.

Other functional studies have imaged the dopamine neurotransmitter system in the striatum of TS adults. Several early studies failed to detect abnormalities in the D<sub>2</sub>-receptor system ( [Brooks et al., 1992](#); [Singer et al., 1992](#); [Turjanski et al., 1994](#)). A subsequent preliminary SPECT study suggested the presence of elevated presynaptic dopamine transporter levels in TS subjects ( [Malison et al., 1995](#)), but this finding was not replicated in a larger TS SPECT study ( [Heinz et al., 1998](#)). Another SPECT study of five adult TS MZ twin pairs reported an increased availability of the D<sub>2</sub>-receptor in the caudate nucleus of all five of the more severely affected cotwins ( [Wolf et al., 1996](#)), and differences between cotwins in receptor availability correlated significantly with the differences between twins in tic symptom severity. D<sub>2</sub>-like receptors, however, were reported to be normal in two subsequent PET studies ( [Wong et al., 1997](#)), emphasizing the need to replicate the findings from the MZ twins. Finally, a PET study reported higher F-DOPA accumulation in the left caudate nucleus and right midbrain of 11 TS adolescents compared with 10 sex-matched normal controls ( [Ernst et al., 1999](#)). The results if replicated suggest the presence of increased DOPA decarboxylase enzyme activity in these regions, which would suggest the presence of either a larger number of dopaminergic synapses or enzyme up-regulation in the caudate and midbrain. Clearly, more research is needed to define the role of the dopamine system in TS.

## Corpus Callosum

The apparent abnormalities in structural lateralization of the basal ganglia in TS prompted the search for abnormalities in other structurally lateralizing neural systems, including the corpus callosum. This structure was found to be smaller throughout all of its subregions in TS adults ( [Peterson et al., 1994](#)). A subsequent study in children, however, found the structure to be larger in children who had "pure" TS (i.e., without OCD or ADHD), with abnormalities confined to the anterior portions of the structure. These seemingly contradictory findings could reflect chance findings owing to methodologic differences, or they could reflect real morphologic differences in the callosum of TS children compared with adults. The latter possibility is strengthened by recent age-specific findings in the cortices of TS children and adults (see the following).

## Cortical Regions

A large anatomic imaging study examined regional cortical and ventricular volumes in 155 TS and 131 healthy children and adults ( [Peterson et al., 2001](#)). The most prominent regional effect in TS subjects was a larger volume of dorsal prefrontal cortex. This effect, however, derived primarily from larger volumes in TS children. Group differences were less prominent in older children owing to an inverse association of age with prefrontal volumes in the TS group. In fact, by adulthood, the TS subjects had smaller dorsal prefrontal volumes. Similar effects that were specific to age group were observed in the parieto-occipital region, where volumes were larger in younger TS children but negligibly larger by late adolescence. In adults, parieto-occipital volumes were still significantly larger in TS men but were significantly smaller in TS women. Group differences were nearly identical in inferior occipital regions, except that volumes were not significantly different for TS men. Because tic symptoms typically attenuate through adolescence, smaller prefrontal volumes in symptomatic TS adults could have contributed to the relatively unusual persistence of their symptoms into adulthood, and then to the subsequent clinical identification and inclusion of those adults in the imaging study.

Prominent sex differences were observed in normal individuals but not in TS subjects in the parieto-occipital region. The finding was strongest in adult subjects. This difference between groups was owing to relatively larger volumes in TS males and smaller volumes in TS females. This finding is consistent with a previously hypothesized involvement of sexually dimorphic brain regions in the pathophysiology of TS ( [Peterson et al., 1992](#)) and may account in part for the predilection of TS to affect males more often than females.

Regional cerebral volumes were significantly associated with the severity of tic symptoms in orbitofrontal and parieto-occipital regions, suggesting that smaller volumes in these regions may provide insufficient inhibitory reserve to help suppress tics. Larger prefrontal volumes in the TS subjects may represent an activity-dependent structural plasticity that could help to suppress tics, consistent with numerous preclinical and clinical studies suggesting that the orbitofrontal region plays an important role in inhibitory control ( [Diamond and Goldman-Rakic, 1989](#); [Divac et al., 1967](#); [Drewe, 1975](#); [Fuster, 1997](#); [Goldman-Rakic, 1987](#); [Iverson and Mishkin, 1970](#); [Luria, 1980](#); [Mishkin and Manning, 1978](#); [Rosvold and Mishkin, 1961](#)). The presence of larger prefrontal volumes that correlate with symptom severity

is also consistent with the prior finding that prefrontal activation is required by TS subjects to suppress their tics ( [Peterson et al., 1998](#)). The larger premotor regions in men compared with smaller volumes in TS boys, in contrast, were hypothesized to represent long-term, activity-dependent effects in the motor system associated with the presence of tics. Longitudinal imaging studies are required to understand better the age-associations seen in TS imaging studies, particularly whether age-related findings represent central deficits or compensatory responses to the longstanding presence of tics and related symptoms.

## PEDIATRIC AUTOIMMUNE NEUROPSYCHIATRIC DISORDERS

Exacerbations of tic and OCD symptoms associated with group A beta-hemolytic *Streptococcus* (GABHS) infections are now typically referred to as pediatric autoimmune neuropsychiatric disorders (PANDAS) ( [Swedo et al., 1998](#)). Recent imaging investigations of PANDAS have reported enlarged basal ganglia volumes ( [Giedd et al., 1995, 2000](#)). Other investigators recently extended this finding and reported that chronic or recurrent GABHS infections were associated with increased basal ganglia volume in OCD and ADHD patients ( [Peterson et al., 2000c](#)). They also observed correlations between antibody titers and basal ganglia volumes. Moreover, at least some forms of immunotherapy (e.g., plasmapheresis) seem to attenuate GABHS-related OCD symptoms ( [Allen et al., 1995](#)), and normalization of basal ganglia volumes may accompany the improvement in OCD symptoms ( [Giedd et al., 1996a](#)). In at least one study, however, the associations between antibody titers and basal ganglia volumes were most prominent in children with ADHD ( [Peterson et al., 2000c](#)), suggesting that basal ganglia enlargement may not be specific to autoimmune subtypes of OCD and tic disorders.

Both increased and decreased basal ganglia volumes therefore have been associated with pediatric OCD and ADHD. This may suggest that nonspecific volumetric disturbances in the striatum may be more relevant for striatal function than any particular direction of change. It may also suggest the existence of distinct neurobiological subtypes of pediatric OCD and ADHD—namely, those that are and those that are not associated with recent GABHS infection.

## MOOD AND ANXIETY DISORDERS

Compared with other areas of pediatric brain imaging, relatively few neuroimaging studies have contributed to the neurobiological understanding of pediatric mood or anxiety disorders, with the exception of research on OCD. In fact, as of this writing, only four published studies and a handful of other unpublished reports have compared brain structure or function between healthy and ill children using modern neuroimaging methods. Two of these studies examined major depression, and one each examined bipolar disorder or generalized anxiety disorder.

The two studies of major depression used structural MRI and MRS to examine brain volumes and neurochemistry in depressed children ( [Steingard et al., 1996, 2000](#)). The volumetric study compared frontal lobe volumes between depressed and healthy subjects ( [Steingard et al., 1996](#)), based on extensive research implicating the frontal lobes in various forms of psychopathology, including major depression ( [Drevets, 2000](#)). This study found reduced frontal lobe volumes in pediatric depression. Of note, this initial study employed relatively older morphometric techniques, and recent studies of depression among adults suggest that structural abnormalities may vary as a function of family history or particular components of the frontal lobes. For example, familial forms of major depression show particularly consistent associations with abnormalities in ventral frontal regions, in the vicinity of the subgenual cingulate gyrus ( [Drevets, 2000](#)). In unpublished work among adolescent twins ( [Botteron, 1999](#)), an association seems to exist between major depression and subgenual cingulate abnormalities.

Consistent with this focus on frontal lobe pathology, the MRS study of depressed children compared choline-to-creatinine levels in the orbitofrontal cortex of depressed and healthy adolescents ( [Steingard et al., 2000](#)). Studies among adults have noted an association between brain choline levels and depression, although associations vary by brain region and disorder ( [Renshaw et al., 1997](#)). The childhood study found evidence of increased brain choline in depressed adolescents. In addition to these studies of unipolar depression, high rates of T<sub>2</sub>-hyperintensities on structural MRI have been observed in bipolar children, consistent with data in bipolar adults ( [Botteron et al., 1995](#)).

The only study of pediatric generalized anxiety disorder reported enlarged volumes of the right amygdala ( [De Bellis et al., 2000](#)). It is worth noting that enlarged amygdala volumes have been reported in studies of adults with bipolar disorder ( [Altshuler et al., 2000](#)) but not in studies of adults with anxiety disorders. Such structural findings have stimulated functional MRI studies of amygdala activity. A relationship between anxiety levels and the degree of amygdala activation has been noted in children during face viewing, a task that normally increases amygdala activity in both children and adults ( [Thomas et al., 2000](#)).

Two areas appear particularly promising for future study in children with mood or anxiety disorders. First, depressed or anxious adults seem to have both structural and functional abnormalities in limbic brain regions involved in memory and attention ( [Drevets, 2000](#)), consistent with neuropsychological studies of attention and memory in mood and anxiety disorders ( [McNally, 1996](#); [Mineka and Sutton, 1992](#); [Moog and Bradley, 1998](#)). Despite evidence from two studies relating mood or anxiety in children to mnemonic dysfunction ( [Moradi et al., 1999](#); [Pine et al., 1999](#)), evidence from other studies suggest that this association may only be found in subsets of depressed or anxious children ( [Frost et al., 1989](#); [McClure et al., 1997](#); [McGee et al., 1986](#)). This might include children with severe, persistent, or comorbid forms of depressive or anxiety disorders. Further neuroimaging studies of limbic structure and function in these children are needed. Second, abnormalities in lateralized brain functions using quantitative EEG and dichotic listening tests have been documented in adults with mood or anxiety disorders ( [Kayser et al., 2000](#)). Similar abnormalities have recently been reported in depressed or anxious children and adolescents (Kentgen et al., in press; [Pine et al., 2000](#)), suggesting that future neuroimaging studies should investigate lateralized neural systems in these disorders.

## POSTTRAUMATIC STRESS DISORDER

The majority of neuroimaging studies of posttraumatic stress disorder (PTSD) have been conducted in adults. Functional imaging studies using PET and SPECT have characterized changes in brain activity associated with exposure to pictures or scripts that recall past traumatic events. These studies suggest that PTSD is associated with changes in key cortical (e.g., prefrontal, cingulate, orbitofrontal) and subcortical (e.g., hippocampus, amygdala) structures known to be involved in the regulation of the stress response and control of memory functions ( [Newport and Nemeroff, 2000](#)). Specifically, when exposed to these trauma-related stimuli, adults with PTSD show increased regional cerebral blood flow (rCBF) in orbitofrontal cortex ( [Rauch and Shin, 1997](#); [Shin et al., 1999](#)), posterior cingulate cortex ( [Bremner et al., 1999a,b](#)), or the amygdala ( [Liberzon et al., 1999](#); [Rauch et al., 2000](#); [Shin et al., 1997](#)). Decreased rCBF is seen in the medial prefrontal cortex of subcallosal gyrus ( [Bremner et al., 1999a,b](#)), anterior cingulate ( [Bremner et al., 1999b](#); [Shin et al., 1999](#)), or hippocampus ( [Bremner et al., 1999a](#)). The relatively small number of subjects included in these studies has contributed to the variability in these findings. Nevertheless, when significant changes in rCBF have been reported in a given brain region, the direction of change has been consistent across studies, and each of the preceding changes in rCBF have been replicated at least once.

The most consistent finding across several structural MRI studies of adults with PTSD has been reduced hippocampal volume ( [Bremner et al., 1995, 1997](#); [Gurvits et al., 1996](#); [Stein et al., 1997](#)). These reduced volumes may represent local tissue atrophy, possibly a toxic consequence of stress-induced elevations in circulating glucocorticoids (e.g., cortisol) and excitatory neurotransmitters (glutamate). No consistent changes in amygdala volume have yet been documented in these structural imaging studies ( [Bremner et al., 1997](#); [Gurvits et al., 1996](#)), despite consistent findings of functional abnormalities in these regions in PTSD.

Only two neuroimaging studies have yet been reported in children and adolescents with PTSD. One was a pilot MRS study of 11 maltreated children with PTSD and 11 healthy, nontraumatized controls ( [De Bellis et al., 2000](#)). Concentrations of *N*-acetyl-aspartate (NAA) were measured in the anterior cingulate gyrus ( [De Bellis et al., 2000](#)), based on findings from adult studies of anterior cingulate dysfunction in PTSD ( [Bremner et al., 1999a](#); [Shin et al., 1999](#)). Reduced NAA concentrations were detected in the maltreated children, suggesting the presence of reduced integrity of anterior cingulate neurons in children with PTSD.

The other was a morphometric study that attempted to replicate the association between PTSD and reduced hippocampal volumes previously reported in adults ( [Bremner et al., 1997](#); [Gurvits et al., 1996](#); [Stein et al., 1997](#)) and in preclinical studies of early stress effects on hippocampal structure ( [Sapolsky, 2000](#)). Regional brain volumes were examined in 44 maltreated children with PTSD and 61 matched controls ( [De Bellis et al., 1999](#)). Unlike the findings from adult studies, hippocampal volumes were normal in children with PTSD. This study did report, however, an overall reduction in brain volume in pediatric PTSD that correlated with various indices of environmental stress ( [De Bellis et al., 1999](#)). Children with PTSD also had smaller medial and posterior portions of the corpus callosum, and volumes of the right and left lateral ventricles were significantly increased over control values.

Relevant to these findings in children with PTSD is an MRI study that examined the effects of early stress on brain structure in prepubescent nonhuman primates ( [Sanchez et al., 1998](#)). Most preclinical studies of early stress have examined the long-term impact of these experiences on brain development in adult animals. In contrast to findings in adult animals, this study of young primates failed to detect hippocampal atrophy. Instead, reduced size of the medial and caudal portions of the midbody of the corpus callosum was detected, consistent with the findings from structural imaging studies of maltreated children ( [De Bellis et al., 1999](#); [Teicher et al., 2000](#)). A recent abstract has reported that psychiatric inpatients with a history of maltreatment had a similarly reduced size of the medial and caudal portions of the corpus callosum compared with controls without a history of early childhood trauma ( [Teicher et al., 2000](#)). The significance of these corpus callosum findings, if they



are replicated, is not clear. Several of the regions that are interconnected through these portions of the corpus callosum are also connected with prefrontal cortical areas that mediate the processing of emotional stimuli and various memory functions that comprise the core disturbances in PTSD ( [Pandya and Seltzer, 1986](#)). If corpus callosum abnormalities contribute to the pathophysiology of PTSD, they may do so via downstream effects on these prefrontal regions. Clearly, more research is needed to understand the neural underpinnings of PTSD in children, especially because the neuroanatomic and functional abnormalities associated with PTSD may differ between children and adults.

## DYSLEXIA

Reading disability is characterized by the failure to develop age-appropriate reading skills despite normal intelligence and adequate reading instruction. Behaviorally, deficits are most evident at the level of single word and pseudoword reading, in which reading disabled (RD) performance is both slow and inaccurate relative to nonimpaired (NI) readers. Many lines of evidence converge on the conclusion that the difficulties with word and pseudoword reading in developmental dyslexia, to a large extent, are manifestations of more basic deficits at the level of rapidly assembling the phonologic code represented by a token letter string ( [Bradley and Bryant, 1983](#); [Lieberman et al., 1989](#)). Given this behavioral literature, the studies selected for discussion here will involve a comparison of RD and NI reading groups on word and pseudoword reading tasks that stress phonologic processing. For a discussion of functional neuroimaging studies that have examined sensory-level processing deficits in developmental dyslexia ( [Demb et al., 1998](#); [Eden et al., 1996](#)) the reader is referred to [Eden and Zeffiro \(1998\)](#). For reviews of research examining differences in brain structure between RD and NI groups, please see [Filipek \(1995\)](#) and Galaburda (1992).

When contrasting the functional organization of the brain in nonimpaired and reading disabled populations, we must turn first to the literature on the neurobiology of skilled reading. Converging findings from a number of laboratories using several imaging technologies fMRI, PET, and magnetoencephalography (MEG) indicates that printed word and pseudoword reading is based in a left hemispheric, posterior reading system consisting of two components, a ventral and dorsal component. The *ventral circuit* includes lateral extrastriate areas and a left inferior occipito-temporal area, where functional imaging studies show robust and early (i.e., 150 to 180 msec) activity as subjects perform word and pseudoword reading tasks ( [Fiez and Peterson, 1998](#); [Frackowiak et al., 1997](#); [Henderson, 1986](#); [Nobre et al., 1994](#); [Puce et al., 1996](#); [Salmelin et al., 1996](#)). The more *dorsal circuit* includes the angular gyrus and supramarginal gyrus in the inferior parietal lobule, as well as the posterior aspect of the superior temporal gyrus (Wernicke's area). This latter temporo-parietal circuit has long been implicated in reading. A large literature associates the acquired inability to read (alexia), for example, with neuroanatomic lesions that are centered most prominently about the angular gyrus ( [Damasio and Damasio, 1983](#); [Dejerine, 1891](#); [Henderson, 1986](#)), a region considered pivotal in carrying out the cross-modal integration of sensory stimuli that is necessary for reading (i.e., the mapping of the visual percept of printed letters onto the phonologic structure of language) ( [Benson, 1994](#); [Black and Behrmann, 1994](#); [Geschwind, 1979](#)). An *anterior circuit* centered in and around Broca's area in the inferior frontal gyrus appears to be associated with, among other things, the sequencing and control of speech-gestural articulatory recoding. This circuit is also involved in silent reading and naming ( [Fiez and Petersen, 1998](#); [Frackowiak et al., 1997](#); [Pugh et al., 1996, 1997](#); [Shaywitz et al., 1998](#)).

Neuroimaging studies of skilled readers reveal task-dependent differences in activity of the dorsal and ventral regions in the posterior left hemisphere reading system ( [Frackowiak et al., 1997](#); [Pugh et al., 2000](#)). These differences prompt speculation about the distinctive roles of these circuits in word and pseudoword reading. The dorsal circuit may play a critical role in extracting the predictable mappings among the orthographic, phonologic, and morphologic forms of words with the lexical-semantic properties of words, binding these properties into integrated mental representations. This circuit may be necessary for rule-based, algorithmic, learning. By contrast, the ventral occipito-temporal area, the point of contact between the ventral visual stream and the middle to inferior temporal lobe, may constitute a linguistically structured, memory-based, fast word identification system (i.e., a template-like device for recognizing word-forms). Research on a large sample of children at different ages suggests that the ventral word form area likely plays an increasingly important role in word identification in NI readers as they develop skill ( [Shaywitz et al., 2000](#)). That work also suggests that the development of this ventral circuit with increasing age and skill depends on the integrity of the dorsal circuit's analytic processing.

### Posterior and Anterior Circuits in Reading Disability

NI and RD readers clearly differ with regard to task-related activity in these dorsal, ventral, and anterior portions of reading circuitry. Functional imaging studies of phonologic processing in RD readers, for example, have repeatedly shown dysfunction of the left posterior hemisphere system in both the dorsal and ventral sites ( [Brunswick et al., 1999](#); [Helenius et al., 1999](#); [Horwitz et al., 1998](#); [Pugh et al., 2000](#); [Rumsey et al., 1997](#); [Salmelin et al., 1996](#); [Shaywitz et al., 1998, 2000](#); [Simos et al., 2000](#)). This disruption is reflected both by a relative under-engagement of these circuits, as well as a reduced functional connectivity between them, specifically when the phonologic decoding of words and pseudowords is required. Dysfunction in the activity of these regions and the associations among them is consistent with a recent anatomic study using diffusion weighted imaging that reported the presence of structural anomalies in white matter tracts within the temporo-parietal region of the left hemisphere (Klingsberg et al., 2000). In a recent examination of children ( [Shaywitz et al., 2000](#)), abnormal activation was observed in RD readers at both dorsal and ventral left hemisphere sites during word and pseudoword reading tasks, confirming that similar abnormalities observed previously in adults are also present in children ( [Rumsey et al., 1997](#); [Shaywitz et al., 1998](#)). This posterior anomaly was confined to tasks of word and pseudoword reading (demonstrating the linguistic specificity of the finding), and it was seen in both adults and children.

In several studies, some of the differences between RD and NI groups in brain activity appears to be compensatory for deficits elsewhere ( [Shaywitz et al., 1998, 2000](#)). On tasks that make explicit demands on phonologic processing (pseudoword and word tasks), for example, RD readers showed a disproportionately greater engagement of inferior frontal gyrus and prefrontal dorsolateral sites than did NI readers ( [Brunswick et al., 1999](#); [Richards et al., 1999](#); [Rumsey et al., 1997](#); [Salmelin et al., 1996](#)).

Evidence of a second apparent compensatory shift (in this case, to the posterior right hemisphere), comes from several findings. In one study ( [Shaywitz et al., 1998](#)), greater right than left hemisphere activation was observed in RD readers, whereas greater left than right hemisphere activation was detected in NI readers ( [Barnea et al., 1994](#)). Additionally, in these same reading tasks, the major posterior circuits in the left hemisphere of RD readers did not correlate with one another as they did in NI readers, suggesting a problem with the connectivity of these circuits. In contrast, RD readers displayed strong correlations at homologs of these sites in the right hemisphere that were more robust than the correlations among these regions in NI readers ( [Pugh et al., 2000](#)). Right hemisphere activation seems to correlate with standard measures of reading performance only in children and adult RD readers ( [Rumsey et al., 1999](#); [Shaywitz et al., 2000](#)), suggesting a compensatory function for the shift to the right hemisphere.

### Summary

Posterior reading circuits including both dorsal (temporo-parietal) and ventral (occipito-temporal) components are disrupted in people who are reading disabled, as indicated by reduced activation as well as disrupted functional connectivity among these areas. Additionally, there appear to be two characteristic compensatory patterns in response to this posterior left hemisphere anomaly: (a) increased reliance on inferior frontal gyrus during reading and (b) an increased tendency to rely on the RH homologs of the dysfunctional left hemisphere posterior circuits.

The studies of children suggest the following model. In the normally developing NI reader, the development of the posterior left hemisphere reading circuits, particularly the ventral occipito-temporal area, is dependent on an organized integration of phonologic, morphologic, and lexical-semantic processing of words within highly overlapping neural circuits. This integration presumably relies initially on the intactness of processing in the temporo-parietal circuit. Deficient dorsal function will fail to support appropriate ventral development. Thus, in the RD reader, temporo-parietal difficulties disrupt the normal developmental trajectory of the occipito-temporal circuits. The shift to inferior frontal sites in the RD child reflects a compensatory reliance on these circuits to support articulatory recoding (covert pronunciation) in an attempt to cope with a problematic phonologic analysis of printed words. A second compensatory shift, from posterior left hemisphere to posterior RH, likely reflects the development of an additional word recognition process that is essentially visual-perceptual—graphemic patterns in the printed word are associated directly with entries in the RD reader's mental lexicon. Thus, these visual patterns do not code the phonologic or morphologic information that the NI reader perceives within the printed word but instead represent the printed word as a nonlinguistic visuo-semantic icon.

## FUTURE DIRECTIONS OF NEUROIMAGING IN DEVELOPMENTAL NEUROPSYCHIATRIC DISORDERS

### Imaging Technologies

The immediate and intermediate future of brain imaging in childhood disorders will be dominated by investigations that employ MRI because of its safety in cross-sectional and longitudinal studies. The use of PET and SPECT will be limited because of their dependence on radioactive ligands and tracers that are



problematic in children, especially normal control children.

Technological advances have intensively driven brain imaging, like most of science. These advances dictate the kinds of scientific questions we are able to ask in our studies. Technological innovations that will affect MRI studies include the application of scanners with higher magnetic field strength, which provide greatly improved resolution and signal-to-noise in the images. MRS and DTI will be applied increasingly to the study of chemical compounds and fiber tract tracing studies, particularly as magnetic field strengths increase to improve the information available from these imaging modalities.

### **Broadening the Scope of the Disorders Studied**

Despite the high prevalence and morbidity of many childhood developmental neuropsychiatric disorders, such as depression, conduct disorder, anxiety disorders, and pervasive developmental disorders, and despite the known importance of biological, environmental, and developmental determinants in each, remarkably few imaging studies of these disorders in children have been reported. These are currently areas of increasingly intense focus, however, and we will soon learn much about the pathophysiology of these childhood conditions.

### **Studying Pathophysiologic Determinants**

Aside from technical advances, new directions in the imaging of childhood disorders will involve the novel application of more standard clinical investigative tools to help delineate through imaging studies the genetic and nongenetic determinants of the neurobiological substrate of childhood disorders. Stratifying subjects with respect to the degree of genetic risk (using family-genetic data or genetic markers) and degree of exposure to known nongenetic risks (such as prenatal and obstetric complications) will permit the ascertainment of the effects of genetic and nongenetic factors on the neurobiological substrate of these disorders.

The effects of these factors will likely be most evident in their interactions for the vast majority of complex neuropsychiatric disorders. A given nongenetic risk factor may exert its effect on neurobiologic substrate, for instance, only in the context of some threshold of genetic risk or vulnerability to developing the disorder. Conversely, nongenetic determinants appear to have varying degrees of importance in the pathophysiology of childhood neuropsychiatric disorders. Exposure to a nongenetic risk factor during a period of developmental vulnerability alone may be sufficient to produce a specific disorder and its corresponding substrate; alternatively, an environmental exposure may predispose to any number of disorders, in which case the child's specific genetic and other constitutional endowments will determine which particular disorder is manifested. Only careful clinical and biological characterization will permit the kind of dissection of pathophysiology that will discern the effects of these numerous determinants on neurobiological substrate and disease phenotype. These studies necessarily also will involve a large number of subjects to permit these associations and interactions to be detected reliably.

### **Longitudinal Studies**

Finally, perhaps the most important scientific information to be obtained in imaging childhood neuropsychiatric disorders will derive increasingly from longitudinal studies of brain structure and function in normal and pathologic development. The importance of studying developmental changes in brain structure and function in normal children for the improved understanding of pathologic brain development cannot be overemphasized. Longitudinal imaging studies are understandably less feasible than simple cross-sectional studies, given the potential difficulties inherent in longitudinal research with patient compliance and current concerns about radiation exposure with PET and SPECT that limit the possibilities of repeated examinations within subjects. Nonetheless, longitudinal imaging studies are necessary to determine whether individual and group differences seen in disease states are a cause or consequence of the disease. Longitudinal studies also are necessary to begin discerning the effects that normal CNS maturation has on the unfolding of the clinical expression of a disease at characteristic times and in characteristic manners at varying stages of development. Sizable longitudinal studies, combined with careful clinical and biological characterization of subjects, thus will permit the beginning elucidation of the mechanisms whereby genetic and environmental risk, in the context of normal and pathologic CNS maturation, interacts to produce a particular neurobiological substrate and subsequently unique clinical phenotype.

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# 10 COGNITIVE-BEHAVIORAL APPROACHES

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A useful theory of child and adolescent development should provide a guiding framework for organizing and conceptualizing information about thoughts, feelings, and behavior. Recently, models that have integrated concepts from different theories offer promising backdrops for understanding the course of human development (Kendall, 2000b). One of these integrative approaches, the cognitive-behavioral model, combines features of learning theory with aspects of human information processing to explain both adaptive and maladaptive behavior during childhood and adolescence. Instead of considering it an exotic, new theoretical model, the cognitive-behavioral approach can be viewed as an evolving theoretical orientation—one that attempts to preserve concepts of behavior therapy (within a less doctrinaire context), while incorporating the cognitive activities of the individual (Kendall and Hollon, 1979). In this chapter, we briefly review the evolution of a cognitive-behavioral approach by: (a) presenting the general principles of the behavioral and cognitive models, (b) discussing how the combination of these concepts contributes to the development of cognitive-behavioral interventions, and (c) considering how the integrative techniques are best applied to the treatment of psychopathology during childhood and adolescence.

Our first section begins with a discussion of the behavioral approach, a framework that conceptualizes human actions in terms of overt behavior, whenever possible. The next section presents the principles of the cognitive model, a perspective that emphasizes the contribution of people's beliefs, expectancies, and other cognitive activities for producing and understanding thoughts, feelings, and behavior. The ideas of the behavioral and cognitive models are combined into an integrative theoretical perspective with the introduction of the cognitive-behavioral model in the third section. To discuss the theoretical orientation of the cognitive-behavioral approach in greater detail, the remainder of the chapter considers the application of cognitive-behavioral therapy (CBT) to human problems. This discussion considers the key elements of CBT including treatment goals and strategies, the role of the therapist, and the conceptualization of therapeutic change.

## PRINCIPLES OF THE BEHAVIORAL MODEL

The behavioral model focuses on the manner in which human behavior is learned or acquired, and encompasses principles, assumptions, and techniques rooted in learning theory. Consistent with this model, behavior therapy, or behavior modification as it is often called, deals with the assessment, evaluation, and alteration of human behavior. It underscores the observable behavior of the individual and the environmental factors that maintain a particular action. Because behavior is thought to occur as a consequence of its having been learned in the past, behaviorally oriented psychologists stress the role of a person's learning history and reinforcement (pleasant events), as well as the role of the current interpersonal and social environment, as factors that contribute to both adaptive and maladaptive behavior.

A behavioral model rests on the primary assumption that behavior develops and is maintained according to the principles of learning (Herbert, 1988; Johnson et al., 1986). As such, the model argues that when other influencing factors are constant (e.g., biology), the differences that exist among people's behavior are the result of learning. Within this perspective, changes in the environmental influences are thought to have a profound effect on the alteration of both normal and abnormal behavior patterns (O'Leary and Wilson, 1987). Interestingly, although researchers during the first half of the 20th century (Pavlov, 1927; Thorndike, 1911; Watson, 1913) pointed to the importance of behavioral principles in shaping human behavior, it was not until the 1950s that mental health professionals began to apply behavioral techniques on a wider scale to the treatment of mental disorders. To date, the techniques embraced by behavior modification have been used for different populations showing a range of maladaptive behaviors and clinical disorders (e.g., affective, developmental, anxiety, conduct, or oppositional-defiant). In addition, the techniques have been used to alter maladaptive behavior or improve the quality of life of individuals outside of the clinical setting; these efforts have included increasing prosocial interactions among children and their peers, helping individuals adhere to medical regimens, and promoting healthy lifestyles involving exercise and nutritious diets (Bellack et al., 1990; Hersen et al., 1994).

Although applicable to a variety of concerns, behavior therapy also has been useful for different types of individuals. These populations include children, adolescents, and adults in everyday surroundings (e.g., school, work, and home), and also specific groups like individuals in psychiatric settings. Given that behavior modification can be applied to a wide range of problems and populations, it is important to note that this approach does not refer to one specific form of treatment. Instead, behavioral techniques have come to embrace several theories about the development and maintenance of maladaptive behavior. As a result, proponents of behavioral techniques do not all offer the same explanation of behavior. With respect to describing behavior, some behaviorists, for example, stress the importance of processes that result from events in the environment (e.g., consequences of behavior, cues, and feedback). In contrast, other behaviorists emphasize the role of processes within the individual (e.g., perceptions, thoughts).

Although behavior modification encompasses a variety of populations, treatment techniques, clinical concerns, and theoretical perspectives, the approach consistently focuses on the development of adaptive functioning in life. Three forms of learning—the roots of behavior modification—have made significant contributions to our current understanding of how behavior patterns develop: classical conditioning, operant conditioning, and observational learning. Given that each form of learning plays a role in the development of later cognitive-behavioral interventions, we now turn our attention to presenting each form in greater detail.

### Classical Conditioning

Among the many influences on behavior modification is the significant work of Russian physiologist Ivan Pavlov (1849 to 1936). Pavlov's seminal research involved the study of reflex responses with animals, specifically dogs. After using food to stimulate different parts of an animal's digestive system, Pavlov found that animals would begin to secrete gastric juices when they merely saw food or heard food being prepared. This finding was significant because it demonstrated that digestive processes could be elicited in the absence of any physical stimulation. Pavlov continued his work using other environmental stimuli, such as sounds and sights, showing that various environmental incentives could elicit reflex reactions such as salivation (Pavlov, 1906, 1927). He argued that the connections between the environmental stimuli and the animals' reactions were the result of learning that took place in the laboratory. This type of learning is now referred to as classical or respondent conditioning.

In general, classical conditioning is interested in stimuli that evoke responses. The process involves a once, neutral stimulus (e.g., light, noise) that comes to elicit involuntary unconditioned responses or reflex reactions (referred to as respondents). The respondents are often considered automatic responses because they are not under the individual's control. In this way, the association between the neutral, unconditioned stimulus and the response is one that is *not* learned. It is possible, however, for reflex behavior to occur in response to a stimulus that would not automatically evoke the response; in this situation, the behavior is considered one that is

learned. To illustrate the difference between an automatic and learned response, suppose that you find yourself stuck in a dangerous location, such as on the center lines dividing traffic on a major highway. In this situation, your reflex reaction to feel frightened would be perfectly normal and adaptive. When, however, people learn to be afraid of certain things and situations when it may not be adaptive or normal to feel frightened, classical conditioning can help explain the development of these types of fears.

To begin, a realistically dangerous situation produces fear without prior learning or conditioning. Because this fear occurs without learning or conditioning, the dangerous situation is called an *unconditioned stimulus* (UCS), and the reaction, fear, is the *unconditioned response* (UCR). They occur naturally. A neutral stimulus does not itself produce a response; however, a neutral stimulus that is repeatedly paired with a dangerous situation becomes a conditioned stimulus (CS) and can itself lead to the UCR, fear. When the neutral situation itself becomes capable of producing fear, the fear it produces is called a conditioned response (CR). It is the conditioned (learned) responses that are of particular interest in psychopathology. For example, a specific piece of music paired repeatedly in a dangerous situation becomes itself capable of producing fear. Consider the two-note theme from the movie *Jaws*. The music becomes a CS because it has acquired the fear-producing properties via conditioning, and the fear it produces is a CR. Thus, through classical conditioning, a stimulus that was once neutral can gain the power to elicit a reflex response or respondent behavior.

Pavlov's work was significant because his findings offered one explanation for how behaviors can be learned. His specific findings, as well as his tightly controlled methods of laboratory investigation, made important contributions to the study of behavior and launched further examinations of learning processes. In one of the seminal investigations of learned behavior, [Watson and Rayner \(1920\)](#) studied an 11-month-old boy named Albert. In their work, Watson and Rayner provided a significant example of how a fear reaction can be elicited via classical conditioning. The investigators paired an originally neutral stimulus (white animal) with a loud noise (unconditioned stimulus) that produced a startle or fear response (unconditioned response). After pairing the white animal with the noise only seven times during the period of 1 week, the investigators could eventually make Albert respond simply by showing him the white animal. Thus, after 1 week, the conditioned stimulus (animal) was able to elicit the conditioned response (fear). In a related line of research, one of Watson's students, Jones (1924a), reported the case of a 34-month-old boy named Peter. As with Albert, a neutral stimulus was repeatedly paired with an unconditioned stimulus, so that that the neutral stimulus—in this case, a rabbit—would elicit a fear response. After the link between the conditioned stimulus and response was successfully established, the association was systematically deconditioned via gradual pairings of the feared object with positive reactions. Through the process of deconditioning, Peter was able to overcome his fear of rabbits. Collectively, the work of Watson, Rayner, and Jones demonstrated that fears could be both conditioned and deconditioned via the principles of classical conditioning, two findings that exerted a significant impact on the study of behavior.

Another contemporary extension of classical conditioning that is of historical significance is the work of Joseph Wolpe. [Wolpe \(1958\)](#) examined an experimentally induced state, experimental neurosis, in which cats showed disruption of behavior, irritation, agitation, and other symptoms akin to anxiety in humans. In an attempt to decrease the anxiety experienced by the cats, Wolpe gradually exposed the animals to situations and cues that elicited decreased amounts of anxiety. He also encouraged the animals to engage in other activities (e.g., playing or eating) while experiencing states of decreased arousal. Based on his observations, he reasoned that anxiety could gradually be overcome if an animal engaged in competing responses while exposed to anxiety-provoking situations. Eventually, Wolpe applied his research findings to human participants and developed a treatment referred to as systematic desensitization for human anxiety. In this procedure, individuals were exposed to a series of situations, either imaginary or real-life, that provoked increasing levels of anxiety. Wolpe trained his clients to engage in deep relaxation so that the representation of situations would only produce mild forms of anxiety. Relaxation would be paired with the anxiety-provoking situations, and over time, the individual would be able to use relaxation to overcome situations with increasing levels of anxiety. Eventually, the client would not be anxious in response to the stimulus that originally produced anxiety because the conditioned relaxation response was incompatible with the fear.

## Operant Conditioning

An alternate form of learning that was under investigation was concerned, not with examining reflex responses, but with evaluating the impact of different consequences on developing new behavior ([Kazdin, 1978](#)). This separate line of inquiry led to what is now known as operant conditioning—the probability that a response will be increased or decreased when followed by reinforcement or punishment ([Thorndike, 1911](#)). According to the principles of operant conditioning, responses are strengthened and more likely to occur in the future when they lead to satisfying consequences. In contrast, responses are not strengthened and are less likely to occur again in the future if they lead to unsatisfying consequences. Edward Thorndike (1874 to 1949), who was interested specifically in how new behaviors were learned, was most responsible for developing the behavioral principles of operant conditioning. In his well-known puzzle-box experiments with cats, he discovered that rewards (e.g., food for the cat after escape) provided consequences that increased learning of the behavior (e.g., successful exit from the puzzle-box). From his careful experimentation and observations, he formulated principles or laws of behavior, the most significant being the Law of Effect (consequences that follow behavior help learning) ([Thorndike, 1932](#)).

BF Skinner, who was influenced by the work of Pavlov and Thorndike, further developed the theory of behavioral consequences. He was interested in the impact of consequences on behavior and used the principle of reinforcement to describe how behavioral consequences can strengthen the probability of a behavior recurring. He pointed out that many behaviors are performed spontaneously (not as reflex responses) and are controlled primarily by their consequences (not by eliciting stimuli). Skinner referred to these behaviors as operants because the responses operated or demonstrated some type of influence on the environment. Examples of everyday operant behaviors include smiling, laughing, talking, walking, or any other response that is influenced or controlled by its consequences (Skinner, 1953). Operant conditioning, then, is the process of learning operant behavior, and operant behaviors increase or decrease as a function of the events that follow them.

[Skinner \(1938\)](#) outlined four major principles to demonstrate how operant behavior can develop and undergo change. The principles describe the relations between overt behavior and environmental consequences and include: (a) reinforcement, (b) punishment, (c) extinction, and (d) stimulus control. The first principle, reinforcement, refers to an increase in the frequency of a response owing to the presentation or removal of an event after the response. For example, when positive consequences follow a behavior, the behavior is positively reinforced or rewarded, and the behavior is more likely to occur again in the future. Indeed, maladaptive behavior, as well as desired patterns of behavior, can be acquired through their consequences. For example, when a child is polite in the presence of houseguests, she may be rewarded with a snack for her good behavior; however, a child who screams or throws a temper tantrum may also get a snack (reward) simply because the parent wants to calm the child down. Because both types of behavior are rewarded, they are both more likely to occur again.

Given that human existence does not proceed from a sequence of one simple behavior after another, it must be the case that not all behavior has to be directly reinforced to be learned. For example, in the case of complex behavior, the process of rewarding successive approximations (i.e., shaping) does not require the learner to produce an entire new response pattern to receive reinforcement. The shaping process is gradual and the reinforcement is provided for several intermediate steps. As the learner masters each successive step, reinforcement shifts so that it is provided only after the new last step in the sequence is established. Thus, a child's disruptive behavior in the classroom may be the result of a shaping process that has taken years to complete, rather than a behavior that was learned all at once. The fact that maladaptive patterns of behavior can be shaped over time has significant implications for child development ([Gewirtz and Pelaez-Nogueras, 1992](#)) and for understanding and treating ([Mash, 1998](#)) disorders.

Another type of reinforcement is negative reinforcement. With negative reinforcement (which is not the same as punishment), the likelihood of a response increases by the removal of a negative or unpleasant stimulus or situation. For example, if you politely ask a roommate to turn off the television so you can study, and your roommate does so immediately, the likelihood of your making a similar polite request in the future is increased. The polite request was reinforced by the removal of the unwanted (aversive) television distraction. A different but related situation characterized by negative reinforcement involves avoidance and escape responses. For example, if a child is continuously teased by her peers, she may try to avoid interacting with her classmates or escape from situations that may lead to uncomfortable scenarios. Eventually, staying away from peer interactions can become associated with anxiety reduction—an avoidance response that prevents the unwanted or aversive emotions, but also has unwanted effects in terms of reduced socialization.

Skinner's other principles of behavior also describe the relations between overt behaviors and environmental consequences. Punishment, for example, refers to a decrease in the frequency of a response owing to the presentation or removal of an event after the response. Thus, if a child is scolded for misbehaving in front of guests (i.e., a negative outcome), the likelihood that the unwanted behavior (i.e., the misbehavior) will occur in the future is reduced. Extinction, the third principle of behavior, refers to a decrease in the frequency of a previously reinforced response owing to the removal of the reinforcing event after the response. For example, a parent may try to extinguish the occurrence of temper tantrums by removing what was once used as a reinforcer (e.g., snack). The act of removing the reinforcing event may work to decrease the unwanted behavior. Skinner's fourth and final principle, stimulus control, refers to a procedure in which a response is reinforced in the presence of one stimulus but not another. This process works to increase the frequency of the response in the presence of the former stimulus, while decreasing the frequency of the response in the presence of the latter stimulus.

As with classical conditioning, the principles of operant conditioning have extended beyond the experimental laboratories from which the laws of behavior originated. Specifically, the application of operant conditioning principles have benefited children and adolescents who demonstrate speech or language delays ([Drash and](#)



[Tudor, 1990](#); [Lovaas, 1977](#)), exhibit social skills deficits ([Elliott and Gresham, 1993](#)), are about to undergo medical procedures ([Horne et al., 1994](#)), and who have been diagnosed with clinical disorders such as autism ([Harris and Weiss, 1998](#)), attention deficit hyperactivity disorder ([Pelham et al., 1993](#)), and obesity ([Williams et al., 1993](#)). Although this portion of the chapter could provide only a sketch of certain learning principles and how they can be applied to human problems, we later address the importance of behavioral principles with respect to assessing, understanding, and treating psychological concerns. For now, it is important to note that the early investigations on conditioning principles sparked researchers to apply principles of learning to a variety of concerns within child clinical populations ([Ullman and Krasner, 1965](#)).

## Observational Learning

Observational learning or modeling is a type of learning in which behaviors are learned by observing others ([Bandura, 1969](#)). Modeling is a primary factor in social learning theory (also known as social cognitive theory) ([Bandura, 1977, 1986; Bandura and Wilson, 1963](#)), a theory that proposes that behavior is the product of both external stimulus events and internal cognitive processes. Social learning theory tries to expand on the cause–effect type of relations described by the aforementioned conditioning principles; it does this by combining the two conditioning perspectives with an understanding of cognitive processes. According to [Bandura \(1977\)](#), three types of regulatory processes govern learned behavior: (a) paired stimulus–response events (as in classical conditioning), (b) environmental consequences (as in operant conditioning), and (c) symbolic cognitive processes. The most significant of the three components are cognitive processes, and specifically those that involve observational learning—the hallmark of social learning theory. For example, a child who views another child being rewarded for talking politely in class will be more likely to engage in similar behavior. In this way, an individual's social context is important because it provides several opportunities for behavior to be observed and eventually imitated.

Two additional features of social learning theory underscore the theory's departure from the more deterministic, cause–effect view of human nature as described by the classical and operant conditioning theories. One concept is the notion that behavior is determined by the interaction of three factors: cognitive, behavioral, and other environmental factors ([Bandura, 1986](#)). This interplay, referred to as “triadic reciprocity,” stresses the importance of all three components for determining behavior. Another distinguishing feature of social learning theory is its emphasis on the capacity for self-directed change (i.e., self-efficacy) ([Bandura, 1977](#)). This notion, which reaffirms the concept of “triadic reciprocity,” carries significant import with respect to understanding the development of behavior patterns and the treatment of maladaptive functioning. Both the ideas of self-directed change and “triadic reciprocity” reappear later in this chapter as important components of cognitive-behavioral theories and interventions. We now turn our attention to presenting the next step in the evolution of the cognitive-behavioral theoretical orientation—a description of the cognitive model.

## PRINCIPLES OF THE COGNITIVE MODEL

From a behavioral perspective, observable phenomena play an essential role in how human dysfunction is assessed and understood. Although individuals' thoughts and feelings may not be completely ignored with respect to clinical problems, emphasis is placed on what a person does, or how a person behaves. Thus, human problems are conceptualized in terms of overt behavior whenever possible. In contrast, cognitive perspectives ([Beck, 1964; Ellis, 1962; Meichenbaum, 1977](#)) assert that people's beliefs, attitudes, expectancies, attributions, and other cognitive activities are central to producing, understanding, and predicting behavior, especially psychopathologic behavior. [Ellis' \(1962\)](#) rational-emotive theory, [Beck's \(1963, 1976\)](#) cognitive theory, and [Meichenbaum's \(1985\)](#) cognitive analysis have played significant roles in generating acceptance of such cognitive phenomena. Theoretical advances of learning theorists, such as [Kanfer's \(1970\)](#) notion of self-regulation and [Bandura's \(1977\)](#) theory of self-efficacy, have also helped legitimize the importance of covert cognitive processes. Cognitive theorists, then, assert that individuals are active participants in their environments, judging and evaluating stimuli, interpreting events and sensations, and appraising their own responses ([Beck and Weishaar, 1995](#)).

Cognitive models argue that cognitive functioning contributes to emotional and behavioral distress. How human beings recognize, perceive, reason, and judge information has come to play an important role in attempts to understand the development of different disorders. According to [Beck \(1967\)](#), the individual develops a schema, or frame of reference, from which each individual uniquely views himself or herself, others, and the environment. This schema, then, affects what an individual perceives, recalls, and prioritizes as important. Beck argues that it is through schemas that an individual interprets the world and derives meaning accordingly. When emotional psychopathology is present, it is hypothesized that the activated schemas are maladaptive in nature. As a result, a person may show systematic biases in information processing, such that he may overgeneralize, selectively attend to, magnify or minimize, personalize, or make arbitrary references about information ([Beck, 1967, 1976](#)). For example, a tendency to blame oneself for mistakes, misperceive interpersonal situations, or think negatively about circumstances without sufficient data, are all examples of dysfunctional cognitive processing often limited to depression.

Cognitive theorists suggest that clinical disorders have specific cognitive profiles as evident in dysfunctional schemas, automatic thoughts, and biased interpretations. For a cognitive therapist, then, psychological interventions focus on the cognitive schemas thought to contribute to the development and maintenance of a particular disorder. In contrast, a behavioral approach underscores observable behavior and environmental factors. It is the combination of the two approaches that provides the foundation for a cognitive-behavioral orientation. This perspective stresses the importance of overt behavior and covert cognition, along with the interaction between the two, for explaining the development and maintenance of emotional and behavioral distress.

## THE COGNITIVE-BEHAVIORAL APPROACH

The cognitive-behavioral approach integrates the two more traditional approaches—the behavioral and the cognitive. It is an integration of the methodologically rigorous and performance-oriented behavioral techniques with the evaluation and treatment of cognitive-mediational phenomena ([Craighead et al., 1994; Kendall and Hollon, 1979](#)). In this way, the cognitive-behavioral model is essentially a two-process approach combining behavioral models with a cognitively mediated model. The model places greatest emphasis on the learning process and the influence of contingencies and models in the environment, while stressing the centrality of the individual's mediating/information-processing style in the development and remediation of psychological distress ([Kendall, 2000a](#)). Thus, the cognitive-behavioral model represents an integrationist perspective ([Meichenbaum, 1977](#)) and considers both children's internal and external environments. The hyphenated “cognitive-behavioral” term represents an integration of cognitive, behavioral, emotion-focused, and social aspects of change. Without a singular allegiance to a behavioral or cognitive model, the cognitive-behavioral perspective includes the associations of behavior and cognition to the emotional state and functioning of the organism in the larger social context.

When considering a cognitive-behavioral approach, it is important to recognize how the term cognition is used and understood within this particular perspective. Although some mental health professionals have viewed cognition as inaccessible, cognitive-behavioral theorists have promoted the notion that cognition can be subdivided for access and understanding. For instance, it has been suggested ([Ingram and Kendall, 1986; Kendall and Ingram, 1989](#)) that cognition is a complex system that includes cognitive content (events), processes, products, and structures that can be distinguished.

### Considering Cognition

Cognitive structures, which can also be referred to as cognitive schemata ([Beck, 1976](#)), refer to memory, specifically information that is internally represented in memory. The notion of a cognitive structure can be viewed as a template that filters certain cognitive processes. With more rigid schemas, children's perceptions become more filtered and potentially more dysfunctional ([Lochman and Dodge, 1998](#)).

Cognitive content refers to the information that is actually represented: the contents of the cognitive structures. Cognitive content may be referred to as one's self-talk or internal speech ([Kendall and Hollon, 1981; Meichenbaum, 1977](#)): comments in which the audience is primarily the person himself or herself, and not just those comments in which the individual is the object of the statement.

Cognitive processes are the procedures by which the cognitive system operates: how we go about perceiving and interpreting experiences. Cognitive products (attributions) are the resulting cognition that emerges from the interaction of information, cognitive structures, content, and processes. After several real or imagined experiences, a person can come to have a characteristic way of making sense of events. Attributions about the event and its outcomes reflect the influence of the preexisting structure. In this model, psychopathology can be related to any problem in each or all of these areas, and effective therapy includes consideration of these factors for each individual client.

Children and adolescents are in the process of developing ways to view their world. Cognitive-behavioral treatments seek to provide educational experiences that attend to cognitive content, process, and product (paying attention to the child's self-talk, processing style, and attributional preferences) to help the child/adolescent build a cognitive structure that will have a positive influence on future experiences. For example, the cognitive schema for the anxious child is threat—threat of loss,

criticism, or harm. The child brings this anxiety-prone structure to an experience and processes the experience accordingly. Cognitive-behavioral interventions provide the arena to challenge the youth's preexisting anxious structure (Kendall et al., 2000d). In-session role-playing experiences and exposure experiences are valuable opportunities for learning in such cases.

For the purposes of applying cognitive-behavioral interventions, it is important to understand the nature of the cognitive dysfunction. Because not all cognitive dysfunction is the same, knowing the nature of the dysfunction associated with specific psychological disorders has important implications for treatment ( Kendall, 1993).

### The Nature of Cognitive Dysfunction

Not all cognitive dysfunction is the same. A distinction can be made between cognitive deficiency (lacking careful informational processing where it would be beneficial) in processing and cognitive distortion (dysfunctional thinking processes) in processing ( Kendall, 1981, 2000a), which can help design interventions that direct themselves appropriately to the area of concern.

Distorted information processing has been implicated in anxiety and depression, in which individuals tend to misconstrue or misperceive their social and interpersonal environment. In these cases, there is active information processing, but it is distorted (irrational, illogical, or crooked). For example, in a series of studies with depressed youngsters, the depressed children perceived themselves to be less capable on several dimensions than their nondepressed classmates when, in fact, teachers reported that the nondepressed and depressed children were indistinguishable on the same dimensions that the depressed children saw themselves lacking (Kendall et al., 1990). Thus, it was the depressed children who evidenced distorted thinking via the underestimation or misperception of their competencies. Additional studies have demonstrated that anxious and depressed youth tend to misperceive threats and danger, misperceive demands of the environment, and make attributional errors (anxiety: Daleiden et al., 1996; depression: Hammen and Zupan, 1984).

In contrast to anxious and/or depressed children, impulsive and hyperactive children tend to evidence processing deficiencies in the form of acting without thinking or performing poorly on a task because of a lack of planning or forethought. Children with deficiencies in processing do not engage in careful information processing and their performance suffers as a result. Their difficulties emerge from failing to stop and think before acting, as opposed to distorted processing of information. Studies have shown that aggressive (Dodge, 1985) and impulsive acting-out behavior is in part related to a lack of self-control, a lack of social perspective-taking, and a failure to employ verbal mediational skills ( Kendall and MacDonald, 1993). Interestingly, research has shown that aggressive youth evidence both cognitive distortions and deficiencies. An example of a deficiency is the limited ability to generate alternative, nonaggressive solutions to interpersonal conflicts ( Edens et al., 1999; Lochman and Dodge, 1998; Nelson and Finch, 2000). An example of a processing distortion of an aggressive youth is the child's tendency to misattribute the intentionality of others' behavior (e.g., construing an ambiguous situation as intentional) ( Dodge, 1985).

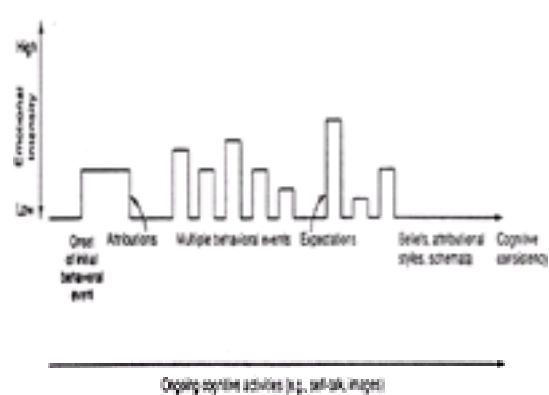
Cognitive-behavioral treatment strategies are modified to account for whether the youth acts without thinking or partakes in action with misguided thinking. Therapy for youth with cognitive deficiencies requires stopping nonthoughtful activity and developing and deploying skills in thoughtful problem solving. Helping youth with cognitive distortions involves the identification of faulty thinking and correction of distorted processing. Recognition of this distinction and the use of interventions that direct themselves appropriately to the needed arena have benefits for participating youth.

### A Temporal Model: Developing Coping Over Time

Empirical investigation continues to document the role of cognitive concepts such as expectations, attributions, self-statements, beliefs, and schemata in the development of both adaptive and maladaptive behavior patterns and the process of behavior change, yet the interrelations of these and other cognitive factors themselves need further clarification.

A model that can be useful in elucidating these relationships is one built along a temporal dimension. The model must take into account and be able to reflect the cognition associated with behavior across time (e.g., cognitions that occur before, during, and after events). Because events do not occur in a vacuum and behavior is determined by multiple factors, the model must allow for feedback that results from multiple, sequential behavioral events. This implies that the cognition before an event varies and the variation is owing to outcomes of previous events. The model must allow for variations in the pre-event cognition associated with the different outcomes (e.g., successful, unsuccessful) of prior events. Furthermore, the model must emphasize the development of more regularized cognitive processing (e.g., cognitive structures, cognitive styles) because repetitions of cognitive-event sequences result in some consistency in cognition.

The proposed model (Fig. 10.1) illustrates the flow of cognition across behavioral events of different emotional intensity. The starting point (on the left) is the initial behavioral event, which ultimately leads to cognitive consistency (on the right) after much repetition. Attributions are the cognitive concepts that are important after the behavioral event. How do children disambiguate the causes of their behavior once it has occurred? In other words, of all possible explanations that can be proposed, how do youth explain their own and others' behavior to themselves? Attributions are temporally brief in that they occur at the termination of the event. It is possible to assess an attribution long after an event, although many factors (e.g., memory recall) may interfere with accurate recall. Typically, the assessment of attributions occurs soon after the behavioral event took place.



**Figure 10.1.** A temporal model of the flow of cognition across behavioral events of different emotional intensity. Self-statements and images occur at any point and can be studied at various points in the temporal flow. Problem-solving processes also occur at various points, especially where conflicts arise.

Repeated behavioral events and the related cognitive processing result in some degree of consistency in both behavior and cognition. The figure illustrates that cognitive consistency (i.e., beliefs, and attributional style, or a cognitive structure) results from multiple events. These cognitive variables (consistencies over time) are more stable than a single attribution. More stable cognitive style variables may be more predictive in a general sense but are less predictive in specific situations than the actual cognition at the time of the specific behavioral event.

When there is an accumulation of behavioral events and event outcomes, the youth experiences more precise anticipatory cognition (i.e., expectancies). Bandura (1977) described expectancies as outcome expectancies and self-efficacy expectancies. Intentions, plans, and commitments are also anticipatory cognition, which may be more stable and consistent over time than situationally specific expectancies. The generalized expectancy is transitional in nature and can be viewed as an attributional style. For example, the generalized expectancy of an external locus of control can be present both before (expectancy) and after (attribution) an event. Preceding the event, the person's external orientation can lead to the anticipation of having a minimal effect: "Why bother to speak up? No one listens to me." After the event, when a decision has been made without the child's input, the event can be attributed to powerful others: "See? The big mouths always get their way."

Emotional intensity is represented vertically in Fig. 10.1. The higher the elevation of the bar indicating a behavioral event, the more emotionally intense the behavioral event. The greater the emotional intensity is, the more intense the experience is; thus, the greater the impact on the development of cognitive structure (schema). Events that are less emotionally intense may have less influence on attributions, future expectations, and memory, whereas emotionally significant events have greater impact on the development of schema and future thinking. Early negative experiences can result in future dysfunctional thinking. Thus, therapy should be emotionally positive and involve experience, leading to coping and adaptive cognitive processing. Effective cognitive-behavioral interventions for youth capitalize on



creating behavioral experiences with intense emotional involvement, while recognizing and addressing the participant's cognition.

## COGNITIVE-BEHAVIORAL THERAPY

The cognitive-behavioral approach to therapy can be defined as a rational amalgam: a purposeful attempt to preserve the demonstrated positive effects of behavioral therapy within a less doctrinaire context and to incorporate the cognitive activities of the client into the efforts to produce therapeutic change ( Kendall, 1993). According to the model outlined by Kendall (1985), CBT: (a) emphasizes both the learning process and the influence of the contingencies and models in the environment, while it (b) underscores the centrality of mediating information processing factors. As such, this integrative therapeutic approach combines the principles of early learning perspectives with aspects of the cognitive model. When treating children and adolescents, cognitive-behavioral strategies include both performance-based procedures and cognitive interventions to produce positive changes in thinking, feeling, and behavior.

Cognitive-behavioral therapy with children, as in work with adults, emphasizes the effects of maladaptive beliefs and attitudes on current behavior. The assumption is made that a child's reaction to an event is influenced by the meanings he or she has attached to the event. Or, in other words, emotional and behavioral responses to events in one's life are a function of how these events are perceived and recalled, in that the events affect one's self-perceptions and the pursuit of one's goals. In turn, these cognitive processes are influenced by underlying beliefs that individuals maintain about themselves, the world, and the future ( Hammen and Goodman-Brown, 1990). Accordingly, the treatment goal is for children to develop a new cognitive structure or a modified existing structure through which he or she can function adaptively in the world ( Kendall, 2000a). In the next section, we describe the key elements of CBT, including treatment goals and strategies, the role of the therapist, and the conceptualization of therapeutic change.

### Treatment Goal: Building a Coping Template

The notion of the schemata (Beck, 1976) or cognitive structure refers to the template through which an individual perceives and makes sense of the world. Cognitive-behavioral theorists have promoted the notion that this template influences what the individual perceives and how the information is processed and understood.

As children and adolescents develop, their style of processing the world changes. The goal of CBT, then, is to build new "coping" templates through education experiences and therapist-coached reconceptualizations of problems. New experiences, with guided processing of the experiences, are thought to help correct detrimental thinking.

What is being suggested is that the existing cognitive structure is not erased but that new skills and means of construing the world are built, and these new constructions come to serve as new templates for making sense of future experiences. Therapy does not provide a surgical removal of unwanted cognitive structures but offers to help to build new schemata with new strategies that can be employed in place of the earlier dysfunctional ones. Existing cognitive structures are incorporated into the new schemas providing the child with response alternatives. Consequently, the therapy helps to discourage the maintenance of dysfunctional schemas.

An effective program is one that intentionally plans and capitalizes on creating behavioral experiences with intense positive emotional involvement, while paying attention to the anticipatory and after-the-fact cognitive activities of the participants. The therapist guides both the child's attributions about prior behavior and his or her expectations for future behavior. Thus, the child can acquire a cognitive structure for future events (a coping template) that includes the adaptive skills and healthy cognition associated with adaptive functioning. Therapy aims to help a child change dysfunctional schemata into new schemata so that the child can identify and solve problems. The child either develops a new cognitive structure or modifies an existing structure, through which he or she can view a formerly distressing event.

### Elements of the Coping Template

Building a cognitive coping template entails the identification and modification of maladaptive self-statements, along with the building of a new way to view situations—a new structure that is based on coping. Cognitive-behavioral interventions promote the development of skills that are necessary for effective coping. Though several types of skill training techniques have been developed ( Table 10.1) (Kendall, 2000c), we focus mainly on a few procedures that have been most often used and studied. The following is an overview of these skill-building procedures, along with strategies that facilitate the child's acquisition of these skills.

Strategy	Specific intention
Coping modeling	Use of coping modeling, with therapist as role model rather than modeling model; the therapist models coping (into problem, behavior, strategy to deal with problem, eventual success); the coping model "thinks" out loud
Building coping template	Changing client's self-talk, reframing of problems, use of schemas as problems to be solved rather than as personal weaknesses
Rehearsal	Use of different contingencies, given the nature of the disorder being treated; modeling the frequency and intensity of self-evaluation, depending on the disorder
Rehearsal—practice procedures	Role-play exercises Multiple opportunities to practice Exposure to realistic experiences Simulated experiences
Effective education	Learning about feelings, self and others
Learning tools	Problem tools (e.g., worksheets) Homework (out-of-session) assignments

**Table 10.1. General Treatment Strategies Used in Cognitive-Behavioral Therapies with Youth**

## RELAXATION

As part of the treatment of psychological distress, cognitive-behavioral therapies offer exercises and opportunities to build skills in the area of relaxation. Relaxation training aims to teach children to develop awareness and control over their own physiologic and muscular responses. In this procedure major muscle groups of the body are progressively relaxed through systematic tension-releasing exercises ( King et al., 1988). By tensing and relaxing various muscle groups, the individual learns to perceive sensations of bodily tension and to use these sensations as the stimuli to relax.

When teaching these techniques, relaxation-training scripts are often incorporated ( Ollendick and Cerny, 1981). For example, a child is taught to tense and relax her stomach by imagining that she is squeezing through a fence or tensing and relaxing her hand muscles by pretending to squeeze the juice out of a lemon. There are substantial data supporting the beneficial results of teaching relaxation skills to children and adolescents ( Ollendick and Cerny, 1981; Koeppen, 1974).

## CORRECTING MALADAPTIVE SELF-TALK

Cognitively oriented therapies work from the assumption that maladaptive thinking is related to maladaptive behavior. By changing faulty cognitive functioning, thoughts can be restructured, so they can help the client function more effectively, rather than contribute to the client's dysfunction. Addressing the self-talk of children during stressful situations can have positive effects on their performance. Although they engage in some positive thought, they also engage in a detrimentally large amount of negative thought that detracts from and undermines their positive thinking. Data from research on young adults demonstrate that clients who are suffering from undue distress benefit from a reduction in their negative thinking ( Kendall, 1985; Treadwell and Kendall, 1996).

## PROBLEM SOLVING

Another component of the cognitive-behavioral approach is problem solving. Problems occur in the lives of all children and adolescents. The ability to solve problems is necessary for adequate adjustment in childhood. The overall goal of teaching problem solving is training children to develop confidence in their own ability to help themselves meet daily challenges. D'Zurilla and Goldfried (1971) have outlined a five-stage problem-solving sequence ( D'Zurilla and Nezu, 1999; Spivack and Shure, 1974). In the first stage, the therapist focuses on helping the child understand that problems are a part of everyday life and inhibit his or her initial impulses (e.g.,



avoidance behavior). In the second stage, the child works to formulate the problem into a workable problem with goals. The third stage involves generating alternative solutions, the core of which lies in brainstorming. Here, both practical and outlandish ideas should be generated. In the fourth stage of problem solving, the child evaluates each alternative, choosing the most appropriate solution, and then puts the action into effect. The fifth and final stage involves verifying the merit of the chosen solution. Training the child to ask himself or herself questions may help the child orient himself or herself in a problem-solving mind set. (Which solution do I think will work best?) In the end, problem solving helps the child become adept at generating alternatives in what may at first appear like hopeless situations.

Children's ability to generate alternatives to a problem and to competently evaluate each option is important for overall psychological health. In the domain of problem solving, it is important to recognize that: (a) children at different developmental levels face different challenges and (b) children vary in their ability to recognize a problem in need of a solution.

In therapy, children practice new skills while receiving encouragement and feedback, which leads to further refinement of those new skills. In addition, appropriate contingencies are established to shape involvement, to promote intrinsic interest, or to motivate clients who would otherwise be disinterested.

We have described a variety of skills typically referred to as "coping skills"—methods to cope with situations and events that would otherwise produce distress. The question remains, how best to transmit these skills to those children who need them most. In the next section, we describe several strategies that facilitate client acquisition of the coping skills. For instance, we discuss features of the therapeutic relationship that foster coping, as well as specific strategies including coping modeling, rehearsal (role-plays), and exposure.

## Treatment Strategies

Several treatment strategies have been developed to maximize the effective communication of the coping skills described earlier. We briefly describe the strategies that are most commonly considered essential in cognitive-behavioral therapies: coping modeling, behavioral rehearsal (role-plays), and exposure.

### COPING MODELING

Modeling derives its conceptual roots from social learning paradigm ( [Bandura, 1969, 1986](#)). The consistent finding that behavior can be acquired, facilitated, reduced, or eliminated through the process of observing others' behavior, and the effects of their behavior, has direct treatment implications. When perceived as a reasonable individual—respected, liked, and possibly even admired (and occasionally associated with positive reward)—the therapist can be a powerful model for nondistorted thinking, careful reasoning, and adaptive behavior.

Modeling is a valuable opportunity for children and adolescents to observe and learn; the therapist plans demonstrations that are focused on the child's specific behavioral and cognitive needs. The therapist can serve as a model for effective coping, not by behaving in an error-free manner (mastery-modeling), but also by exhibiting misbehavior, lack of attention, and cognitive misperception, while displaying strategies to correct these difficulties (coping modeling).

### REHEARSAL: ROLE-PLAYS

Practice is an essential strategy for enhancing learning and coping skills. Role-plays let both the therapist and child experience how it feels to utilize each of the alternative problem solutions that have been generated for a specific problem. These role-plays provide opportunities for the therapist to guide the experience and reinforce progress; this guided and reinforced practice is important in the remediation of cognitive, behavioral, and emotional difficulties. Role-playing can start with minimally stressful situations and, as the child masters the skills, move on to progressively more challenging situations. The therapist can begin with imaginal scenes and then move to real-life sessions that can be arranged in the office, home, school, or community.

### EXPOSURE

Exposure entails placing the child in a fear-provoking experience, either imaginably or *in vivo*, to help him or her acclimate to the distressing situation and to provide opportunities for the child to practice coping skills within simulated or real-life situations ( [Francis and Beidel, 1995](#); [Marks, 1975](#)). Exposure in one form or another has been the basis of treatment of several anxiety disorders for some time. Historically, variants of exposure include implosion and flooding. <sup>1</sup> Flooding involves exposing the client to the actual anxiety-provoking stimuli. Some treatment research has shown that flooding is helpful in the treatment of the more complex anxiety disorders such as agoraphobia and obsessive-compulsive disorders. In CBT, preparation for the exposure task, the exposure task itself, and postexposure discussions provide opportunities to address coping skills. Youths with anxiety, for example, can learn via exposure tasks that their predictions or fears are not always accurate, that they can experience and survive anxiety, and that they then have data to use to challenge anxious thoughts in the future.

Although we have described specific strategies for promoting change, CBTs are neither monolithic nor narrow-minded ( [Mahoney, 1977](#)). Quite the opposite is true: There are treatment strategies that appear in therapies for the various disorders; however, there are no rules carved in stone, and the emphasis on cognitive information processing within a context that uses social rewards and behavioral procedures to modify maladaptive methods of adjusting is intentionally flexible.

To assume that there is a simple monolithic "right" way to think, behave, and feel is to make a fatal error. In fact, quite the contrary is true ( [Kendall, 1992](#)). The human experience, including childhood and adolescence, is replete with opportunities for a diversity of thoughts, feelings, and actions. The definition of what is "normal" is broad and inclusive: Thoughts, feelings, and actions are abnormal only when they are maladaptive for the individual or interfering or destructive for others. Interventions are not designed to make the child conform to a single-minded definition of appropriate childhood behavior. Rather, interventions are designed to remove detrimental cognitive, affective, or behavioral styles that children may be developing and to offer—at an early point in life—valuable educational experience that can modify unwanted features of their developmental trajectory.

Interventions with youth are perhaps best when they mesh effectively with the normal developmental trajectory. In the target years of development, the move toward autonomy and independence is a central developmental challenge. A decided strength of the cognitive-behavioral strategy is that it works in a collaborative problem-solving fashion with the youth and, correspondingly, fosters independent development and prosocial behavior change. These intervention strategies are used to maximize the effective communication of the coping skills described earlier. Aside from specific strategies, the therapeutic relationship is critical in fostering the effective communication of these skills.

## Therapeutic Posture

The "posture" (referring to one's mental attitude) of the cognitive-behavioral therapist working with youth can be described using the terms "consultant," "diagnostician," and "educator." As a consultant, the therapist is a person who does not have all the answers but has some ideas worthy of trying out and some ways to examine whether or not the ideas have value for the individual youth. The consultant therapist helps create behavioral experiments to test some of the dysfunctional beliefs of the client. Telling a child or adolescent exactly what to do is not the idea: rather, the therapist gives the client an opportunity to try something and helps him or her to make sense of the experience. The therapist, as a consultant-collaborator, strives to develop skills in the client that include thinking on his or her own and moving toward independent, mature problem solving. The youngster and therapist interact in a collaborative, problem-solving manner.

As a diagnostician, the therapist integrates data, and, judging against a background of knowledge of psychopathology, normal development, and psychologically healthy environments, makes meaningful decisions. The posture associated with "diagnostician" is one of being confident to go beyond the verbal report and/or behavior of the client and his or her significant others. The cognitive-behavioral therapist serves as a diagnostician by taking into account the various sources of information (e.g., child, parent, school) and, judging against the background of knowledge, describes the nature of the problem and an optimal strategy for its intervention.

As an educator, the therapist is involved in teaching interventions for learning behavior control, cognitive skills, and emotional development in determining the optimal ways to communicate to help young people to learn. A good therapist, like a good educator, stimulates youths to think for themselves. A good educator observes the student, helps to maximize strengths and to reduce hindrances, and accepts that individuals can and should do things differently.

The posture of the cognitive-behavioral therapist working with children and adolescents is one that has a collaborative quality (therapist as consultant), integrates and decodes social information (therapist as diagnostician), and teaches through experiences with involvement (therapist as educator). A high-quality intervention is one

that alters how the child makes sense of experiences and the way the child will behave in the future.

## Developmental Considerations

The exact intervention method that is most effective is not necessarily the same for all children. When treating adolescents and children it is essential to be aware that they are not homogeneous groups. Children are developing organisms, which implies that they are ephemeral and evanescent beings. They are not stable, consistent, or unchanging. Numerous features of each child are not necessarily stable from pretreatment to posttreatment. Change over time owing to maturation alone is one aspect of the “developmental uniformity myth.” A second aspect of this myth is the assumption that children of different ages with the same behavioral problems are alike ([Kendall, 1984](#)). Clinicians need to be aware of the error in assumptions, and clinical interventions should be adjusted accordingly for different developmental levels. Furthermore, the same behavioral problems at various developmental levels may have varying effects on the child's social and interpersonal world.

The following example emphasizes the importance of considering developmental level in clinical interventions used with children. Systematic desensitization ([Wolpe, 1969](#)), a forerunner of more modern approaches, involves having the client experience deep muscle relaxation in the presence of gradually increasing exposure to feared stimuli. Muscle relaxation is an important part of the intervention. Interestingly, outcome literature of systematic desensitization with children has been unclear ([Hatzenbuehler and Schroeder, 1978](#)). It is possible that muscle tension is not uniformly associated with anxiety across various developmental levels. [Katz and colleagues \(1980\)](#) demonstrated that children between the ages of 10 and 18 experience muscle tension but that children under the age of 6 years do not. Thus, age may be considered a moderator in systematic desensitization.

Cognitive-behavioral interventions are designed with developmental considerations in mind. For example, a young cognitively immature child might benefit less from long, didactic sessions and would be better served by brief, active sessions, often with the cognitive content of the session occurring *in vivo*. Another important developmental consideration is sensitivity to the context of the developmental challenges facing the child. Therefore, a cognitive-behavioral therapist seeing a child with separation anxiety at 9 years of age intervenes within the appropriate developmental challenge, such as issues of autonomy. With older children, other themes (e.g., peer-related, achievement related) present themselves as more appropriate for intervention ([Southam-Gerow et al., 1997](#)).

## Parental Role

The cognitive-behavioral perspective considers the importance of the social context because behavioral patterns in the external world and cognitive interpretations in the internal world pertain to social and interpersonal contexts. The social context for children includes both family and peers. Satisfactory relations with peers are a crucial part of children's adjustment. Thus, an understanding of the role of peer relationships within a child's environment is necessary for meaningful assessment and treatment. Children learn about rules and roles for social interaction within the family context, making the family a powerful socialization agent.

The importance of parents in therapy for children and adolescents is clear. Increasingly, clinicians and researchers are focusing on the role of families in the treatment of childhood disorders ([Faubert and Long, 1991](#); [Sanders, 1996](#)). Adding a family management component to individual cognitive-behavioral therapy for children can be helpful ([Barrett et al., 1996](#); [Kazdin et al., 1992](#)). A family management component encompasses contingency management strategies to support and encourage courageous behavior and extinguish maladaptive behavior. Helping parents become more aware of their own emotions, teaching problem-solving skills, conflict-resolution training, and encouraging listening skills are some of the techniques used to help parents become more involved in their child's treatment and progress. Parents can serve as consultants by providing valuable information about the nature of their child's psychological problems and they can also help assist in the implementation of program requirements. In addition, parents' own maladaptive behavior and cognition can contribute to the child's distress. Changes in the family system are implemented in conjunction with promoting the child's skill building.

## Conceptualization of Change

Children and adolescents with behavioral and emotional difficulties have associated maladaptive qualities in cognitive information processing. Modifications in cognitive processing are in order for the depressed adolescent who is misattributing negative outcomes to internal-global-stable features, as well the impulsive or hyperactive child who is active in behavior but deficient in planned forethought. How might we conceptualize the needed changes?

Some cognitive distortions, as noted, require modification. New experiences, with guided processing of the experiences, help reduce distorted thinking. Thus, the existing cognitive structure is not being erased, but new skills and means of construing the world are built, and these new constructions come to serve as new templates for making sense of future experiences. For example, in the case of an aggressive child with a tendency to misinterpret the intentionality of others' behaviors, being bumped may be interpreted as being pushed maliciously by another child. With new skills such as enhanced problem-solving abilities, corrective of self-talk, and improved inhibition of maladaptive responses, the child can learn to adjust his or her interpretation and reaction (e.g., “maybe he tripped and lost his balance” or “stop and think before reacting”).

Therapy offers exposure to multiple behavioral events with concurrent cognitive processing, such that new cognitive structures can be built over time. Future experiences are construed differently as these new conceptions are incorporated into the child's overarching view of the world and his or her place in it. Using newly acquired skills and constructed schemata, the individual moves forward to face and confront new challenges in ways that manage former maladaptive tendencies.

## CONCLUSION

We have outlined a cognitive-behavioral theory in which behavioral events, associated anticipatory expectations and postevent attributions, ongoing cognitive information processing, and emotional states combine to influence behavior change. Relatedly, the theory adapts to the different challenges facing different levels of development. The theory is problem-solving oriented, deals directly with cognitive information processing, incorporates emotional and social/interpersonal domains, employs structured and manual-based procedures, and emphasizes performance-based interventions. Cognitive-behavioral therapy places greatest emphasis on helping youth advance their cognitive information processing in social contexts by using structured, behaviorally oriented practice, while concurrently paying attention to the participant youth's emotional state and involvement in the treatment.

The cognitive-behavioral model includes the relationships of cognition and behavior to the affective state of the organism and the functioning of the organism in the larger social context. Social/interpersonal factors play a crucial role in the design of and outcomes from therapy. Acknowledgments of peer and family contributions to child and adolescent psychopathology, however, far outweigh the currently available research database; the need for further inquiry into these areas cannot be overemphasized.

Cognitive-behavioral approaches to interventions for children and adolescents have been applied to a wide range of topics ([Kendall, 2000c](#); [Meyers and Craighead, 1984](#)), including anger ([Lochman et al., 2000](#)), attention deficit hyperactivity disorder ([Braswell and Bloomquist, 1991](#); [Hinshaw and Erhardt, 1991](#)), anxiety disorders ([Kendall, 1994](#)), medical and dental stress ([Melamed et al., 1984](#); [Varni et al., 2000](#)), depression ([Stark et al., 1991](#)), and impulsivity ([Kendall and Braswell, 1985](#)). Cognitive-behavioral approaches also have been used with children with chronic illness ([Walco and Varni, 1991](#)), depression ([Stark et al., 2000](#)), and learning disabilities ([Keogh and Hall, 1984](#); [Wong et al., 1991](#)). Inclusion of the parents ([Braswell, 1991](#); [Braswell and Bloomquist, 1991](#)) and families ([Barrett et al., 1996](#)) of youth has also been undertaken from the cognitive-behavioral perspective.

Cognitive-behavioral therapy with children and adolescents is promising in that it explicitly recognizes the importance of cognitive, behavioral, affective, and socioenvironmental variables in the etiology and maintenance of emotional disorders. Moreover, it is consistent with contemporary integrative models of behavioral change and maintains the objective, empirical focus that has served as the foundation of both behavioral psychotherapy and child psychiatry.

<sup>1</sup>In implosion, the therapist and child attempt to create feared images that are dramatic and exaggerated. As this imaginal exposure is prolonged, it is expected that extinction of the fear would occur. There is only limited research to support this approach, and its overall effectiveness has been questioned, especially with children (Rimm and Masters, 1979).

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# 11 ATTACHMENT THEORY AND RESEARCH

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Freud described the mother–infant relationship as “unique, without parallel, established unalterably for a whole lifetime as . . . the prototype of all later love relations” (1940, p. 45). But he was not the first to describe how important the mother–infant relationship is for psychosocial growth. For centuries parents have recognized their special place in the lives of young children, and because infants are so vulnerable and dependent, philosophers, practitioners, and (later) psychologists have been concerned with the quality of parental care that provides a secure foundation for healthy psychological development. It is only in the past two to three decades, however, that their ideas have been systematically tested by developmental scientists, and the interesting and provocative findings from their studies have contributed to making attachment theory one of the most exciting fields of contemporary developmental study, addressing enduring questions concerning infant–parent relationships. Moreover, the applications of attachment research to understanding and treating conditions of developmental psychopathology, and describing the relational needs of children, adolescents, and adults in our society underscore the practical as well as theoretical value of this literature. In a sense, attachment theory and research have provided developmental scholars with an unprecedented opportunity to empirically evaluate theoretic views of the importance of parental care for early psychological development, and explore diverse important applications of this research.

Attachment can be defined as an enduring emotional bond uniting one person (or animal) with another, and attachments are commonly manifested in efforts to seek proximity and contact to the attachment figure, especially when the individual is under stress. Theories of attachment underscore its evolutionary origins to promote the protection and nurturance of the young and, thus, contribute to species survival. Applied to human behavior, attachment is also regarded as an important psychological catalyst for the early emergence of trust in others and understanding of self, and as the basis for risk for psychosocial difficulty if attachments are disorganized or dysfunctional. As a consequence, attachment theory unites interests in evolutionary biology, developmental psychology, and clinical concerns in understanding early parent–offspring bonding.

In succinctly summarizing this exciting field, my purpose is to provide an orienting introduction to the theoretical views, research methodologies and findings, ongoing controversies, applications, and emergent trends that characterize contemporary attachment theory and research. (The reader is also directed to major papers and current review sources for further information.) In so doing, my goal is to highlight both what we do and do not know about the origins, correlates, and sequelae of attachment relationships in early childhood, and their relevance to applied problems of early psychological development. The emerging literature on attachment in adolescents and adults is also profiled. Much has been learned about the nature and importance of early attachment relationships and, combined with the provocative conceptual views from attachment theory, there are many exciting ideas to guide current applications and future research. At the same time, recent research has also shown that when we consider seriously the mutual influences of the quality of parental care, child's characteristics, ecology of early development, and consistency of caregiving influences over time, attachment processes are far more complex than traditional theoretical views might portray. It is this complexity and the research agenda it provides that make this field of research both exciting and compelling.

## THE THEORETICAL LEGACY: DEVELOPMENTAL PSYCHOLOGY, EVOLUTIONARY BIOLOGY, AND OBJECT RELATIONS THEORY

Attachment theory has roots in the psychoanalytic emphasis on the enduring significance of the early mother–infant relationship, together with subsequent studies showing that this affectional bond does not necessarily depend on the provision of food, warmth, or other physical necessities (Harlow, 1958), and the insights of developmental psychology (Karen, 1994). To create a new conceptual synthesis, John Bowlby (1969/1983, 1973) borrowed from evolutionary theory to argue that infant–mother attachment develops not only because of the mother's satisfaction of physical needs but also species-typical behavioral patterns that evolved to promote infant survival. In the Savannah grasslands of human evolution, Bowlby reasoned, there would have been survival advantages for vulnerable and defenseless human young to seek the protective proximity of responsive conspecific adults, especially when they were distressed, alarmed, or in danger. Such adults function as a “secure base” for the infant, and processes of evolutionary adaptation may, thus, account for why infants develop emotional attachments to responsive adults that motivate such proximity and contact-seeking efforts. Young who are motivated to stay close to such adults in these circumstances are less likely to fall victim to predators, become lost, starve, or suffer other calamities that, in the end, will undermine reproductive success, and thus it is normative for infants to develop attachments to their caregivers in species-typical rearing conditions. In a sense, Bowlby proposed that infants are predisposed virtually from birth to respond socially to social partners as part of this survival-enhancing motivational system, and he borrowed ideas from engineering control systems theory (updating the hydraulic energy model of psychoanalysis) to explain the dynamic functioning of this system. Finally, Bowlby provided a developmental perspective to his analysis by using insights from developmental psychology (especially Piaget's cognitive theory) to explain the growth of the baby's discrimination of and emotional investment in the parent, the increasing sophistication of the infant's attachment behavioral repertoire, and the child's developing understanding of the relationship with the parent.

Bowlby's theoretical insights are consistent with research evidence concerning the development of attachments in the first year of life. Emotional attachments to familiar caregivers become apparent between 6 and 12 months of age as infants gradually construct expectations for the behavior of other people and are becoming socially and behaviorally more competent (Thompson, 1998). As a consequence, well before the first birthday, infants are showing their preferences for adults to whom they have developed attachment relationships. These attachment figures include mothers and fathers, of course, but they also include regular baby sitters, grandparents, day care teachers, and others who assume a frequent role in the child's nurturance. Bowlby's theory is also consistent with research evidence showing that virtually all infants normatively develop attachments to their caregivers. The innate impetus toward emotional attachments is poignantly evident in some young children who have been raised in extremely deprived or abusive conditions, but afterwards manage to quickly develop close bonds to the nurturing adults who subsequently care for them, although the quality of their attachments may be disordered (Chisholm, 1998).

Individual differences in the child's perception of the parent as a “secure base” are also important and as a clinician, Bowlby was concerned with the long-term consequences of variations in the quality of infant–parent attachment. He and another early attachment theorist, Mary Ainsworth (1973), argued that the quality of these attachment relationships varies on a dimension of security, and that infants develop security in their caregivers based on their prior experiences of sensitive, helpful, responsive care (cf. Erikson's [1963] concept of “basic trust”). The secure attachment that results is adaptive—in both an evolutionary and psychological sense—because it derives from the infant's confidence that his or her help-seeking signals will receive a prompt and appropriate adult response. If infants experience sensitive care only inconsistently or sporadically, or if their care is regularly insensitive or unresponsive, they develop insecure attachments. An insecure attachment is *not* the same thing as having no attachment at all to a caregiver, however, because a young child who is uncertain, even doubtful, about the caregiver's helpfulness nevertheless derives important emotional support from the caregiver's presence that would not be derived from the company of someone to whom the child had no attachment at all. This underscores how important it is to respect children's emotional attachments to caregivers, even caregivers who are insensitive, because of the emotional resources such attachments provide to young children.

Bowlby (1973) believed, nevertheless, that many adult psychological disorders could be traced to the early development of insecure attachment. Indeed, in his view, an important part of the therapeutic process is to assist the client in developing secure attachments (such as to the therapist) while examining the contemporary ramifications of earlier, insecure attachment relationships. Such a view recently has been revived in current studies of adults' representations of their early experiences of childhood care (Dozier et al., 1999; Main, 1991).



The formulations of object relations theory thus can be found in Bowlby's portrayal of the sequelae of early attachments, and attachment researchers have sought to understand both the behavioral and representational outcomes of attachment security in early childhood. According to current theorists ( [Bretherton and Munholland, 1999](#); [Thompson, 1998](#)), attachment relationships exist not only as a behavioral system characterizing infant–parent relationships but also as a conceptual system governing significant social ties throughout the life span, with the emotions and expectations (secure or insecure) arising out of infant–parent relationships forming the basis for rudimentary conceptual representations of self and others in the years that follow. These “working models” or “states of mind” ( [Bowlby, 1973](#); [Bretherton and Munholland, 1999](#); [Main, 1991](#)) of self and significant caregivers are portrayed by theorists as interpretive filters through which self-understanding and understanding of important relationships are processed, while operating generally out of conscious awareness. Because of this, they are thought to be relatively stable over time, and individual differences in working models may foster consistent, long-term characteristics in parent–child relationships and, as they are maintained, also intergenerational continuities in caregiving tendencies as offspring internalize roles of caregiver and care recipient ( [Sroufe and Fleeson, 1986](#)). Such a view is also consistent with the notion that psychosocial development is characterized by continuity in adaptation in which a child's success in meeting early developmental challenges (such as forming a secure attachment) provides a strong or weak foundation for subsequent developmental challenges ( [Sroufe, 1996](#)). In each case, a secure attachment in infancy provides the basis for more successful psychosocial functioning in later years and conceptions of self as lovable and others as trustworthy, whereas an insecure attachment affords a much weaker psychosocial beginning and different forms of self and other understanding. In these ways, concepts from object relations theory significantly inform attachment theorists' views of the sequelae of early secure or insecure attachments.

Taken together, Bowlby's theoretical insight was to wed contemporary thinking in developmental psychology with evolutionary biology to provide fresh explanations for important psychoanalytic insights concerning the importance of the mother–infant relationship. Moreover, by bridging formulations in object relations theory, attachment theory also offers valuable perspectives into clinical problems and issues of lifelong adaptation. By and large, Bowlby's formulations have served the field well for more than three decades; however, recent advances in three areas add fresh insights and theoretical challenges.

First, in evolutionary biology, current thinking emphasizes that organisms are equipped with a variety of adaptive behavioral patterns employed contingently based on ecologic demands and other life circumstances ( [Chisholm, 1996](#); [Simpson, 1999](#)). Thus contrary to the view that evolutionary processes have equipped infants with a single adaptive attachment pattern (i.e., a secure attachment) that is based on a species-typical style of sensitive parental care, different patterns of infant attachment behavior can be considered adaptive in different caregiving contexts and in relation to different styles of parental care. This has important implications for cultural studies of attachment, and the interpretation of “insecure” attachment patterns in cultural contexts very different from the United States middle class.

Second, in cognitive developmental psychology, research scientists have moved beyond Piaget's theory to newer formulations that offer a far richer portrayal of the mental life of young children. Thus, the development of early “working models” of self, others, and relationships from attachment theory can be informed by recent studies in the growth of event representation, autobiographical memory, theory of mind, and other cognitive processes that develop significantly during the preschool years ( [Thompson, 1998, 2000](#)). One implication of this research literature is that the representations that guide enduring understandings of self and others probably emerge in early childhood, not infancy, and are influenced by parent–child discourse and other relational influences ( [Thompson, 2000](#)). This broadens understanding of how parental behavior influences attachment security and its sequelae in early childhood.

Finally, research on the ecology of early care reveals that significant changes have occurred in the contexts of early childhood development during the past three decades, and raise new questions about the diversity of attachment relationships, construction of internal working models, and continuity and change in attachment security. Young children now become attached to many caregivers in the family, child care setting, and elsewhere, and it is yet unclear how these relationships make independent or overlapping contributions to the internal representations that constitute working models of self and others ( [Berlin and Cassidy, 1999](#); [Howes, 1999](#)). Furthermore, young children now more often experience changes in family circumstances, including residential relocation, changes in care arrangements, and marital separation and divorce, that can challenge the stability of parent–child relationships and alter attachment security and the working models with which it is associated ( [Thompson, 2000](#)). The implications of these facets of the ecology of early care constitute one of the more important research topics for attachment theorists.

## RESEARCH METHODS

The value of these theoretical formulations would have remained limited, however, without the emergence of empirical tools for assessing individual differences in the security of attachment. There currently exist well-validated measures of attachment security in infancy and early childhood, newer (but less well evaluated) measures of attachment in older children and adolescents, and exciting advances in assessing adult “states of mind” concerning attachment ( [Crowell et al., 1999](#); [Solomon and George, 1999](#)).

### The Strange Situation

Along with her contributions to the conceptualization of these differences, Ainsworth's major contribution to attachment research was the creation of the Strange Situation procedure ( [Ainsworth et al., 1978](#)), a 21-minute laboratory assessment that has become the predominant procedure for assessing the security of attachment in infancy. It is difficult to overemphasize the contribution of this assessment procedure to attachment research. Because it provided developmentalists with a semistandardized, easy to use, and readily scorable means of studying a phenomenon that was long assumed to be fundamentally important to early sociopersonality development, the availability of the Strange Situation (together with Bowlby's provocative theorizing) virtually unleashed an unprecedented amount of research on attachment. This reliance on a single methodology also permits the comparability of diverse studies on attachment using different samples observed in the Strange Situation; this is a benefit rarely enjoyed by students of early social development.

The Strange Situation is designed to create a condition in which critical features of an infant's expectations for the caregiver's helpfulness and accessibility will be revealed behaviorally. Because attachment behavior is instigated when babies are distressed or alarmed, the Strange Situation creates gradually escalating stress for the baby to heighten the child's need for the parent's assistance over the course of seven 3-minute laboratory episodes. After a brief introduction to the setting followed by an initial episode in which the infant plays with toys in the mother's presence (episode 2), a stranger enters the room and plays with the baby (episode 3), after which the mother leaves the baby in the company of the stranger (episode 4). Mother and child are reunited (and the stranger departs) in episode 5, but stress is further incremented by a second separation period with the baby alone (episode 6), and the stranger's subsequent return to the room while mother remains absent (episode 7). Mother and infant are reunited a second time in episode 8, at which time the stranger again departs. Thus, by altering the social setting of the laboratory playroom, the Strange Situation heightens the child's need for the parent's support and assistance, and variations in the security of the infant's attachment to the parent are thought to be revealed most clearly. (See [Ainsworth et al., 1978](#), for further details.)

Attachment researchers focus particular attention on the two reunions (episodes 5 and 8) during which these variations are most apparent. Infants who are deemed securely attached (group B) greet the parent's return with relatively unequivocal pleasure, conveyed either through proximity and contact-seeking efforts or distal interactive behavior (e.g., smiling, vocalizing, and so on). These behavioral reactions are thought to reflect underlying confidence that the parent will be accessible and helpfully responsive to their needs for comfort or assistance. Alternatively, infants may be deemed insecurely attached and assigned instead to one of three other groups. Insecure-avoidant (group A) infants either conspicuously avoid the parent during reunions or, more commonly, mingle avoiding or ignoring the parent with more positive reunion greetings (e.g., a markedly delayed look and proffer of toy). Insecure-resistant (group C) babies combine proximity- and contact-seeking activities with angry, rejecting reunion behavior, or alternatively show extreme passivity during reunions. Finally, insecure-disorganized/disoriented (group D) infants are characterized during reunions not by any distinct behavioral style but by incoherent, confused, or inconsistent behavioral patterns (e.g., approaching parent with head averted) ( [Main and Solomon, 1990](#)). In each case, the behavioral reactions indexing insecure attachment are thought to be reflections of uncertainty or ambivalence concerning the parent's helpfulness following stressful separations, or an expectation that the parent will be unhelpful during reunion.

In most middle-class samples, the large majority (approximately 65%) of infants are deemed securely attached; this proportion tends to be lower in lower-income samples and is markedly depressed in many clinical samples. This is true whether infant–mother or infant–father attachments are studied or, for that matter, attachment relationships with any regular caregiver in the baby's life. As attachment theory predicts, infants develop emotionally meaningful attachments to each of the variety of adults who assume a regular caregiving role, including mothers, fathers, and regular baby sitters and day care workers ( [Howes, 1999](#)). Although these attachments usually vary independently in their security (e.g., an infant may be secure with mother and insecure with father, or the reverse), there is sometimes convergence between these different attachment relationships that may reflect the influence of temperament or other influences ( [Fox et al., 1991](#)).

### Attachment Q-Sort

Research using the Strange Situation accounts for most of the literature concerning the security of attachment in infancy, but this procedure is limited to samples of infants between 11 and 24 months of age. After 2 years, brief separations from the mother do not have the same impact on the child's attachment behavior because



separations are usually much less stressful. As a consequence, attachment researchers have explored several alternative methodologies that are appropriate for older children or can be used with a broader range of ages. Because they assess attachment security differently, these alternative measures capture somewhat different features of secure or insecure attachments than are revealed in the Strange Situation ( [Thompson, 1998](#)).

One alternative is a parent- or observer-report instrument called the Attachment Q-sort, in which individuals who are familiar with the child can systematically describe the child's attachment-related characteristics ( [Waters and Deane, 1985](#)). The Attachment Q-sort consists of 90 brief behavioral descriptors written on cards ("easily comforted by adult") that are sorted by the rater into one of nine piles, depending on how accurately they describe the child. Thus, cards sorted into pile 1 are "very much unlike the child," whereas those put into pile 9 are "very much like the child," with gradations between these anchors. After the sorting is completed, cards are scored (i.e., cards in pile 9 are each assigned a score of 9, cards in pile 8 receive scores of 8, etc.), and the entire card set can then be correlated with a criterion Q-sort that describes the characteristics of a securely attached child. The higher the correlation, presumably, the more securely attached a child is. Although the Q-sort approach is necessarily susceptible to the biasing influences of any observer-report procedure—that is, it indexes an observer's perceptions of the child's behavior, not necessarily the behavior itself—it benefits from the observer's detailed and long-term knowledge of the child and provides the most versatile means of indexing attachment security in children of various ages. Indeed, the Q-sort developed by [Waters and Deane \(1985\)](#) is suitable for children from infancy through early childhood and can be used in longitudinal as well as cross-sectional studies. In infancy, Q-sort scores often concord well with Strange Situation classifications ( [Vaughn and Waters, 1990](#)). However, defining a suitable threshold correlation with the criterion Q-sort for a securely attached child can be challenging, because the Q-sort yields a continuous score. Some researchers use an empirical threshold from previous attachment research (e.g., children with the highest 65% of the correlations in the sample are regarded as securely attached), whereas others establish an a priori threshold (e.g., a correlation of 0.36 or larger), whereas still others take advantage of analytical benefits of the continuous security score and do not try to categorize children at all.

### Other Approaches for Older Children and Adults

Other researchers have sought to develop direct behavioral measures of attachment suitable for older children ( [Greenberg et al., 1990](#)). Among the more notable efforts is a procedure for 6 year olds devised by Main, Cassidy, and their colleagues ( [Cassidy, 1988](#); [Main and Cassidy, 1988](#)). The procedure begins with the family together watching a short film of a child during a 10-day separation from his parents and is followed by an hour-long separation of parents and child during which the child plays with a female stranger and responds to a "separation anxiety interview." Observations of the subsequent parent-child reunion provide the primary basis for classifying 6 year olds' attachment security, borrowing the strategy of the Strange Situation. Attachment classifications for 6 year olds include a secure category as well as insecure-avoidant and insecure-ambivalent (or resistant) classifications. New for this age are additional classifications for insecure-controlling and insecure-unclassified attachments ( [Main and Cassidy, 1988](#)). Other researchers use a conventional, or modified, Strange Situation procedure with altered rating conventions for evaluating the attachment security of older children observed in this procedure.

Other researchers rely on self-report instruments with older children. Examples of these include the Security Scale, by which grade-school children report on the extent to which they perceive a parent as responsive, open to communication, and a reliable source of help in times of distress ( [Kerns et al., 1996](#)), and the Inventory of Parent and Peer Attachment, a measure for adolescents to describe the extent of their trust, communication with, and alienation from parents and close peers ( [Armsden and Greenberg, 1987](#)).

Finally, attachment theorists with a special interest in adolescents' and adults' representations of attachment processes have developed an interview procedure called the Adult Attachment Interview that systematically assesses adults' "states of mind" concerning attachment. These include recollections of the earlier parent-child relationship they experienced, including memories of security and comfort, acceptance by their own parents, feelings of rejection, fears of abandonment, and similar phenomena, and reflections on how these earlier experiences affect present caregiving and attachment relationships ( [Hesse, 1999](#); [Kobak et al., 1993](#); [Main, 1991](#)). Based on the content, emotional tone, and discourse quality of their responses to interview questions, adults are classified as either "autonomous," "dismissing of attachment," "preoccupied with attachment," or "unresolved" to characterize the adult's representations of relationships with each parent.

Research using the Adult Attachment Interview has yielded provocative findings indicating that differences in adults' representations of attachment are associated with variations in parental sensitivity and the security of attachment in offspring. Adults with autonomous (i.e., secure) representations tend to be more sensitive and to have infants with more secure attachments, compared with adults with dismissing, preoccupied, or unresolved classifications ( [van IJzendoorn, 1995](#)). It is yet unclear whether this arises because early patterns of care as a child have long-term effects on parenting capacity—consistent with current interest in the intergenerational transmission of parenting ( [van IJzendoorn, 1992](#))—or whether an adult's attachment representations are composed of the mature self-referential beliefs and attitudes that are shaped throughout life and guide parenting in complex ways ( [Baldwin, 1992](#); [Fox, 1995](#)). Both processes are likely involved and, it is hoped, will be elucidated in future research employing the Adult Attachment Interview and other measures of parenting beliefs ( [Thompson, 1998](#)).

These exciting methodologic advances attest to the fact that attachment researchers have moved beyond their initial concern with infancy to consider attachment processes throughout the life span. Moreover, studies using the Adult Attachment Interview and the Attachment Q-set indicate that these measures have promising validity and reliability and suggest important conceptual and developmental links to processes of attachment in infancy, although attachment security remains a developmentally dynamic process. This methodologic literature is promising, but the focus of the research review that follows is with the more substantive literature on attachment in infancy and early childhood, appraised primarily in the Strange Situation procedure. However, this research raises significant issues about attachment processes that have lifelong importance.

## SECURITY OF ATTACHMENT: ANTECEDENTS, STABILITY, AND CONSEQUENCES

Consistent with traditional formulations of child-parent attachment and the theoretical model developed by Bowlby and his followers, most of the research on the security of attachment has focused on several basic questions. (For comprehensive reviews of this research, see [Belsky and Cassidy, 1994](#); [Cassidy and Shaver, 1999](#); [Colin, 1996](#); [Lamb et al., 1985](#); [Thompson, 1998](#).) First, what are the antecedents of individual differences in attachment security, and in particular, are they associated with variations in the caregiver's sensitivity, helpfulness, and accessibility, as predicted by attachment theory? Second, once consolidated, how stable are differences in the security of attachment over time? In other words, does attachment security or insecurity become an enduring feature of child-parent relationships, or does it change and, if so, under what circumstances? Third, what are the predictable sequelae of a secure or insecure attachment in infancy or early childhood? Equally important, by what processes do these consequences occur? The reader will recognize that although these issues are central to the formulations of attachment theory, they also encompass longstanding theoretical questions concerning developmental continuity and the importance of early experiences ( [Thompson and Lamb, 1986](#)). This helps to account for the widespread interest of developmental scholars in attachment research.

### Antecedents

Consistent with theoretical expectations, attachment researchers have found that infants who become securely attached have experienced more optimal maternal care during the first year than do those who become insecurely attached. In general, the mothers of securely attached infants tend to act more positively, harmoniously, responsively, and sensitively toward their babies compared with the mothers of insecurely attached infants ( [Belsky, 1999](#); [De Wolff and van IJzendoorn, 1997](#); [Isabella, 1995](#)). Using more extreme comparison groups, a number of researchers have found that the large majority of infants experiencing parental maltreatment are insecurely attached, when compared with matched nonmaltreated control groups. Moreover, experimental intervention studies designed to improve the sensitivity of maternal care often succeed in doing so, and this can result in an increase in the attachment security of offspring ( [van den Boom, 1994](#); [van IJzendoorn et al., 1995](#)). These findings substantiate the validity of the Strange Situation as an index of infant-mother attachment as well as confirming several important predictions of attachment theory concerning the association between maternal sensitivity and infant security. Finally, as noted earlier, there is also evidence that the parents of securely and insecurely attached infants themselves differ in their security and in their recollections and representations of the care they received as children, which may contribute to the sensitivity of their care of their own offspring ( [van IJzendoorn, 1995](#)).

Although the quality of early care is an important influence on the security of attachment, it is obviously not the only important influence. Others include the caregiving involvement of the father and the extent of parental satisfaction with the marital relationship, the impact of socioeconomic stress on the family, social support to the mother, and the infant's own temperamental characteristics ( [Belsky, 1999](#); [Thompson, 1998](#); [Vaughn et al., 1992](#)). These findings indicate that the security of attachment is shaped not only by the quality of maternal care, but also by the reciprocal influences of each parent, the baby, the family network, and the broader ecological circumstances in which they live. The importance of adopting a broad, rather than narrow, view of the antecedents of attachment security is underscored by a metaanalysis of attachment research by [Goldsmith and Alansky \(1987\)](#), who found that maternal caregiving quality and infant temperament alone each accounted for a meaningful but small proportion of the variance in the security of attachment ( [De Wolff and van IJzendoorn, 1997](#)). Although the emotional climate of maternal care is important, that climate is influenced by many other aspects of the child's life circumstances ( [Thompson, 1997](#)).



This view is affirmed by cross-cultural studies of attachment, which have found considerable variation between cultures and subcultural groups in the relative proportions of secure and insecure attachments ([Thompson, 1998](#); [van IJzendoorn and Kroonenberg, 1988](#); [van IJzendoorn and Sagi, 1999](#)). For example, insecure-avoidant attachments are uncommon but insecure-resistant attachments occur more frequently than in the United States among samples of infants raised in Japan and on traditional Israeli kibbutzim. By contrast, avoidant attachments are more common among West German infants than US infants. However, there is also considerable variation within, as well as between, these cultural groups. Why do these differences exist? Most researchers believe that cultural differences in the normative conditions of early infant care (e.g., frequency of contact with strangers, regularity of separations from mother) and/or in child-rearing prescriptions (e.g., parental independence-training of young offspring) may help account for these differences. Because the Strange Situation, which was originally designed for middle-class infants in the United States, includes experiences with strangers and separations, it may provide an unusually stressful experience for some infants from other cultures, such as Japanese infants who have had little prior experience with maternal separations. As a consequence, the Strange Situation may not appropriately or validly assess the security of attachment for infants from these settings. Some babies may act insecurely when they are overwhelmingly distressed, for example, but the infant–parent relationship may nevertheless be secure. These studies not only suggest, therefore, that cultural and subcultural child-rearing norms may foster meaningful variations in normative patterns of attachment, but also that an infant's experiential background must be considered very carefully in evaluating the validity of the Strange Situation as an assessment of the security of attachment.

### Stability Over Time

Despite theoretical expectations that early attachments, once consolidated, should remain relatively stable over time because they take shape during a period of heightened sensitivity to early caregiving influences, the empirical evidence is actually quite mixed. Although some studies show that infants or young children retain the same quality of attachment over many years, others indicate that attachment security can change over a period of a few months. In studies of infants from lower-income as well as middle-class homes, for example, the proportion of infants with stable attachments over as little as a 6- to 8-month period in the second year ranges from 48% to 96% in test-retest assessments ([Thompson, 1998](#)). It appears that the security of attachment can, and does, change normatively in early childhood.

Equally important, however, is additional research evidence suggesting that changes in attachment are theoretically predictable: They are linked to circumstances that might precipitate changes in the infant–mother relationship. In a study of low-income families ([Vaughn et al., 1979](#)), for example, attachment security changed in response to financial, legal, and social stresses on the family; in a study of middle-class families ([Thompson et al., 1982](#)), changes occurred in response to significant transitions in caregiving arrangements for the baby; in a third study ([Teti et al., 1996](#)), changes occurred when a younger sibling was born. More broadly, there is considerable research evidence that parental behavior toward offspring changes considerably throughout childhood owing to the maturing capabilities of children, and changes in the life circumstances of parents, that may significantly alter parental sensitivity and parent–child relationships ([Holden and Miller, 1999](#)). In short, changes in the security of attachment are normative and are linked to many kinds of family events that might create a renegotiation of familiar patterns of mother–infant interaction. This is, in fact, exactly what we would expect to find if the Strange Situation sensitively appraises normative fluctuations in attachment relationships in infancy, although considerably more research is necessary to specify the conditions underlying change, and continuity, in the security of attachment.

Such a conclusion is unsurprising, given the variety of antecedent influences shaping the security of attachment outlined earlier. Because infants are embedded in a complex social ecology, changes in that ecology are likely to influence their attachment relationships. These findings indicate that the security of attachment reflects the current—but not necessarily enduring—status of the infant–parent relationship, and efforts to predict whether the current status of this relationship will endure or change must take into account the consistency of the conditions of care experienced by the child. As a general rule, therefore, flexibility rather than resiliency of early attachments seems to be true, and although this view is contrary to some theoretical expectations, it is optimistic with respect to the possibility of intervening into dysfunctional infant–parent dyads by changing familiar patterns of interaction or the broader ecological conditions in which they live ([Lieberman et al., 1991](#)).

### Attachment and Later Development

What difference does a secure or insecure attachment have for later development? Do secure children grow up to become more sociable, well-adjusted children compared to those who were insecurely attached as infants? The association between attachment security in early childhood and later psychosocial functioning is among the most important topics of attachment research because of theoretical views that a secure mother–infant bond establishes a foundation for subsequent psychosocial growth, and that the initial attachment relationship shapes representational working models of self, others, and relationships that color later social interactions. On the other hand, development is complex, and because of the multifaceted, cumulative dynamics of psychosocial growth, few early sociopersonality measures have been found that predict long-term developmental outcomes. This might also be true for the security of attachment.

There is now evidence, however, that a secure attachment in early childhood has predictable outcomes ([Belsky and Cassidy, 1994](#); [Thompson, 1998, 1999](#)). The most important is that it foreshadows a more cooperative, harmonious parent–child relationship in the years that follow. This is important, because parent–child comity makes the child more responsive to the parent's socialization efforts and more likely to adopt the parent's viewpoints, values, and goals ([Waters et al., 1991](#)). There is also evidence that securely attached young children socialize more competently and are more popular with well-acquainted peers during the preschool years, and establish warmer relationships with teachers, camp counselors, and others whom they know well in the years to come ([Sroufe et al., 1993](#); [Thompson, 1999](#)). Securely attached children may also respond more positively to unfamiliar people (e.g., new classmates, a substitute teacher), but the positive or negative expectations for close relationships inspired by a secure or insecure attachment are most apparent in children's encounters with familiar partners. There are also recent findings suggesting that security of attachment is associated with different kinds of self-concept, emotional understanding, friendship conceptions, conscience and moral understanding, memory processes, and other representational processes in children, especially when they are assessed contemporaneously with attachment security. The emerging conclusion that securely attached young children see themselves and others more constructively, and have more sophisticated emotional and moral understanding, is consistent with the notion that attachment is associated with unique working models of self and others ([Thompson, 1999](#)). The research evidence is very weak, however, concerning whether attachment security is associated with features of personality organization or development ([Thompson, 1998, 1999](#)). Perhaps this is because personality is a complex phenomenon that is affected by many factors besides attachment security.

These interesting findings are qualified in several ways. First, the developmental influence of attachment security in early childhood wanes over time: Its effect is most apparent during the preschool years, but its influence becomes less evident with increasing age ([Sroufe et al., 1990](#); [Thompson, 1999](#)). This can occur because of subsequent developmental experiences, or because of changes in family circumstances that alter the sensitivity of parental care (such as marital discord between parents) ([Cummings and Davies, 1994](#)). In these circumstances, the effects of an initially secure attachment on subsequent development are diminished when parents become markedly insensitive and no longer support relational trust or, conversely, when an increase in sensitivity inspires positive developmental outcomes in offspring who were initially insecure ([Erickson et al., 1985](#)). Although some studies reveal a lingering influence of early security of attachment many years later, most suggest that the developmental consequences of early security or insecurity depend on consistency and change in the continuing quality of parent–child relationships. The strongest associations between attachment security and other sociopersonality variables are apparent when both are assessed at the same time, although their causal association is more difficult to discern in these circumstances.

Second, many other influences besides a secure or insecure attachment affect developing sociability, self-concept, relationship representations, and personality organization. Bowlby himself recognized that attachment is only one of several dimensions of the parent–child relationship and, indeed, at the same time that attachment is developing, young children are also learning the skills of cooperation, compliance, and conflict resolution with parents ([Thompson, 1999](#)). This means that even when attachment security is developmentally influential, its impact is in the context of multidetermined developmental processes in which other factors also make significant contributions ([Sroufe et al., 1999](#)). Moreover, with increasing age, new developmental experiences subsume the influences of earlier experiences, like a secure attachment, and may alter their influence on sociopersonality development. Third, and finally, there are some suggestions in the research that the influence of attachment security may also be mediated by a child's temperament, such that children with particular temperamental profiles are differentially susceptible to the effects of a secure or insecure attachment ([Belsky, 1997](#); [Kochanska and Thompson, 1997](#)).

The research on attachment and later development thus indicates that a secure or insecure attachment has diverse but contingent consequences for young children. On one hand, there is increasing evidence that attachment is associated with the growth of sociopersonality characteristics and understanding that are consistent with the view that attachment shapes social behavior and emerging working models of self, others, and relationships. These outcomes are especially apparent early in life. On the other hand, the effects of early attachment security are not necessarily enduring, tend to diminish over time, and emerge in concert with many other developmental influences. It thus appears that early in life—before representational capacities have become sophisticated and well consolidated—young children rely on the continuing harmony of their relationships with parents to develop and maintain positive working models of self and relationships. The security of attachment in infancy is a marker of the quality of parent–child interaction that must be maintained early in life to support positive developmental outcomes. At later ages, as representational capacities become more enduring and self-maintaining, these working models then become incorporated into the network of self-referential beliefs that older children and adolescents begin to construct from a variety of developmental experiences, and become more independent of the current quality of

parent–child interaction. These constitute the basis for the “states of mind” concerning attachment that are assessed by the Adult Attachment Interview and related measures.

## ATTACHMENT AND CLINICAL INTERVENTION

Given the amount of interest they have generated among developmental scholars, it is perhaps unsurprising that formulations of attachment theory have found important applications in a variety of human services fields, including clinical psychology and psychiatry, social work, preventive mental health services, and even family law. This is because attachment theory and research help to articulate the relational needs of young children, adolescents, and adults and describe how early relational experience can have broader consequences for development and personality functioning.

One of the most important applications of attachment theory is to the understanding and treatment of child clinical disorders. This is especially so because the field of early mental health is still in its infancy, and the growth of research on attachment relationships has coincided with the emergence of theoretical and clinical models of psychological disorders in infancy and early childhood. Because attachment researchers are concerned with the formative effects of early patterns of infant–parent interaction, the emergence of early representations of self and others, and the prediction of later behavioral problems from early social relationships, there is a natural affinity with the etiologic, diagnostic, and therapeutic concerns of child clinicians.

There are important conceptual contributions of attachment theory to an understanding of early problems of developmental psychopathology ( [Belsky and Nezworski, 1988](#); [Greenberg, 1999](#); [Rutter, 1995](#); [Sroufe et al., 1999](#)). These include insights into young children's relational needs, such as the importance of the caregiver's warmth and sensitivity, the developmental significance of a young child's trust in the caregiver's helpfulness and accessibility (especially under stress), the ways that relational experience “goes underground” to provide a foundation for representational systems of self-awareness and social understanding, the impact that early relational experience can have on subsequent relationships by shaping a young child's expectations for people and responses to them, and the ways that early relationships can be affected by broader features of the child and family social ecology. Other conceptual contributions of attachment research to child clinical work include an understanding of the nature of development, especially the realization that developmental history interacts with (and can be transformed by) subsequent experience in shaping a child's current functioning, and the importance of risk and protective factors provided by close relationships to the development of mental health. Attachment theory also teaches that early experiences rarely lead directly to later psychopathology, but instead inaugurate developmental processes that may be maintained (or altered) by subsequent experiences ( [Sroufe et al., 1999](#)). Each of these formulations, arising from attachment theory and research, can provide a valuable perspective for thinking about young children's clinical disorders and how best to provide treatment. These ideas are also consistent with the valuable insights offered by the field of developmental psychopathology ( [Cicchetti and Cohen, 1995](#)).

Attachment theory and research is also relevant to understanding specific clinical disorders, because of the importance of relational influences to their origins, maintenance, and remediation. Attachment theory has been applied to understanding conduct disorders, social withdrawal and inhibition, anxiety disorders, childhood depression, and other early clinical problems ( [Greenberg, 1999](#); [Zeanah, 2000](#)). In each case, disturbed caregiving relationships are often one of the significant etiologic features that, in concert with other risk factors (such as temperamental vulnerability) contribute to the onset and maintenance of psychopathology. There is also emerging understanding of the varieties of “disorders of attachment” that arise most significantly in conditions of aberrant care, such as parental maltreatment, institutional rearing, or marked neglect. Disorders of attachment can be manifested in behaviors such as fearfulness and hypervigilance, indiscriminate sociability, aggressive or controlling behavior, blunted emotional responsiveness, or markedly ambivalent or contradictory social behavior ( [Lieberman and Zeanah, 1995](#); [Lyons-Ruth and Jacobvitz, 1999](#); [Zeanah et al., 1994](#)). Although these dysfunctional behavioral patterns are clearly related to more than just disturbed attachment relationships, attachment theory and research offers valuable insights into their etiology and treatment.

Attachment research also has important implications for the treatment of these developmental disorders of early childhood, including underscoring the importance of a family systems perspective, attention to the representational facets of early psychological problems, and understanding the impact of early relational experience on subsequent relationships ( [Crittenden, 1992](#); [Erickson et al., 1992](#); [Fraiberg, 1980](#); [Lieberman and Zeanah, 1999](#); [Marvin, 1992](#); [Zeanah, 2000](#)). More speculatively, attachment theorists have also extended their clinical implications to therapeutic practice with adults, drawing on Bowlby's original formulations concerning the lifelong importance of relational security, the reestablishment of secure working models in the context of a therapeutic relationship, and the association between insecure attachment and defensive styles ( [Slade, 1999](#)).

These clinical applications of attachment theory and research contribute to fulfilling Bowlby's original vision of creating a developmental theory of early relationships that would update and extend the psychoanalytic model and its therapeutic implications. But this is still a young field of inquiry and considerable conceptual and empirical work remains to understand fully how attachment concepts are relevant to problems of developmental psychopathology. Although insecure attachment is probabilistically related to less optimal developmental outcomes than is secure attachment, for example, it is not clear that attachment insecurity places a baby “at risk” for later problems or warrants special intervention ( [Nezworski et al., 1988](#); [Peterson, 1987](#)). Although the Strange Situation has proven to be a valuable research tool in studies of early attachment, clinicians should be cautious in making individualized assessments of infant security or parenting sensitivity based on the Strange Situation alone because of the many factors that can influence any particular assessment in this procedure ( [Rutter, 1995](#)). These are two examples of the problems commonly encountered in translating exciting research findings into individualized assessment and therapeutic implications.

More broadly, there are at least two other issues from the attachment literature requiring greater reflection as this research informs clinical practice. First, the results of research with nonclinical samples, reviewed in the preceding, indicate that there is considerable flexibility, rather than resiliency, to attachment system functioning in the early years of life. Rather than foreshadowing inevitable subsequent difficulties, an insecure attachment is related only conditionally and provisionally to later psychosocial problems ( [Bates and Bayles, 1988](#); [Erickson et al., 1985](#); [Fagot and Kavanagh, 1990](#); [Lewis et al., 1984](#)). Moreover, early attachment foreshadows later developmental outcomes primarily when there is consistency in caregiving influences over time that help to maintain and support early characteristics in offspring. Thus, there is considerable value to considering the extent to which attachment security reflects a characteristic of the child, or of the parent–child relationship, how this changes developmentally, and its implications for treatment of early clinical problems. There is also value in reconsidering the confidence with which clinicians can estimate future developmental outcomes for individual children based on early attachment security or insecurity.

Second, although the concept of “working models” of self, others, and relationships derived from attachment theory is heuristically powerful, very little is known about the developmental origins of individual differences in these representations in children and adults, or how they can best be assessed ( [Thompson, 1998, 2000](#)). When a young child responds to a semiprojective story or provides an account of family experience, for example, it typically reflects many things, of which enduring understandings of self and others is only one contribution. The need for further research on the development and assessment of early attachment-related representations is underscored by the importance of careful, cautious thinking concerning their lifelong significance. Even though there are influential theoretical models positing that adult representations of attachment are directly linked to early experiences of care—and help to account for interest in the “intergenerational transmission” of attachment security—these theoretical models have not yet been adequately tested. This warrants caution in making assumptions about the bases of these attachment representations, which are likely to be quite complex, or about their long-term developmental significance.

There are clearly many valuable applications of attachment theory and research to the concerns of child clinicians, and these applications are already apparent in the design of early interventions and growing understanding of problems of early mental health. Although the formulations of attachment theory are of considerable heuristic value, however, substantially more research (with clinical as well as nonclinical samples) is required to specify more clearly the meaning of security or insecurity of early attachment and its contemporary and subsequent significance.

## CONCLUSIONS

One clear conclusion from the attachment research is that support for relational trust is essential not just in infancy, but throughout the life course as young children, older children, adolescents, and adults are continuously seeing secure relationships and reworking their working models of attachment. As a consequence, developmental researchers, clinicians, and other practitioners have a considerable research agenda for building on the provocative findings of studies reviewed in this chapter. Given the continuing contributions of Bowlby's insightful theory, there is good reason for optimism concerning the future contributions of attachment theory and research.

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# 12 THEORIES OF COGNITIVE DEVELOPMENT

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## Part 1: Developmental Theories

### COGNITIVE DEVELOPMENT AND THE CHILD PSYCHIATRIST

In his essay, "The Basic Functions of a Child Psychiatrist in Any Setting," J. Cotter [Hirschberg \(1966\)](#) proposed

the child psychiatrist brings to any setting his [her] awareness of the child's age in relation to the child's thinking, feeling, and behaving. . . . [and] . . . his [her] awareness of . . . the characteristics of the child's thinking at various stages of growth ( [Hirschberg, 1966](#), pp. 360, 363).

He was alluding to the fact that children organize their experiences of events and of themselves as agents in different ways at different phases of their life. Normative life events (separation from caregivers, birth of a sibling); disruptive ones (abuse, death of a parent); and ongoing conditions (deafness, attentional disturbances) impact differently on the developing child as infant, toddler, preschooler, school-age child, and adolescent. Recurring or significant experiences become structured knowledge about the world and what can be expected from it. Knowledge structures that help the child anticipate, and thereby manage, common events become self-reinforcing and enduring. Cognitive developmental theories help child psychiatrists understand how children organize experiences, good and bad, to create a coherent sense of themselves in their world, be it functional or problematic.

### TERMS: COGNITION, DEVELOPMENT, AND COGNITIVE DEVELOPMENT

#### Cognition

Put simply, *cognition* is the faculty of knowing—the making and using of knowledge. It includes the faculties of attentiveness, information processing, intuition, memory, skill, and many others. *Cognitive psychology* explores how knowledge is deployed both to adapt to circumstances and alter them. *Cognitive developmental psychology* examines how the mind organizes experience into structured knowledge and how knowledge structures, in turn, organize and reorganize the mind from infancy through adulthood. This chapter outlines the principal theories of cognitive development and demonstrates their relevance to the development of the whole person and the puzzling clinical problem of autism.

Examined more closely, cognition is such a protean idea in psychology that it has been hard to comprehensively define it; indeed, [Flavell \(1993\)](#) contends that *all* psychological activity may be cognitive in some sense. Nevertheless, the outlines of cognition as distinct from other faculties of the mind can be drawn from comparing metaphors used to describe it ([Lakoff and Johnson, 1980](#)). For example, [Gregory \(1987, p. 149\)](#) sees cognition as "the use and handling of knowledge." In Gregory's metaphor knowledge resembles a *substance* and cognition its application. Other writers ( [Flavell et al., 1993](#); [Vygotsky, 1934/1986](#)) consider cognition to be "higher mental processes." Here the focus is on mental processes (thinking) rather than mental content (knowledge). In counterpoint to Flavell's assertion, Sandler avers "there is no such thing as a purely cognitive or intellectual process" ( [Sandler, 1987](#), p. 247). For example, [Bruner \(1990\)](#) sees cognition as "meaning-making" that has affective, social, and subjective connotations. Similarly, Piagetians propose that "in order to know objects the subject must act upon them" ( [Boden, 1994](#), p. 8). Clearly, learning depends on the input and feedback generated by actual encounters with the world; however, by what mechanisms do emotions and actions contribute to the origins of thinking? If Sandler is right, can "thinking" be reliably distinguished from "doing" and "feeling"?

Well, perhaps. Philosophers and cognitive scientists define cognition to be "any instance of a mental operation that displays *intentionality*" ([Dunlop and Fetzer, 1993](#), p. 24). Intentionality is "a property [of] any mental state that is representative, or ('about' something—whether or not that something actually exists. . . . Intentionality is uniquely characteristic of minds. . . . Beliefs, memories, goals, desires, expectations, etc. all display intentionality" ( [Dunlop and Fetzer, 1993](#), pp. 67–68). In the language of cognitive science, "Insofar as the information contained in and processed by an information-processing system is *about* anything—that is, insofar as it functions representationally—the states and processes of that system are intentional" (italics added) ( [Stillings et al., 1987](#), p. 317). By contrast, neither feelings nor actions are necessarily intentional.

Coming at affect and cognition another way, Piaget proposed that cognition organizes and channels the psychological energy generated by affect as a car engine



organizes and directs the energy provided by burning gasoline (Cowan, 1981). Piaget's distinction is a qualitative one; cognition is structure, and affect is energy. Yet again, those who view cognition as the processing of information, distinguish it from sensation and perception quantitatively, by the time it takes to process a stimulus from input to output. The more time taken, the more complex the presumed mediating events, the more cognitive the process. Cognition can be distinguished from perception by its processing time and from affect and sensation by its intentionality. In summary, cognition entails *content* and *process, structure, and complexity*: That is, knowledge (content) and thinking (process) are both structured and complex.

## Development

The term *development* denotes a sequential increase in the structural and/or functional complexity of a system. *Structures* are inferred organizational properties that underlie any given behavior. These organizational properties are assumed to change systematically with age. Development of structure means that a system becomes organized in a more complex way, whereas development of function implies that it can coordinate and integrate more complex arrays of information: A change in functional or *processing* complexity entails an increase in structural complexity and vice versa.

Heinz Werner (1948) proposed development to be an alternation of *differentiation* and *integration* (or *coordination*). For example, in biology, unspecialized precursor cells differentiate during development into several types of specialized offspring. These integrate their specialized functions to form organs that, at a higher level, organize themselves into organ systems and, in turn, into organisms. In mental terms, perceptions and symbols are *internalized*—reconfigured internally as *mental representational structures*. *Representations* are “the internalized schemas or frames of reference which the child uses in his interaction with the external world” (Deregowski, 1977, p. 219). They may be in the forms of logic, rules, concepts, images, analogies, and neural connections (Thagard, 1996). Simple representations combine to form complex arrangements that, in turn, differentiate and recombine into ever more integrated and complex structures that then influence the selection and handling of further input.

## Cognitive Development

As there are differences about what cognition is, so there are differences as to how it develops. Some see cognitive development as the increasing speed and sophistication of mental activity, independent of knowledge (Sternberg, 1984); others believe that the contents of knowledge determine cognitive development (Chi, 1988). Some include affect to explain cognitive development (Case, 1985), others do not (Anderson, 1983). Some (Piaget) emphasize the development of the internal, formal, self-constructed qualities of thought; others (Vygotsky) highlight the developmental role of external, social–interactional exchanges. Some see cognition as *domain-specific*. For example, Howard Gardner (1985, 1999) proposed several distinct domains of cognition (“intelligences”) musical, logical, verbal, spatial, kinesthetic, intrapersonal, interpersonal, and others, all quite independent of each other. Those who see cognition as domain-specific often take a *nativist* position; cognitive capacities are innate and development is the biologically preordained coming online, with age, of specialized processing units (modules) minimally influenced by experience or each other. By contrast, both Piaget and Vygotsky see cognition as *domain-general*; that is, as a property of the whole mind. In this *constructivist* (or *constructionist*) view, development takes place as the child internally reconstructs her or his lived experience. Development in one domain informs development in others.

### COGNITIVE DOMAINS: SPECIFICITY VERSUS GENERALITY OF DEVELOPMENT

In cognition, a *domain* is “the set of representations sustaining a specific area of knowledge (language, number, . . . and so forth). . . . A *module* is an information processing unit that encapsulates that knowledge and the computations on it” (italics added) (Karmiloff-Smith, 1992). Information processing follows the general model of a computer based on a *central processing unit* (CPU) that can be deployed to perform computations for a great variety of purposes. The same CPU can run a graphics program, a word-processing program, or a spreadsheet according to the software installed. If we call each application a domain of knowledge, the computer's operating system is domain-general, whereas each software application is domain-specific. Each software application can be seen as a module dedicated to organizing and assimilating informational input into its domain's representational structure as image, text, chart, or whatever. In human cognition, modules are “subsets of our neural networks . . . genetically prestructured for processing different kinds of input information: for example language . . . or mathematical information” (Lee and Das Gupta, 1995, p. 20). In the strong version of this view, modules are “inborn, automatic, . . . unconscious and embodied in dedicated neural mechanisms” (Boden, 1994). They are “cognitively impenetrable”; that is, “other parts of the mind can neither influence nor have access to the internal workings of a module, only to its outputs. . . . Input systems [modules] are inflexible and unintelligent . . . but they are just what a young organism needs to get initial cognition off the ground speedily and efficiently” (Karmiloff-Smith, 1992, pp. 2–3). The whole configuration of operating system plus software makes up the computer analog of a person's *cognitive architecture*.

### DEVELOPMENT AND STAGES

Often a developmental theory is presented as a series of *stages*. Stage theories imply discontinuous development: Periods of stability and consolidation alternate with periods of instability and transition. They also imply qualitative changes in cognition from stage to stage. The notion of stages is controversial; some believe stages reflect a natural rhythm in the developing organism while others see them as artifacts of the method or bias of the investigator. Theories based on a biological metaphor (e.g., Piaget's), tend to be set in stages, whereas nonstage theories usually have a social-learning pedigree.

Each stage organizes all cognition in a characteristic way at any point in the life span in a Piagetian-type stage theory. Each stage entails the hierarchic integration and transformation of the preceding ones; each earlier one becomes a subset of the next later one. Stages succeed one another in an invariant sequence of transformations; a step cannot be skipped nor does the culture change the sequence. Stages are universal; the culture may vary the content but not the structure. Finally, each stage achieves its full range only on completion; most activities in a stage are incomplete and contribute to the final version. The theories of Piaget, the neo-Piagetians (Case), and some Piaget-based social-cognitive (Selman) and moral (Kohlberg) theories are stage theories.

Nonstage theories are represented in this chapter by information-processing (Anderson, 1983) and learning-based theories (Carey, 1990; Ceci, 1996; Chi et al., 1988). In these, development is continuous; they do not distinguish points of qualitative change in cognition nor alternating periods of relative stability and instability. They explain the apparently discontinuous changes of cognitive development as ever-increasing processing efficiency. They do not require invariant sequencing or central processing structures that organize disparate domains of knowledge according to a single blueprint.

### The Six Theories

Sternberg and Berg (1992) identify six distinguishable theories of cognitive development—Piagetian, contextualist, or sociocultural (Vygotsky), information-processing (IP), neo-Piagetian, knowledge-based, and psychometric. The first four are detailed under their own headings in this chapter, whereas knowledge-based frameworks are mentioned more briefly. The reader is referred to Sternberg and Berg for discussion of psychometric theory. In cognitive development, what is innate, constructed, domain-specific, and systemwide, weave in and out through all theories and are not considered separately. The reader is referred to Sternberg (1999) for a current, exhaustive review of the controversies in the field.

## JEAN PIAGET: THE GENEVA SCHOOL

### Piaget's Goals and Methods

Piaget was the first to systematically study the “qualitative development of intellectual structures” (Flavell, 1963, p. 15). As a child, he had been a somewhat solitary prodigy with an interest in natural history; he published his first article, on a species of sparrow, at age 10. He discovered philosophy and psychology in adolescence and, after graduating in biology, worked with Simon in Paris, France, doing standardized intelligence tests on children. He became interested in the reasoning children of different ages used to explain their answers, both correct and incorrect, and proposed a connection between the development of logical thinking in children and the philosophical problem of knowledge—epistemology. He proposed that logic was the basis of all cognitive structures; he called this approach *genetic epistemology*.

Curious about the evolution of the adult capacity for logical thinking, he interviewed children of different ages about their ways of knowing, applying techniques from psychoanalysis and clinical psychology. This *clinical method* allowed more flexible interaction than did formal laboratory procedures. Piaget augmented his early experimental work by systematically observing his own children in their home and posing naturalistic tasks at intervals throughout their childhood. On the basis of these observations, and other, more formal ones, he made inferences about the development of children's capacity to think in terms of formal logic.

## Heredity and the Functional Invariants

Piaget believed psychological organization is the end toward which biological development is directed ( [Langer, 1969](#)). The notions of specific and general heredity were the biological bases of the theory. *Specific heredity* allotted to each member of a species a common physical apparatus that would function in a consistent, species-specific manner in an adequate environment ( [Ginsberg, 1985](#)). *General heredity* ensured what Piaget called the *functional invariants: organization and adaptation*.

### ORGANIZATION: SCHEMES AND STRUCTURES

Piaget proposed that humans are innately predisposed to organize whatever is taken in by the nervous system. In doing so the individual constructs *schemes* that are “the internal representation . . . of some generalized class of situations, enabling the organism to act in a coordinated fashion over a whole range of analogous situations” ( [Gregory, 1987](#), p. 696). Schemes combine to form ever more comprehensive mental structures organized in a stage-specific logical form. For Piaget a structure is [first] a:

unified *whole* whose parts can be identified only in relation to each other and their place in the overall structure. . . . [second] structural alterations are . . . orderly *transformations*. . . . Finally, structures are *self-regulating* . . . in that the nature of the whole is conserved by adaptive compensatory transformations among the parts ( [Boden, 1994](#), pp. 8–9).

### ADAPTATION: ASSIMILATION, ACCOMMODATION, AND EQUILIBRATION

Adaptation is subdivided into the complementary processes of *assimilation* and *accommodation*, by means of which the organism both creates and adapts to new knowledge. During assimilation, the organism adapts by organizing novel stimuli according to schemes already in its repertoire. Accommodation occurs when the cognitive apprehension of a novel stimulus causes the scheme to expand or alter its organizational structure. An infant with a scheme for grasping will attempt to grasp everything in sight, that is, to assimilate newly encountered objects as “things-to-be-grasped.” At the same time, different shapes and sizes of objects oblige the grasping scheme to enlarge and diversify so as to accommodate the variety of things-to-be-grasped. These processes work together with each new encounter until the novelty, which has temporarily unbalanced the cognitive organization, has been adapted to, a new equilibrium is established, and new knowledge has been created. The rebalancing tendency is *equilibration*, which is the fundamental developmental transition process within stages. Each new equilibrium structure is challenged and unbalanced by the next inassimilable novelty. Structure and novelty contend until a new equilibrium is achieved and the process reiterates. It is this *dialectical* process by which the child constructs and reconstructs her knowledge.

### Piaget's Stage Sequence of Development

Piaget proposed that cognitive development is a progression of structural changes. The child continuously adapts to and organizes events by assimilating and accommodating. Schemes are constructed, enlarged, combined, and coordinated to form ever more comprehensive and complex structures. These structures ultimately form a *structure-of-the-whole* that, in each stage, organizes experience across all domains of knowledge. The process is repeated in four stages as the old structure is folded into the newly forming one. Stage transitions—qualitative changes of cognitive structure—involve what Piaget called *reflective abstraction*.

By [which] process the subject extracts certain characteristics of [established cognitive] activities and uses them for new goals . . . in the sense of a . . . reconstruction and reorganization on the higher plane of what is transferred from the lower plane ( [Sinclair, 1992](#), pp. 224–225).

### THE SENSORIMOTOR STAGE (BIRTH THROUGH 18 TO 24 MONTHS)

The infant's first strategies for organizing her or his experience are such innate reflexes as grasping and rooting. During this stage, the infant transforms her or his reflexes into self-generated schemes of action. However, sensorimotor cognition is still nonrepresentational; it can only operate on an environment that is immediately apprehended by the senses and the motor system. By the end of this stage, the development of mental representations permits the transition from a physical to a psychological basis for thought.

The transformation of reflex to scheme begins as the neonate, enacting its reflexive organization, interacts with the environment. As something touches its palm, it grasps; as something touches its lips, it sucks, and so on. The more experience the child has of grasping, the stronger the grasping tendency. The stronger this tendency is, the more things the child will grasp and the greater the variety of things assimilated as “graspable.” The child accommodates by using different grasps for different things. As the grasping begins to differentiate, a scheme of grasping is constructed. Early in the second month, elementary schemes emerge as the infant experiences feedback from its own body. For example, although thumb-sucking behavior was innate, it is now repeated in an intentional way. Piaget called these first schemes *primary circular reactions*—“primary” because they involve the infant's own body, circular because an action–reaction loop is set up. During the middle third of the first year, the infant begins to interact with the external world in the same way, constructing *secondary circular reactions*. For example, coordination of two schemes, such as gazing and reaching, begins to occur. A random arm movement causes a mobile over the crib to move, creating an interesting visual effect. Soon the movements become less random. The infant's arm causes the mobile to move, and the pleasure generated causes the infant to do it again, establishing circularity. Toward the close of the first year, the infant begins exploring, rather than merely responding. In one celebrated sequence, Piaget interposed his hand between that of his infant son, Laurent, and a matchbox that fascinated him. At 7½ months, after earlier unsuccessful trials, Laurent actively attempted to push aside the obstructing hand to reach for the matchbox ( [Piaget, 1952](#)). Using the “means” of removing the obstacle, the hand, to attain an “end,” the matchbox, marks the beginning of complex intentional behavior and an appreciation of causality. During the first half of the second year, infants become more intentional in their explorations. Instead of seeking to reproduce exactly the previous effect, they deliberately vary the procedure so as to cause slight differences each time. Laurent, dropping different objects, varied the position of his arm on release each time. He would repeat the procedure exactly for a few trials, as if to confirm an observation, and then resume his variations. “This interest in novelty for its own sake is called a *tertiary circular reaction*” (emphasis added) ( [Ginsburg and Opper, 1969](#), p. 58).

Children begin to use things to represent other things in the last half of the second year, giving rise to pretend play and more sophisticated language use. Piaget called this new capacity the *symbolic (or semiotic) function*. The transition to symbolic mental representation begins with symbolic physical representation. Piaget's daughter Lucienne, at the age of 16 months, was presented with a matchbox containing a watch chain. She had learned from previous experience either to reach in and remove it or invert the box to make it fall out. This time Piaget narrowed the opening, so that she could not reach in, nor would it fall out. Lucienne stared at the narrow opening and opened her mouth wider and wider. Piaget saw this as a sensorimotor representation of her awareness of the opening and the space beneath it. “Soon after this phase of plastic reflection, Lucienne pulls so as to enlarge the opening. She succeeds and grasps the chain” ( [Piaget, 1952](#), pp. 337–338). Piaget posited that Lucienne's imitative mouth-opening was an analog of the box opening, and by *interiorization*, such sensorimotor analogs become thought as the child's interaction with caregivers provides symbols—words—to represent and express this knowledge; however, toddlers first assimilate language into their own schemas before using it in a conventional way. Piaget wrote of T. at nearly 18 months, “No more” meant going away, then throwing something on the ground and was then used of something that was overturned (without disappearing). He, thus, said ‘no more’ to his blocks” ( [Piaget, 1951](#), p. 218).

### Critique of Piaget's Sensorimotor Stage

Much work has been done on infant cognition over the past 20-odd years using techniques unavailable to Piaget. One is a type of habituation paradigm that electronically monitors eye gaze. The hypothesis is that an infant loses interest in, and stops looking at (habituates to) a stimulus if it has already formed a representation of it. For example, 7-month-old children who had been shown sequentially an array of different stuffed animals more quickly habituated to a novel one compared to other infants who had been exposed to the same toy or none at all. On this basis, the authors argue that very young infants have the cognitive capacity to form the category of “stuffed animals,” irrespective of particulars ( [Cohen and Caputo, 1978](#)). Because infants by 6 months are capable of thought, as memory, reasoning, and problem-solving, sensorimotor thought appears not to be the necessary developmental precursor of representational (symbolic) thought and “[Piaget's] idea that the capacity for symbolic mental representation only emerges. . . at 18 months appears to be wrong” ( [Goswami, 1998](#)).

### THE PREOPERATIONAL STAGE (2 THROUGH 5 TO 7 YEARS)

Achievement of the symbolic function ushers in the preoperational period that is representational but not yet logical. Piaget seemed to dwell more on children's cognitive limitations during this period than on their accomplishments because he was particularly interested in the development of logical thought.



## Object Permanence and Centration

At 10 months Jacqueline could retrieve a toy Piaget had twice put under a cover as she watched. However, if he subsequently put it under an adjacent cover, also as she watched, she looked under the first cover the moment the toy disappeared from view. At 18 months, playing the same game with a coin, Jacqueline watched as her father concealed the coin in his hand, moved his hand from A to B to C, and showed it to her, empty. She pushed aside his hand and searched sequentially in A, B, and, triumphantly, C. For her, the object now has permanence beyond sensorimotor apprehension.

In Piaget's theory, the achievement of *object permanence* is a Copernican event for the toddler in that he or she can now manipulate images and words internally; however, the relationship of one representation to another is not yet fixed, and magical thinking is the norm. "This kind of reasoning proceeds from particular to particular. *Centering* [which follows] on one salient element of an event, the child proceeds irreversibly to draw as conclusion from it some other, perceptually compelling happening" (emphasis added) (Flavell, 1963, p. 160). The child's understanding of things, once subject to its sensations and motor activity, is now dominated by its perceptions. Piaget argued that children tend to center their thinking on one dimension of a problem and have difficulty systematically coordinating it with another; in other words, they process only one dimension of a multidimensional stimulus. At 2 years, 7½ months, Jacqueline saw her younger sister in a bathing suit and cap and asked, "(What's the baby's name?)" Her mother explained that it was a bathing costume, but J. pointed to L. herself and repeated the question several times. But as soon as L. had her dress on again, J. exclaimed very seriously, "(It's Lucienne again,)" as if Lucienne had become a different person on changing her clothes (Piaget, 1951). Jacqueline had centered on the novel clothing and could not "decenter" so as to recognize her sister.

## Egocentrism

An important extension of centration is the controversial notion of *egocentrism*, the apparent inability to see things, at first perceptually and later cognitively, from another's point of view. In a famous experiment, Piaget stood children on the opposite side of a tabletop model of three quite different mountains. He asked each child to indicate how the child thought he, the experimenter, saw the mountains in relation to one another. Young preoperational children maintained that the experimenter saw the model as they did in spite of their 180-degree difference in perspective. It seemed as if the child believed her own point of view to be the only possible one.

Some have extrapolated perceptual egocentrism into the social-cognitive arena and suggested a concomitant conceptual and affective egocentrism that limits the preschool child's capacity for cognitive empathy—understanding another's psychological point of view. This skill is known as *social perspective taking* (Selman, 1980). Elkind (1970, 1978, 1985) proposed that interpersonal egocentrism accounted for limits in social cognition at different stages of development. He saw egocentrism as a way of measuring how a child is moving toward adult thinking along three parameters—distinguishing transient from permanent phenomena, objective from subjective phenomena, and universal from particular phenomena.

## Critique of Egocentrism

Donaldson (1978) challenged Piaget's assertions about early egocentrism because she believed the tasks posed were too abstract and unfamiliar. She cited a study in which preschool children were given a modified version of Piaget's "mountains" task without changing its logical structure. The task was presented in the form of a story/game of hiding a boy doll from policemen dolls standing at various vantage points. Under these conditions, decentration is found in children as young as 2 years pointing, among other things, to the power of a familiar framework such as a game or story as an aid to reasoning. Other studies show the capacity for both perceptual and conceptual perspective taking at 3 years (Borke, 1975; Zahn-Waxler et al., 1977).

The degree of egocentrism attributed to a preschooler thus depends on how the task is presented. If small variations in experimental procedure yield larger variations in the age at which a task is mastered, a Piagetian age-specific logical structure is harder to defend. By contrast, information processing methodology determines how many discrete operational steps are needed to solve a problem. Looked at this way, many logically similar tasks have very different cognitive demands. Sociocultural theorists would interpret the discrepancy between the "mountains" task and the "boy-hiding-from-policeman" version as supporting their developmental premise that children learn by having their problem-solving efforts "scaffolded" by helpful adults, often as narrative.

## THE CONCRETE OPERATIONAL STAGE: 6 TO 11 YEARS

What are the "operations" to which toddlers are "pre" and that for schoolchildren are "concrete"? A preoperational child, shown a collection of colored wooden blocks and asked if there are more wooden blocks than red ones, may reply that there are more red ones. A concrete operational child who can classify understands that "red blocks" is a subclass of "wooden blocks" and is not fooled. A preoperational child, told that Billy is taller than David and that Susan is taller than Billy, may not be sure that Susan is taller than David. A concrete operational child can arrange a series of things according to any one of their several properties, or, in Piaget's terms, can *seriate*. He can form one series of Susan, David, and Billy according to height and a different one according to weight, given the information. A preoperational child who sees a liquid being poured from a short, broad container into a tall, slender one is liable to center on the dimension of height and proclaim the latter to contain more liquid, even though he can see that the same amount of liquid went into both containers. A concrete operational child can mentally reverse the act of pouring and understands that the volume was conserved. For Piaget, a *mental operation* is "any representational act which is part of an organized network of related acts" (Flavell, 1963, p. 166). The three discussed in the preceding—*classification*, *seriation*, and *conservation*—are among the most important mental operations. With classification, recursive relations can be constructed, and concepts can be nested vertically in a branching hierarchy. Seriation permits things to be ordered horizontally in lists and rows according to their properties. Conservation stabilizes transformations because the child can "reverse the film" and mentally restore the transformed situation to its original form, conserving continuity. Once stability of operations has been achieved, the child's representations are connected systematically and logically rather than in the inconstant, intuitive way of the preschooler. As concepts begin to override percepts, the world ceases to be magical and takes on order and predictability. The limit of concrete operations is that children can only operate on things that are present or experienced.

## Critique of Preoperational and Concrete Operational Stages

Piaget's second and third stages have encountered less experimental disconfirmation than the first stage; however, many so-called concrete operational tasks can be performed at ages earlier than Piaget believed. For example, infants of 3 months demonstrate object permanence (Baillargeon and DeVos, 1991). Yet, to be fair, Piaget himself did not insist that his stages were tied tightly to particular ages. So too, in spite of his brief in favor of domain-general cognitive structures, he allowed that similar processes might come on line at different times in different cognitive domains or even within the same one (Chapman, 1988).

## THE FORMAL OPERATIONAL STAGE (11 YEARS TO ADULTHOOD)

An observer may ask a young subject to indicate whether what she (the observer) says about a solid-colored poker chip she holds is true, false, or uncertain. She may say, "Either the chip in my hand is green, or it is not green," or, "The chip in my hand is green, and it is not green." During some trials the chip is concealed, and during others it is not. School-age children tend to depend on the physical, visible evidence of the actual chip to judge the truth of the statements—the "greenness" standard. Adolescents can focus on the formal propositional nature of the statements themselves and recognize that the first statement is true, and the second false, irrespective of the color of the chip (Osherson and Markman, 1975).

This experiment captures an essential difference between the thought processes of a concrete operational school-age child and a formal operational adolescent. The adolescent's thought is not necessarily structured by the percept or the concept of the actual object in question. Where the younger child can form hypotheses about *what is* (*empirico-deductive* reasoning), the older can hypothesize about *what could be* (*hypothetico-deductive* reasoning). Formal operations are the culmination of cognitive development for Piaget because they form the backbone of logical, and, hence, scientific, thought; however, others believe that cognitive development extends beyond formal operations (see Summary).

## Summary of the Piagetian System

Piagetian theory is domain-general and constructivist; it draws attention to the commonalities of cognition across domains at a given age and the child as active constructor of her or his own experience. Piagetian theory also explains why children can solve some logical problems spontaneously at a certain age but not earlier, even with instruction. It does not explain discrepancies in performance on logically similar tasks in different domains nor does it account for improvements, with instruction, in performance on tasks that were theoretically beyond the logical competence of the child.

Piaget's work evokes a metaphor of the child as scientist, exploring her environment and constructing representations, based on logical principles, which organize her



experience. Representations are tested by their ability to assimilate novel experiences. If they cannot, accommodation helps form more adequate ones and cognitive equilibrium is restored. More complex representations and combinations of representations extend her range of interactions and increase encounters with novelty, and so on. Interestingly, this image of a somewhat solitary investigator with the world for a laboratory resembles Piaget himself as a child.

## General Critique

Is Piaget's theory still relevant? David Cohen believes "it is time psychologists ceased being so obsessed with him. It is not that he should be burned but that he should be buried" (Cohen, 1983, p. 152). By contrast, Mike Anderson (1992) contends, "as yet, nothing has replaced Piagetian theory as a general theory of cognitive development." In Margaret Boden's view, "Piaget was often wrong [and] often importantly right" (Boden, 1994). So, then, where was he wrong and where right?

Piaget's position that "knowledge is . . . actively constructed by a dialectical process of assimilation and accommodation" is widely accepted (Boden, 1994, p. xi). However, his bias toward *output systems*, that is, the child's actions on the environment, contrasts with current infant research on innate, domain-specific input systems (modules) by way of which an infant is biased toward processing some aspects of the environment while ignoring others. Such inborn cognitive constraints direct attention to the most developmentally vital qualities of the environment such as human faces and speech. Piaget did not dwell on the infant's predisposition for the uniquely human environment but built his edifice on logico-mathematical structures. "Of all the constructs in Piaget's theory, none has been subjected to more persistent criticism than the notion of a general logico-mathematical structure . . . acquired by an autoregulative process in which children reflect on the adequacy of their existing logical structures and assemble new ones by differentiating and reorganizing their elements. . . . As new data were collected it became apparent that children's intellectual processes were far more content, context and culture specific than he had suggested" (Case and Okamoto, 1996, p. 189). Simply because children's thinking could be parsed in terms of logical structures does not necessarily mean that is how children represent things to themselves. Arguably, "Piaget's theory was better equipped for representing the structure in the mind of logicians than the structure in the minds of young children" (Case, 1992a, p. 6). Finally, Nelson faults him for attending hardly at all to the vital role played by language in cognitive development (Nelson, 1996, p. 3).

## VYGOTSKY: THE CONTEXTUALIST APPROACH

### Vygotsky's Premises

The Russian psychologist Lev Vygotsky was born the same year as Piaget but died much earlier at 38. He was broadly educated in literature and philosophy and a committed Marxist. He worked during the early years of Soviet communism when the relationship between the transformation of the individual and that of the social order was a central philosophical and political issue. Where Piaget saw young scientists exploring the world of things, Vygotsky saw children immersed in an intricate sociocultural system based on a shared set of symbols for organizing and interpreting experience. In his view, the child developed by imitating and internalizing culturally based forms of cognitive mediation—words, for example—used by others in interaction with him. Therefore, "higher" cognitive processes appear first on the social-cultural (*interpsychological*) level and only then on the individual (*intrapyschological*) level. In other words, whereas "Piaget believed that intelligence matured from the inside and directed itself outward. . . . Vygotsky. . . believed that intelligence begins in the social environment and directs itself inward" (Sternberg, 1990, p. 242).

### The Genetic Method

The foundation of Vygotsky's thought is his *genetic method*. As in Piaget's genetic epistemology, the term "genetic" means to understand a phenomenon in terms of how it developed. For Vygotsky, therefore, before one can understand individual development, one must appreciate both the evolutionary origins of human biological organization and the historical origins of one's culture. On this basis, Vygotsky rooted cognitive development in three *genetic domains*—phylogenesis (evolution), cultural history, and ontogenesis (individual development)—each with its own line of development. Developmental change in each line is owing to the emergence of new forms of *mediation*; that is, *tools* and *signs*. For example, the emergence of tool use in primates marked a phylogenetic discontinuity where, from that point on, anthropology explained human development better than biology.

### Internalization, Semiotic Mediation, and Decontextualization

In Vygotsky's terms, both culture and cognition are *semiotically mediated*; that is, expressed through and shaped by, signs and symbols. Tools (implements) are used in the physical world to shape materials, and by analogy, psychological tools (signs) are used internally and shape the form of thought. Cognitive development takes place when the culture's psychological tools, mainly language, are shared with, and *internalized* by the child. Vygotsky examined the representational systems . . . needed to participate in . . . [social] processes—hence, his emphasis on the internalization of speech. In contrast, Piaget's focus on the young child's interaction with physical reality led him to examine the representational systems required to manipulate objects (Wertsch, 1989). For Vygotsky, internalization is "the process of gaining [internal] control over external sign forms" (Wertsch, 1989) or, less technically, "the ingrowing of lived experience into personal meaning" (Frawley, 1997). Developmental transitions occur with a change in the form of semiotic mediation. "Internalisation changes the complexity of what is done. . . . Culturally provided skills are generally more sophisticated than the skills which they replace" (Meadows, 1993, p. 241).

Vygotsky noted that each person is born with a set of biologically given *elementary* mental processes (memory, attention, and so on) analogous to the same capacities in other species. Cognitive development is the super session, in each domain, of elementary mental processes by *higher*, culturally based ones. "The purpose of higher thought is the control of thinking and action, and internalization and mediation are means to achieve this goal" (Frawley, 1997, p. 97). *Elementary attention*, for example, is stimulus-bound and reacts automatically to signals from the environment. The higher level, *voluntary attention* is semiotically mediated—regulated by *inner speech*—selective and self-activated. In the same way, elementary, holistic, *integral perception* becomes the higher, sequential, *analytic perception*. Similarly, *early memory* becomes *logical memory* with "the internalization of a language with which to represent logical problems" (Case, 1985, p. 72).

Higher mental functions are subdivided into *rudimentary* and *advanced* ones according to their degree of "*decontextualization* of mediational means" (emphasis added) (Wertsch, 1989, pp. 56–57). Decontextualization simply means the capacity to use cultural signs independent of any particular referent or setting. For example, when a child first learns to count, she can do so only with particular things; numbers (the "mediational means") cannot be manipulated without a concrete referent. This is a rudimentary higher process. Numbers must be decontextualized before she can understand  $2 + 2 = 4$ , irrespective of what is counted. Decontextualization allows the individual to attain *advanced* forms of higher mental functioning that, in the history of populations, provides the possibility of sociocultural change.

### Higher Thought: Social Speech, Inner Speech, and Metacognition

Prior to their coming together in cognition, Vygotsky believed thought and speech had independent origins. In the first two years of life, children use vocal activity for social contact and emotional expression, so-called "preintellectual speech." Nevertheless, during this period, they can problem-solve and act in goal directed ways unmediated by language—"preverbal thought." However, the child's language soon begins to include "syntactic and logical forms that have parallels in the child's problem-solving activity but are not linked to them in any systematic or useful way" (Meadows, 1993, p. 245). This *egocentric speech* (speech not directed at another) "is found in the transition from [social] to inner speech" (Wertsch and Tulviste, 1996, p. 57). It develops as children start to use language to help with problem solving and to:

talk themselves through problems or . . . count by using their fingers as aids. Finally such aids are internalized and problem-solving thought uses internal dialogue, while language can be used more to reflect on and develop thought than as a prop to support problem-solving (Meadows, 1993, p. 245).

In other words, the internalization of language gives rise to *metacognition*—thinking about thinking. Such higher thought allows one to reflect on how to use one's problem-solving capacities in a particular context, to frame a situation to oneself and to deploy one's cognition appropriately.

Vygotsky believed inner speech to be essential for higher thought. For example, when a child assembling a puzzle asks himself, "Now where does that piece go?" he is orienting his thought toward the next necessary step of the task, no doubt having internalized similar comments made to him as social speech by a helpful adult. Young children "must essentially talk to themselves using their speech to guide thought and behavior. With development, the self-regulatory function of language changes so that children can direct their behavior using inner speech" (Bjorklund, 1989, p. 141). It is important to emphasize that, by internalization, the child has appropriated social speech in the service of her or his own cognitive agenda every bit as much as her or his thinking has been assimilated into the cultural norms. Inner speech—in the sense of metacognitive self-talk—is the means of problem-solving central to cognitive behavior modification (Meichenbaum, 1977).

Social speech has an earlier, *indicative* and a later, *symbolic* function. Indicative speech is highly contextualized; adult, child, and object must all be physically present, as when an adult uses words to draw the child's attention to a particular object in their shared environment. By contrast, symbolic speech abstracts (decontextualizes) properties of an object so that it may be categorized with others of its kind. Further abstraction allows connections among categories and so on. To understand how this faculty develops, Vygotsky posed a block-sorting task to children of different ages. On the bottoms of a particular category in an assortment of blocks of different colors, shapes, and sizes, he wrote a nonsense word. He upturned one and asked children to sort those that they believed would have the same word on the bottom. When each child was done, he showed her the bottom of an unselected piece; either with the same word on it but differing in some way or one that seemed to resemble those selected but had a different word on it, and asked her to try again. He determined that young children tend to sort into idiosyncratic, unorganized heaps, older ones into complexes where there is an obvious relation among the blocks but not a "logical" one and finally, in the oldest, sufficient capacity for generalization and decontextualization allows sorting on the basis of true concepts. "Higher, uniquely human forms of psychological social interactions are possible only because thinking reflects reality in a generalized way" ( [Vygotsky, 1934/1986](#), pp. 11–12). "The stages in the development of *generalization* are directly linked to stages in the development of social interaction" ( [Wertsch, 1989](#), p. 96).

The internalization of speech and its decontextualization forms and transforms cognition. As the child internalizes, decontextualizes, and generalizes she gains control over her cognition. She becomes more aware that he is thinking and problem solving and others are too (see Theory of Mind). This awareness increases cognitive control, efficiency, and effectiveness. She can interact with the culture in ever more sophisticated ways as a storehouse for vast amounts of symbolically mediated knowledge. In doing so, she is not only acquiring knowledge but also developing cognitively.

### The Zone of Proximal Development

Vygotsky's model of development highlights the connection between knower and learner that begins when a child encounters a problem that he or she cannot solve. The adult has to set up an interpsychological space such that the child can, with help, learn to solve the problem. Internalization allows the child to solve the same problem the next time aided only by inner speech. Generalization allows him or her to approach other, similar, problems. Early on, the intersubjective space through which learning passes is context-bound and informal; later, with a shared set of linguistic meanings established, learning can be more formal and decontextualized, as in school; however, it may be structured,

the distance between the actual developmental level as determined by independent problem-solving and the level of potential development as determined through problem-solving under adult guidance or in collaboration with more capable peers is termed the *zone of proximal development* ( [Vygotsky, 1978](#), p. 86).

In order to understand the role of verbal mediation in . . . intrapsychological functioning, [Vygotsky] argued that one must examine speech on the interpsychological plane. In particular . . . the use of directives or commands by adults to regulate children ( [Wertsch, 1989](#), p. 92).

Experimenters who looked at the techniques of effective mother–child instructional interaction noted that the most successful mothers titrated their level of involvement to the child's apparent need. When the child began to fail, a little more instruction was added; as the child succeeded the parent stood back. "One sets the game, provides a *scaffolding* to assure that the child's ineptitudes can be rescued by appropriate intervention, and then removes the scaffold part by part as the reciprocal structure can stand on its own" ( [Bruner, 1983](#), p. 60). Scaffolding is useful for instruction and also provides a narrative structure so that, in a clinical setting, a child can be helped to become aware of what she knows but could not say. Inner speech is a form of self-scaffolding. For example, suppose a 6-year-old girl tells her father that she has misplaced her boots. Although he does not know where they are, he can help her to reconstruct her activities throughout the day until the moment when she remembers where she left them ( [Wertsch, 1988](#)). Even without the necessary information, the father had a systematic strategy for accessing it. The child had the information but, as yet, no strategy. Next time she misplaces something, she may say to herself, "Now, let's see, where did I have it last?"

Because an adult and a child operating in the zone of proximal development often bring divergent situation definitions to a task setting, they may be confronted with severe problems of establishing and maintaining *intersubjectivity*. The challenge to the adult is to find a way to communicate with the child such that the latter can participate . . . in interpsychological functioning and can eventually come to define the task setting in a new, culturally appropriate way ( [Wertsch, 1989](#), p. 161).

Creating an intersubjective field with a child also happens to be one of [Hirschberg's \(1966\)](#) "necessary functions" of the child psychiatrist.

### CRITIQUE OF VYGOTSKY

[Ceci \(1996\)](#) faults contextualist theories in general because they inadequately account for individual intellectual differences. Nevertheless, although some disagree with Vygotsky as to whether internal structure is the product of social interaction or precedes it, few have dismissed the role of adult–child interaction in cognitive development. Vygotsky's developmental transition mechanisms entail co-construction between adult and child and internalization by the child: Cognitive structures do not have to originate primarily by self-organization. In this way, Vygotsky and other contextualists explain, quite economically, how children in any given culture come to similar cognitive stances at approximately the same age. *Individualist* versions of cognitive development invoke biologically based invariant structures (Piaget) or innate modular structures (Fodor) to account for such convergences.

### Thought as Narrative

Jerome [Bruner \(1990\)](#), for decades the major American exponent of the sociocultural position, views the child as a natural maker of meaning and, thus, an apprentice storyteller. He was dismayed that the "cognitive revolution" had reduced cognition to computation and that the computer metaphor had centered development on the private "processing of information" rather than the public and interactional "construction of meaning." He proposed a "cultural psychology" to be "organized around those meaning-making and meaning-using processes that connect [humankind] to culture. . . by virtue of participation in culture, meaning is rendered *public and shared*" ( [Bruner, 1990](#), pp. 12–13). In Bruner's view, the computational model left no room for intentionality without which the individual cannot experience herself as an agent and without a sense of *agency* she or he will not have a sense of personal meaning. Bruner concerned himself not with objectified *behavior* but intentional *action*, specifically, with "action situated in a cultural setting, and in the mutually interacting intentional states of the participants" ( [Bruner, 1990](#), p. 19). These interacting intentional states form the culture and provide an interpretive system for the making of meaning. The form of this interpretive system is *narrative*.

Narrative implies a sequence of happenings, told from someone's point of view, that explains events in terms of the intentional states of the actors. It is a culture's way of interpreting events, including *noncanonical*—extraordinary—events: It is both everyday conversation and the nightly news. Narrative "mediates between the canonical [expected] world of culture and the more idiosyncratic world of [one's] beliefs desires and hopes" ( [Bruner, 1990](#), p. 52). People make meaning as narrative both by innate predisposition and cultural facilitation. There are "certain classes of meaning to which humans beings are innately tuned and for which they actively search" ( [Bruner, 1990](#)). "Social understanding, however abstract it may become, always begins as praxis [enacted experience] in particular contexts . . . the child learns to play a part in everyday (family drama) *before* there is any telling or justifying or excusing required" ( [Bruner, 1990](#), p. 85; [Dunn, 1988](#)). Margaret [Donaldson \(1978\)](#) and others showed how logically competent very young children can be when propositions are embedded in a story with familiar elements. Similarly, some have proposed that children younger than 4 years do not have a "theory of mind"—a representation of what another person may be thinking—because they do not seem to understand deception in stories told them by an experimenter ( [Leslie, 1987](#); [Perner, 1991](#)). However, Chandler found that, if children were participants in the deception, they showed an awareness of others' false-beliefs between 2 and 3 years ( [Chandler et al., 1989](#)). Others dispute this ( [Perner, 1991](#), p. 311).

As the infant interacts with its caregivers, Daniel [Stern \(1995\)](#) suggested that she or he constructs a network of representational formats that he calls *schemas-of-being-with* [another person]. These schemas are in the form of perceptions, concepts, sensorimotor events, event sequences, and affects (as *temporal feeling shapes*, representations of instances of rising/falling affective experiences). Initially they do not form a whole but

from the interplay, coordination, and integration of these lower-level processes, a more global mental event emerges: an emergent property of the mind, which has coherence and sense in the context in which it emerges ( [Stern, 1995](#), p. 89).

Stern calls this the *pronarrative envelope*:

a narrative-like mode of thought that concerns motivated, goal-oriented behavior. . . . When the [infant's] motive or desire is enacted in an interpersonal situation, it creates, subjectively, a narrative-like structure. As the motivated event moves in time toward its goal, it generates a dramatic line of tension,



which is an essential temporal feature of the narrative-like structure . . . as well as a narrative-like mode of perception ( [Stern, 1995](#), p. 89).

Stern's elegant model, of which this is the merest sketch, shows how self representations emerge from interpersonal interaction, are organized in narrative form, and provide the foundation of each individual life narrative.

So then, both the culture as a whole and children as individuals organize experience as narrative and the two levels of meaning-making interact recursively. The culture teaches a child to tell a good story as part of growing up. Most responses to questions in the to-and-fro of the family are, and are expected to be, in the form of a story, often to justify one's actions or displace blame onto the nearest sibling. Katherine Nelson described young Emily's evolving narrative competence between 18 months and 3 years of age ([Nelson, 1989](#)). Time and again, her evolving speech seemed "impelled by a need to get things organized in an appropriate serial order, to get them marked for their specialness to take some sort of stance on them" ([Bruner, 1990](#), p. 89). "In time she imports another genre into her narratives—problem-solving" ([Bruner, 1990](#), p. 94). This last is a direct challenge to the Piagetians and information processing theorists for whom problem solving is the foundation of cognitive development.

## Language and Cognitive Development

Katherine [Nelson \(1996\)](#) contends that virtually all theories of cognitive development underemphasize the vital role of language:

In each [theory] the independence of cognition from language has been maintained. There seems to be an implicit assumption that language is [only] a communicative tool . . . [it] is assumed to exist as a separable system either . . . internally as a mental organ, or . . . externally as a means of expression and reception of language-neutral "information." . . . the increasing acceptance of the modularity of mind has the effect of shunting language off into its own impenetrable module ([Nelson, 1996](#), p. 4).

Nelson proposes that any firm distinction between communication and representation is misleading. Beyond the most basic formats, representation is in the form of language: One does not have a "higher-level" representation and then reconfigure it as language, language is a necessary component for *any* reflective cognition.

In her *experiential view*, she proposes that infants prioritize the organization of what is presented predictably and/or intensively in their human environment. Nelson calls these early organizational structures *situational models*, which are "generalizations of pattern and reorganizations of experience" ([Nelson, 1996](#), p. 6). These models provide a context for action in classes of situations, informed by an appraisal of the "fit" between the model and specifics of the situation at hand. As specific situational models accumulate, the child begins to construct a *world model* that ever more comprehensively guides his actions across situations. Nelson calls these generalized models *mental event representations* (MERs) that are "individual constructions of social situations." Individual constructions, however, are not enough. "The basic mammalian individual representation system . . . [is] . . . solipsistic and egocentric" so, absent social influence on developing MERs through interactions in language, they will remain unaligned with those of others and ineffective as guides to action in a human environment.

Although Nelson draws much from Vygotsky, she does not agree that the mind is originally a social construction. Rather, it begins as an individual mind as the child constructs his or her world from direct experience but becomes ever more socialized and acculturated with development. The young child enters the social-cognitive world by *participatory interaction* with other children and adults, "taking part in activities without a full understanding of what the activity was about or how it was structured" ([Nelson, 1996](#), p. 19). As the child grows, his individual mind becomes ever more consonant with those of others as he compares and aligns his constructions with them and forms his representations as language. Nelson calls this process *collaborative constructionism*, distinct from both the individual constructionism of Piaget and the social constructionism of Vygotsky.

## THE INFORMATION-PROCESSING APPROACH

### The Child's Mind as a Computational Device

The third contemporary explanation of cognitive development is that of information-processing (IP) theory. Information-processing psychology is a part of the larger field of *cognitive science*, a new discipline based on computer modeling and comprising artificial intelligence (AI), psychology, linguistics, neuroscience, philosophy, and anthropology. The central hypothesis of cognitive science is that, "thinking can best be understood in terms of representational structures in the mind and computational procedures that operate on those structures" ([Thagard, 1996](#), p. 10).

Information-processing theory has only recently offered explanations of children's thinking and therefore is a newcomer to the family of developmental theories. Information-processing theory is not tied to the work of a single pioneer investigator, so it is not as unitary as Piaget's or Vygotsky's. Information-processing theories of development, "focus on the information that children represent, the processes that they use to transform the information, and the memory limits that constrain the amount of information they can represent and process" ([Siegler, 1991](#)). The goal is to construct a comprehensive model of the mind—a cognitive architecture—that accounts for the "sort of general information-processing capacities a mind must have in order to do the many things it does" ([Stillings et al., 1987](#), p. 17). The mind is seen to be, essentially, a problem-solving device, and cognitive scientists have constructed computer-based cognitive architectures to model human thought ([McClelland et al., 1986](#); [Newell, 1990](#)). Among many other intriguing innovations are IP models of Freud's Oedipus complex ([Fischer and Watson, 1981](#)) and Piaget's object permanence ([Franklin, 1995](#)). The developmental task of IP theory is to account for the enormous increase, from birth to maturity, in the complexity of a person's knowledge organization and processing capacity.

### Computational Transformation: Data, Information, and Knowledge

In the world of artificial intelligence, *data* are simply "facts without context in a form . . . that can be entered into a computer" ([Thro, 1991](#), p. 76). Once entered, "Human organization and interpretation give data context and meaning, producing *information*" ([Thro, 1991](#), p. 76). If information may be distinguished from data by meaning, knowledge may be distinguished from information by longevity and purpose: *Knowledge* is the "representation of facts (including generalizations) and concepts organized for future use, including problem-solving" ([Gregory, 1987](#), p. 410). In summary, the transition from data to knowledge involves increasing contextualization and more sophisticated rules of application.

A data-processing system—in our terms, a cognitive architecture—must be able to transform input into an internal representation it can operate on; that is, to *encode*. It must be able to hold new information "on screen" for appraisal and transform it into knowledge by connecting it with prior knowledge, in addition to the storage capacity to keep the new knowledge for future use. By analogy, in order to learn, children must first be able to transform data into information by encoding them as mental representations. Children then must hold the representation in active memory while retrieving relevant knowledge from their accumulated store. The new information is brought together with existing knowledge, and if it is deemed important to the ongoing life of the child, the information will be stored with related knowledge for future use. The input system is then freed up to take in new data.

Information-processing theorists and Piagetians agree that children actively modify their own cognition, but disagree as to how. The former analyzed standard Piagetian tasks, for example, the balance-beam task, in terms of the problem-solving steps required instead of analyzing their logical form as Piaget did. In this task children of different ages are asked to predict which side of a balance beam will go down as weights and their distances from the fulcrum on each side are varied. The child's performance is measured in terms of processing capacity relative to *processing demands*—the number of discrete cognitive operations—of the task. The older the child the better is the ability to predict correctly as variations of weights and distance become more complex. These age differences suggest that many tasks that shared a similar logical form imposed quantitatively different processing demands. Many cognitive architectures have been constructed to explain how children go about solving such problems; a sample is presented in the following.

### THE CLASSICAL PROCESSING MODEL

In 1956 [George Miller](#) wrote the landmark paper, "The Magical Number Seven Plus or Minus Two: Some Limits on Our Capacity to Process Information." He argued that

humans are limited to remembering approximately seven items—plus or minus two—in their immediate memory. However, by *chunking* items into composite units, a large amount of information could be contained in seven units (italics added) ([Miller, 1989](#), p. 276).



For example, it would be too difficult for most people to repeat back the 13 digit series 7472462121945 without a strategy. However, if one associated 747 with the aircraft, 246 with the first three even numbers, 212 as the boiling point of water in 8°F, and 1945 as the end of World War II, the original 13 units become 4 and the task is manageable. Chunking is a

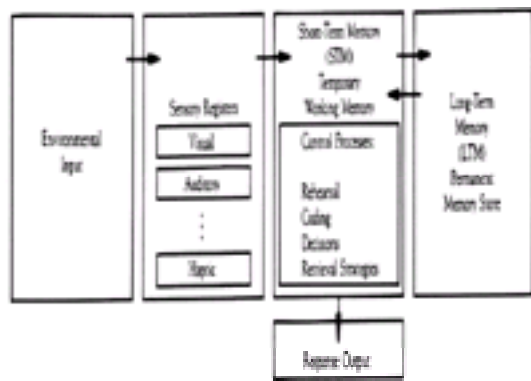
continuous, automatic, experience-based learning mechanism . . . that operates when impasses are resolved . . . [and] . . . converts goal-based problem-solving into long-term memory. . . . Chunking does not just reproduce past problem-solving; it transfers to other analogous situations . . . [it] . . . applies to all impasses (Newell, 1992, p. 430).

Miller's paper gave the newborn movement a name and delineated its central metaphor-processing capacity. He identified "immediate memory" as the locus of information processing and proposed chunking to be the principal strategy for increasing available processing space. Chunks are aggregates of related facts, concepts, or percepts. Chunks become larger and more complex with experience and are often hierarchically integrated, one inside another, like Russian dolls, so as to take up less processing space. Development may be seen as the increasing capacity of "immediate" or *short-term memory*—now refined and expanded as *working memory*. If so, is this owing to biological maturation or increasingly effective and sophisticated strategies to chunk information? Clearly, one's general knowledge base and specific knowledge of memory strategies influence this capacity (Chi et al., 1988).

#### THE DEVELOPMENT OF PROCESSING CAPACITY

Development in IP models is toward faster processing and greater organizational complexity of cognitive architecture. A cognitively mature person can manage more information per unit of time and deal better with complex and ambiguous information than an immature one. Infants cannot handle complex information as well as older children because they would have to "attend to and interrelate more pieces of information than their working memory capacities can handle" (Flavell et al., 1993, p. 85). With experience, elements of a task are chunked into aggregates and processes automatized, which allows more attention for conscious, reflective thinking and problem solving.

Let us follow an item of information through the system according to a version of this classic model (Atkinson and Shiffrin, 1968). The stimulus, for example an auditory or visual sensation, passes first into a modality-specific *sensory register*—"echoic store" for sound, "iconic store" for sight—where, in milliseconds, it is scanned for pattern recognition, tagged as relevant, and transferred into *short-term memory*. In an adult, it may stay there up to 15 to 30 seconds in the form of five to nine (seven plus or minus two) units. (It may stay longer if some deliberate strategy—a *control process*—is used to keep it active. One such process is "rehearsal," wherein a person might repeat the new information to herself or himself until it is either used or retained.) This new information may: (a) be interpreted as insignificant and forgotten, (b) cause a behavioral response, (c) be stored in *long-term memory*, or (d) remain in short-term memory and undergo further processing. These possibilities may be represented as a flow diagram (Fig. 12.1).



**Figure 12.1.** Flow of information through the memory system. (From: Atkinson RC, Shiffrin RM: *The Control of Short Term Memory*. Scientific American, Inc., 1971. All rights reserved.)

In the classic model, the place where new information is held, awaiting further processing, is simply short-term memory. This concept has recently become the more comprehensive *working memory* (Baddely, 1986). Briefly, it consists of an attention-like, central executive in the dorsolateral prefrontal cortex that manages both an articulatory loop system associated with the reception of phonological information and the production of speech, and a visual-spatial scratch pad for encoding images (Eysenck, 1990). Working memory is the principal arena of cognition and its processing capacity determines the sophistication of problem solving. Changes in this capacity account for much development in many IP theories (Case, 1985).

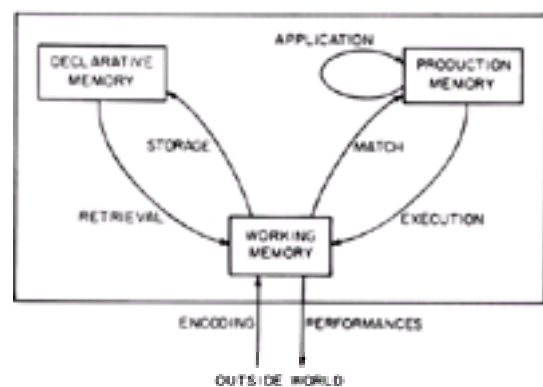
#### SEMANTIC ORGANIZATION: NETWORKS AND PRODUCTION SYSTEMS

Information processing also may be rendered as a series of *production systems*, as in a computer program (Anderson, 1983; Newell, 1990). Production systems are rule-based mental representations in the form of condition-action ("if . . . then") statements stored in long-term memory. If condition A is held in working memory and matches a rule for action in long-term memory, then the unit acts to produce an output that in turn may trigger another production system, and so on. For example, a student reading a text may bring into operation the production system "If the sentence is important, then underline it." This may be followed by a superordinate rule such as, "If the book isn't yours, don't mark it up." There can be a great many of these nested within one another to make up a "program" of great recursive complexity. A more current version of the flow-diagram representation described in the preceding is a network type of architecture also based on production systems. One such is the propositional network of Newell and Simon (1972) called SOAR (Franklin, 1995; Newell, 1990); another was developed by Anderson (1983), which he called adaptive control of thought (ACT).

Anderson proposed an architecture based on two fundamental types of knowledge—*declarative knowledge* and *procedural knowledge*. Declarative knowledge is *knowing that*; it is knowledge of facts and concepts, such as the date and significance of an historical event. It may be stored as language-like representations—propositions—or perception-like representations—images (Stillings et al., 1987, p. 18). Procedural knowledge is *knowing how* to do things, such as read a book or ride a bicycle. It contains elements of declarative knowledge that have been *proceduralized*. That is, copies of elements of declarative knowledge have been integrated into production systems so that one can access this information from production memory automatically and instantaneously rather than consciously searching declarative memory each time the information is needed. Once proceduralized, however, retrieval of these declarative elements requires conscious, focused attention; that is, *controlled* processes. Procedural knowledge operates by *automatic* processes that are unconscious and make no demands on working memory (Schneider and Schiffrin, 1977). Developmentally, the more we proceduralize our knowledge, the more we can do simultaneously and the more complex the tasks we can manage.

As in the preceding, let us follow an item of information through this system. To begin with, an external stimulus or an internal computation is encoded in working memory, which, in ACT, is not separate but designates the currently active part of declarative memory. Information may enter declarative memory as a proposition, image, or representation of a sequence of events. The declarative system is made up of propositions ("I need to study tonight to pass tomorrow's exam") and production systems form the procedural component ("if invited out tonight, then decline").

For a production to apply, the clauses specified in its condition must be matched against the information active in working memory. This information is part of the system's *declarative* component . . . [and] must be retrieved from long-term memory (Anderson, 1983, pp. 10–11) (Fig. 12.2).



**Figure 12.2.** A general framework for the adaptive control of thought production system. (From: Anderson JR. *The Architecture of Cognition*. Cambridge, MA, Harvard University Press, 1983, p. 19.)

If more than one rule may apply, as in the preceding with the “underlining” dilemma, preference may be given to the one that most closely matches the goals currently in working memory (therefore, underline), or because whenever a rule is applied successfully it is strengthened and more likely to be called up again, the prohibition against defacing library books might override the immediate goal (therefore, do not).

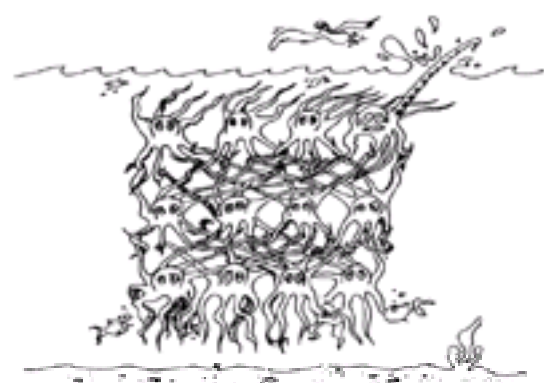
Knowledge is represented in declarative memory as a network of *nodes*, and relations among them, or *links*. In Anderson’s scheme, “The nodes of the propositional network stand for ideas, and the linkages among the nodes represent associations among those ideas” (Best, 1989, p. 249). Retrieval of ideas or images from long-term memory involves *spreading activation* (Collins and Loftus, 1975; Quillian, 1968).

Activation controls the rate of information processing . . . Activation spreads through the declarative network along paths from original sources to associated concepts. A piece of information will become active to the degree that it is related to current sources of activation (Anderson, 1983, p. 86).

The “firing” (or not) of a node influences the ongoing direction of the activation. Particular nodes may be more or less easily involved in this chain reaction, depending on the relevance of their content to the current situation and the closeness of their association to already activated nodes.

### NEURAL NETWORKS AND CONNECTIONISM

Anderson’s production system model has been criticized as too “computerized” and remote from the way people actually think (Gardner, 1985). In ACT, as in the classical model, “sets of symbols are moved about from one memory store to another, and are processed by explicit rules applied in sequence” (Eysenck, 1990, p. 66). This “top down” view has been called *representationalism*, and it is contrasted with *connectionism*, which is “bottom-up” (Frawley, 1997, pp. 68–86). Initially, it seemed promising to build a model of cognition based on rules and symbols. However, when programmers first tried to set up software to do things that brains seemed to do easily, they discovered that the simpler the task for a human the harder it seemed for a computer. A program for college-level calculus was easier to write than for grade-school math and it was even more difficult to write one to manipulate an infant’s blocks. So too, when programmers tried to write rule-based programs based on human experts’ experience-derived knowledge in their field, they found that the experts could not articulate their rules to solve any but the simplest problems; they seemed to operate more by a combination of intuition, prototypes, and rules with multiple exceptions than formal logic. These setbacks moved some cognitive scientists to look to the structure of the brain to inform the design of their cognitive architectural software. These revolutionary cognitive architectures are called *neural networks* (Fig. 12.3).



**Figure 12.3.** A whimsical depiction of the layers of a neural network. (From: Kosslyn SM, Koenig O: *Wet Mind: The New Cognitive Neuroscience*. New York, The Free Press, an imprint of Simon and Schuster, 1992. Reprinted with permission.)

The basic design of a neural network is quite simple; a layer of input processing units, or neurons, a layer of connecting neurons and a layer of output neurons (Fig. 12.3). Data are taken in and passed to the connecting layer that has a myriad of interconnections among its elements.

Each neuron takes in signals from other neuronlike components, adds them up, and decides on the basis of the answer whether to send out a signal of its own. In a way the neural units . . . are analogous to people in a jury talking amongst themselves, trying to influence each other to decide one way or another. . . . (Allman, 1989, p. 12)

At some point, output ensues. The relative success or failure of the output in solving the problem at hand constitutes new input and particular connecting pathways are strengthened (*weighted*) or weakened as a consequence (connectionism). In terms of Hebb’s Law, “neurons that fire together, wire together” (Hebb, 1949). Like humans, the system learns from examples, by trial and error; however, it seems likely that infants come into the world with many preweighted structures.

Knowledge is represented as the sum of all these connections at any given moment. It is therefore *distributed*, that is, spread throughout the network; there is no central processor nor distinct memory bank. Unlike a modular system whose components are cognitively impenetrable, all components of such an *interactionist* system are mutually accessible. So too, most or all elements of the system are necessarily active at the same moment as activation spreads among millions of neurons simultaneously, that is, the system works in parallel rather than in sequence. Given these characteristics one of the most developed connectionist architectures is called *parallel distributed processing* (PDP) (McClelland and Rumelhart, 1986). By entering lists of verbs and their past tenses, Rumelhart and McClelland (1986) “taught” a neural network system to change the present-tense form of a verb to its past-tense form and found that, “though it wasn’t designed to do so, this neural network made the same kinds of mistakes that children make when they are learning verbs” (Allman, 1989, pp. 36–37). In other words, “connectionism says that the nature of the brain really matters” (Lakoff, 1995, p. 123).

Neural networks explain better than conventional, rule-based architectures how we make inductive inferences and generalize. As the number of input neurons is virtually limitless all sorts of subtle contextual variations can be encoded so as to enormously increase the capacity to adapt to shifting circumstances and act on incomplete information. Connections resemble rules in that antecedent circumstances influence subsequent ones but the influence is virtually indeterminate because one can never be sure, from instance to instance, how all the inputs will add up. Connections are probabilistic rather than deterministic; rules impose hard constraints, connections soft ones. Parenthetically, neural networks also undergird applications of “fuzzy logic,” a new form of artificial intelligence that allows nonhuman systems essentially to program themselves, taking as input partial truths and incomplete information the processing of which has hitherto been the unique property of the human mind (Kosko, 1993; McNeil and Freiberger, 1993).



Connectionist models may explain more precisely Piaget's concepts of assimilation and accommodation.

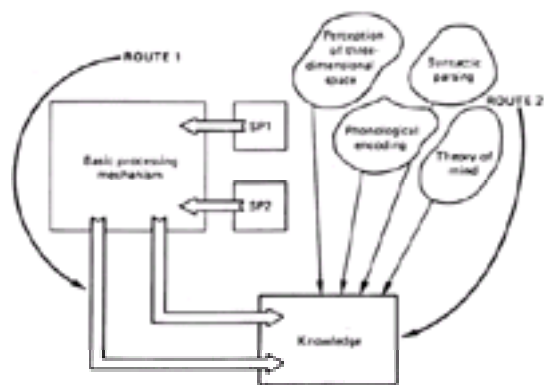
Assimilation can be interpreted in terms of the tendency of an interactive network to settle into the most appropriate of its stable ( *attractor*) states . . . when input is presented to it; in Piaget's language this is the schema to which the experience has been assimilated. Accommodation can be interpreted as the changes in activations as well as weights that occur in order to assimilate the experience . . . these processes can actually be designed into networks and observed under a variety of scenarios ( [Bechtel and Abrahamsen, 1991](#), p. 271).

For a detailed and current overview of neural networks and cognitive neurodevelopment, the reader is referred to [Green and associates \(1996\)](#), [Johnson \(1997\)](#), or [Spitzer \(1999\)](#).

#### MIKE ANDERSON'S MINIMAL COGNITIVE ARCHITECTURE

Australian Mike [Anderson \(1992\)](#) has proposed a "minimal cognitive architecture" that accounts both for individual differences in intelligence and cognitive development on the basis of an interaction of generality and modularity in mental functioning. We encounter a similar synthesis in the neo-Piagetian model of Robbie Case.

He begins by noting that individuals differ in data processing speed, measured neurophysiologically as inspection time (IT), reaction time (RT), and average evoked potential (AEP). Average evoked potential is derived mathematically from electroencephalogram (EEG) tracings, whereas IT and RT are measures of how much time it takes subjects to make choices on the basis of rapidly presented sensory stimuli. These measures may represent a sort of general neuronal efficiency and all correlate well with IQ and other measures of cognitive ability. A person's relative performance on cognitive tests remains more or less constant throughout life so that differences in processing speed are likely biologically based. Anderson postulates a wired-in *basic processing mechanism* (BPM) that carries out general computation and varies in speed from person to person. This would account for some individual differences in intelligence ( [Fig. 12.4](#)).



**Figure 12.4.** A diagram of Mike Anderson's minimal cognitive architecture. SP, specific processor. (From: Anderson JR: *The Architecture of Cognition*. Cambridge, MA, Harvard University Press, [1983](#), p. 107.)

On the other hand, there are some things that everyone can do about the same, irrespective of IQ, and that computers find very difficult. For example, virtually from birth, all can extract the features of three-dimensional space, encode phonologic information and organize our own and others' utterances as language. These abilities do not correlate with processing speed, and Anderson suggests these are, like the BPM, part of our genetic inheritance; however, unlike the BPM, they do not vary in efficiency but operate in a modular, all-or-none fashion. They come on line at different points with age.

People differ in their talents and, often, a particular talent is associated with others: Some people are better at art, others at math, and if they are good at math, they are likely to be musical. Indeed, during World War II, musicians were put to work as cryptographers simply because they were handy but they turned out to have unusual gifts for unraveling codes. Standard IQ tests recognize such differences by sorting cognitive abilities into two piles—verbal and performance. Correspondingly, Anderson proposes two *specific processors*, which are "function-specific knowledge-acquisition mechanisms" ([Anderson, 1992](#), p. 118). One such generates problem-solving algorithms for verbal/propositional input (SP1) and the other for visuospatial input (SP2). Specific processors are *experience-dependent* in that they need experience to develop as processing mechanisms. They must "borrow" from the general computational capacity of the BPM to process information so their development is also dependent on the BPM's processing speed. By contrast, modules are *experience expectant*; that is, they need only exposure to an experience, perhaps during a sensitive period, to unfold. Knowledge is acquired either by thought-mediated learning (Route 1, [Fig. 12.4](#)) or by the age-related activation of phylogenetically given modules (Route 2, [Fig. 12.4](#)).

Finally, there are things that some cannot do irrespective of intelligence. For example, dyslexics, even those with a high IQ, cannot decode certain types of language-based information. In some cases, there may be a fault in SP1, in others a defect in the module for phonologic encoding. These possibilities are not mutually exclusive as there are types of dyslexia that are better explained as processing problems and others better explained as pure or modular defects.

For Mike Anderson, "the maturation of modules is the primary cause of cognitive development" ([Anderson, 1992](#), p. 207). In his scheme, development happens either by: (a) the acquisition of new competencies, or (b) the elaboration of knowledge ([Fig. 12.4](#)). For example, the maturation of language-related modules brings a new set of processing competencies to all.

Suddenly having access to linguistic representations will result in a discontinuity of development that is unrelated to individual differences in intelligence. However, elaborating one's knowledge of the world by using this new competence—for example, by building vocabularies—will be constrained by the basic processing mechanism and the specific processors. Thus individual differences in intelligence will have an effect on the elaboration of knowledge but not on the acquisition of new competencies ([Anderson, 1992](#), p. 121).

#### Modules and Learning: Representational Redescription

Annette Karmiloff-Smith posed the following question to herself: Does human cognitive change affect all domains of knowledge simultaneously or does development occur in a domain-specific fashion? ([Karmiloff-Smith, 1992](#), p. 1). In brief, her answer is "both"; however, rather than hardwired, prespecified modules, she posits "a process of *modularization*. . . [whereby] . . . the mind becomes modularized as *development proceeds*" ([Karmiloff-Smith, 1992](#), p. 4). She acknowledges innate cognitive predispositions to preferentially attend to certain classes of stimuli; however, "this endowment interacts richly with, and is in return affected by, the environmental input" ([Karmiloff-Smith, 1992](#), p. 5).

When the innate predisposition is specified in detail, it is likely that the environment acts simply as a trigger for the organism to select one parameter or circuit over others [assimilation?]. . . . By contrast, when the innate predisposition is specified merely as a bias or skeletal outline, then it is likely that . . . [the environment] actually influences the subsequent structure of the brain [accommodation?] ([Karmiloff-Smith, 1992](#), p. 15).

*Representational redescription* (RR) is Karmiloff-Smith's hypothetical mechanism that accounts for the output of modules being used by more general processes to create representations accessible to other parts of the cognitive system [both within and beyond domains] by making implicit representations explicit. "A specifically human way to gain knowledge is for the mind to exploit internally the information that it has already stored (both innate and acquired), by redescribing its representations" ([Karmiloff-Smith, 1992](#), p. 15).

Representational redescription begins with *implicit* (proceduralized) knowledge. A child learning the piano begins by keying each individual note from the sheet music. With practice she learns whole phrases and strings them together until she can play the piece pretty much automatically. Karmiloff-Smith calls this *behavioral mastery*, represented as a connectionist network. Such basic networks are still quite rigid; the pianist cannot yet start anywhere in the piece nor perform variations on a theme. Only representational flexibility and control will permit these latter two skills to develop. In Karmiloff-Smith's terms, representational redescription has to take



place; that is, representations coded in a specified way have to be translated into a common language so that other cognitive components can “understand” them.

In this model, development takes place in three recurrent phases. In phase 1, learning is data driven—the child learns which note on the music sheet corresponds to which key on the keyboard. Phase 1 culminates in behavioral mastery as in the preceding. In phase 2, “internal representations become the focus of change” (Karmiloff-Smith, 1992, p. 19). The child begins to experiment with the music, which introduces new errors and inflexibility. Finally, in phase 3, external data and internal representation are back in synchrony and a new level of mastery is achieved that allows for creativity and thematic variation. The formats that undergird these observable phases are characterized as levels I(implicit), E(xplicit)1, E2, and E3. “These [formats] do not constitute age-related stages of developmental change. Rather they are parts of a reiterative cycle that occurs again and again within different microdomains and throughout the developmental span” ( Karmiloff-Smith, 1992, p. 20).

Information at level I is “not available to other operators in the cognitive system” ( Karmiloff-Smith, 1992, p. 20). Soon, the internal dynamics of the cognitive system, operating unconsciously, begin to generate level E1 representations, a “new compressed format of the procedurally encoded representations at level 1” (Karmiloff-Smith, 1992, p. 21) that have lost some specificity of detail. A zebra could now be more abstractly represented by a child as alternating black and white stripes allowing a perceptual/conceptual link to a (European) sign for a crosswalk depicting the same motif. A child without E1 representation would not understand why such a crosswalk is nicknamed a “zebra crossing.” However, at level 1, the child still does not have conscious access to the representation and cannot speak of it. (It should be noted that as one level emerges out of the preceding one, the original remains; the child can still recognize a zebra: Phenomena are represented at multiple levels simultaneously.) Representations at level E2 are in a similar code to those at E1, but the child is conscious of them. For example, spatially encoded E2 representations still cannot be verbalized but could perhaps be drawn or diagrammed. Finally, at level E3,

knowledge is recorded into a cross-system code . . . close enough to natural language for easy translation into storable, communicable form. . . some knowledge learned directly in linguistic form [may be] immediately stored at level E3 ( Karmiloff-Smith, 1992, p. 23).

Where there was a dichotomy between procedural and declarative knowledge Karmiloff-Smith has created a continuum.

This movement from implicit information embedded in an efficient problem-solving procedure, to rendering the knowledge progressively more explicit . . . is what I think development is about (Karmiloff-Smith, 1992, p. 17).

In RR,

cognitive change involves movement from the procedural to the computationally accessible (where procedures may be recombined or broken apart) and then to the consciously accessible (where the knower can consciously reflect on and manipulate procedures) . . . it is not the operations of the cognitive system that change, but the kinds of representation that are available to the system. In this view, cognition is a self-organizing system that is success-driven rather than failure-driven, as in Piaget's theory ( Nelson, 1996, p. 14).

### Information-Processing and Piagetian Models Compared

Cognition entails mental representation in both Piagetian and IP models, but representations are organized differently in each. Piaget attempted to explain achievements across cognitive domains by way of age-specific logical structures. However, Piagetians have been obliged to recognize that the theory did not account well for uneven performance across tasks with the same apparent logical structure—the phenomenon of *horizontal décalage*. For example, a child may be able to pass a conservation of number problem at age 5 but not pass a conservation of volume task until age 9 or 10. Information-processing theorists (and knowledge-based theorists) emphasize task and domain specificity; that is, the encoding of the task as of one type or another draws together and organizes knowledge to solve it. Cognitive representations have their origins and applications in particular domains. For example, the capacity to perform certain mental operations in the domain of mathematics does not mean that the child can transfer this capacity to the spatial domain: Décalages are discrepancies in domain-specific knowledge that may also reveal discrepant processing demands, concealed by apparent similarities in logical structure.

### Information-Processing and Sociocultural Models Compared

William Frawley (1997) charges that IP theory privileges explanations of cognition as private, internal computations and gives little account of how the computational mind is influenced by its real-life interpersonal situation. However, before anyone performs computations to solve a problem, he has to decide what the problem means in his situation and from what position, if at all, he will respond. This is called *orienting* to the problem and differs from *representing* to oneself the elements of the problem. Orientation is a context-dependent form of metacognition. That is, any instance of computation requires an appreciation of what is the most relevant computation in the moment: The determination of relevance is very hard for autistic children, as we shall see.

### Critique of Information-Processing as a Developmental Theory

The IP approach has been described as:

more a methodology (derived from task analysis) than a theory. . . . There is no agreed-upon nexus between the information-processing parameters and their biological substrate nor is there a description of the contextual mechanisms by which these parameters are initially shaped and later elicited ( Ceci, 1996, p. 197).

Patricia Miller (1989) noted that, although changes in processing capacity have been clearly described in adults as they go from novice to expert, development from child to adult might not proceed in the same way. Siegler does not see this to be a problem. Investigators, he believes, “can better understand the development of children's thinking when [they] know where that thinking is headed” (Siegler, 1986, p. 63). Miller also felt that there has been relatively little application of IP theory to the understanding of emotions, motivation, and social development. Recently, however, IP theory has been brought to bear on explaining autism and Case's neo-Piagetian theory, which has IP components, has been applied to social and emotional development ( Case, 1988).

## NEO-PIAGETIAN THEORY

### The Response to Piaget and Responses to the Response

Case (1992a) summarized the recent history of, and intellectual influences on, the most current theories of cognitive development. First, in reaction to the inadequacies of Piaget's “monolithic, universal and endogenous” system ( Case, 1992b, p. 10), other investigators—*empiricists*—began to study task-specific learning and avoided hypothesizing about how the development of different competencies might resemble one another. As one would expect, these projects explained some of what the Piagetian enterprise could not, but did not account for the similarities among children of divergent cultures and common, age-specific constraints on learning. This impasse prompted three, quite different, responses.

Nativists proposed that cognitive development was based on the maturing of neurologically based modules that biased children's attention to certain types of stimuli (Herschfeld and Gelman, 1994). In the strong version of nativism (Fodor, 1983), there is little development per se, simply the coming on line of more advanced modules. In the weaker version, modules change and develop under the influence of external data and internal differentiation and coordination of elements (Karmiloff-Smith, 1992).

*Knowledge-based* theory put forth a model of development similar to the novice-to-expert transition in adult learning. Development is learning-based, domain-specific, and cumulative. As children acquire more experience in a domain:

they begin to form new connections (whether conceptual, procedural or purely associationistic) among the basic elements of which the domain is comprised. These . . . lead to the integration of knowledge structures that were previously discrete. Once integrated, new knowledge structures or networks lead to new [problem-solving] strategies . . . and to new memorial capacities ( Case, 1992a, p. 14).

These knowledge structures are domain-specific conceptual networks that become ever more integrated until adult-level cognition is achieved ( Case and Okamoto,

1996). The distinguishing feature of knowledge-based cognitive developmental theory is that content influences process; the more one knows in a given domain the better one can think about it (Carey, 1990). Micheline Chi, a pioneer in this approach, did a fascinating study on a 5-year-old dinosaur expert demonstrating reciprocity between knowledge structures and learning strategies (Chi and Koeske, 1983). Similarly, it was supposed that an adult-like understanding of the inevitability, universality, and finality of death was achieved no earlier than 9 years of age in a sequence of age-linked stages (Nagy, 1948); however, Bluebond-Langner (1977) showed that leukemic children, who discovered with each clinic visit that fewer of their fellow-patients remained, acquired a full adult understanding much earlier.

Finally, *neo-Piagetian* groups proposed that there are domain-specific cognitive structures that develop more or less independently. Like task-specific structures, they are dependent on the context of learning and the child's previous experience. Like Piagetians, their application is broad within their domain, and they have common organizational features across domains. They are not based on logical operations (*syntactic* content) as are Piaget's, but on information (*semantic* content). Their pattern of development is hierarchical and recursive, and they are constrained, at any given age, by maturational limitations on the size of working memory and/or the speed of processing.

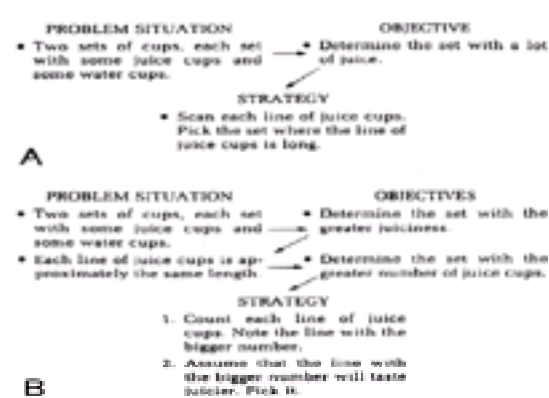
### Robbie Case

A well-regarded example of the neo-Piagetian synthesis is the work of Robbie Case (1985, 1992a, Case and Okamoto, 1996). Originally, Case supported the Piagetian notion of age-related, general cognitive structures evolving in an invariant sequence but also believed that problem-solving strategies, and working memory limitations had to be taken into account. Significantly, he incorporated affect as an important element in his approach. More recently (Case, 1992a, Case and Okamoto, 1996), his work has addressed the question of domain specificity and modularity. That is, is the mind best understood as a general IP device that stretches across domains to approach problems with the same tools or is it a system of phylogenetically shaped modules whose components are each dedicated to processing only particular kinds of input? Or is it both?

### EXECUTIVE CONTROL STRUCTURES

Case (1985) proposed that a child's cognitions are composed of schemes representing "recurrent patterns of stimulation" called *state representations*, and schemes representing mechanisms of transforming these states, called *operations*. The activation of a scheme is attended by a positive, negative, or neutral affect. From the second month of life, children have some control over and attempt to optimize their affective experience. They do this by organizing schemes to represent: (a) a particular, recurrent state (the *problem representation*), (b) an anticipated state of greater pleasure (the *objective*), and (c) the operation that will produce this transition (the *strategy*). These three elements are combined to form *executive control structures*, which are basic problem-solving structures analogous to production systems.

Case applied the concept of executive control structures to the well-known "juice problem" (Noelting, 1975). In this problem, children are asked to compare two lines of small cups, each line containing some cups filled with juice and others filled with water. One line has a large number of juice cups and a few water cups. The other has a small number of juice cups and the same number of water cups as the first, so that it is shorter in length than the other line. The children are asked which line, when mixed together, will taste "juicier." Four-year-old children guess (correctly) the first, because "there's a lot there." This is the earliest that children can solve this problem. At 6, children can go one better. In this situation, the lines of cups are made of equal length, and the same question is posed. By counting juice cups on each side, the children confidently pick the side with the greater number. The executive control structures assembled for the juice problem, for each age group, appear in Fig. 12.5.

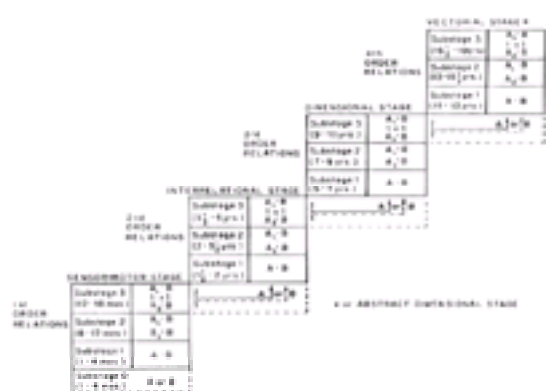


**Figure 12.5.** The executive control structure for solving the juice problem. **A:** Four-year-old control structure. **B:** Six-year-old control structure. (From: Sternberg S (ed): *Mechanisms of Cognitive Development*. San Francisco, WH Freeman and Company, 1984. Reprinted with permission.)

The 6-year-old child's structure is actually two hierarchically integrated structures. It is more quantitatively fine-tuned than the earlier one, more "dimensional." Case (1984) used this analysis to contrast the cognitive organization of 4-year-old children with that of 6-year-old children. For Case, the "4 to 6" transition is a stage transition, one of three linking his four stages. At 6, two different, earlier executive control structures have been coordinated and hierarchically integrated. As a result, a qualitative differentiation occurs wherein a new way of thinking emerges—a *quantitative dimension* of thought. This capacity to dimensionalize, as it were, applies not only to the task at hand; once this dimensional structure is established, the child applies it to a wide variety of tasks. Case calls this higher-level knowledge organization a *central conceptual structure*. It is central in that it can be applied to a broad range of tasks, analogous to, although perhaps less comprehensive than, Piaget's structures-of-the-whole.

### CENTRAL CONCEPTUAL STRUCTURES

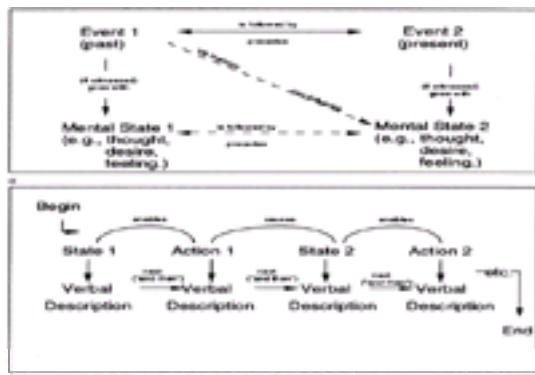
Executive control structures are task and culture specific as in the IP and sociocultural models, respectively. As in the Piagetian model, Case's are transformed according to a "general and universal" sequence of stages throughout development. This takes place stepwise in four age-specific stages (*sensorimotor*, *relational*, *dimensional*, and *vectorial*), as depicted in Fig. 12.6. Case's sensorimotor stage, like Piaget's, describes a physical, sensory representational system. The relational stage is characterized by a global, nonquantified worldview of "up-down," "big-little," "high-low," and the like. It is "either-or," "pass-fail." The dimensional stage is full of measurement and shadings of "this much" more or "that much" taller. The vectorial stage allows the representation of real or hypothetical quantity, which has, as it were, both magnitude and direction. It permits the representation of a multiplicative relationship between two forces.



**Figure 12.6.** The neo-Piagetian system of Robbie Case. (From: Case R: The whole child: Toward an integrated view of young children's cognitive, social, and emotional development. In: Pellegrini AD (ed): *Psychological Bases for Early Childhood Education*. New York: John Wiley and Sons, 1988, p. 159.)

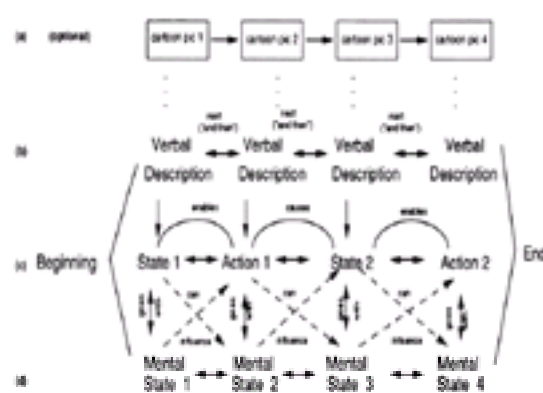


Case's most recent work further explicates central conceptual structures and details the mounting experimental evidence in support of the notion in three domains—mathematical, spatial, and social (Case and Okamoto, 1996). The last is presented in some detail because it is the most germane to child psychiatry. The research stimuli are narratives in four-panel cartoon format depicting event sequences and characters' reactions. Four-year-old children understand how mental states can be modified by external events (Fig. 12.7a). They also understand familiar event structures (scripts) (Schank and Abelson, 1977) and the normal sequence of their unfolding (Fig. 12.7b). However, they cannot yet bring the two "files" together and infer how the mental state of the actor in the script might influence the next event in it.



**Figure 12.7.** Central narrative structures at about 4 years of age. (predimensional/interrelational stage). A: The inner state schema that allows children to infer the thoughts and feelings of others. B: The event sequence schema that permits children to verbalize familiar social scripts. (From: Case R, Okamoto Y: The role of central conceptual structures in the development of children's thought. *Monogr Soc Res Child Dev* 61:10, 1996.)

Between the ages of 5 and 6 years, children become capable of answering such questions in an "intentional" fashion. . . . They have merged the two original files and have formed a superordinate structure (Fig. 12.8). . . . Children can now think of any familiar human activity as a coordinated sequence of events involving two components: a "landscape of action," which is the behavioral component of an event sequence, and a "landscape of consciousness," which is the internal or "intentional" component (Case and Okamoto, 1996).



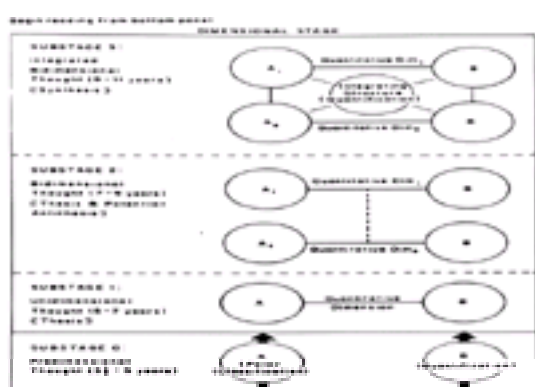
**Figure 12.8.** Central narrative structures at about 6 years of age (unidimensional stage) shows a merging of the two schemes in Figure 12.7. (From: Case R, Okamoto Y: The role of central conceptual structures in the development of children's thought. *Monogr Soc Res Child Dev* 61:11, 1996.)

Further changes take place between 6 and 10 allowing yet more coordination and integration of multiple and conflicting characters, mental states, and story lines. The central conceptual structures for mathematical or spatial cognition in 6-year-old children have the same general layout as the one for social cognition. This resemblance makes Case's point that, in spite of the domain specificity of each, there are common structural elements that gesture toward a domain-general process.

### The Mechanism of Development: Stage and Substage Transition

Stage transition occurs as executive control structures assembled during the previous stage, different in form and function, are hierarchically integrated. Stages are distinguished from one another by the kind of elements of which the executive control structures are made up. For example, when the counting element is integrated with the relative amount element, a whole new type of element—a dimensional one—has been constructed. This is the difference between a 4-year-old and 6-year-old child's approach to the juice problem.

Substage transition also occurs by integration of executive structures, but the integration need not be hierarchical, nor need the elements be different. Within each stage, there is a universal, age-specific sequence of three substages (*unifocal*, *bifocal*, and *elaborated coordination*) differentiated by the number of bits of information they represent, and how these bits are organized. Within the dimensional stage, substages unfold, coordinating two, then three, then four elements. This allows more complex dimensionality in that different sets of counting and relative amount units can be coordinated but does not produce a qualitative transformation as at the stage shift from relational to dimensional thought (Fig. 12.9). Stage transition (Fig. 12.6) is the natural outcome of substage transition (Fig. 12.9). Substage transition is the natural outcome of a growth in the capacity of working memory. Working memory is made up of space for executing cognitive operations and space for short-term storage and retrieval of relevant, but temporarily inactive, elements of the problem.



**Figure 12.9.** Substages within Case's dimensional stage. (From: Case R: The whole child: Toward an integrated view of young children's cognitive, social, and emotional development. In: Pellegrini AD (ed): *Psychological Bases for Early Childhood Education*. New York: John Wiley and Sons, 1988, p. 157.)

Logically, working memory can grow by either some sort of maturational increase in short-term storage space or becoming able to process more efficiently, so that less space is taken up by executing cognitive operations. Although practice does increase precision of representation and operational efficiency, it cannot entirely account for the process of development. Physical maturation, perhaps in the form of increasing myelination, may be ultimately rate limiting. Case (1992b) describes some promising correlations between his developmental timetable and frontal lobe EEG changes. The processes that move development along and their



synchronization may account for cognitive similarities between ages 4 and 10. First, “the tracts connecting the two hemispheres become increasingly myelinated . . . and the activity in the two hemispheres becomes better differentiated and integrated” ( [Case and Okamoto, 1996](#), p. 16). So too, the fibers connecting the frontal and posterior lobes show a strong pattern of dendritic growth between 4 and 10 and the two parts become more synchronous in activity. Second, schooling ensures that children in most cultures are taught mathematics and reading for about the same time each day; therefore, it is no surprise that reasoning about calculation and narrative keep pace.

[Case \(1992\)](#) stays true to his neo-Piagetian roots but incorporates elements from neo-nativism theory (modules), neo-associationist theory (domain-specific learning), and contextual theory (sociocultural influence on cognitive structures). He begins with the premise that children's cognition is innately modular and domain-specific including social, verbal, numerical, and spatial domains, at least. At the heart of children's conceptual systems is the central conceptual structure, which undergoes qualitative shifts at certain ages throughout development. Central conceptual structures are semantic networks that need domain-specific experience to develop both the nodes and links between them. Culturally specific experience, especially academic learning, becomes more important in filling these structures as the child becomes more cognitively advanced. Although there is no intrinsic limit to the growth of these structures in adulthood, there are maturational constraints on the rate of development of children's central conceptual structures. These constraints limit the capacity of executive structures and/or working memory. Finally, consonant with classical Piagetian theory, these structures are actively constructed by the child; grow by differentiation and coordination (integration) of existing structures; undergo qualitative changes by structural reworking; and become more powerful, abstract, and complex with development.

Case proposes a multilevel cognitive system, with levels ranging from the very general to the specific. . . . An increase in capacity ( *general* system-wide change), along with . . . particular experiences . . . leads to a change in the central conceptual structures. These . . . are at an *intermediate* level of generality; each is a representational system of a domain of knowledge such as number or space. These structures interpret specific tasks and affect the problem-solving procedures for these tasks ( *specific*-level change). By including systems that vary in generality, Case accounts for both the evenness and unevenness of cognitive development ( [Flavell et al., 1993](#), p. 14).

## Part 2: Applications of Cognitive-Developmental Theory to Affective Development and Clinical Practice

### A NEO-PIAGETIAN VIEW OF SOCIALEMOTIONAL DEVELOPMENT

#### Cognitive Dimensions of Early Affective Relationships

A neo-Piagetian viewpoint has been brought to bear on early object relationships and “the vital issues that must be negotiated at different stages of development, if these early relationships are to form a basis of healthy functioning in later life” ( [Case, 1988](#), p. 155). Two of the most important questions on which the thinking of psychodynamic psychiatrists and cognitive developmentalists is converging are: (a) How do children represent the affectively powerful transactions that take place with their earliest caretakers? and (b) How do these representations evolve in the course of children's development? For a start, it is assumed that the cognitive construction of both the social and inanimate world is subject to constraints based on the child's general level of cognitive development.

In particular, it is assumed the *level of relationship* children can encode, and the *complexity of event* they can understand which entails such a relationship, increases with age at approximately the same rate, and according to the same epigenetic schedule ( [Case, 1988](#), p. 162).

#### A Social-Cognitive View of Empathy Development

Parental empathy is absolutely necessary for the social, personal, and moral development of the child ( [Feshbach, 1987](#); [Fonagy et al., 1999](#)). [Hobson \(1993\)](#) cites an innate incapacity for affect perception and responsivity as the fundamental reason why autistic children cannot fathom the minds of others (see the following). Gender differences in the development of empathy ( [Hoffman, 1977](#)) are also fundamental to women-centered developmental theories in general ( [Jordan et al., 1991](#)) and moral ones in particular ( [Gilligan, 1982](#)). Empathy has both an affective and cognitive component: It is both a “vicarious affective response to another person” and “the cognitive awareness of another person's internal states” ( [Hoffman, 1984](#), p. 103). The cognitive aspect of empathy is somewhat like social perspective-taking, so that “empathy development must correspond at least partly to the development of a cognitive sense of others” ( [Hoffman, 1984](#), p. 108).

#### A NEO-PIAGETIAN STUDY OF EMPATHY

[Bruchkowsky \(1989\)](#) offers a developmental analysis of empathy based on Case's theory. In her study, children of 4, 6, and 10 years were presented with videotapes of peers enacting emotionally distressing situations. One situation depicted a little girl, Mary, and her dog playing together; shortly afterward, the dog was killed off screen by a car. The subjects were asked, “How do you feel?” “How do you think Mary is feeling?” and “Why is she feeling that way?” Analysis of facial expression during the showing was performed to cross-validate the verbal responses.

Bruchkowsky found no significant age differences in the accuracy of emotional attributions: The children did not differ in terms of what they appeared to feel, what they said they felt, and what they said the other felt. As predicted, 4-year-old and 6-year-old children could attribute only one affect, whereas 10-year-old children could attribute two or more. Bruchkowsky found a “stable developmental progression” in children's reports of why Mary felt as she did, that is, their *empathic cognitions*. Some 4-year-old children could not construct any explanation for their affective labeling of the other's reaction. They would say things like, “Mary was sad because it was raining.” These children were considered “pre-empathic.” Most 4-year-old children, however, were able to offer an explanation for Mary's feelings. They focused on external and salient features of the situation: “Mary was sad because her dog died.” These children could coordinate: (a) Mary's perceived affective reaction with “sad” and (b) “sad” with “her dog died.”

Most 6-year-old children referred to an internal dimension of the other's affective experience in addition to the external event: “Mary was sad because her dog died, and she will miss it.” Ten-year-old children focused on and coordinated two intrinsic dimensions of the other's affective experience: “Mary was sad because her dog died and she loved him and she will miss him.” Some offered as well a coordination of two opposing affects over time: “Mary was sad because at first she was playing with her dog and she was happy, then her dog was run over by a car and she was sad.” In summary, only external cues elicited a response at first, then one internal state was inferred, then two were inferred and coordinated in an elaborated manner. Age differences in children's empathic cognitions depended on a progressive increase in the capacity to coordinate from one to two to four elements. Case's theory, as an example of a cognitive developmental theory designed on the basis of problem solving with physical tasks, also can provide an account for the development of social relationships as well as their maldevelopment ( [McKeough et al., 1994](#)).

### THE DEVELOPMENT OF SOCIAL COGNITION

#### Social versus Nonsocial Cognition

Social cognition is distinguished from nonsocial cognition by the inherent intentionality of humans; people—including oneself—think, feel, and cause things to happen. Both oneself and others may conceal intentions and harbor ambivalences. Thoughts about others are virtually always informed by affects, the social context of interactions, and the need to separate one's own thoughts from those of others. This similarity between the knower and the known poses new cognitive challenges. Working out issues of cause and effect where one's way of thinking influences the way one's object of thought thinks and behaves is quite different from sorting out causality with crib mobile or hidden coins. Other human beings can establish “mutually intentional relations” with the child ( [Stern, 1985](#), p. 124ff).

It is this mutuality of conduct and communication that distinguishes social from merely physical events and that engenders (and requires) a special sort of understanding. The developmental study of social cognition is, in part, a study of this understanding as it grows and changes in the child ( [Damon, 1981](#), p. 159).

There is reciprocity between social and cognitive development: Cognitive skills mediate social interaction, which, in turn, provides the context for further cognitive development.

On the other hand, social cognition holds to many of the same developmental sequences as nonsocial cognition. For example, it proceeds from centration on particular external, perceptually salient events and objects of thought to decentration and the capacity to comprehend multiple interacting factors and internal, conceptual events. The child becomes able to conserve the identities of things and people—the self no less than others—over time and through transformations. Abstract and hypothetical thinking about self and others allows inferences about present relationships and internal activities, as well as hypotheses about future states

of the self, those of others, and relationships.

### The Structure of Social Perspective Taking

One form of social cognition is *social perspective taking*, which entails the capacity to conceptualize: (a) self and other and (b) the relationship between them. [Selman's \(1980\)](#) Piagetian model depicts a stage-developmental sequence of *concepts of persons* (e.g., undifferentiated) and *concepts of relationships* (e.g., egocentric). The capacity for social perspective taking has implications for the development of all social relationships, particularly empathic understanding and moral reasoning.

#### LEVEL 0: UNDIFFERENTIATED AND EGOCENTRIC PERSPECTIVES (3 TO 6 YEARS)

A preschool child's concept of persons does not differentiate objective-physical from subjective-psychological states of self and other. There is confusion between acts and feelings and intended and unintended behavior. Concepts of relations are egocentric in that the preschooler attributes his or her own perspective to the other person. The other is distinguished from oneself, but no commonality is established.

#### LEVEL 1: DIFFERENTIATED AND SUBJECTIVE PERSPECTIVES (5 TO 9 YEARS)

The child's concept of persons now differentiates the physical from the psychological. The other is seen as having a "unique, subjective, covert psychological life" ([Selman, 1980](#), p. 38), although it is seen as unitary, in that multiple feelings and internal conflicts cannot be recognized. The child's concepts of relations are one-way rather than mutual. It is the physical entity of a gift that makes someone happy, not the thought behind it; the intentionality of giving is not yet appreciated. Subjective attributions are made about another if the other is in a situation familiar to the child, by the reasoning "same situation equals same viewpoint."

#### LEVEL 2: SELF-REFLECTIVE/SECOND-PERSON AND RECIPROCAL PERSPECTIVES (6 TO 12 YEARS)

The later school-age child can see his or her own thoughts, feelings, and actions from another's perspective. This second-person perspective allows for reciprocity. However, although each can see self and other, he or she cannot yet see the relationship system between self as an entity distinguishable from the individuals who constitute it. Actions can be seen as different from intentions, and feelings can be seen as multiple and contradictory. Concepts of relations are reciprocal in that the child can differentiate self from other at a psychological level and not merely at the level of overt behavior. Attributions are made with uncertainty because the other is now seen as having an inner life distinct from one's own, and may dissemble.

#### LEVEL 3: THIRD-PERSON AND MUTUAL PERSPECTIVES (10 TO 15 YEARS)

Adolescence awakens the possibility of taking an observer position to the two-party reciprocity of the previous stage. This third-party perspective allows a clearer vision of the mutuality of individual perspectives. Adolescents "see themselves as both actors and objects, simultaneously acting and reflecting on the effects of action on themselves, reflecting upon the self in interaction with the self" ([Selman, 1980](#), p. 39). Thus, the observing ego is born. The concept of relations is a third-person or "generalized other" perspective from which one can coordinate, simultaneously, the perspectives of all parties in interaction, including the self and the social system in which all are embedded.

#### LEVEL 4: IN-DEPTH AND SOCIETAL-SYMBOLIC PERSPECTIVES (12 YEARS TO ADULTHOOD)

One gradually learns that the observing ego cannot always comprehend the complexity of internal relations, that something is always hidden. At this stage the idea of a personal unconscious emerges. When younger, a child may have been able to understand that someone may do something he does not want to; now the child can see that the other may not understand why. Relations are understood to go beyond the third-party stance of the previous stage and can be seen as systems of perspectives in relation to one another. This allows for a *generalized other* perspective of the interplay of multiple social forces and of the participation of self and other in the formation of this "generalized other" point of view. Coming full circle from infancy, one can recognize the possibility of an inner, unspoken mutuality among people.

[Selman and Schultz \(1990\)](#) applied the stages of social perspective taking to observations of children forming peer relationships and negotiating (or not) conflicts that arise in working together. If one child has something the other wants, the child at level 0 will act impulsively and, for example, try to take it away. At level 1, a child will likely try to influence the other, perhaps by a demand, to get what he wants. The effect of the loss on the other is not a consideration. At level 2, the child is aware he will have to influence the other's mental state and attempt to persuade or bargain. At level 3, the child can reflect on both their mental states and suggest compromises that may satisfy both. This work provides a useful guide to social-skills training and education.

### The Development of Emotional Understanding

[Nannis \(1988\)](#) has analyzed the sequential development of the ways that children reason about their feelings. For very young children, feelings are seen as inherent in the situation eliciting them. Getting a gift creates happiness in itself; the inner response of the child is not invoked as an explanation. The control of the feeling is equally external; it is the gift that causes a smile in the recipient rather than her sense of pleasure. Next, feelings are seen as internal, although located somewhat concretely within the child, almost like a body organ. The child experiences little control because the body is represented as a receptacle into which feelings are put. At the next point in development, feelings are understood as being diffuse within the person rather than concrete things in a concrete body. Feelings are now experienced as being influenced by the self; they have become conceptualized. Finally, the most complex conception of affects moves the self to center stage. "Feelings involve the regulation and integration of internal processes and external events. Feelings are part of a system with universal laws or principles. The self has gone from receptacle, to regulator, to mediator" ([Nannis, 1988](#), pp. 102–103). There are close parallels between this sequence and that of Selman's stages of social perspective taking.

The development of emotional understanding parallels the cognitive capacities of the child in other domains. Beyond that, the subject of emotional development is beyond the scope of this chapter. The reader is referred to [Saarni and Harris \(1989\)](#) or [Sroufe \(1995\)](#) for an overview.

### The Development of Moral Reasoning

In general terms, moral development involves increasing the complexity of one's perspective taking, sense of intentionality, and capacity for coordinating complex and often contradictory arrangements of factors. It also entails a diminishing of one's egocentrism, and a better capacity to interiorize one's own and others' motives. Piaget applied his ideas on the development of logical thought to the evolving capacity of the child to make moral judgments. He proposed stages of moral reasoning to parallel his stages of cognitive development. These were elaborated extensively by [Kohlberg \(1969\)](#) and critiqued trenchantly from a feminist point of view by Carol Gilligan. Moral development is discussed elsewhere in this volume (see [Chapter 19](#)).

### Cognitive Developmental Explanations of Autism

Ever since [Kanner \(1943\)](#) described autism, as a "primary disturbance of affective contact," the nature of the developmental deviation has been an enduring clinical puzzle; although autism has a genetic-neurological basis, the question remains by which psychological pathways it is expressed. There is much to support Kanner's intuition of a primary disturbance of affective development ([Hobson, 1989, 1993](#)); however, recent work makes an equally strong case for a primary disturbance of cognitive development ([Baron-Cohen, 1995](#); [Baron-Cohen et al., 1985, 2000](#); [Bruner and Feldman, 1993](#); [Frith, 1989, 1991](#); [Happé, 1991](#); [Perner, 1991](#); [Sternberg, 1987](#)).

#### THE NATURE OF SOCIAL RELATEDNESS IN AUTISM

Autism entails characteristic impairments in the capacity for social relations. Interpersonal communication, if present, is repetitive and self-absorbed or instrumental and literal, without regard for social context; that is, the *pragmatics* of communication are impaired. Pragmatics include tailoring one's speech to a particular listener, turn-taking in conversation, recognizing what is appropriate to say in a given context, staying on topic, and cueing the listener to changes in topic ([Baron-Cohen et al., 2000](#), p. 13). An example of impaired pragmatics is [Langdell's \(1980\)](#) finding that autistic children unable to modify their account of an event even when they knew



their listener had witnessed it. Particular autistic children may learn to communicate with others, but “an inability to participate in two-way reciprocal social interaction persists throughout the lives of autistic people” ( [Baron-Cohen, 1988](#), p. 384).

#### A PRIMARY DISTURBANCE OF THEORY OF MIND

[Baron-Cohen \(1988, 1989\)](#) believes that a disturbance of affective development is not the primary psychological mechanism in autism. Autistic children have emotional responsivity and they do not completely avoid eye contact or physical closeness with others; they simply do not seek it out as normal children do ( [Hermelin and O'Connor, 1970](#)). Autistic children can also show preferential attachment ( [Sigman et al., 1986](#)). Rather, [Baron-Cohen and colleagues \(1985\)](#) argue that, in autism, an innately impaired capacity to develop a *theory of mind* (ToM) is the primary deficit. A child who has a ToM understands that “people have minds and mental states, and that mental states relate to behaviour” ( [Baron-Cohen, 1993](#), p. 59). Without a ToM, children cannot represent to themselves the mental states—the acts of representing—of others and use that to predict their behavior: They cannot *metarepresent* and, as we shall see, cannot therefore have a ToM.

One of the precursors of ToM is *shared attention* manifested by *protodeclarative pointing*. The infant, knowing from early on how to track the mother's gaze, coordinates this with pointing toward something he wishes to draw to the attention of his mother—“Look at that!” The infant's aim appears to be to influence the mother's attention so that the two can share their interest in the same object. Shared attention implies that the infant realizes that his mother has a mental state that he wishes to align with his own. Shared attention is conspicuously absent in populations of 18-month-old children who have are likely to be later diagnosed with autism ( [Baron-Cohen et al., 1996](#)). Autistic infants, however, may display *protoimperative pointing*—“Get it for me!”—intended to influence the mother's behavior rather than her mental state ( [Baron-Cohen, 1995](#)). [Baron-Cohen and associates \(1992\)](#) used the CHAT—the CHecklist for Autism in Toddlers—to pick up deficits in shared attention, protodeclarative pointing, social interest, and pretend play in 18-month-old children at high genetic risk for autism. None of the controls failed on more than one of the key items, whereas four of the 41 high-risk children did. At 30 months these four were found to be autistic.

Children soon come to understand that they share with others the capacity for intentionality, the mind's inherent tendency to form mental representations. The shared attention of mother and child as they point together is based on their mutual representation of each other's intentions. Insofar as children can imagine what another thinks and apply this knowledge socially, they can be said to have a theory of mind. Mike [Anderson \(1992\)](#), Alan Leslie, and others have suggested that there may be a “theory of mind module” that is dysfunctional or absent in autistic people. Simon [Baron-Cohen \(1995\)](#) has proposed a stepwise developmental schedule of such a module (see the following).

Baron-Cohen and others devised an ingenious experiment to look at children's theory of mind ( [Baron-Cohen et al., 1985](#)). Normal children and children with Down's syndrome and autism, matched for mental age (MA), watched a scenario in which two dolls, Sally (beside a basket holding a marble) and Anne (beside a box) are depicted together. Sally puts the marble in her basket and leaves the scene. While Sally is “off stage,” Anne removes the marble from Sally's basket and puts it in her box. Sally returns, and the child is asked, “Where will Sally look for her marble?” Most of the nonautistic children answered correctly, “in the basket.” By contrast, 80% of the autistic children indicated the box (as would children under 4 years). The authors believed the results demonstrated that autistic children have a specific problem inferring the mental states of others, irrespective of their intellectual capacities.

What of the 20% of autistic children who passed the Sally-Anne ToM experiment? [Baron-Cohen \(1989\)](#) called the capacity to attribute belief to another “first-order belief attribution” and recognized that some older autistic children could do this. He devised a more complex study to explore “second-order belief attribution”; that is, the ability to represent to oneself what a second person believes a third person believes. The scenario was a toy village with a park, two houses, a church, and four people. John and Mary are in the park when the ice cream man comes along in his van. John wants an ice cream but has no money and has to go home to fetch some. The ice cream man assures John that he will still be at the park when John returns: Mary stays in the park. As soon as John leaves, however, the ice cream man decides to drive to the church to try and sell ice cream there. On the way, he passes John and tells him where he is going. Mary, who sees nothing of this, goes later to John's house, and his mother tells her that he has gone to buy ice cream. The question is then posed to the child, “Where does Mary think John has gone to buy ice cream?” To answer correctly, the child has to understand what Mary believes about where John believes the ice cream man is. None of the autistic children (mean age 15 years, mean nonverbal MA 10.7 years) answered correctly, whereas 90% of normal (age 7) and 60% of children with Down's syndrome (age 14, MA 6.8 years) got it. Because Baron-Cohen controlled for memory capacity, intellectual ability, and motivational factors, he postulated a specific delay in the development of a second-order theory of mind in autism.

To investigate the specificity of this cognitive limitation, [Baron-Cohen and colleagues \(1986\)](#) showed three disassembled four-panel comic strips to the same three groups of children. The children were told to make a story by sequencing the panels. Properly ordered, each strip demonstrated a specific kind of causal reasoning. The first (a “mechanical” story) depicted a balloon carried away from someone's hand, landing in a tree and bursting when punctured by a sharp branch. The second (a “behavioristic” story) involved a child going into a candy store, buying candy, and leaving. The third (a “mentalist” story) showed a child putting a piece of candy in a box then going out to play. Unseen by him, his mother removes the candy and eats it. The child returns, opens the box, and looks surprised ( [Fig. 12.10](#)). Despite a lower mental age, children with Down's syndrome could sequence the mentalistic story better than children with autism. The two groups performed the same on the behavioral one, and the Down's group worst on the mechanical one. Frith concluded that *mentalizing* is a different sort of reasoning from causal or behavioral reasoning. Mentalizing includes both the ability and the drive to “predict relationships between external states of affairs and internal states of mind,” not unlike intentional reasoning ( [Frith, 1989](#), pp. 156–157). The ability to mentalize implies a theory of mind.



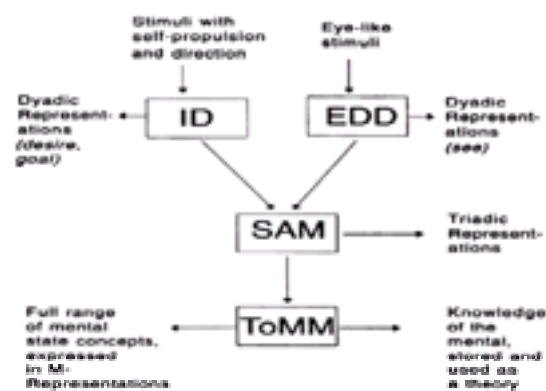
**Figure 12.10.** Cartoon strips illustrating the three types of causal reasoning. (From: Frith U: *Autism: Explaining the Enigma*. Oxford, UK, Blackwell Scientific, [1989](#), p. 164.)

#### DOMAIN SPECIFICITY: A THEORY OF MIND MODULE?

[Baron-Cohen \(1995\)](#) argued for a sequence of development that might give rise to a theory of mind module (ToMM). The centerpiece is the idea that the capacity for shared attention depends on a specific mechanism that he calls a SAM, a *shared attention mechanism*. The SAM has been assembled from two more basic, earlier mechanisms, an *intentionality detector* (ID) and an *eye direction detector* (EDD). The ID appears in infancy: It is a module that “will interpret almost anything with self-propelled motion or anything that makes nonrandom sound, as a query agent with goals and desires” ( [Baron-Cohen, 1995](#), p. 32). The EDD:

detects the presence of eyes or eye-like stimuli, it computes whether [another's] eyes are directed toward it or toward something else, and it infers from its own case that if another's eyes are directed at something then that organism sees that thing. ID interprets stimuli in terms of the volitional states of desire and goal. EDD interprets stimuli in terms of what an agent sees ( [Baron-Cohen, 1995](#), pp. 38–39) ( [Fig. 12.11](#)).





**Figure 12.11.** The mind reading system. (From: Baron-Cohen S: *Mindblindness: An Essay on Autism and Theory of Mind*. Cambridge, MA, MIT Press, 1995, p. 32.)

There is much evidence that infants of 6 months show an active preference for eye-gaze directed toward them. So too, children of 3 can easily figure out what another agent can and cannot see depending on that person's location vis-à-vis some target object; however, the infant can construct only dyadic representations using these two detectors. That is, they can only specify the relations between two entities—agent-self or agent-object. The SAM can specify relations among three things—agent, self, and object: a triadic representation. This permits protodeclarative pointing; the infant is now aware that she or he and mother are both looking at the same thing. Baron-Cohen argues that the ID, EDD, and SAM combine to form the ToMM that generates a theory of mind. In autism, this module does not come on line at the expected age because the failure to develop a SAM precludes the formation of a ToMM. (Note that there is a progression of complexity of representation from two uncoordinated skills, ID and EDD, to the triadic SAM. Case's view of cognitive development as an increasing capacity for coordination and integration nicely explains such a progression.)

Even this most refined theoretical version of ToM, elegant as it is, cannot be taken as proven. In its original form, ToM could not explain how symptoms of disturbed social cognition in autism were detected before age 4 when, presumably, the ToMM was well established. Baron-Cohen's explanation is that ToMM has precursors going back to shared attention seen between 9 and 14 months and that these have failed to progress to SAM much less to ToMM. Bailey and colleagues point out that:

it has not yet been possible to demonstrate a causal relationship between a candidate precursor and later ToM skills. The fact that children with autism show deficits both in precursor behaviours (for example, joint attention or imitation) and in ToM ability does not demonstrate a causal link (Bailey et al., 1996).

#### CRITIQUE OF THEORY OF MIND AS AN EXPLANATION FOR AUTISM

Since the first report of ToM failure in Baron-Cohen, Leslie and Frith's sample of autistic youngsters there have been several attempts to replicate their findings: Some did and some did not. Dahlgren and Trillingsgaard (1996) pointed out that, in earlier studies, between 38% and 60% of autistic children passed first-order false-belief tasks. Leslie and Frith (1988) discovered that success among autistic subjects at false-belief tasks correlated with verbal MA and chronologic age. There have been several other studies that report some autistic subjects as able to pass second-order false-belief tasks (Bowler, 1992; Ozonoff et al., 1991a). Ozonoff and colleagues (1991b) found that Asperger's syndrome (AS) children were better at false-belief tasks than high-functioning autism (HFA) children; another study found no difference.

The success of some autistic-spectrum children on standard ToM tasks has brought into question the specificity of ToM impairment as an explanation for the social cognitive deficits in autism. Nevertheless, impaired ToM remains a robust finding in autism but "is not universal since a small proportion of able children and older people with autism pass the tests" (Bailey et al., 1996). That said,

it is still an open question whether "theory of mind passers," who have IQs within the normal range, have developed theory of mind competence or whether they make use of some alternative strategy to solve the experimental tasks. The latter seems likely, since most of these individuals still seem to suffer profound social impairment in everyday life . . . among "passers" there is a small subset who may have genuine mentalizing abilities, and a larger subgroup for whom a [non-mentalizing] strategy seems a better explanation for task success (Bailey et al., 1996, p. 102).

However, explaining away what appears to be mentalizing in HFA or AS people on the basis of a putative "alternative strategy" risks the charge of circular reasoning.

Baron-Cohen and associates (1997) argued that standard ToM tasks were not subtle enough to capture their mentalizing deficits. Happé (1994) and Baron-Cohen's group (1997) report on two more advanced ToM tasks showing that HFA/AS adults show significant impairment in mentalizing. In Happé's task subjects read a short story about a prisoner of war being interrogated about the location of his army's tanks. The interrogators assume the prisoner will lie. In an effort to save his comrades the prisoner tells the truth. Subjects were asked where the enemy will look for the tanks and why. Happé believes this task requires a "third-order" ToM. Not surprisingly, HFA/AS subjects did worse than controls. In Baron-Cohen and associates' new ToM test, normal IQ HFA/AS adults were matched with patients with Tourette's syndrome and normals. The task was to identify, from a photograph of the eye area alone, complex emotions such as concern versus unconcern, serious versus playful, and so on. Baron-Cohen and colleagues have repeatedly demonstrated the unique significance of the eyes in mind reading by infants, toddlers, and preschoolers (Baron-Cohen, 1995). The HFA/AS subjects did significantly worse than either control group; these latter two did not differ. Nevertheless, Baron-Cohen still feels it would be easier to pass this test than to function in society where the cues are rapidly shifting. For example, all his HFA/AS subjects told him that going to movies was pointless because they cannot adequately keep track of characters' motives. Bailey and coworkers (1996) agree with Baron-Cohen that the ultimate test of one's social cognition is in everyday social interchange and even ToM task passers remain decidedly odd in ordinary settings.

Another complication in fathoming HFA/AS passers relates to the methodology of ToM studies. Charman and Campbell (1997) observe great variation among normal children in ToM tasks and "small changes to the wording or conduct of the task has . . . significantly affected performance." A recent test-retest study had shown only poor-to-moderate reliability within the same group of children given three versions of standard false-belief tasks over a 3-week span (Mayes et al., 1996). Children with general cognitive delays may also fail mentalizing (ToM) tasks (Yirmiya et al., 1998). However, Baron-Cohen argues that

people with autistic-spectrum conditions appear to have mentalizing difficulties for different reasons from . . . people with mental retardation or those who are blind or deaf, since a deficit can be revealed even with the highest-functioning individuals with an autistic-spectrum condition, in whom general comprehension problems can be ruled out (Baron-Cohen et al., 2000, p. 15).

He cited the case of an eminent mathematician with Asperger's disorder who failed a ToM task that normal children easily pass.

#### LANGUAGE AND THE NORMAL DEVELOPMENT OF THEORY OF MIND

Katherine Nelson (1996) points out that false-belief tasks rely on the child's understanding and use of mental-state words. Such understanding and use cannot be assessed without reference to the way in which these words are learned and deployed. Children of 2 and 3 years old can operate in a social domain with empathy and consideration of others before they can use mentalizing verbs with precision. Because they hear others use them, such young children can use "think" and "know" in speech but cannot explain the difference between them: The child may have the *language* of mental states before she has the *concept*. Two- and 3-year-old children are much more sophisticated in understanding the feelings and thoughts of others within their family relationships where it is familiar and their personal interests are at stake (Dunn, 1988).

Children of parents who use mental state words and concepts in conversation with them pass false-belief tasks earlier and have more advanced perspective-taking skills than those whose parents do not (Dunn et al., 1991). For example, deaf children of hearing (but not of deaf) parents have difficulties in understanding and signing mental-state words such as "feeling," "wanting," and so on (Peterson and Siegal, 1995). Deaf parents are skilled enough with signing that they spontaneously use mental state expressions in their communications; hearing parents who are less skilled in signing do not. These findings would support a knowledge-based explanation of, at least, some theory of mind attainments. Recently, Peter Fonagy proposed that mothers of infants who had a well developed *reflective function* (mentalizing capacity) as measured by the Adult Attachment Interview, were more likely to foster secure attachment in their infants. He further suggested that

development of a more mature reflective function is a central goal of psychotherapy ( [Fonagy et al., 1999](#)).

#### AUTISM AS A FAILURE OF NARRATIVITY

Jerome [Bruner and Carol Feldman \(1993\)](#) allow that there is a failure in the autistic child's theory of mind, but they propose a more fundamental deficit. They point to evidence that the autistic child

is incapable of (or highly resistant to) organizing interpersonal encounters into a canonical form that captures . . . regularities in the way in which people's intentional states are situated in typical situations, are expressed in typical action sequences, and require reciprocal response from those who are interacting with them ([Bruner and Feldman, 1993](#), p. 286).

If so, autistic children cannot create, with others, zones of proximal development and are deprived of the interpersonal scaffolding that promotes the development of internalized cognitive structures: In this case, the structures that enable one to construct a coherent self-narrative.

Structures of canonical sequences, or *formats*, depend on the capacity to consistently represent the other's mental state. Bruner and Feldman do not believe, however, that the innate capacity to intuit another's mental state, which they believe to be impaired in autism, entails a theory of mind. They suggest that a theory of mind might follow from the application of the child's innate predisposition to interact and develop representations of cultural formats. "Some autistic children could follow narrative sequences, even those involving intentional states, when such narratives were well scaffolded by pictures and stories told by others" ( [Bruner and Feldman, 1993](#), p. 287). They propose that intelligent children and adults reason their way through interpersonal situations as others would reason through a math problem; they "convert the personal world of intention-regulated social experience into an impersonal world of causally-driven events" ( [Bruner and Feldman, 1993](#), p. 288). In other words, going back to the reasoning task using comic strips (see the preceding), these people could often successfully apply mechanical or behavioral thinking to an intentional situation and so obscure the fact that they were not truly thinking in an intentional way. These children always seem "out of sync" in social exchanges because it takes more time to reason through interpersonal situations than to intuit a response.

#### SUMMARY: CHILDHOOD'S END

As a child develops cognitively, she or he can move beyond the observed to the inferred, beyond percept to concept, beyond concrete to abstract, and beyond intuitive knowing to conscious reflection. She or he becomes aware that events can have internal and symbolic meaning beyond their external and literal significance. As she or he invests events with meaning, the child can go beyond content to appreciate formal similarities, patterns, and processes. As the child reflects on patterns and processes, she or he can reflect on systems, including social systems. Older children can reflect on their own patterns and processes and how they relate to those of others and the larger social world.

As a child becomes aware of himself or herself as part of a larger world—call it decentration, disembedding, or social perspective taking—the universal enters the personal: The "I" becomes at once smaller and larger. With differentiation, the subjective "I" encompasses less of an ever more complex array of possibilities; yet, as a child internalizes personal experience, the sense of self grows in ways hitherto unimaginable. However, in each moment of transition, he or she may become aware that, in order to gain a new sense of self, an old one, usually quite comfortable, must be given up. This may be frightening; the more so because the child does not know why he or she feels anxious and caregivers often are equally puzzled. Yet the adults that form the child's "holding environment" must be intuitively if not consciously aware of these developmental anxieties and support the child in his or her journey from the global undifferentiated "I" of infancy to the smaller but more integrated "I" of true adulthood.

Development also entails an ever-increasing capacity to draw distinctions. Drawing distinctions sets up tensions that, when resolved, are replaced by new distinctions and tensions. Integrating the new into the familiar and transforming the familiar into the new, creating more complex familiarities, is the task of a lifetime. The dialectics of development—differentiation and integration, assimilation, and accommodation, being "a part of" or "apart from"—operate throughout the life cycle.

Later adulthood often brings awareness that life is more process than achievement. For this awareness to happen, Michael [Basseches \(1984\)](#) believes that formal operations, Piaget's last word, must be superseded by "dialectical" thinking. Formal thinking ultimately requires static entities and fixed relationships: It is designed to avoid contradiction. Dialectical thinking does not push for closure; it needs contradiction and creates a lifelong interplay of contrasting ideas ( [Carse, 1986](#)). By old age, the self may become less dependent on externals and integrity comes to rest in a sense that one's life is "the accidental coincidence of but one life cycle with but one segment of history; and that for [one] all human integrity stands and falls with the one style of integrity of which [one] partakes" ( [Erikson, 1959](#), p. 98).

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# 13 CHILD PSYCHOANALYTIC THEORIES OF DEVELOPMENT

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The central concern of the theory of child psychoanalysis is the understanding of emotional life—the experiences of love, hate, pain, pleasure, longing, guilt, and concern—and their representation in the child's mind. Psychoanalytic investigation focuses on how children develop into individuals with their own minds, feelings, and desires and how they become engaged with their parents and others who become important to them. The emphases of child analysis are the internal and private experiences of children, from the first months of life through adolescence; the ways in which bodily and environmental processes influence inner life and the child's perceptions of the outer world; the ways in which children represent their experiences and selves; and the mutual interactions between reality and fantasy, inner and outer, adaptation to the shared world of reality and appreciation of personal desires and needs. The theory of child analysis involves cultural and personal history—familial and social influences that have their origin before the birth of the child and that are conveyed to the child through the particular social expectations and caregiving of parents and society, as well as the enduring effects of experience and the continuities and discontinuities in the child's outer and inner life.

Psychoanalytic theories of development begin with the infant's somatic experiences and processes, bodily sensations, and ability to attend to and regulate the impact of internal and external stimulation. With maturation, these inherent functions become elaborated into increasingly sophisticated forms of organization of the child's perceptions of himself or herself and others. The physical requirements of the young infant and the parents' emotional investment in the infant's well being are paradigmatic for the unfolding relationships that serve as the context in which neurologically based systems unfold. At each phase of development, maturing biological demands and capacities assume meaning and find expression in the constructs, including the conscious and unconscious fantasies, that the child develops to make sense of himself or herself and the greater world. The child's functioning at any one point reflects the mutual interactions among many forces; in turn, the child's history and current functioning set the stage for the emergence of capacities and vulnerabilities in subsequent phases of personal development ( [Erikson, 1959](#); [Freud, 1966](#); [Klein, 1958](#)).

Throughout childhood, the processes of maturation and development reflect the interaction between biology and endowment—genetically transmitted characteristics, constitution, and the programming for physical change—as well as experience (in the family and broader social world) and the workings of the child's mental apparatus, as such. The charting of the mutual influences between psyche and soma and their representation in mental life provides a framework for understanding both normal development and illness ( [Freud, 1965](#)).

## DATA OF CHILD PSYCHOANALYTIC THEORY

Child psychoanalytic theories of development have grown out of clinical experiences and research with children in varied settings, including: (a) the home, well-baby clinics, school, and group living arrangements ( [Bowly, 1969, 1973](#); [Freud and Burlingham, 1973](#); [Hellman, 1962, 1983](#); [Mahler et al., 1975](#); [Stern, 1985](#)); (b) institutional settings ( [Provence and Lipton, 1962](#); [Robertson and Robertson, 1958, 1971](#); [Spitz, 1945](#)); (c) medical or rehabilitative settings ( [Burlingham, 1961, 1979](#); [Earle, 1979](#); [Fraiberg, 1968](#); [Freud, 1952](#); [Furman et al., 1968](#); [Moran, 1984](#); [Schowalter and Lord, 1972](#)); (d) the course of determining custody and placement disposition ( [Goldstein et al., 1996](#); [Solnit et al., 1992](#); [Wallerstein, 1996](#)); and (e) child psychoanalytic treatment ( [Anthony, 1980](#); [Bornstein, 1953](#); [Freud, 1945](#); [Frijling-Schreuder, 1969](#); [Marans, 1993b](#); [Meers, 1970](#); [Neubauer, 1987](#); [Sandler et al., 1975](#); [Target and Fonagy, 1994](#)).

Similar to Freud's earliest work, child psychoanalytic theories of development have evolved primarily out of a clinical perspective on infants and children with difficulties. Within the context of clinical care, psychoanalytic observations of children focus on the interplay of features of emotional presentation, cognition, modes of communication, physical activity; themes of play; and discussion and patterns of interaction. However, whether in the "unstructured" hours of ongoing psychotherapy or in observations of children in other settings, child psychoanalytic investigators generally proceed by careful observation of the ways in which the child's interests, concerns, and patterns of functioning unfold. They integrate the data with information from caregivers about current and past life experiences. The data are used to develop multiple hypotheses regarding: (a) implications of the child's biological "equipment" and its impairment on the tasks of development, (b) areas of strength and preferred modalities for adaptation, (c) specific developmental tasks and various ways in which they are negotiated, (d) areas of vulnerability in the path of development, (e) implications of various life events on the course of development, (f) the nature of unconscious conflict, and (g) defense activity and conflict resolution.

Psychoanalytic theories of development have evolved over the past decades in response to new observations and concepts. Using available observational and historical information, the psychoanalyst attempts to reconstruct a narrative history of the child's life—a portrait of the child's inner experiences, relationships, and modes of representing himself or herself and others. There is no one uniform, systematized theory in which all major concepts are formally defined; rather, child psychoanalysis as theory consists of a range of related theoretical perspectives. Although these share an orientation on inner emotional experience and developments, they differ in particular conceptual emphases to a greater or lesser degree. Relating these perspectives to a specific child or phenomenon may highlight theoretical divergences or may provide a sense of the complexity of development and emotional life.

## CATEGORIES OF EXPERIENCE

Psychoanalytic concepts of early development have long been organized according to a hierarchy of dominant body zones. The delineation of oral, anal, phallic, narcissistic, and oedipal phases was meant to map out processes of development from the infant's earliest dependence on the mother to the adult's relative independence and autonomy. The goal of these theoretical constructs was to provide a framework for organizing observations. However, the broadening scope of observations of the development of relationships, concepts about the self, cognition, and the like, highlighted a tension within the theory between the notion of continuity and discontinuity of experience. Although there are nodal points that mark out different experiences in different periods of life—reflecting a degree of discontinuity—the concept of "stage" as defined by body zones is limited when we observe the overlapping of psychological experiences from one period of life to another, or, rather, the underlying continuities. The goal of contemporary "phase theory" is to explicate major modes of psychological organization and changes from one epoch to another. In addition, the theory tries to describe and understand the personal and shared meanings of experiences and tasks that are salient in a given period of life, as well as those that continue to exert influences from earlier periods.

### 0 to 18 Months ("Oral" Phase)

Contemporary psychoanalytic theories focus on the ways in which biological processes in this earliest period of development come to have psychological meaning in the infant's rudimentary sense of the self as distinct from others. That is, how do the infant's bodily sensations contribute to the emergence of an internal,



psychological “self” that experiences the body and increasingly guides its actions?

During the first months of life, the mouth plays a central role in shaping the infant's earliest images of himself or herself in the context of the world. In addition to its role in eating and sustaining life, the baby uses his or her mouth—in the activities of sucking, licking, and biting—as a central organ of perceiving, regulating, and altering sensations. In the absence of hunger, the infant mouths fingers, toes, toys, pacifiers, and mother's breast to self-soothe, at once experiencing the various physical properties and decreasing his or her distress. Similarly, his or her crying when uncomfortable or making high-pitched squeals to attract mother's attention leads him or her to represent the communicative power of the mouth.

The experiences associated with orality—pleasure in the satisfaction of urges and discomfort when satisfaction is not immediately available—continue to be central to psychoanalytic conceptualizations about early development. In addition, the contributions of early infant research ( [Emde, 1982](#); [Klaus and Kennel, 1976](#); [Spitz, 1965](#); [Stern, 1985](#)) have focused on a broader range of inborn processes and their influence on the infant's earliest experience of the body and emergence of a “self.” The study of variations in the sensorimotor system (sight, audition, reflexes, muscle tone) and state regulation (sleep/wake cycles, quiet/alert periods, withdrawal, gaze, responsivity to comforting) have emphasized the contribution of these “nonoral” intrinsic factors to early patterns of mediating endogenous/instinctual or external/environmental stimulation ( [Mayes and Cohen, 1994](#)).

From the beginning of life, the infant's relationship with his or her caregivers organizes the ways in which bodily requirements, inborn processes, and constitutional and instinctual urges find expression. The mother's investment in the infant's bodily needs sensitizes her to the ways she can help diminish the potential for discomfort. Maternal involvement and intimate contact with the infant's body—feeding, cleaning, holding, cooing, and so forth—reflect the mother's earliest attachment ( [Bowlby, 1969](#); [Freud, 1974a](#); [Greenacre, 1957](#); [Klein, 1958](#); [Mahler et al., 1975](#); [Osofsky, 1995](#)); she “reads” the infant's cues according to the recognition of specific sources of pleasure and discomfort. In addition, the mother attributes emotional meaning to gross and subtle changes in the infant's presentation. This guides her response and progressively conveys to the child a sense of meaningfulness of her actions and affection. Inborn hypersensitivities to various stimuli—touch, sound, gastrointestinal, and the like—complicate the mother's task of learning how to understand and establish reliable means of responding to her infant's needs. The infant's neurologic maturation—for example, decreasing the prominence of colic or ease of startling and becoming distressed by noise—and the parent's increased experience often offset difficulties in the early parent–child relationship. It is important to note that the tremendous variation of inborn characteristics in state regulation may play as crucial a role in the infant's early adaptation as the contributions of possible conflicts the mother or father may have about various aspects of parenthood ( [Furman, 1992](#); [Kestenberg, 1956, 1980](#); [Winnicott, 1962](#)).

The parent's efforts to maximize the infant's comfort and to relieve his or her distress take over when the infant is not yet able to soothe himself or herself. By ensuring that the baby is not overwhelmed by discomfort, the parent contributes to the child's experience of effectance; *his* or *her* activities can make things happen that alter the child's own bodily sensations. Repetition of patterns of parental care, in turn, influences and reinforces those behaviors of the infant that promote pleasure and satisfaction of needs. Although diminishing physical tension (between need and gratification) and maximizing pleasure appear to be essential tasks during this period of development, experiences of frustration and discomfort are equally significant in the baby's maturation and development. In fact, the sequence of discomfort followed by relief is mediated, in part, by the absence and appearance of the caregiver and contributes to the infant's growing capacity to more clearly delineate between “me/not me.” The discrepancy between the experience of need and satisfaction may include nuances of interaction—failure to elicit a smile, a cuddle, soothing or playful tone of voice, or physical proximity, as well as hunger or physical discomfort from a soiled diaper. Beginning with the buccal mucosa as a junction between inner and outer, sensations associated with repeated sequences of coordinated activity—sucking, biting, licking, swallowing, looking, listening, touching—foster the infant's capacity to locate and identify the origins of percepts and contribute to the delineation of body boundaries. Although the infant's recognition of the mother's face, for example, is observable from the earliest weeks of life ( [Greenspan, 1989](#); [Mayes, 1989](#)), by the middle of the first year his or her response to her is no longer dominated by the satisfaction of bodily needs. With the appreciation of physical separateness, the infant links the pleasure associated with her ministrations to the person of the mother in her own right. Her presence itself is a source of pleasure and satisfaction: her absence is a source of anxiety. He or she will look at mother to see if a situation is safe or dangerous, and by this process of social *referencing* of the meaning of situations, the baby will be socialized into the family. He or she will learn about the parents' specific ways of responding (muting or exaggerated, calm or fearful), including how they deal with closeness and separation. The child's protests at being handled by others, his or her distress on seeing people other than mother (stranger anxiety), and his or her upset on separations from her stem from the infant's feeling that “not here” is “gone forever.” With the delineation of boundaries, the infant views mother as a bridge between himself or herself and the world. In her absence, he or she may appear panic stricken and “disoriented,” as if he or she had lost the connection with his or her newfound world and with emerging feelings of “self” ( [Fraiberg, 1959](#)). From within the dyadic relationship, how the mother acts on leaving and how she feels on return exacerbate or diminish the child's worries.

Games of peek-a-boo, experimenting with hiding and finding objects, and repeated experiences of parents coming and going help the infant on his or her way to establishing object permanence ( [Beebe and Lachmann, 1998](#); [Piaget, 1952, 1954](#); [Zelnick and Buchholz, 1990](#)), the ability to conceive that things continue to exist even when hidden, that “not here” is “maybe there.” The ability to mentally “hold on to,” or internally represent images of the parents in their absence contribute to the infant's exploration of the world beyond the lap as the child begins to stand, cruise, and then take his or her first steps. The toddler's ability to move away from the parents under his or her own steam provides a vivid experience of physical separateness from them well beyond the earlier activities of averting gaze or arching away from mother's body when held. Although needing periodically to check back with the parents or “refuel” ( [Mahler et al., 1975](#)), the toddling infant's explorations contribute to his or her inner definition of “self;” that is, what the toddler can do in relation to his or her body and the central figures in his or her life ( [Freud, 1965](#)). Increased dexterity, language acquisition, and rudimentary cause and effect thinking provide the tools for this research. Optimally, the young child's investment in these burgeoning capacities leads to the child's “love affair with the world” ( [Greenacre, 1957](#)), in which the pleasure of mastery facilitates the elaboration of these capacities. Alternatively, gross disruptions in the dyadic relationship—including prolonged separations between mother and child, mother's emotional unavailability because of severe depression, or physical abuse—may lead to the earliest expressions of psychopathology, such as failure to thrive, anaclitic depression, hypersensitivity to stimulation, and extreme wariness in interactions with others.

The child's psychological differentiation and separation from the parents is accompanied by tension, sadness, and anxiety as well; he or she may sometimes find “walking away,” or being walked away from, a painful experience. The young child often turns to a specific soft blanket or cuddly toy, specially invested with attributes of the mother, to hold and fondle in her absence. Unlike the real mother of separation, these transitional objects ( [Winnicott, 1953, 1965, 1971](#)) can be controlled and literally held on to by the toddler when the mother is too far away. The capacity to evoke mental images of the self and parents and memories of satisfaction of needs and possible sequences of events are crucial achievements in this phase and have broad implications for the young child's capacity to remember and anticipate interactions with the world. From suckling at the breast to first steps and from random smiling and cooing to words and conversation, the infant's developing organization of “self” as distinct from the body is matched by an accompanying elaboration of mental schemes—conscious and unconscious fantasies—of his or her relationship to the central figures in his or her life. The increased specificity of feelings, both pleasurable and frustrating, associated with the parents during infancy form a foundation on which internal mental structures and attachments to others gain complexity in subsequent phases of development.

### 18 to 36 Months (“Anal” Phase)

This phase of development obviously owes its designation to the role of elimination and toilet training in early childhood. The capacity to control defecation and urination and the demands of the parents to do so have been seen as paradigmatic for a range of tasks and struggles the child encounters with regard to his or her body, relationships, impulses, and fantasies.

In addition to the toddler's continued exploration of the world around him or her, heightened anal sensations in the period of development also promote the toddler's appreciation for the separateness between himself or herself and caregivers. Central nervous system maturation underlies the increased awareness of rectal pressure and anal sensitivity; psychologically, the child's attention becomes focused on yet another area of the body where there is an interface between inner and outer. In defecation and urination, the child experiences a complex process involving bodily feelings, self-control, action, and perception. He or she feels and sees how what is originally inside can become outside, along with a sense of what was previously hidden being public and shared. Defecating and urinating are no longer simple processes that occur in response to physical pressure; they assume meaning as sources of pleasure that the toddler can control.

The ability to label and identify parts of the body is concurrent with the ability to regulate pleasurable sensations through holding and releasing feces and urine; these capacities permit the elaboration of an internal set of representations of the self. A distance is established between the body and the self that experiences the body. As the child represents himself or herself as an object that can be perceived, the child naturally begins to compare his or her body with that of others. This capacity to compare leads to the child's appreciation of anatomic differences between the sexes and child and adult.

Recognition of separateness and specific differences in the body and abilities between the child and adult caregivers (and other children) is crucial in the child's development of “reality testing” and sense of self. In addition, this recognition is also a source of considerable conflict and anxiety. Under the sway of powerful sexual and aggressive desires, the toddler seeks immediate satisfaction of urges that his or her developing capabilities alone cannot always deliver. In addition to the

motivation provided by need satisfaction—seen throughout development—the child now experiences the satisfaction of his or her needs as related to his or her own degree of control and effectiveness. The child's awareness that he or she does not share the powers of the parents increases the intensity of the wish for effectiveness; awareness of dependency is often at odds with the wish for autonomy and omnipotent control. Parents are then frequently confronted with a toddler who is inconsolable about his or her inability to accomplish a given task or satisfy a need. The child may loudly protest what he or she can not accomplish alone and angrily refuse all parental attempts of assistance. The young child crumpling to the floor in tears is a poignant illustration of the struggle between the competing aims of independence and dependence. In this phase, anxiety experienced on occasions of separation from parents is an additional marker of the child's comparison of his or her competence with the parents' and his or her continued need to rely on them for care, safety, and affection. In earlier separations the infant was frightened and disoriented because of an inability to conceptualize the existence of the mother or father in their absence. In this later period of development, however, the child may believe that by his or her voracious demands, anger, frustration, and moves toward independent power the child has become the agent of his or her destruction. Until reunited with mother, the child imagines that she has gone away forever and feels completely alone, bereft, and guilty.

In this period, words can now substitute for actions; remembering simple sequences of events can help the child anticipate the immediate future, and increased coordination can broaden the range of pleasurable physical activities. These capacities promote the young child's sense of effectiveness and offer potential diversion from the intensity of needs and impulses that were previously satisfied only by the parents' immediate response. The child can now tell the mother that he or she is hungry instead of simply crying. And when told by the mother to "wait for a few minutes," the child may be able to mobilize defenses against feeling hurt and sustain the wait by rolling cars on the floor, knowing that "a few minutes" means not too long. Alternatively, the child may again use language to implore mother to hurry or crash the cars or other toys as a way of displacing or deflecting the anger felt toward her for not responding to his or her needs quickly enough.

Although the child is able to tolerate greater amounts of frustration, relative to earlier phases, parental demands to do more or wait just a bit longer introduce conflicts that further the cause of mediating between impulses and action. The child's fear of losing the parent as the result of destructive urges and the wish to please the parent and receive praise are powerful contingencies that often fly in the face of aims of immediate gratification and absolute autonomy. The toddler's alternation between compliance with and obstinate defiance of parental rules, and the attendant tantrums, expresses struggles between passivity and activity, love and hate. As parents expect more from a child in the areas of toileting, self-feeding habits, waiting for help, and attention, for example, they are setting goals for the child to reach. When they are achieved, the child is proud of himself or herself and enjoys the admiration of the parents; when the demands are too difficult, the child may feel frustrated, humiliated, or in danger of losing the parents' love.

Development in language and symbolization, object permanence, and cause and effect thinking sets the stage for the child to elaborate the "representational world" (Fonagy, 1997; Fraiberg, 1969; Sandler and Joffe, 1962; Slade, 1999; Stern, 1988). Within psychoanalytic theory, "self" and "object" representations refer to the organization of the variety of composite images of himself or herself and others that the child has internally constructed on the basis of experiences, urges, and feelings. These concepts are never articulated in consciousness as a sum total but, rather, are expressed as parts in the child's ever-changing fantasies, attitudes, and behavior. At times, the child may comply with the parents' requests in response to his or her inner view of them as wonderful and all-giving. Alternatively, the child's tirades against them may reflect a feeling that they are frustrating figures who are set on depriving him or her of pleasure. They may be seen as sources of safety and comfort or as obstacles in the path toward independence. Simultaneously, the child has rapid shifts in feelings about himself or herself—at one moment victorious and able, at another moment frightened and helpless. Moreover, the child may oscillate between views of himself or herself and parents as all good or all bad, determined by experiences of the child's own and by his parents' loving and destructively hostile urges.

Although the child struggles—internally and externally—with these varying attitudes, the flexibility of the representational world produces greater self-reliance. Now the child's ability to recall loving images of the parents in their absence allows for longer periods of independent activity, sustained by the confident expectation that needs ultimately will be satisfied (Mahler et al., 1975). Second, internalized standards, both prohibitive and encouraging, serve as referents in determining consequences of action (i.e., trial action in thought). Finally, in the absence of real gratification of wishes, impulses and feelings can find expression and discharge in the manipulation of self-object interactions in fantasy. Here, *conscious fantasizing* involves daydreams in which real experiences can be altered or replaced entirely by more gratifying, wish-fulfilling imaginative scenarios. These mental operations involved in fantasizing support the child's capacity to tolerate increasing frustration, whether from his or her own limitations or those imposed by the environment. The young child can now "hold on to" a variety of representations and "play" with them, or pretend. He or she can use imagination for escaping from unbearable disappointment in reality, altering current feeling states, and planning courses of future action. In a broader context, *unconscious fantasies* and their conscious derivatives give expression to the child's deeply felt longings, conflicts aroused by forbidden urges, attempts at resolving conflicts, and preparation for or postponement of action. As such, fantasies may be a source of pleasure and anxiety. When unconscious fantasies give form to unacceptable impulses and wishes, defensive operations are employed to disguise them before they find expression in conscious thought.

In this phase, fantasies and activities that serve to diminish anxiety expand and become more identifiable to the observer when they are at odds with reality or internalized standards. The child's growing disaffection with messiness and disorder, for example, is a signal accomplishment in the anal phase and reflects an active repudiation of pleasurable activities that are in opposition to parental attitudes (reaction formation). By complying with these external demands, the child avoids disapproval and reaps the satisfaction of adult approval. Initially having to yield control of his or her body to the expectations of others, the child's growing identification with parental demands and responses make them his or her own. Although vulnerable to reexternalization, the conflict between competing aims is now internal, belonging to the child alone. Anxiety associated with the dangers of aggressivity and from the conflict between loving and hating may be dealt with via various imaginative mental processes for mobilizing fantasies, which counteract other more frightening fantasies or modulate anxieties in other ways. These "defensive processes" or *mechanisms of defense* (Cramer, 1991; Freud, 1965; Kernberg, 1994) include the child's disavowal of a particular feeling or aspect of reality (denial), a sense that the feeling belongs to someone else (projection), or shifting the target of feelings from parents to himself or herself, others, or a toy (displacement). Hostile impulses that run counter to the love the child feels toward parents, for example, are disowned. Being fearful of others' hatred or frightened of monsters and noises in the night is a preferable alternative to the imagined destruction of loved figures on whom the child depends. These mental activities allow the child to express competing feelings of love and hate without needing to relegate them to completely separate images of himself or herself or the parent (Blum, 1997; Furst, 1998; Marans et al., 1993c; Mayes and Cohen, 1993; Parens, 1979). Real competence in communicating and acting on needs independently and the continued experience of the parent's availability promote a psychological "rapprochement" (Mahler et al., 1975). The child can begin to tolerate an ambivalent attitude toward himself or herself and the parents, between love and hatred, and between total dependence and self-reliance. In this phase, the child's recognition of others as separate entities and ability to appreciate the specificity of his or her own feelings toward them extends to a capacity to empathize with their feelings as well. These achievements are seen in expressed concern for and questions about parental moods and in the designation of feelings to figures used in play.

In the first year of life, physical objects were mouthed and handled as the infant explored their properties as well as the bodily sensations aroused in the process. These activities, as described earlier, aided the infant's delineation of body boundaries (Hoffer, 1949). In the latter part of the second and in the third year, the child uses toys and other play items for the extensive representation and elaboration of daily experiences and fantasies, as well as engaging the caregivers in pleasurable interaction. Moving cars across the floor, carrying and feeding baby dolls, manipulating puppets, and imitating parental activities constitute rehearsals, reworking of experiences, and trying on of new roles. They also are precursors to the development of imaginative and pretend play in which the child will employ complex narrative structures or story lines to elaborate these roles and fantasy scenarios. At age 3, there is a gradual move in the child's view of other children as playthings or things that get in the way of personal pursuits. Increasingly, children not only play side by side but turn to one another as companions and partners in shared activities that are more fun because they are social. Here, the capacity to generalize symbolic representation and empathize with the feelings of others serves as a common basis on which the fantasy configurations of each partner can be mutually enriched and enacted in play.

The achievements in this phase of development expand the young child's range of possible pleasures and the complexity of conflicts. Increased capacity for independent functioning is a source of pride in mastery, whereas the appreciation of separateness and reality highlights limitations and the vulnerability of being little and dependent. Ownership of the body and insistence of bodily urges (drive pressures) air in conflict with the child's sense of the contingent nature of the relationship with his or her parents. That is, *the push* to give expression to his or her own will is at odds with the desire to please the objects of his or her love by submitting to their requirements. As external controls and rewards become internalized, so too do the capacities to retain and elaborate various representations of self and significant others. Whether serving configurations of fantasy or memories of reality, the capacity for representation offers the young child an inner frame of reference that can be a resource for increased frustration tolerance, substitute forms of gratification, trial action in thought, self-esteem, and the companionship of important people regardless of their presence or absence. As the central tasks of the anal phase of development merge into those of subsequent stages, the groundwork is laid for further elaboration of relationships and capacities both within and without the context of the child's family.

### 36 to 48 Months ("Genital-Narcissistic" Phase)

A transitional phase of development has been posited between the anal and oedipal periods. This phase is characterized by an increasing crystallization of gender and concurrent preoccupation with appearance, anatomic differences, and sensations arising from the phallus and testicles in boys, and the vagina and clitoris in girls.



The child's assumption of a specific gender identity—the core sense of being a boy or a girl—obviously has its roots in the first year or two of life. Early gender identification during infancy is the result of multiple, interacting forces, including differential treatment of girls and boys by parents and others; the anatomically distinctive sensations arising from the genitals in the course of parental handling, elimination, and self-stimulation (including penile erection, clitoral stimulation, and the sensations accompanying defecation and urination); and other biological factors, including genetic and endocrine influences on brain and behavior. By age 3, children generally are quite clear that they are either a girl or boy and are aware of the types of play activities, dress, and the like expected of children of their sex. Attempts to alter assigned sexuality after this time, for example, for children with anomalous genitalia, usually are not successful. As a psychologically organizing internal construct, however, gender may not be sharply dichotomous, for either boy or girl, during the first years of life or even much later. Indeed, psychoanalysts have long appreciated that there is a spectrum of gender-related experiences throughout the course of development that may be considered normative bisexuality—the desire for some of the attributes and opportunities of the opposite sex.

Consistent with the psychoanalytic theory of psychosexual stages as developed in relation to the oral and anal phases, “phallic narcissism” ( [Burgner and Edgcombe, 1975](#)) has been used to designate the central zone of sexual sensations during this phase of development. However, the emphasis on the phallus does not recognize the girl's proud and pleasurable experiences of her genitals or the anatomic distinctions that are felt by both girls and boys. A more suitable designation for this psychosexual phase, which arises around age 3, might thus be “gender identification, genital narcissism,” a period in which children become progressively more aware of the pleasurable sensations in their genitals as well as tensions arising from the genital zone, take pleasure in displaying and being appreciated for their genitality, and experience themselves as having a core gender identity, that is, when all goes well, consistent with their anatomic identity. During this phase, both boys and girls may show off their genitals (enjoying how far they can urinate or lifting their skirts) and engage in more focused and sometimes more persistent masturbation (occasionally leading to genital irritation and a cycle of concern by both parents and child). Although the genitals may be a leading edge of narcissistic investment, during this phase, children feel a generalized pride in their bodies and what they can do in rough and tumble play, learning new skills on the jungle gym, displaying their fine and gross motor skills and coordination, and gaining parental attention through exhibitionism (such as dressing up in mother's clothes, clowning, or performing new feats of skill).

As with each phase, there are dilemmas faced by children during this period that are directly related to the developmental achievements. A child whose self-esteem is closely related to the beauty, power, and pleasures of the body may feel hurt, rejected, and enraged by not being noticed or mirrored by his or her parents or from failure in learning or performing some tasks for which the child may still be too immature or small. Although the anal phase was characterized by struggles concerning control, the phase of genital (bodily) narcissism may be burdened by struggles with siblings with whom the child may feel rivalry and with parents concerning issues of competence. The child may be unmindful of or angered by the recognition of his or her personal limitations. The parents' imposition of rules or even their offers of help may be felt by a child as demeaning, and sensitive parents may try to disguise their assistance or attribute the achievements to the child to foster the narcissistic delight in mastery.

There is a gradual transition rather than a sharp boundary between the psychological issues of gender identified, genital narcissism—proud exhibitionism, delight, pleasure, and sense of achievement as a girl or boy—and the issues of the oedipal phase. In some ways, this boundary is crossed as boys become more worried about the loss of their prized genitalia and girls express more concern about the adequacy of theirs, along with a range of increasingly complex feelings about the roles of the male and female parents.

#### 4 to 6 Years (“Oedipal” Phase)

Within contemporary psychoanalytic theory, *oedipal phase* refers to a range of concerns about loving, sexual/rivalry, and aggressive attitudes toward the same/opposite sex parent, which are correlated intrapsychically to the consolidation of mental structures. The achievements of this phase are a consequence of the advance of multiple forces—cognitive, affective, social, bodily—and provide a template for perceptions about the self and intimate relations with others for future development. If the child was an “explorer” in earlier periods of development, the oedipal child is best described as a “scientist,” curious, developing hypotheses, and experimenting in thought and play about: (a) his or her relations with parents, (b) their relationship with each other, and (c) how his or her mind works.

#### PRECONDITIONS

During the first years of life, the child moves through several phases of development, which have been characterized in relation to the “leading” bodily zone (oral, anal, and genital). Within psychoanalytic theory of development, the various tasks, concerns, and processes of these phases lead to a phase of mental integration involving economic factors (biologically driven urges), the direction of love and hate, and the relationship between unconscious and conscious thoughts and feelings in the context of the realities of daily life. The preconditions for the child's successful move into the oedipal phase and its optimal negotiation include overlapping, mutually influencing “preoedipal” achievements in areas such as the following:

1. Social: Good primary attachments to parents, representations of mother and father as separate and good, sense of self as the locus of initiatives that are successful, and capacity for empathizing with the feelings of others.
2. Cognitive: The ability to represent abstractions and call on these in assuming various social roles, taking the perspective of others, tolerating mixed feeling about self and others, appreciating the rights and responsibilities of oneself and others, and sensing that oneself and others can act and feel differently at different times.
3. Moral: A developing sense of right and wrong, good and bad, recognizing the link between actions and their consequences in relation to parents (approval and pleasure versus disapproval and anger) and the self (well-being and pride versus shame and guilt).
4. Physical: The capacity for fine and gross motor coordination for pleasurable and playful activities such as running, jumping, and climbing, as well as self-care (toileting oneself, brushing teeth, hand washing) and imitating adult behavior in play (cooking, driving a car, caring for a baby, building).
5. Emotional: The development of a wide range of feelings (including excitement, happiness, sadness, remorse, fear, disappointment, pride, anger, envy, love, and hate) that can be distinguished, expressed, and communicated to others.
6. Mental: The capacity for remembering personal history, realizing that dreams and imagination come from within oneself, perceiving complex social situations involving causality and time sequences, and developing narrative structures that organize inner and external experiences.
7. Biological: A hypothesized biological state that is analogous to the maturation of psychosexual zones (oral, anal, and genital) and to biological changes seen in puberty, which is now represented primarily in relation to emotions—intensified affective and sexualized longings (love) and raw expression of rivalry and aggression (hate). These are directed to the same/opposite sex parents during the oedipal phase and later are expressed in adolescence toward peers.

When the preconditions are in place, the 4- to 6-year-old child moves naturally into the oedipal phase, as evidenced by new integrations of self and others. During this period, the child recognizes that he or she is the child of his or her parents and that the parents have a relationship to each other that is not solely related to their roles as parents. Appreciating their strong feelings for each other, the child wishes to remain with them as in his or her previous fantasies—the center of their lives. The child knows that this position can be recaptured, if only for a while, through being very good, attractive, and clever or through being very naughty, destructive, and demanding. In addition, the child understands that he or she cannot be the only person for mother or father. Ultimately, the oedipal phase child recognizes the complexity of mother and father as full people. Whereas previously they were seen as either all good or all bad, depending on the child's state of frustration or satisfaction, increasingly, as a result of improved cognitive abilities, he or she feels the need to understand them as autonomous individuals, separate and in relation to himself or herself, to each other, and to others both in and outside of the family.

#### TRIADIC RELATIONS AND THE PRIMAL SCENE

The classical oedipal phase within psychoanalysis in its boldest expression related to one aspect of the Greek myth: the murder of the father by the son and sexual union between son and mother. This mythic beginning for the theory of the oedipal phase had the virtue of drama and suggestiveness but the vice of oversimplifying the phenomenology. The child in the oedipal myth did not know who was being murdered, and the remorse came later. The struggles of the oedipal phase are painful just because of the child's knowledge and continued love for both parents. He or she is confronted with amorous longings and rival wishes *and* the desire to work out possible compromises—how to be powerful and central, feel proud of achievements, and at the same time have the continuing secure presence of both parents ([Marans et al., 1993c](#)).

Crucial to the challenges of this phase is the fact that the girl's wish to exclusively possess her father, and the boy, his mother, cannot and will not find immediate gratification in reality. In order to sustain his or her quest, the oedipal child must be capable of suspending disbelief of denying that he or she is small and cannot enjoy the privileges and pleasures of the grownups ([Freud, 1974a](#)). However, the child is burdened in this quest by anxieties and real deficiencies that must be denied, lessened, or worked through if the child's invincibility is to be maintained ([Peller, 1954](#)). Boys and girls must find ways to explain why they do not win the “exclusive” position in the opposite-sex parent's life in spite of attempts to woo the parent (by impressing the parent with physical and intellectual competence as well



as charm).

Oedipal children are confronted by their exclusion from the parents' special relationship even though each parent may acknowledge how big, strong, attractive, or smart the young suitor might be. A hallmark of this phase is the child's capacity for *triadic relations*: the representation of the parents' relationship with each other and with him or her as *their* child. The child's theories about the parents' relationships with each other in his absence (*primal scene fantasies*) are based on observations of their affective ties as well as on projection of the child's own intense sexual and aggressive urges. Primal scene fantasies are a source of excitement and danger—in the child's view of sexual intercourse as violent—and of narcissistic hurt as well, representing the ultimate adult activity from which the child is excluded. Oedipal children must turn from a passive role in which they might fall prey to dangers and disappointments to an active one in which they can feel as though they are the masters of their own fate ([Freud, 1917](#)). In this task, they increasingly turn to fantasy and its expression in play.

#### REPRESENTATIONS IN PLAY

The child of this age has an increased capacity for reality testing; in addition, he or she continues to have the facility for magical thinking as well as more advanced capacities for guiding integration of competing wishes and aims. His or her ability for daydreaming, fantasy, and pretend play allows greater access to wishes that do not need to be compromised too quickly by reality. The child is thus protected in fantasy from the recognition of ultimate frustration. An increased capacity for symbolic representation opens the door to more elaborate forms of binding anxiety and expressing bodily urges in thought and imaginative activities ([Marans et al., 1993a](#)). Advances in cognitive and motor skills yield functional pleasure, minimize anxieties, and compensate for felt inadequacies in comparison with the adults with whom he or she competes ([Peller, 1954](#)).

Oedipal children can move fluidly through a range of themes determined by the urgency of what is uppermost in their minds, whether exciting, pleasurable, or fearful. The child's push toward mastery combines with curiosity to arrive at solutions in play in which theories may be tested and explored. Do babies come from eating special, magical foods? Do mommy and daddy hurt each other when they are in their bed together without me? ([Cohen et al., 1987](#); [Marans, 1993b](#)).

The oedipal child's play, in particular, reflects a plasticity of representations employed in the service of creating and maintaining the illusion of wish fulfillment and invincible mastery. The guiding motif of the child's creativity is contained in the oedipal wish and in attempts to ward off its attending dangers and disappointing confrontation with reality. The child in this phase of development is able to juggle the inconsistencies he or she perceived both *within* fantasies and *between* fantasies and reality. There is equal flexibility as the constituent themes of the oedipal phase seek expression in fantasy and play. At one moment, aggressive competition with the rival may be uppermost as the child assumes the role of the strongest, most attractive and competent member of the family. The same role may simultaneously give expression to the child's exhibitionistic impulses or wish to protect and care for the object of his or her longings. The play may easily shift from themes of power and strength to scenarios involving the production and feeding of babies. Repeated crashing of toy cars may at one moment express the child's concerns about aggression and bodily intactness. With additional features and elaboration, the same play may articulate the child's fantasies about sexual intercourse. In this latter context, the child's curiosity about the contents arid activities behind closed dollhouse doors or in closets and bedrooms may at once give vent both to sexual excitement and disappointment at being excluded from parental activities. Equally, increased interest in theories regarding the origin of babies, sexually exciting sensations focused on genitals, and anatomic differences and comparisons of physical attributes between sexes and between adult and child versions (same sex) are expressed in the oedipal child's fantasies and imaginative play ([Marans et al., 1993a](#)). Reality events in the child's life—accidents and injuries, as well as surprises and pleasures—are dexterously integrated into fantasies as they stimulate, organize, and accentuate the specific themes with which the child struggles ([Caper, 1996](#); [Cohen et al., 1987](#); [Neubauer, 1987](#); [Solnit, 1987](#)).

#### Infantile Neurosis

The powerful wish to get rid of the same-sex parent and assume his or her role in an exclusive, intimate relationship with the opposite-sex “partner” is also fraught with tension as the child struggles with love and hate felt toward the rival. The experience of hurt, tension, and uncertainty during this phase leads to the achievement of internalized conflict. The child now has the capacity for neurosis, that is, an internal struggle between opposing forces (wishes versus personal values, desires versus parental attitudes and opposing desires). The *infantile neurosis* is theoretically the paradigmatic form for age-appropriate mental disorders engaged by oedipal phase structures. It is expressed by the appearance of regressive behaviors and fears—obsessive-compulsive symptoms, enuresis, encopresis, thumb-sucking, phobias, separation difficulties, nightmares, frequent battling with parents—that are common in this period of development and serve the child's attempts to withdraw from or displace the conflicts aroused by the intensity of his or her oedipal longings. Similarly, in imaginative play, the child will change the narrative script when he or she is unable to maintain an adequate distance from painful reality or anxiety stemming from conflicts. For example, the role of a given protagonist may be elaborated in order to add dimensions that would compensate for the child's intruding sense of vulnerability (e.g., the policeman who is battling robbers is transformed into Superman). Alternatively, the specific role and theme in the play may be abandoned altogether as the child shifts to ones more congenial to the maintenance of pleasure and the prospect of mastery. This shift might be seen in the child's assuming the voice and behavior of a baby, in provocative messing, or in destruction of play materials. However, the child might equally turn to an intensification and insistent involvement in the fantasy, becoming narrower in his or her range of play while trying to overcome anxiety in the repetition of a singular theme.

In these varied ways, the child's internal conflicts and their representation in fantasy, symptoms, and imaginative play constitute the child's research about the self and others. Expanding experience in a broader social context (chiefly through the introduction of nursery and school programs) fosters increased psychological independence. Organized and regular group experiences offer a broader context in which the conflicts and tasks of the oedipal phase of development may be worked through. The capacity and opportunity to engage with peers increases the range of play activities in which fantasies and skills may be “tried on.” Cooperative play (in which role assignment, turn taking, and mutual elaboration of narrative/story line are component parts) often revolves around themes regarding:

1. Exploration of and acquisition of knowledge about physical properties of things and various characteristics of the environment
2. The child's sense of place within the family
3. Size, strength, capacities/competence
4. Theories and feelings about babies and their origins
5. Anatomic differences and attributes of both sexes
6. Curiosity and excitement associated with genital sensations
7. Competition and rivalry
8. Destructive aggression
9. Consequences of wishes and actions

Relationships with children and adults outside of the family capitalize on and promote the youngster's greater frustration tolerance, reliance on increased intellectual and physical capacities, and confidence in the ability to function—with pleasure—in the absence of parents. In addition, the world beyond the family affords the child with a broader range of stimulation and opportunities for using new skills such as reading, writing, and problem solving in the classroom setting.

Although giving rise to considerable anxiety, the oedipal child's experiments in thought and pretend play serve to integrate representations of the self and others. The conflict between loving and hating leads the child to further consolidate and integrate the images of the “good” and “bad” parents into representations of them as whole, autonomous people who have relationships beyond the dyadic parent–child configuration. In muting the intensity of oedipal longings, the child strives to live up to the ideal of being like the rival parent (ego ideal) and to identify with their rules and values (superego). These achievements of internalization allow the child to desist in overt rivalry with the same-sex parent, to feel that he or she has not lost out in the struggles but has in fact taken on the role of the parent internally, and serve as reparative structures for anxiety, frustration, and the hurt narcissism that accompanied the more raw competition. The child's feelings of wounded pride and anxiety serve as “signal affects” and alert the child to how he or she is feeling in response to specific thoughts and actions (in the past, present, and future) and lead to the child's capacity to observe himself or herself and to establish theories of how his or her mind works.

#### THEORIES OF MIND

The “theories” the child develops organize internal experience of affects, bodily urges, memories, parental rules, and praise and personal ideals and provide structure and cohesion for the child's perceptions of self and others. They are tested and elaborated in fantasy, play, and daily life and draw on the many new roles the child assumes in relationship to the parents, as a friend to peers, as a sibling, as a student in the classroom, and the like ([Klin et al., 2000](#); [Marans, 2000](#); [Mayes and Cohen, 1994](#)). The child's theories of how his or her mind works—what makes the child worry, and how does the child enhance good feelings about himself or herself through fantasy, imaginative play, and direct action—lead to decreased egocentricity and increased self-observation ([Marans et al., 1993c](#)). The ability to manipulate ideas and feelings in preparation for action and the capacity to take the perspective of parental representations allow the child to postpone gratification, to balance urges with ideals that are now the child's own. Achievements in delaying immediate satisfaction and identifying the principles of justice/morality are expressed in the

child's social play with companions, where he or she is able to see the world from the view of the other—not grabbing toys, taking turns, playing by implicit rules, developing narratives from shared fantasies—and accepting the fact that he or she is not alone in having important needs. The child's new capacities are also apparent in the child's recognition of the difference between play and work and in his or her sense that industriousness and perseverance brings their own rewards and personal satisfaction. Although parents' approval and praise (and the admiration of other adults and peers) remain important to the oedipal child, the child's ability to refer internal theories about himself or herself—sources of pleasure, memories, ideals, positive and negative consequences of thoughts and actions—allows the child to give up a singular reliance on external sources of reward/response for the regulation of self-esteem ( [Marans, 1993b,1993c](#); [Mayes and Cohen, 1994](#)).

### 7 to 10 Years (“Latency” Phase)

The term latency refers to a decrease in the prominence of preoedipal and oedipal striving in terms of both intensity and direction. Although they do not go completely underground, the degree of preoccupation with sexual impulses and interests that are explicitly connected with the assumption of parental roles is significantly diminished. In fact, it would seem that in the sexual curiosity, excitement, and joking that can be observed among latency-age children, notions about parents as sexual objects/beings must be denied or avoided. Activities and attitudes seen as infantile—dependency on parents, fearfulness, and the like—also need to be avoided at all costs. In the face of conflicts established in the preoedipal and oedipal phases, infantile and incestuous longings are repressed, and a widening array of adaptive defenses, including intellectualization, humor, identification, obsessional interests, and sublimation, are utilized to support the diversion and alteration of the original impulses ( [Becker, 1974](#); [Bornstein, 1951](#); [Etchegoyen, 1993](#); [Freud, 1968](#); [Marans and Dahl, 1998](#); [Sarnoff, 1976](#)).

The continued consolidation of intellectual, sensorimotor, and social skills and opportunities affords the latency child with a variety of pathways for the sublimated expression of urges that dominated earlier phases of development. For example, friendships and group affiliation can serve as an alternative to the exclusive, close tie to the parents while further facilitating the child's exploration of the world outside of the family. The intense interest and curiosity about the origins of babies, the intimate life of parents, and the mysteries of sexual differences can now be employed for and shaped by the challenges of learning that occur in both school and extracurricular activities. Competitive strivings central in the oedipal phase of development can find expression in games and relationships with peers. Exhibitionistic impulses find a broader range of vehicles and audiences in the performance of intellectual tasks and mastery of athletic and creative pursuits. In addition, the demands and rewards associated with a relatively stable conscience (or superego) are increasingly exercised in the latency-age child's interests and activities. Although not without breaches, as seen in cheating and insensitivity to peers and family members, insistence on following the “rules of the game,” empathy with the feelings of others, and a clear sense of right and wrong are hallmarks of the child's establishment in latency. Similarly, descriptively obsessional interests in orderliness, collecting (as in hobbies), and details of functional relationships and properties of physical phenomena serve reaction formations that derive from both benign and prohibitive aims of the superego. These elaborations of defensive and adaptive functions are only possible with and equally reflective of the development of more sophisticated cognitive processes involving the introduction of operational thought.

Optimally, the latency-age child achieves greater autonomy with respect to aspects of daily living such as hygiene, dressing, looking after possessions, and the like. The ability to engage in operational thinking and problem solving, in conjunction with the capacity for increased frustration tolerance, broaden the range of potential achievements and attendant satisfaction. These accomplishments enrich the child's interaction with peers, who are viewed as partners and “best friends” as well as new objects of identification, admiration, and competition.

The central threat to the latency-age child is the reemergence or breakthrough of the original sexual and aggressive fantasies of the oedipal phase, particularly when associated with the impulse to masturbate. Sleeping difficulties; nightmares; worries about burglars, bodily injury, and death; and the ease of regression to earlier modes of relating to parents (struggles over food, self-care, household responsibilities, and the like) may be some of the behavioral phenomena that accompany the child's attempts to defend against and give expression to the residual or persistent intensity of oedipal conflicts in the early period of latency. The elaboration of cognitive capacities, peer group involvement, and academic and extracurricular interests facilitates the late-latency child's sublimation of previous trends in action and fantasy and promotes greater distance from the original objects of oedipal longings. Alternately, these achievements are subject to neurotic interference; school failure is not necessarily an outcome of learning disabilities, and isolation from the peer group is not alone determined by athletic skills, looks, or abundance of toys.

### Adolescence (12 to 20+ Years)

Adolescence is one of the most dramatic phases in the course of development, marked by profound changes in biological, psychological, and social functioning. Preceded by a period between ages 10 to 12 in which there is renewed interest in anatomic differences, sexual curiosity, and masturbation (preadolescence), the transition into *early adolescence* (12 to 14 years) is introduced by the endocrinologic and biological processes of puberty. With much greater intensity, these processes focus the young adolescent's attention on concerns about bodily changes and sexual sensations. The primary and secondary sexual characteristics—growth spurt; voice changes; menarche; nocturnal emissions; breast development; and pubic, axillary, and facial hair—associated with pubertal maturation are often greeted with a mixture of pleasure and trepidation. Preoccupation with comparisons of the rate of changes in others; the intensification of exciting genital sensations and the compelling need for masturbation as a source of relief; and the increased conscious awareness of sexually arousing yet conflictual fantasies, precipitating the urge for and accompanying masturbation, may all arouse a tremendous amount of anxiety for the young adolescent ( [Blos, 1970](#); [Dahl, 1993](#); [Kestenberg, 1980](#); [Laufer, 1989](#)).

Although physical maturation heralds the entry into manhood and womanhood, the changes can be experienced as happening too quickly or not quickly enough. These feelings may be manifested in the exhibitionism, embarrassment, and secretive behaviors seen in the young adolescent's attitudes toward his or her body. In either event, pubescent children may be impressed and made anxious by the lack of control of their own bodies. Menarche and the first nocturnal emission are dramatic, paradigmatic events for the real absence of control that characterizes this phase of development. The young adolescent's relationships with his or her parents may be equally tumultuous as rapid shifts between longings to remain close and dependent and requirements for privacy and strivings for autonomy are experienced. These struggles are often played out around old issues having to do with bodily care and hygiene, cleanliness and orderliness of personal property, insensitivity to the feelings and needs of others, and intense preoccupation with immediate satisfaction of one's own needs ( [Blos, 1962](#); [Eiguer, 1996](#); [Erlich, 1993](#); [Freud, 1958](#); [Hauser and Smith, 1991](#); [Laufer, 1985](#); [Ritvo, 1984](#)).

By *mid-adolescence* (14 to 16 years) maturation levels off; all girls are now in menarche, boys are able to ejaculate, and both have attained secondary sexual characteristics. Physical maturation potentiates the conflictual nature of incestuous fantasies that reemerge during adolescence. Unlike 4- to 6-year-old children, the young person by mid-adolescence is confronted with the reality that he or she now possesses the equipment necessary for carrying out both sexual and aggressive wishes. The adolescent's provocative and battling stance toward the same-sex and both parents reflects renewed oedipal competition and repudiation of both oedipal and preoedipal longings. Disregard for family rules at this point may reflect desperate attempts to achieve emotional distance from parents. However, behaviors that “test limits” are now often implicit invitations for parents to remain involved and take charge; the adolescent's subsequent outrage maintains his or her explicit objection to any parental interference with his or her freedom.

The complexity and difficulties of family relations further stimulate and support the adolescent's activities outside of the home. He or she finds refuge from the claustrophobic pull of family ties in intellectual, athletic, musical, and political interests and potentially in illicit drug use, drinking, and crime as well. Similarly, as the adolescent withdraws from his or her parents, the intensity of the attachment to them is shifted to peer group relations, including same-sex and opposite-sex relationships. Opposite-sex pairing and dating serve as: (a) sexual and emotional experiments in intimacy, (b) a gauge of masculinity and femininity, (c) an opportunity to try on (in reality as opposed to imaginative play) adult relationships, (d) an escape from fantasied or real incestuous and homosexual activity, or (e) a marker of conquest and competence, to be appreciated by an audience of peers. In addition to tighter affiliations with peers, social expectations and the push for mastering anxiety associated with dependency on parents spur the adolescent toward real independent functioning. In this, the adolescent must recognize that the craving for independence implies truly relying on his or her own abilities rather than on parents.

By late adolescence (17 to 20+), involvement in relatively stable peer groups, academic and extracurricular activities and interests, some degree of financial reward for responsibilities associated with jobs, and the like reflect the adolescent's emancipation from the roles and requirements of earlier phases of development. The degree of successful engagement in life beyond the relationship with the parents may be a gauge of the extent to which the adolescent is able to emotionally disengage from and mourn the passing of his or her reliance on these powerful figures.

There is perhaps no more powerful example and test of the adolescent struggle for autonomy from parents—the breaking of preoedipal and oedipal ties—than the fantasied and real search for a new, typically heterosexual partnership. In late adolescence, the young person is very aware of the difference between the love felt toward a new heterosexual partner and the earlier experiences of experimentation. The girl is no longer primarily interested in conquests or simply owning and overpowering the man; the boy is no longer singularly guided by his need to prove his powers of seduction, his masculinity, and his repudiation of homosexual aims. Both are caught up in the desires that had been previously in the domain of oedipal fantasies. Longings for physical intimacy, for exclusive attachment, and to protect and be protected, to admire and be admired, can find full expression in the courtships of adolescence. This new experience of closeness and trust is the essential



climate for surrender to the heterosexual partner in which earlier components of the attachment to parents—dependency, passivity, exclusivity, jealousy, physical intimacy, and so on—may be subsumed. Again, the extent to which these and other longings (such as unresolved childhood wishes to possess the anatomy and prerogatives of the opposite sex) remain predominantly and conflictually tied to their original objects, the parents, determines option for and depth of new heterosexual partnerships.

Although many, and perhaps most, adolescents experience some degree of sexual interest in individuals of the same sex and may engage in some degree of arousing physical contact (such as mutual masturbation or sexual display), their fantasy lives and primary objects of sexual arousal are heterosexual. Typical homosexual experimentation occurs in the context of primarily heterosexual interests. During the usual course of development, heterosexual orientation appears to be based on biological factors that are shaped during gestation and the first months of life and that are reinforced by social experiences in the family and peer group, as well as the developmental sequences of the psychosexual phases. A sizable number of adolescents, however, are primarily involved in thinking about and being aroused by individuals of the same sex; they are not turned on by individuals of the opposite sex. For some, the recognition of the direction of their attraction is clear during the earlier school-age years (latency); for others, the intensity and singularity of homosexual object choices become clear only during early adolescence or somewhat later. There are probably a variety of determinants of homosexual orientation in both males and females; biological factors have been suggested, as well as interactions between constitutional and familial patterns of interaction. There is also a range of variants of homosexuality in adolescence, which for clinical purposes need to be differentiated: Homosexuality may represent a defense against aggression or a negative resolution of oedipal conflicts (i.e., focusing longings for intimacy with the same-sex parent as a way of warding off anxiety associated with incestuous wishes involving the opposite-sex parent), it may be an exaggeration of bisexuality or emerge in specific environmental contexts (e.g., in seminaries or boarding schools), or it may represent the adolescent's immutable and well-integrated sexual orientation, as authentically a part of the adolescent's self as is heterosexuality.

The developmental tasks for homosexual adolescents are complicated by both internal and external factors. The youth's discovery that he or she is excited by and attracted to individuals of the same sex runs counter to what the adolescent may have expected and the experiences of his or her peer group. This feeling of being different compounds the usual feelings of estrangement and secrecy of adolescent sexual development. The homosexual adolescent also usually has no peer with whom to share, in locker room or girl-to-girl intimate chats, the emerging sense of being a sexual person. The homosexual adolescent also lacks socially sanctioned opportunities for sexual experimentation, for finding suitable intimacy, for using the pairing of adolescence to move away from the parental ties. In his or her loneliness, the homosexual adolescent may try to deny sexuality altogether and attempt to be celibate; may attempt to engage heterosexual friends in sexual relations that the other youngster feels initially as experimentation but that for the homosexual takes on more intense, crush-like importance; or may feel the need to engage in brief encounters that may be dangerous psychologically and with the increased risk of human immunodeficiency virus (HIV) infection ( [Downey and Friedman, 1998](#); [Friedman and Downey, 1993](#); [Isay, 1989](#)).

The capacity for more mature, intimate relationships in the latter part of adolescence by no means guarantees their permanence. The task of consolidating the sense of self continues to find expression in shifting interests and ideals; the extent to which they mesh with those of the chosen partner, in part, determine the success or failure of the relationship at any given time. In addition, the amount of emotional energy available for intimate ties with others may fluctuate as the adolescent becomes increasingly involved in making plans for the future. That these may involve the young person's physical departure from home and community often contributes to the end of significant intimate relationships. Even more important is the adolescent's psychological departure from and break with the familiar, as he or she moves further into the realization of independent strivings and autonomous functioning. These are not without considerable practicing, uncertainty, and anxiety, as the adolescent anticipates the breadth or limitations—determined by individual strengths as well as multiple social factors—of opportunities available in the world beyond the family.

Adolescence has long been described as a period of inner turmoil in which the onslaught of biological changes, intensified sexual and aggressive urges, and shifting social relations and responsibilities make the search for a cohesive self a ubiquitous, crisis-laden struggle. Given the reality of the multiple variations in the appearance and behavior of teenagers observed on the streets and in the malls and schools of inner cities, working-class neighborhoods, and upper-middle-class communities, can there be a central theory of adolescent development? Although there are "typical" preschool- and school-age children, what can be said about adolescent development that is true to the tremendous variations seen in this period? The task of a psychoanalytic theory of adolescent development is to delineate those processes that: (a) are generally found in adolescents, regardless of surface differences in behavior, and (b) allow for the broad variations in personal and social situations, explaining why adolescents operate in the context of their families and neighborhoods on the one hand, and in relation to their personal, inner lives on the other. Although there are other approaches to understanding the problems and achievements of this phase of development, the psychoanalytic task is primarily to understand the meaning of the inner experiences of adolescents and their links to behavioral and life choices.

## PSYCHOANALYTIC PHENOMENOLOGY OF ADOLESCENCE

From the psychoanalytic perspective, the phenomenology of adolescence centers around an array of reorganizations of inner life. The primary reorganization concerns the representation of the bodily and sexual experiences: Early in puberty, the child initially feels that these new experiences are external to the self, but when all goes well, he or she gradually integrates the mature sexual body as a source of pride and pleasure. Concurrent with the reorganization of the representation of the body in its mature sexuality and size, there are psychological reorganizations in relation to drives (especially the modulation of aggression and the fusion between aggression and tenderness), values (especially the assumption of personal values that differ from those of parents), concepts of time (especially the adolescent's sense of having a personal history and a future that he or she can more or less envision), and the self and relations to others (especially the possibility for sharing intimate experiences outside the family and the giving and receiving of sexual pleasure). Each of these reorganizations rests on the achievement of various preconditions during earlier phases of development, good enough physical and cognitive functioning, ongoing support from the family and other adults, particularly teachers, and good fortune in finding friends and avoiding irreparable injury.

Although most adolescents do not experience psychiatric disorders ( [King and Apter, 1996](#); [Rutter et al., 1976](#)), this phase represents a period of increased prevalence of a broad range of psychiatric and behavioral disturbances, including affective disorders, anorexia, suicide, and accidents. For children who are vulnerable because of preexisting difficulties, such as chronic medical illness, emotional difficulties, cognitive delays, or the burdens of poorly resolved earlier neurotic conflicts, the entry into adolescence, with the push toward autonomy and new social expectations, may herald major upheavals in previously stable patterns of medical care or parent-child relations.

### The Experience and Representation of the Body

Preoccupation with the body is a prominent characteristic of adolescence. Normal young adolescents devote considerable attention to ensuring its attractiveness to others. Yet the body may also be punished, mutilated, and destroyed by the adolescent who is experiencing turmoil. The balance between the adolescent's love and hatred for his or her body, between self-care and self-destruction, may shift rapidly, repeatedly, and precipitously. The adolescent uses his or her body as the stage for enacting pride or shame, joy or despair, a sense of being beautiful or horrid. The body may be seen as another, to be treated respectfully or thoughtlessly; as a persecutor, to be ignored or attacked; as a lover, to be caressed and narcissistically exhibited.

Psychologically, the adolescent may experience the rapid changes in his or her body in a passive mode that may reverberate with earlier experiences of humiliation or dependence. Here again, the adolescent may feel that he or she is forced to "learn to accept" what is not under his or her control. The adolescent may try to deny what is happening to his or her body and hold on to the interests and social forms of latency. Or he or she may feel comfortable with its increasing potentialities, secure that what nature has in store is consistent with previous good experiences. Or, most commonly, the adolescent may react to the changes in body and drives with a mixture of anxiety and exhilaration, of acceptance of what is in store along with a push toward mastery in which passive is turned into active. In trying to take active control over the passive experiences of puberty, the adolescent may try to improve on nature—to decorate his or her body with hairdos, special clothing, makeup, and tattoos; to improve the appearance of the body's shape by weight lifting, dieting, or gaining weight to increase muscle bulk; or to distort the body's appearance, to make what the child feels is passively weird (in his or her own eyes) into actually weird, through second-hand clothing, wild hair, multiple earrings, long hair, or whatever else the peer group, at a particular moment, has defined as hip, in, or cool. Through such stage design, the adolescent reorganizes his or her representation of the body and the meanings of its various parts in relation to others, including same- and opposite-sex peers and parents. When not carried too far, the experimentation allows the child to take responsibility, to own his or her body in its new form, smells, and modes of arousal.

For the vulnerable adolescent, the manipulation of the body as a thing may become a preoccupation. The adolescent may feel that life is unbearable because her nose is too long, his penis is too small, or she or he is too tall or short. The reorganization of the body representation may also become enmeshed in the reorganization of family relations or other changes in self, leading, along with other determinants, to anorexia, bulimia, self-mutilation, or obesity. When the body becomes the expression of neurotic and intrafamilial conflicts, the reorganization of the self revolves around representations of the body: who owns the body, who is responsible for it, and who enjoys its pleasures and feels its attacks.



## Sexuality: from Autoeroticism to Shared Intimacy

Psychoanalytic theory emphasizes the role of sexuality—of pleasurable experiences through the body—from the first months of life. Autoeroticism and sexuality continue throughout each of the psychosexual phases, with pleasures felt in specific zones (oral, anal, and genital), and in the activity of the body and its musculature as a whole. Puberty heightens the role of sexuality in conscious and unconscious life and focuses sexual feelings on the genitals. As with sexuality in general, psychoanalytic theory recognizes the child's ability to provide himself or herself with sexual pleasures through his or her own activity and fantasy, from the first years of life. With puberty, sexual self-gratification through masturbation and self-regulated fantasy becomes central to the definition of selfhood and the relations between self and others. Adolescent masturbation is an achievement: It includes physical maturation and the capacity for erection/ejaculation or vaginal/clitoral orgasm, the capacity to imagine and sustain fantasies that are pleasurable enough to lead to climax, and the maturity to delay satisfaction, to plan for and carry out masturbatory activities at suitable, private times.

Adolescent masturbation, as a physical act, is accompanied by rich internal processes that have been conceptualized as “masturbatory fantasies.” The adolescent is aware of the conscious story, major themes and images, and cast of characters. Often, these conscious elements are based on current experiences with peers, popular movie star images, and materials such as magazines and the like, as well as on powerful experiences that the adolescent may actually have had earlier (including beatings, active or passive sexual encounters, and primal scene fantasies and observations).

Psychoanalysts such as the [Laufers \(1987\)](#) have postulated that adolescents generally have a fixed and limited number of central masturbatory fantasies—fantasies that are not fully conscious or conscious at all and that underlie the structure or affects of the conscious fantasies. These central masturbatory fantasies may provide a paradigmatic form or emotional tone to the experienced or conscious fantasy—for example, sadomasochistic features, incestuous impulses, teasing, perversity, or passive longings—that may be uncomfortable or the source of anxiety within the conscious masturbatory experience. The central masturbatory fantasies are integrative; they bring current sexual urges together with the child's earlier experiences with being cared for by the mother (tactile, oral, anal), his or her fantasies during the oedipal phase and in relation to primal scene, and the pleasures of gender identification genitality and latency age masturbation and excitements.

When development along the line from autoeroticism to adolescent sexuality has gone well, masturbation provides opportunities for rehearsing sexual behavior, expressing and receiving affection, and trying on the different active and passive roles needed for mutuality in love-making (including how to hold back on orgasm until just the right moment). Probably few adolescents are completely without guilt concerning masturbation; understanding the sources of this guilt reveals the ways in which adolescents may feel that sexuality is the appropriation of parental rights, that their fantasies run counter to their values, especially in relation to aggressively being out of control or oedipally prohibited. Adolescents who are too anxious about sexuality or too frightened by aspects of their masturbatory fantasies may try to abstain completely; other adolescents may compulsively and guiltily engage in masturbation, increasing their guilt and, thus, the tension that leads to masturbation. Through trying to titrate their involvement in masturbation, adolescents reorganize their representations of control over their sexual desires. They learn they can turn on, delay, and discharge their sexual feelings, more or less at will.

## Reorganization of the Self

The “self” has many meanings: It can be understood theoretically as the locus for the integration of intention, abilities, desires, and values; as a mental structure that coordinates the other agencies of the mind; as an internal, conscious and unconscious “representation” of the individual's feelings, ambitions, and goals; or as a component of an individual's experience, quality of experience, and personal identity, which has continuity over time, space, and feeling states. The concept of self has a sense of individuality, distinctiveness, and boundaries—of the separateness of the person from others. Being true to oneself is a value, but self-valuation carried to self-preoccupation is self-absorption and selfishness at the expense of others. On the other hand, giving oneself to others and feeling other's needs as though they were one's own may be enriching of the self as long as one does not lose track of one's own self-interests. The concept of self is recursive, as captured by phenomena such as self-consciousness and self-awareness, as well as by processes such as taking, or not taking, care of oneself.

Adolescents take themselves seriously: They wonder who they really are, how they are different from their parents, how they became who they find themselves to be, what they are becoming. They enjoy losing themselves in crowds and finding themselves in “deep and meaningful” relationships. They search for their true values and refuse to compromise, fearful that any loss of the high ground will lead to the slippery slope of loss of their hard-earned individuality. They become abstractly conceptual, wondering about how their minds work, their values, authenticity, and politics. They may feel that they are obligated and may even have the ability to make a difference, to reduce the suffering or wrongs of the world; or, just the opposite, they may explain to themselves that, “the world being the way it is,” there is nothing they can do but simply get by, get even, go along, drop out, or have a good time. Whatever side of the polarity of involvement in the broader social world that they select, they are likely to think about their choices “philosophically,” in categories of value and history rather than solely or consciously in relation to what their parents believe and they have been taught.

This reorganization of the self involves a heightened cathexis or direction of attention to the internal world (e.g., feeling states and impulses); alternations in the regulation of narcissism; and, concurrently, reevaluation of relationships with friends and partners in intimacy (object relations). These closely intertwined processes are closely associated with sexuality. For example, the adolescent's experience of a crush—of the enchantment of another—at the same time expresses and affects several domains: The adolescent recognizes the power of the other; he or she feels a loss of self-sufficiency and self-control; he or she experiences an enhancement of self through fantasied union with the desired other. Through idealizations and crushes, the adolescent finds ways of dealing with the depressive feelings associated with loss of earlier relations with the parents. When these intertwined processes of reorganization are successful, and the adolescent is fortunate, the adolescent increasingly feels a consolidation of his or her personal identity as a sexual, competent, and reliable individual.

Relations with parents provide a testing ground and launching pad for the reorganization of the adolescent's self. By approaching his or her parents with worries and then striking out at them for intruding or not understanding, the adolescent, who may seem at such times to be paranoid, learns how far he or she has moved toward feeling secure in his or her own abilities to deal with impulses and frustration. Adolescents need to learn how far they can go on their own and also how they can return, when need be, to the security of the preadolescent relations with the father and mother. As they move away and back toward the dependency, adolescents may provoke battles about issues that are of relatively little obvious consequence. In the process of negotiating rules about when to be home at night, whether he or she has carried out the garbage as promised, and the appropriate length of hair or density of makeup, the adolescent boy and girl learn about the modulation of their rage at their parents (in their role as symbols of their own dependency longings) and whether the parents can survive their aggression (in their role as representatives of the adolescent's own abilities to hold together when overwhelmed). When parents break down under the pressure of unremitting aggression, adolescents do not feel victorious. They may feel panicked. And in their distress, they may feel compelled to attack, insisting that the parents be strong, or withdraw in an angry and depressive huff.

Within the family, the adolescent's struggle for self-definition may set off varied reverberations with parents. Parents may feel envious of the youngster's freedom and experimentation, particularly sexual freedoms, or may anxiously overreact to their fantasies of what their child is engaged in. The mother who pounds at the bathroom door when her son stays inside too long knows, and yet does not know, what has happened to her little boy. The sexually mature adolescent daughter may arouse incestuous fantasies in her father, who then, through projection and displacement, engages her defensively in battles about boyfriends and morality. Parents may establish rules that are harshly enforced as externalizations and barriers to the enactment of forbidden (and not fully unconscious) fantasies. Alternatively, parents may vicariously enjoy the exploits of adolescence and too easily relinquish the parental role; they may be too understanding or may implicitly encourage their child's promiscuity, drinking, and fast driving while explicitly condemning him or her for getting into trouble.

## Adolescent Disturbances

The phenomenologic processes of adolescence that move toward consolidation of the self in its many facets also are vulnerable to multiple different types of disorder, from within the adolescent's own bodily changes and mental processes and from the outside world.

There are special burdens to adolescent development that arise from the timing of puberty or its results: precocious puberty, which marks out a child as sexually mature or too tall before he or she is ready, or delayed puberty, which prevents a child from entering fully into the experiences of his or her peers in the discovery of sexuality. There are other burdens that represent the carrying over into adolescence of the sensitivities, conflicts, and poor resolutions of earlier phases: inability to tolerate frustration, predisposition to anxiety, tendency to deal with tension by impulsive activity, and lack of an internal sense of security in relations with parents and within the self. And there are burdens that arise specifically in response to the emergent issues of adolescence: the strength of sexual drives, preoccupation with fantasies, guilt over sexuality and aggression, and troubles coping with the maturing body.

Given the complexity of the developmental tasks involved in reorganization of the self and its representations of the body and others and the modulation of aggression, sexuality, and narcissism, it is no surprise that there is a marked increase in many psychiatric disorders concurrent with puberty. These include affective disorders, particularly depression; personality disorders, which reflect difficulties in self and object representations; disorders of appetite (such as anorexia, and bulimia); and frank schizophrenia. Adolescents also demonstrate breakdowns that elude categorical diagnosis and include dramatic and life-threatening symptoms in various combinations: self-destructiveness, brooding despair, complete social withdrawal, sexual promiscuity and perversity, addictions, immobilizing preoccupations, seeking out and experimenting with danger, and the like. Psychoanalytic investigation of these disorders involves placing the child's inner experiences in the context of his or her personal development and understanding the developmental meaning of the symptoms and activities. Because adolescents are often deeply interested in understanding the lives they are living, they can be engaged, as younger children may not, in reconstructing their histories and reflecting on their experiences; thus, therapeutic work may be in keeping with the developmental tasks faced by all adolescents of finding new representations through reorganization of the self.

Just as adolescence presents difficult tasks of development for each agency of the mind, the achievement of adolescence, as defined by the success of reorganizations, leaves the adolescent with a sense of pride and vigor. Competent, attractive adolescents evoke adult fantasies concerning youth and potentiality, as well as envy and yearning. The image of adolescence in literature—adventurous, seeking, seductive, vulnerable, and emotionally alive and open to new experience—captures the achievements of adolescence and also how the development of adolescents, as the next generation, is intertwined with that of their parents, as reminders of fulfilled longing as well as bearers of hope.

## PSYCHOANALYTIC THEORY: DOMAIN, CONCEPTS, AND THEORETICAL INTENTIONS

The primary domain of psychoanalytic theory is the child's inner world, mental functioning, and internal experiences, as they elaborate over the course of development. First, psychoanalysts are interested in the personal, private, and intimate experiences of life—emotions, feelings, thoughts, wishes, dreamlike states, daydreams, worries, yearnings, pleasures—and how the person becomes aware of these experiences, allows for their unfolding, pushes them outside of consciousness, shares, and hides them. Second, psychoanalysis is interested in behavior—what children do—and the relations between the inner world and action, particularly how the child experiences his or her own action and the actions of others. Psychoanalysis focuses on the child's understanding of his or her own life, of the ways in which the child comes to represent and make sense of his or her inner world and the minds and actions of others. In these ways, psychoanalysis is particularly concerned with highly individualized, detailed, and rich descriptions of mental processes. The major concepts of psychoanalysis are aimed at providing categories and approaches for such descriptions, charting the major domains of experience over the course of development.

Closely related to this descriptive and in many ways historical undertaking, psychoanalytic theory also concerns origins and determinants. The “explanatory” theories of psychoanalysis consist of a range of perspectives, some of which are already outlined by Freud in his classical writings and others that have been described by theorists over the past decades. Freud's primary perspectives were the cathartic (the purging of the mental apparatus of negative experiences by abreaction, closely related to hypnosis and the so-called talking cures), economic (the distribution of psychic forces and the effects of their being pent up or discharged), dynamic (the conflict between wishes and prohibitions and their expression in symptoms), topographic (the distinction between conscious, preconscious, and unconscious mental processes), and structural (the functions of the ego, id, and superego) theories. These theories have been applied and remain useful within child psychoanalysis as well. They have been augmented by perspectives that emphasize development (the emergence of new structures and the changes in each of the classical perspectives over the course of maturation), developmental lines, the organizing role of the self, the importance of social relations (object relations), and the complexities of proverbial experience and mother—child relations.

Alongside the psychological perspectives to explanation or understanding of mental life, psychoanalytic theory has recognized the importance of general theories of psychology ([Fonagy, 1991](#); [Fonagy et al., 1993](#); [Mayes and Cohen, 1994, 1996](#); [Osofsky, 1982](#)), originally conceptualized as autonomous ego functions and adaptation ([Hartmann, 1939](#)), and the role of biological processes—genetic ([Mayes, 1999](#); [Peterson et al., 1995](#)), endocrine ([Moran, 1984](#); [Moran and Fonagy, 1987](#)), and constitutional ([Mayes and Cohen, 1993](#))—and the general health and experiences of illness ([Goldberger, 1995](#); [Lewis, 1994](#); [Moran, 1984](#)) of the individual throughout the course of maturation.

The range of concepts within psychoanalysis is thus enormously broad, encompassing descriptive, historical, and narrative conceptions; multiple perspectives on causation and explanation; and associated conceptions of the roles of learning, adaptation, the environment, and the physical body in mental life. Core psychoanalytic concepts can be illustrated by the central role of conscious and unconscious fantasy as a common pathway for the representation and integration of experiences.

Within child psychoanalysis, conceptual disagreements have revolved around emphasis, timing, and the terminology used to describe normal development and its relationship to clinical phenomena. In this area, the contrasting views of Anna Freud and Melanie Klein and their adherents have been central in the development of child psychoanalytic theory.

With her monograph, *The Ego and Mechanisms of Defense* (1966), Anna Freud (1895 to 1982) became a major contributor to the emerging field of ego psychology and established a new, developmental viewpoint on the broad range of mental functions (intellectualization, sublimation, displacement, reaction formation, identification, turning passive into active, humor, etc.) that individuals can mobilize to reduce anxiety, cope with psychic conflict, or modulate the pain associated with failures in the environment or in one's own achievements. Her work was closely related to the work of Heinz Hartmann, whose *Ego Psychology and the Problem of Adaptation* (1939) was the first systematic exposition of psychoanalytic ego psychology and the concept of the nonconflictual sphere of functioning (memory, reality testing, perception, etc.).

Over the course of decades, Anna Freud articulated a broad, descriptive approach to the charting of the course of children's development and the contributions of biological and experimental determinants. In particular, she emphasized the importance of direct observation and the need to relate theory to observable phenomena; she eschewed speculation about inner life and fantasies that could not be confirmed by the child's own constructions in play or speech. For Anna Freud, conflict among the agencies of mental functioning is not expressed only in symptoms but also in the course of adaptation. She conceptualized the mutually enriching roles of sexual and aggressive instinctual urges in many areas of life; these unfold in relation to the child's parents and the realities of the child's satisfactions and the quality and continuity of responsive caregiving. Anna Freud gave a role to anxiety and tensions as expectable during the course of development, with each developmental phase having modal developmental tasks and specific, normative types of anxiety. She strove to create a developmental point of view, analogous to Sigmund Freud's structural, dynamic, topographic, genetic, and adaptive viewpoints, that recognized that along with maturation there are changes in the functioning of the basic mechanisms of the mind (the functions of the ego, the available mechanisms of defense, the complexity and symbolization of fantasy, the experience of the superego and guilt, etc.). This developmental perspective distinguished disturbances of development (in the emergence of basic capacities for reality testing, memory, social relating, synchronization of functions, unfolding of cognitive abilities, and the physical apparatus) from disturbances that reflected internalized conflict and neurosis.

Psychoanalysts influenced by Klein have felt that Anna Freud was excessively interested in the “surface” markers of children's fantasy life, that she paid too much attention to the influence of the external world of the child, and that she overemphasized the role of adaptation in the course of development. In addition, Kleinian critics feel that Anna Freud undervalued the implications of primitive unconscious fantasies occurring in the first year of life and question the usefulness of her formulation in conducting “deeper” clinical work, particularly with the most severely disturbed patients. Indeed, throughout her career Anna Freud emphasized that the child patient continued to live within the family and that the realities of this life were critical to his or her psychological development, as were the realities of his or her physical and intellectual endowment. As such, the fantasy life of the child was described by Anna Freud as reflecting the mutual influences of factors from these domains and organized according to the unfolding hierarchy of mental functions.

Beginning her work in child analysis at roughly the same time as Anna Freud, Melanie Klein (1882 to 1960) emphasized that instinctual life was shaped from its initiation by the mother—child relationship, not only by the contributions of particular erotogenic zones, and that aggressive as well as sexual forces were operative from within the first months of life. Klein portrayed the inner life of infants as actively organizing their experiences of sexual and aggressive urges in specific and rich fantasies ([Klein, 1984a, 1984b](#)). She described the earliest phase of life in relation to “paranoid” feelings and being persecuted and the next phase centering around the child's “depressive” guilt for aggression directed at the mother ([Klein, 1984b](#)). The working through of this normal depressive phase was, in Klein's theory, an essential step in early development and was a continuing process at various points in life. An important aspect of working through the depressive position was not only acknowledgment of responsibility but also the psychological process of “reparation,” of making amends to the fantasied mother who was the object of the infant's attacks. Unlike for Anna Freud, for Melanie Klein, envy and jealousy as well as the components of the oedipal phase were present during the first year of life. In her broadest metapsychology, Klein adhered to and elaborated Freud's “dual instinct theory” of primary life (eros) and death (thanatos) drives and their instantiation in primitive emotional development, whereas other psychoanalysts have jettisoned Freud's conceptualization of the death instinct.

In large part, controversies stirred by Klein's work related to the precocious timetable she envisioned for the emergence of capacities for fantasy and complex conflict



and her use of pathologic terms to describe normative developmental phenomena. Her ideas, however, provide psychoanalysis with an array of clinically useful constructs concerning fragmentation, splitting, projective identification, and the central role of aggression in fantasy as substrate for experiencing the world and organizing the self. Clinicians working with severely disturbed patients (such as schizophrenics) and in situations where individuals are regressed in their functioning (such as borderline patients in analysis and psychotherapy) have found her ideas especially useful.

At present, there is no fully integrated metapsychology that systematically relates the different levels and approaches to psychoanalytic explanation. Instead, the varying theories and concepts resemble a complex historical geography, rather like an atlas in which topographic, hydrologic, agricultural, military, population, transportation, and other multicolored maps are presented, page after page. There is no single, correct map of the earth; rather, there are different maps for different purposes. Nor is there a unifying geography of the economic, dynamic, topographic, structural, and developmental contours of the mind over the course of individual development. Instead, psychoanalysis provides the explorer with varied sets of signposts and pathways, with assistance in finding one's way into and out of the otherwise overwhelmingly complex and confusing spaces of the child's inner life.

Psychoanalytic theory has perhaps made its major contributions in relation to understanding the difficulties of children suffering from emotional disorders. The validation of psychoanalytic understanding occurs most convincingly in the context of psychoanalytic therapy. In fact, much of psychoanalytic research has involved reporting clinical material from single cases or groups of children with related problems. Critics argue, however, that psychoanalytic propositions employed in treatment have been developed with a lack of scientific rigor ( [Grunbaum, 1984](#); [Kazdin, 1988](#); [Popper, 1963](#)). The broad justification for the use of single case narratives as a database for theory is that they provide a detailed account of the treatment process that is especially suitable for capturing the complexity, variation, and individuality of a particular child's inner life. The single case report generally attempts to highlight and explicate variables that are likely to be seen in other cases. Problems involved in demonstrating the generalizability of findings from single cases include: (a) reliability of the observations, (b) inability to replicate the findings by going back and looking again, and (c) how clinical material should be presented (e.g., verbatim accounts of single hours versus broad descriptive phenomenology versus an emphasis on metapsychological terminology) ( [Marans, 1989](#)). Finding ways of reliably documenting observations for critical study is an important task for psychoanalytic investigators.

For the last several decades, academically oriented child analysts have recognized the limitations of the narrative, single case report and have attempted to develop: (a) schemes for classifying and organizing data, such as the Hampstead index ( [Sandler, 1962](#)) and the Hampstead Diagnostic Profile ( [Freud, 1965](#)), (b) controlled studies of psychoanalytic treatment and outcome ( [Heinicke and Ramsey-Klee, 1986](#); [Moran and Fonagy, 1987](#)), and (c) reliable ratings of children's presentations of clinical and experimental settings using direct observation and recording of children's sessions with analysts ( [Cohen et al., 1987](#); [Marans, 1993b](#)). In a major study of over 700 hundred cases of child psychoanalysis and psychotherapy conducted at the Anna Freud Centre, charts were reviewed and assessed across multiple domains. Examining changes in *Diagnostic and Statistical Manual*, third edition, revised ( *DSM-III-R*) diagnoses and levels of adaptation as measured by the Children's Global Assessment Scale (CGAS), the study identified predictors of improvement and showed that severe or pervasive pathology requires intensive analytic treatment ( [Target, 1998](#); [Target and Fonagy, 1994, 1997](#)). In addition to this retrospective chart review approach, researchers at the Anna Freud Centre have developed a manual of child psychoanalytic treatment and devised a prospective, controlled study that examines both process and outcome in three groups of treatment subjects.

Common to each of these research efforts is the development of measures that are based on operational definitions of theoretical constructs derived from clinical observations. Continued development of strategies that combine the use of narrative process notes, videotaped clinical encounters, manual treatments, and the application of nonanalytic, standardized measures of diagnosis and adaptive functioning (e.g., the Vineland Adaptive Behavior Scales) ( [Sparrow et al., 1984](#)) further psychoanalytic efforts at investigating the developing mind of the child. As psychoanalytic knowledge continues to be enriched by detailed case reports, child psychoanalytic research has developed increasingly sophisticated approaches to systematic research methodology. However, enormous challenges remain as psychoanalytic researchers attempt to retain the breadth and complexity of the psychoanalytic conceptual system while trying to achieve greater clarity and precision in describing the phenomena associated with the inner life of the child as well as the child's experience of the external world.

In addition to its value within the field of psychotherapy, psychoanalytic theory has provided developmental psychology with many major concepts and hypotheses, including the central importance of early mother-child experiences, attachment and separation, the role of anxiety and mechanisms of defense in normal development, and the developmental point of view on emotions and internal representations of the self and others ( [Mayes, 1999](#)). Systematic research outside of the therapeutic situation has supported the heuristic value of many of the concepts of psychoanalytic clinical theory. There is thus a mutual enrichment between the clinical and empathic study of individual children and rigorous research paradigms. In the future, psychoanalytic theory will continue to be elaborated by intensive, well-reported clinical investigation with individual children, as well as by advances within developmental biology, child development, and the neurosciences ( [Fonagy, 1999](#); [Leckman and Mayes, 1998](#); [Solms, 1997](#)).

Similarly, the application of child psychoanalytic principles of development outside of the consulting room continues to lead to the creation of innovative programs and interventions that address a broad range of problems confronting children and their families. The legacy of applied psychoanalysis in the war nurseries operated by Anna Freud and her colleagues during World War II ( [Freud, 1973](#)) is continued in the pioneering work of the School Development Project initiated by James Comer, who has employed psychoanalytic principles of child development in a hugely successful program that engages parents, teachers, and mental health professionals in transforming schools into forums for enhancing self-esteem and family empowerment ( [Comer, 1991](#); [Comer and Haynes, 1990](#)). As a national model for collaboration between mental health and law enforcement professionals, the Child Development-Community Policing Program, trains police officers in psychoanalytic principles of development and human functioning and establishes a 24-hour per day consultation service for children and families exposed to or involved in violence in their homes, schools, and neighborhoods. The program extends the effectiveness of police, as well as juvenile probation officers, child protective service workers and educators in their interactions with children and families while increasing the knowledge of their psychoanalytic colleagues about the psychological phenomena associated with violent trauma and about new ways of delivering psychological services for inner-city children and families ( [Marans and Cohen, 1993](#); [Marans et al., 1998](#)). Similarly, psychoanalytic attention paid to victims of violence and abuse ( [Pynoos, 1988, 1989](#); [Terr, 1991](#); [Twemlow, 1995](#); [Twemlow et al., 1996](#)) and international work with children caught up in the terror of war ( [Apfel and Simon, 1996](#); [Garbarino et al., 1992](#)) have made enormous contributions to our understanding of the developmentally specific and complex phenomena associated with trauma and posttraumatic stress disorder. Where the work of the Robertsons and Edith B. Jackson was responsible for the ways in which we view and deal with the hospitalizations of pediatric patients ( [Robertson and Robertson, 1958](#)), the development of consultation liaison services as part of standard pediatric practice ( [Lewis, 1991, 1994](#)) continues to draw on and develop our understanding of the synergistic relationship between physical and psychological health.

The field of child psychoanalysis encompasses a broad range of clinical inquiry, theoretical conceptualizations, and their applications drawing on detailed observations of the interaction of endowment, maturational and developmental forces, affect regulation, cognitive capacities, and social relationships. Whether the psychoanalyst meets with the child in the consulting room, on pediatric wards, in institutional settings, in custody/placement evaluations, or on the streets of our inner cities, the work is defined by an appreciation of the complex relationship between the inner life of urges, conflicts, defenses, and sense of self that are represented in fantasy, modes of relating, behavior, and adaptive capacities. By attending to the details of the children's experience of themselves and their perspectives of the world and others, psychoanalysts will continue to contribute to our knowledge about the developing mind of the child and enhance the ways in which clinical practice, program development, and social policy optimize children's developmental potential.

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## 14 THE CONTRIBUTION OF TEMPERAMENT TO DEVELOPMENTAL PROFILES

Jerome Kagan

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[Three Approaches to the Study of Temperament](#)

[Thomas and Chess](#)

[Mary Rothbart](#)

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One of the certainties in the study of nature is that the meaning of the first words applied to a phenomenon will change as scientists gather new evidence and, as a consequence, invent new integrative ideas. The sense meaning of temperament, coined over 2,500 years ago, was a set of biologically based features that influenced an individual's acute affects, moods, and behaviors. Hippocrates and Galen would have called these qualities heritable if the concept had existed in ancient Athens and Alexandria. The referential meaning of temperament was limited to a small number of qualities, believed to be universal, for which there was obvious variation among individuals. The set of temperaments proposed first by Hippocrates, and later elaborated by Galen, referred to variation in affect and action profiles that contemporary scholars might call extroversion, frustration tolerance, energy level, and neuroticism. Galen called them sanguine, choleric, phlegmatic, and melancholic. These four temperamental types were created by crossing the natural properties of warm versus cool and dry versus moist, which were presumed to be based on the balance, within each individual, of blood, phlegm, and yellow and black bile. The melancholic was dry and cold because of an excess of black bile; the sanguine individual was hot and moist because of excess blood. Despite the apparent crudity of these ideas, they contain the two current features of the term "temperament"; namely, inherited biologies that influence characteristic moods and behaviors.

Although many American psychologists and psychiatrists minimized the influence of biology on behavior from 1910 to the late 1950s, scholarly return to this topic accelerated following the writings of [Thomas and Chess \(1977\)](#) and remarkable discoveries in the neurosciences. Psychiatrists and psychologists now appreciate that genetic variation can affect the function of many molecules, as well as the structure of microanatomic circuits, which modulate psychological function.

These facts have led to the reasonable suggestion that the term "temperament" refers to the many sources of variation in affect, mood, and action influenced by genetic processes that emerge early in life. The large number of molecules that affect the central nervous system guarantees that there will be a large number of temperaments. Some are obvious, like an active or a fearful child; some are subtle. For example, the concentration of neuropeptide Y in the medulla, which is believed to be heritable, influences cardiovascular function and, therefore, has implications for an individual's feeling tone ( [Stornetta et al., 1999](#)). However, the history and culture of the individual determine the interpretation imposed by someone who is experiencing feedback from the heart.

The context of development always influences the expressed behavioral phenotype. American adolescents who possess a temperament marked by relaxation, low level of arousal, minimal fear to novelty, and high sociability are likely to become leaders and vocational successes if raised in middle-class homes that reward achievement. However, children with the same temperament have a higher probability of becoming delinquents or criminals if raised in broken homes in economically disadvantaged urban ghettos. Two different personality outcomes can emerge from the same initial temperamental bias. All adult personality profiles are combinations of initial temperaments combined with the effects of experience. The only time one might come close to detecting a temperamental bias relatively free, but never completely, of experience is in early infancy before social encounters have had a chance to shape the biological disposition into a personality trait.

There have been two different approaches to the study of temperaments in infants and young children. One group of investigators adopts an empirical strategy unconstrained by strong theoretical biases. A much larger group of scientists, who adopt an a priori style, posits abstract categories based on theoretical assumptions.

[Thomas and Chess \(1977\)](#), for example, define temperament as the style of a person's behavior. [Goldsmith and Campos \(1990\)](#) regard temperament as a set of processes that modulates an emotional profile. [Cloninger \(1987\)](#) believes that the avoidance of danger, seeking of novelty, independence, and social rewards constitute primary temperamental types, although these are usually ascribed to adults. [Buss and Plomin \(1986\)](#) posit emotionality, activity, and sociability as the three major temperaments. However, emotionality in this system refers primarily to anxiety, fear, and depression rather than anger, surprise, shame, disgust, or joy. The sociability factor tends to confound a desire to be with people with the feeling of uncertainty or confidence when with unfamiliar people. The activity factor seems to refer primarily to psychological energy because the answers that best define this construct are: "I am very energetic," "I'm always on the go," and "I like to keep busy all the time."

These types are inferred from factor analyses of questionnaires that ask about qualities that can be described with words from everyday conversations; that do not violate most individuals' ethical sensitivities; and that do not probe deep beliefs, loyalty to relatives, or a preference for hot or cold foods (as Chinese psychologists might do). If Nepalese psychologists constructed personality questionnaires, they would have asked different questions and inferred different temperamental types.

Moreover, a temperamental factor cannot emerge from factor analyses unless there are a large number of questions that reflect the same type. If, for example, there is only one question that measures a significant but rare characteristic and the answer to that question is not correlated with others, it will not emerge as a temperamental factor. A factor analysis of the symptoms of all adult diseases would reveal factors for respiratory problems, cancer, and cardiovascular illness because each has a large number of correlated symptoms. Glaucoma would probably not emerge as a factor because it is less frequent and has a smaller number of symptoms. Although the work of the past 30 years has been useful, new strategies are necessary.

The definition of a personality trait as a stable disposition that is influenced by temperament is unduly permissive and could include unusual musical or athletic ability. Most scientists and clinicians do not regard these talents as having a temperamental contribution because of the implicit consensus that affect states are a salient component of every temperament. Musicians and athletes do not possess a particular set of affective states.

There is also implicit agreement that categories of psychopathology are not the products of temperamental biases, even though they are stable, under genetic control, and can emerge during childhood. For example, Williams' syndrome is not a temperamental category, despite the fact that it is owing to a chromosomal anomaly, because these patients have seriously compromised cognitive abilities. Therefore, the meaning of temperament held by most psychiatrists and psychologists refers to a stable profile of action and mood for which there is variation, a biological foundation, emergence during childhood, and a link to particular affective profiles.

It is likely that future scientists will parse the term "temperament" into different categories depending on the source of the biological variation, whether the varied brain neurochemistries, microcircuitry, or a combination of both. However, those categories remain unknown; hence, this chapter reviews what scientists have learned and accommodates to the concepts and methods characteristic of past research.

### THREE APPROACHES TO THE STUDY OF TEMPERAMENT

## Thomas and Chess

An important reason for the return of interest in temperament after World War II was the influential work of Alexander [Thomas and Stella Chess \(1977\)](#). These psychiatrists conducted lengthy interviews regularly with well-educated parents of young infants and used these data to infer nine temperamental dimensions. The nine dimensions refer to activity level, regularity of bodily functions (especially hunger, sleep, and elimination), initial reaction to unfamiliarity (particularly approach-withdrawal to unfamiliar objects or people), ease with which the infant adapts to new situations, responsiveness to subtle stimulus events, the amount of energy in the infant's behavior, dominant mood (especially whether the child is primarily happy or irritable), distractibility, and attention span-persistence. Reflection on these dimensions, supplemented by factor analytic work, motivated them to infer three more abstract profiles that they regarded as categories. The easy child, the most frequent type and comprising about 40% of their sample, has regular body activity and approaches unfamiliar objects and people with a happy mood. The second category, comprising about 15% of their sample, is slow-to-warm-up to strangers and does not adapt easily to change. The third category, about 10% of their sample, is called difficult. These children show minimum regularity, are frequently irritable, tend to withdraw from unfamiliar events and people, and are poorly adapted. These children were most likely to develop psychiatric symptoms during later childhood and adolescence.

When Thomas and Chess evaluated these subjects during early adulthood, using interviews and questionnaires, they found no relation between the possession of an easy or difficult temperament during the first two years of life and later adult adjustment, although those who had been classified as difficult during the preschool years seemed less able to cope with stress than others. Finally, Chess and Thomas introduced the important concept of goodness of fit, meaning a match between the temperament of the child and the family's notions of the ideal child. They suggested, correctly, that investigators and clinicians must know the match between the family ideal and the child's temperament in order to predict future personality development and the emergence of possible pathology.

## Mary Rothbart

Mary [Rothbart's \(1988, 1989\)](#) bold ideas penetrate contemporary discussions of infant temperament. Rothbart believes that infants vary on two primary temperamental dimensions, which she calls ease of arousal (or reactivity) and self-regulation. The signs of reactivity include both motor behavior and physiologic responses. Self-regulation refers to the processes that modulate (either facilitate or inhibit) reactivity, and they include attention, approach, withdrawal, attack, inhibition, and self-soothing ([Rothbart, 1989](#)).

The notion of ease of arousal is intuitively attractive. Infants differ in their reactivity to stimulation; indeed, even fetal movements are moderately stable over gestational weeks 20 through 35 ([Eaton and Saudino, 1992](#)). Rothbart suggests that the specific origin of the arousing stimulation, whether visual, auditory, or olfactory, is relatively unimportant in classifying an infant as high or low in reactivity. However, this claim is vulnerable to challenge because 4-month-old infants who cry in fear to a recording of an unfamiliar woman's voice without the visual support of a face are different behaviorally in the second and third years from 4-month-old infants who cry to a moving mobile but not the female voice ([Kagan, 1994](#)). [Goldsmith and Campos \(1990\)](#) reported no correlation between the infant's tendency to cry when placed on the visual cliff and to a stranger.

In addition to the importance of the incentive stimulus, the behavioral signs of reactivity are important in judging an infant's ease of arousal. Infants usually vocalize to an auditory stimulus but cry and struggle when their arms are restrained. Thus, one potential problem with the abstract concept "ease of arousal" is that it ignores both the nature of the arousing stimulus and the specific behavior provoked.

The concept of self-regulation also has problems because most infants do something when arousing event occurs; therefore, infants self-regulate to varying degrees. A 1-year-old child who retreats to his parent when a stranger enters the room may be different temperamentally from one who displays a wary face but does not retreat, even though both children are regulating the uncertainty generated by the approach of the stranger. The data suggest that the former child is more fearful in the second year than the latter. Further, it is not clear whether a child who cries to a stranger, compared with one who does not, is less able to regulate fear or is simply more fearful.

Although self-regulation appears to be a useful way to describe the infant, it may be less appropriate for older children because the cause of an emotional reaction in older children is not an external stimulus, but a cognitive interpretation. Most 2-year-old children freeze and stare when an adult dressed as a clown enters a room where they have been playing because the clown is a discrepant event, not because it is an intense stimulus. Further, it is not obvious that the immobility is a self-regulating reaction for the freezing in place may not reduce the child's level of arousal or uncertainty. Indeed, staring at the clown while clutching at the mother's hand might increase the child's level of uncertainty.

The construct of regulation is related to Freud's suggestion that humans seek quiescence and a low level of internal arousal. It is surprising that this idea remains popular, for children prefer to run rather than to sit and to explore rather than to play quietly. Freud made the idea of nervous energy—*vis nervosa*—more attractive to most readers at the turn of the 19th century when he replaced the concept of "depletion of *vis nervosa*," which 19th century physicians believed caused symptoms, with the concept of anxiety.

Because anxiety is obviously unwelcome, any coping response could be called a regulation or defense. This notion, which remains popular among psychiatrists and psychologists, renders the concept of self-regulation appealing.

## Jerome Kagan's Concepts of Inhibited and Uninhibited Children

Avoidance of or approach to novelty and challenge, a concept similar to the Thomas and Chess category of slow-to-warm-up and Bates' idea of adaptability to novelty are two popular temperamental characteristics ascribed to children after the first birthday. The empiric evidence is most extensive for sociability versus shyness with strangers.

Longitudinal data from the Berkeley Guidance Study revealed that boys who were very shy in late childhood, compared with their more sociable peers, married, became parents, and established a career later in adult life. Shy girls failed to develop a career and terminated work once they married or had a child but, unlike the males, married at normative times ([Caspi et al., 1988](#)).

Over 1,000, 3-year-old New Zealand children were rated on a variety of characteristics following a 1-hour interaction in a laboratory. About 15% of the sample was very shy, whereas 30% were sociable. Fifteen years later, at age 18, the subjects filled out a personality questionnaire. The adolescents who had been rated as shy when they were 3 years old described themselves as cautious and likely to avoid dangerous situations ([Caspi and Silva, 1995](#)).

However, it is important to note that a shy posture with children or adults can have different antecedents and, therefore, different meanings. A 4-year-old child in a social setting might play alone because the child: (a) is uncertain in an unfamiliar setting, (b) is concerned over being evaluated by the other children, (c) prefers to play alone, or (d) has experienced traumatic fear-arousing encounters with other children and therefore has a conditioned avoidance to peers. The research of Rubin and his colleagues is exemplary. [Rubin \(1993\)](#) makes a distinction between the child who plays alone and shows signs of anxiety, and an equally solitary child who is actively engaged in activities and does not show signs of anxiety. Both types are stable over time, but the former more often stare at peers and resemble children called behaviorally inhibited ([Coplan et al., 1994](#)).

[Kagan \(1994\)](#) invented the temperamental categories of inhibited and uninhibited to the unfamiliar to capture the differences between these two types of children. The former profile, which emerges in the second year, is characterized by avoidance and suppression of spontaneity to unfamiliar objects, people, and situations. It is important to note that shyness with strangers is only one feature of the category of inhibition. Inhibited children react to many different forms of unfamiliarity with avoidance, distress, or subdued emotion as they pass the first birthday and the developmental stage when discrepancy elicits uncertainty. The contrasting group, called uninhibited, is characterized by approach behavior to the same unfamiliar events. These behaviors are easily quantified, have been observed in animals, emerge early in development, and have implications for later social behavior and therefore adaptation.

## LONGITUDINAL STUDY

A large group of 4-month-old, middle-class, white infants were administered a series of visual, auditory, and olfactory stimuli ([Kagan, 1994](#)). About 20% of the infants showed vigorous motor activity, muscle tension, and frequent crying to the battery of unfamiliar stimuli. It is assumed that these infants, called high reactive, were born with a low threshold of excitability in the amygdala and its projections to the bed nucleus, ventral striatum, hypothalamus, cingulate, central gray, and brain stem.



About 40% of infants display low levels of motor arousal and minimal irritability to the same battery. These infants are called low reactive, and we assume that they were born with a high threshold of excitability in the amygdala and its projections. Of the 450 infants assessed at 4 months old, 250 of these children were evaluated again when they were 14 and 21 months old. The children came to the laboratory and were exposed to a variety of unfamiliar events including social and nonsocial incentives. About one-third of the high reactive infants were extremely fearful or avoidant, whereas only 3% were fearless. By contrast, one-third of the low reactives were fearless in the second year and only 4% showed high levels of fear. Thus, high versus low reactivity in infancy predicts an inhibited versus uninhibited profile in the second year. At 4½ years of age, the high reactives were emotionally subdued; they talked and smiled infrequently compared with the low reactives while they interacted with an unfamiliar woman examiner.

The two groups also differed in their social behavior with two other unfamiliar children of the same sex and age while the trios of children played in a laboratory playroom with all three mothers present. About two-thirds of the children who had been low reactive, but less than 10% of those who had been high reactive, were very sociable with the unfamiliar peers. However, 40% of the high reactives but only 10% of the low reactives were very shy.

These children were seen again when they were 7½ years old. First, questionnaires and telephone interviews with the mothers and teachers of the children were used to determine which children met criteria for anxious symptoms such as fear of harm, dark, animals, separation, and extreme shyness with strangers. Forty-three children who were classified as having anxious symptoms were compared with 107 control children from other temperamental groups who did not meet criteria for either anxiety, conduct disorder, or attention deficit disorder. The children who had been high reactive infants were most likely to have developed anxious symptoms—45% of high reactives, compared with only 15% of low reactives, had such symptoms. The best predictor of the development of anxious symptoms was the 4-month temperamental profile of high reactivity and not level of fearful behavior in the laboratory at 14 and 21 months of age.

Assessment at 13 years of age of another independent cohort of inhibited and uninhibited children, selected originally at 21 or 31 months, revealed that 61% of those who were inhibited in the second year, compared with 27% of those who were classified as uninhibited, were diagnosed as having social anxiety ( [Schwartz et al., 1999](#)). These two groups did not differ in degree of performance anxiety, separation anxiety, or specific phobias, suggesting specificity between possession of an inhibited temperament in early childhood and risk for later social anxiety during adolescence.

However, only 18% of the high reactive infants were consistently inhibited over all of the assessments; that is, they showed a high level of fear at 14 and 21 months, shy behavior with unfamiliar children at 4½ years of age, and anxious symptoms at 7 years. But, not one high reactive infant developed the complementary profile of consistently uninhibited, bold, and fearful behavior from 14 months through 7 years. By contrast, 29% of the low reactive infants were fearless in the second year, extremely sociable at 4½ years, and had no anxious symptoms at 7 years; only one low reactive infant developed a consistently inhibited profile. The infant temperaments of high and low reactivity constrain seriously the likelihood of developing the opposite temperamental profile.

The 23 high reactive infants who developed anxious symptoms were different from the 27 high reactives who did not have such symptoms with respect to facial skeleton, diastolic blood pressure, and magnitude of cooling of the temperature of the fingertips while listening to a series of digits they had to remember. The last two reactions imply a more reactive sympathetic nervous system. The magnitude of asymmetry in the skin temperature of the fingertips of the index fingers of the left and right hands is an index of sympathetic reactivity. A large asymmetry is the result of differential constriction of the arteriovenous anastomoses in the fingertips. The index fingers usually have much larger temperature asymmetries than the other fingers, and the asymmetry typically favors a cooler left hand. The high reactives had larger asymmetries than the low reactives.

The suggestion of greater sympathetic reactivity for high reactives is also supported by the fact that inhibited, compared with uninhibited, 5- and 7-year-old children showed greater pupillary dilation, greater cardiac acceleration, and larger changes in blood pressure to appropriate stressors. Moreover, more high compared with low reactive infants had high fetal heart rates (over 140 bpm) a few weeks before birth and higher 2-week sleeping heart rates while being held erect. Spectral analysis of the infants' sleeping heart rates while erect revealed that high, compared with low, reactive infants had greater power in the low frequency band (between 0.02 and 0.10 Hz). This result, too, suggests greater sympathetic reactivity among high reactives ( [Snidman et al., 1995](#)).

The fact that the spatial skeleton differentiated the anxious from the nonanxious high reactives is in accord with an earlier report revealing that the 14- and 21-month-old infants in this sample who had a narrow facial skeleton were more fearful in the second year than those who had broad faces ( [Arcus and Kagan, 1995](#)). These results imply that the genes that control the growth of the bones of the upper face, which are derivatives of the neural crest and therefore ectodermal in origin, are correlated with the genes that contribute to the development of inhibited behavior.

#### CEREBRAL ASYMMETRY

There is considerable evidence to support the hypothesis that the right frontal area participates more fully in behaviors linked to unpleasant emotional states. Further, visual events symbolic of threat or harm are perceived more accurately, produce larger autonomic reactions, and a larger negative wave at 400 msec in the event-related potential when presented to the left visual field ( [Roschmann and Wittling, 1992](#); [Wittling, 1990](#)). One measure of degree of asymmetry of frontal activation is the difference in degree of desynchronization of alpha frequencies in the electroencephalogram (EEG). Young children who are confronted with a stressful event that increases uncertainty (e.g., maternal absence) show greater activation on the right, compared with the left, frontal area ( [Dawson et al., 1992](#)). In addition, inhibited, compared with uninhibited, children in the third year of life showed greater EEG activation on the right frontal area under resting conditions ( [Davidson, 1994](#)), and high reactive infants who become inhibited at age 2 showed greater activation of the right frontal area at 9 and 24 months ( [Fox et al., 1994](#)).

An unpublished evaluation of our longitudinal sample at 10 years of age corroborates this claim. Asymmetry of alpha power in the EEG during baseline conditions revealed that more high than low reactives had greater activation on the right, compared with the left, frontal area at 10 years of age. Moreover, girls who showed greater activation on the right side were emotionally subdued as they interacted with a female examiner.

The basis for the asymmetry in desynchronization of alpha frequencies is unclear. It could be due either to an asymmetry in projections from limbic or brain stem areas to each of the hemispheres or to inherent differences in intrahemispheric patterns of arousal. In any case, it appears that greater EEG activation on the right compared with the left frontal area is associated with an inhibited style.

#### BRAIN STEM REACTIVITY

An intriguing characteristic of the 10-year-old children who had been high reactive infants involves the brain stem auditory evoked potential (BAER). The Wave 5 component of the BAER is generated by the inferior colliculus to a series of clicks. Activity in the amygdala can influence the excitability of the inferior colliculus and, as a result, produce a Wave 5 magnitude greater than the typical mean of about 0.3 to 0.4 mv. The high reactives, as a group, had a significantly larger Wave 5 component than low reactives ( [Woodward et al., 2000](#)). This result implies greater amygdala activity in the high reactive group.

However, some high reactives who were fearful in the second year and had a large Wave 5 were not exceptionally shy in the laboratory at 10 years of age and told an interviewer that they were neither shy nor anxious. This fact suggests that there can be dissociation between the biological processes that are the presumed foundation of a temperamental category and the contemporary behavioral phenotype.

The possibility of a dissociation between physiology and behavior is illustrated in the data from one high reactive girl who was highly fearful at 14 months, moderately fearful at 21 months, and one of the girls who cried in reflex fear when, during the 21-month assessment, a person dressed as a clown entered the room where she was playing. This 10-year-old girl had the largest frontal asymmetry value, favoring a more active right side, of the entire sample of over 100 children and displayed a Wave 5 value in the 99th percentile. But this girl was relaxed and spontaneous in the laboratory and appeared to the examiner and staff as a mature 10 year old who showed minimal behavioral signs of uncertainty or tension.

Jung acknowledged this distinction between internal biology and external phenotype when he differentiated between *anima* and *persona*. These data remind us that experience can change an early temperamental profile characterized by fear and linked to the excitability of the amygdala to a more normative profile without eliminating completely the excitability of the limbic structures that were the foundation of the infant behavior.

The data from this longitudinal study permit an evaluation of the accuracy of the maternal description of their children's behavior. The mothers of the 10-year-old children were interviewed and, as part of the interview, completed a Q-sort of their child for aspects of inhibited behavior (e.g., becomes quiet in unfamiliar places, is shy with other children and adults, worries about the future), as well as items for uninhibited behavior (e.g., makes friends easily, is easygoing, is a leader with peers, likes to try new things, remains cool under pressure). We compared the contemporary and longitudinal data for the high and low reactives who were described by



their mothers at 10 years of age as equally uninhibited. The children who had been high reactive were more fearful in the second year and, at age 10, had higher resting heart rates and were more subdued with the examiner compared with the low reactives. Thus, the maternal descriptions did not reflect the child's history or behavior in the laboratory.

## HERITABILITY

The profiles of inhibited and uninhibited to the unfamiliar appear to be heritable. Identical twins are more similar in their display of inhibited or uninhibited behavior during childhood than fraternal twins ([Emde et al., 1992](#); [Matheny, 1983, 1990](#)). The heritability of these two temperamental categories at 14 and 21 months, based on direct observations, was between 0.5 and 0.6 ([Saudino and Kagan, 2001](#)). It is relevant that adult social anxiety is three times more frequent in the first-degree relatives of patients with social phobia than in the first-degree relatives of controls.

Although genes make a modest contribution to both infant reactivity and inhibited and uninhibited profiles they also share power with experience. Over one-third of infants who had been high reactive were not exceptionally fearful in the second year and a few were fearless. Observations in the homes of 50 high and 50 low reactive, first-born infants revealed that the mother's behavior with the infant affected the probability that a high reactive infant would become an inhibited toddler. A nurturing parent who consistently protected her high reactive infant from all stressors made it more, rather than less, difficult for that child to control an initial urge to retreat from strangers and unfamiliar events. Equally loving mothers who set firmer limits for their children, by making age-appropriate demands for cleanliness and conformity, helped their high reactive infants overcome their tendency to become inhibited ([Arcus, 1991](#)). Thus, the envelope of the developmental trajectories for these two temperamental groups is not fixed. A child's experiences can mute or enhance an initial temperamental disposition. Infants born with a physiology that biases them to be high reactive at 4 months, but who subsequently experience a supportive environment that helps them overcome fear, need not become timid, shy, inhibited children.

## SOURCES OF EVIDENCE

It is a truism that the meaning of all scientific statements is a function of the sources of evidence. This maxim is also true of the work in temperament. Thomas and Chess recognized that their nine temperamental dimensions and three temperamental categories were a partial function of the nature of their evidence, which consisted primarily of interviews with parents. [Akson and colleagues \(1999\)](#) extracted two types they called temperamental from maternal verbal descriptions of 488 children at both 3½ and 4½ years of age. Application of the statistical technique called configural frequency analysis revealed that about 10% of the sample was low on control of behavior and high on both approach to novelty and irritability—these children could be similar to the Chess and Thomas category of “difficult child.” The second temperamental type, also about 10% of the group, showed the opposite profile of high control combined with low values on approach behavior and irritability—these children seem to be highly socialized and well behaved. Extremely shy compared with sociable children (inhibited or uninhibited children) were not represented in these two categories. This excellent investigation illustrates the earlier statement that all scientific claims depend on the methods used.

The strategy of asking parents to describe their children is reasonable when psychologists are initially probing a complex area. Unfortunately, parent report, whether interviews or questionnaires, is vulnerable to a number of serious biases. The most obvious is the fact that parents typically focus on qualities most relevant to the ease or difficulty of caring for the child, and these qualities may not be components of the most important temperaments.

Verbal reports, whether answers in interviews or check marks on a questionnaire, are characterized by special features that are not characteristic of the natural phenomena that the sentences are supposed to describe. First, human languages are not particularly good at describing internal feeling states. Words such as afraid, happy, and angry fail to provide information on cause, intensity, or duration. The primary function of language is to communicate information about the world, tell others how to perform skilled actions, and communicate rules of conduct. Most words describing behavior imply a static phenomenon and fail to capture the changing, dynamic nature of many events because humans are concerned with whether or not a particular goal was attained, rather than the cascade of events that led to goal attainment; however, rapid change is a primary characteristic of emotional states.

Second, a problem with verbal report is that individuals usually apply, implicitly, the contrast of good versus bad when they categorize people, objects, and events. Therefore, parents impose an evaluative construction on the behavior of their children that represents what they regard as the ideal. Specifically, a parent who values sociability and does not want a timid child is apt to deny shy behavior in his or her child. This evaluative frame influences a person's answers to all questions.

Third, words have quasilogical relations with each other. The relations between words that are antonyms are one example. The term “sweet and sour” are semantic opposites implying that both sensations could not exist simultaneously, even though there are some prepared dishes that provoke both taste sensations. Further, humans are sensitive to the logical consistency of their answers to a series of questions. A mother who reports that her daughter is usually sad will resist acknowledging that the child is occasionally happy and smiling. There is no such demand for consistency in a person's behavior, physiology, or stream of conscious feelings.

Fourth, most English words and sentences refer to discrete categories, and there are few words with blends. For example, there is no word in English that describes the feeling generated when one hopes for good news about a loved one in the hospital but fears the worst.

Fifth, every sentence has an implicit comparison idea. For example, a parent who reads on a questionnaire, “Does your child like to go to parties?” will unconsciously compare that preference with others. If one parent compares going to parties with an activity that the child dislikes, while another parent compares it with one that the child also prefers, the former is more likely to endorse the item than the latter, although both children might enjoy parties to an equivalent degree.

More important, individuals from different social class and ethnic groups do not always extract the same meaning from a word or sentence. For example, mothers who never attended college describe their infants as less adaptive than college educated parents ([Spiker et al., 1992a](#)). Mothers experiencing stress have a lower frustration tolerance and, therefore, are prone to exaggerate their infant's irritability. Depressed mothers with their first child describe their 6-week-old infants as more irritable than experienced mothers or mothers free of depression ([Greene, 1991](#)).

In one study, the parents of 9-months-old infants were asked to rate their infants on fearfulness, smiling, duration of attention, and activity level. Agreement between mothers and fathers was relatively low for these traits. Agreement was especially low for judgments of activity level because the fathers interpreted high activity to reflect a positive emotional mood, whereas the mothers of the same infants interpreted the word as implying a tendency toward anger ([Goldsmith and Campos, 1990](#)).

More seriously, scientists cannot ask parents about qualities that are not observable. There is a small group of children who are minimally irritable, smile frequently, have a low heart rate, minimal muscle tension, and show greater EEG activation in the left, compared with the right, frontal area. A psychologist evaluating the entire corpus of information would be motivated to invent a new temperamental category to reflect this combination of features. Parents do not have access to the biological information and therefore cannot be asked to rate their children on this temperamental type.

Finally, the order in which questions are asked can influence parent's answers. If a parent is asked first about a child's undesirable qualities, the replies given on the rest of the questionnaire will be different from those given if the first questions asked about the child's positive qualities ([Schwarz, 1999](#)).

These problems with questionnaires and interviews imply that those who rely only on questionnaires to study temperament will be forced to restrict their temperamental constructs to a very small number of easily understood ideas, such as level of activity, shyness, or attentiveness, and will fail to detect many significant temperaments. If Thomas and Chess had gathered extensive behavioral observations on their sample, they would have inferred slightly different temperamental categories.

One set of authors summarized the problems associated with asking parents to describe their children. What is clear from the present results is that current methods have limitations that must be recognized in order to forestall both poor decision making with respect to individual children in a clinical setting and misleading interpretations in research studies ([Spiker et al., 1992b](#)). A personal anecdote is relevant. One mother of a child in our longitudinal study who described her child as outgoing and sociable to one of our interviewers wrote a letter to our staff a few days after she had watched her daughter interact with two unfamiliar girls in one of our laboratory playrooms. This was a real eye opener for me. I always thought my child was outgoing and sociable. I now realize that after watching her that she is actually uncomfortable with new people.

This is not the first time self-reports have been criticized. Over 60 years ago, a team of child psychologists noted a poor relation between what actually happened

during the first year of an infant's life and the mother's descriptions of those events when the children were 21 months old ( [Pyles et al., 1935](#); [Yarrow et al., 1970](#)).

If talking to parents about their children's behaviors and moods were such an accurate source of information, the field of personality development would be one of the most advanced domains rather than one in disarray. Our limited progress implies that verbal statements describing the behaviors of others has some, but limited, value because those statements are partly constructions.

History casts aside popular methods in all sciences. Archaeologists now use carbon dating, not informed intuition, to establish the age of a fossil, and evolutionary biologists quantify blood proteins, in addition to anatomy, when they assign an animal to a species. The recognition that questionnaires and interviews, when used alone, are not sufficiently sensitive indexes of all the important temperamental categories should be regarded as progress. Psychologists and psychiatrists hold a deep unquestioned belief in the validity of what people say about themselves or their children. The serious limitations on verbal report should motivate scientists and clinicians to gather behavioral and biological data in addition to self-reports. New temperaments will be discovered when test performances, biological measures, and direct observations of behavior are added to the information that is gained from questionnaires and interviews.

## ARE TEMPERAMENTS CONTINUA OR CATEGORIES?

The decision to regard a temperament as a continuous dimension or a qualitative category is a controversial, but theoretically significant issue. Thomas and Chess regarded the easy, difficult, and slow-to-warm-up children as representing categories. Rothbart regards reactivity and regulations as dimensions. Most psychologists prefer continua to categories for several reasons. First, the use of inferential statistics is the mark of the sophisticated social scientist. Because correlation coefficients and analyses of variance should be computed on continuous variables, investigators find it useful to assume that the entire range of values for a dependent variable is controlled by the same set of causes and is a function of different natural or experimental conditions. Most statistical procedures, such as regression, assume that the forces that produce the distribution of values for a set of variables vary only in magnitude and act similarly on the entire range of scores.

[Meehl \(1995\)](#), who has written persuasively on the utility of considering some profiles as qualitative categories, has described an analytic technique that rests on the assumption that changing magnitudes of association between two indicators of a personality type with respect to a third indicates whether the population consists of two qualitative types or only one type. Application of this analysis to infant behavioral data from our laboratory revealed that some infants are members of a discrete behavioral category and are not on a continuum with other children ( [Woodward et al., 2000](#)). Latent class analysis is another technique that can be used to detect temperamental categories ([Loken, unpublished](#)).

The most important argument for categories over dimension is that nonlinear functions are common in the life sciences. Unique functional relations between variables often emerge at transition points because a small number of subjects with special qualities possess either high or low levels of a variable. These individuals are likely to be members of a category. One example comes from our laboratory. We examined the distribution of average standard scores for heart period and heart period standard deviation for 200, 14-month-old children during an initial sitting baseline. The distribution was divided into quintiles so that a low score represented a very high and stable heart rate (low vagal tone), while a high score represented a low and variable heart rate (high vagal tone). The frequency of smiling to an adult examiner during the subsequent hour of testing was very similar for the children in quintiles 2 through 5, but significantly less frequent for the children in quintile 1 who had the highest and most stable heart rates. Thus, the relation of heart rate to smiling was nonlinear.

## THE MEANING OF AFFECT

Because every temperamental category has an affect state as one of its features, it is important to appreciate that four different phenomena are often awarded the same emotional label. The affect of anxiety provides an illustration, but the following discussion is relevant to all current emotional concepts.

A person's verbal judgment that he is anxious, but given without the presence of any accompanying change in physiology, is one definition of anxiety. It is common for individuals to say that they are anxious over speaking in public, flying, or their health. But if scientists had measured their physiology at the time they made the statement—or even when they are in a situation they said they feared—they would not have detected any particular change in physiology. Let us call this meaning of anxiety “subjective judgment.”

A second meaning is a subjective judgment of being anxious accompanied by a change in physiology, but not the physiology that scientists believe is appropriate. For example, an individual with a viral infection may feel tense and in an attempt to understand the intrusive bodily sensations might decide that she is worried about her job. Let us call this meaning “constructed anxiety.”

The third meaning is the one scientists who work with animals use most often. An event has provoked activity in the limbic structures that are regarded as appropriate indexes of a state of fear, primarily activation of the amygdala and its projections. However, the individual either does not detect the consequences of these physiologic changes or, if detected, awards them another meaning. Let us call this “physiologic anxiety.”

The fourth referent is the one scientists and clinicians would like to believe occurs when the term “anxious” is applied to a person. An event or thought has provoked the physiologic state that is assumed to accompany anxiety and the individual detects and interprets those changes as implying that they are in an anxious state. Let us call this meaning “anxiety.”

Investigators who use questionnaires and interviews rely on the first meaning of subjective judgment; those who work with animals usually adopt the third meaning of physiologic anxiety. There is probably minimal correspondence between these two meanings. That is, the state ascribed to a rat that freezes in a place where it experienced electric shock may not resemble the state of an adult who reports on a questionnaire that he is afraid of spiders.

In addition, we must differentiate between acute emotions provoked by a transient specific event (being pushed while waiting in a line) and chronic moods that are not reactions to a specific external circumstance. Four different emotional constructs are generated by crossing the distinction between detected versus undetected changes in physiology with the distinction between acute emotions and chronic moods. Each of these four phenomena requires a different name. An adult or child temperament usually refers to a chronic mood based on a physiology that is not always detected by the individual.

One reason why there can never be a determinant relation between an underlying biology and a temperamental profile is that historical and cultural contexts influence the interpretation the adolescent or adult imposes on detected changes in physiology. The journals of the writer John [Cheever \(1993\)](#), who died in the second half of this century, and the biography of William James' sister, Alice James ( [Strouse, 1980](#)) who died 100 years before, suggested that both writers inherited a very similar diathesis that favored a melancholic, dysphoric mood; however, Cheever acquired his premises about human nature when Freudian theories were popular. As a result, he assumed that his dour mood was owing to his experiences during childhood, and he used psychotherapy to overcome the conflicts he imagined his family had created.

Alice James believed, with most of her contemporaries, that her dour mood was owing to her inheritance rather than the way she was treated. After trying many forms of somatic therapy, she concluded that because she could not change her genes she wished to die. The historical context of these two creative individuals influenced the interpretation each imposed on their feelings, the coping strategies they selected, and the quality of their emotional lives.

The effect of culture on the interpretation of one's feelings is seen in the comparison of American social phobics, who say why they avoid social situations in order to prevent feeling anxious and embarrassed over being evaluated by others, and Japanese patients, who suffer from what Japanese psychiatrists call “tiajin-kyo-fusho.” The Japanese patients, too, avoid unfamiliar people and social gatherings. However, the Japanese patients explain their behavior by saying that they do not want to cause any psychic disturbance to others. Cultures also influence the content of obsessions. The obsessive thoughts of American patients with obsessive-compulsive disorder usually focus on dirt, contamination, and aggressive and sexual intentions. The most frequent obsessions of patients living in communities in the Middle East involve religious ideas, such as blasphemy, because loyalty to the Islamic faith is a central concern in these regions.

## OTHER TEMPERAMENTS

### Infant Irritability

Variation in infant irritability, which dominates most discussions, is a popular target of study because the referent is obvious, easy to measure, of concern to American



parents, and moderately stable over time.

Differences in the frequency and duration of crying are preserved over the first few months. Irritable newborns smiled and vocalized less often to adults at 4 months than nonirritable newborns ([Birns et al., 1969](#)). Irritable infants, especially those who also show vigorous motor activity to stimuli, often develop a reserved, fearful style in the preschool years.

However, variation in infant crying is not always predictive of later behavior because the bases for the behavior change. A low threshold of responsivity to stimulation causes irritability in the infant. Irritability in a 1-year-old child is influenced in a more important way by reactivity to discrepancy. The major reasons for crying among 3-year-old children include frustrations and parental rewards for crying. Thus, investigators should not be surprised that the predictive correlation between irritability at 1 month and 3 years is low.

### Smiling

Smiling is a less frequent target of study than crying because it is less salient to parents and has no analog in animals. However, variation in this response appears to be heritable ([Freedman and Keller, 1963](#)), stable from 3 months to the end of the first year ([Kagan, 1971](#)), and infants who show greater EEG activation in the left frontal area, compared with the right, are more likely to smile ([Fox et al., 1994](#)).

The tendency to smile to nonsocial stimuli might be a component of temperament. [Kagan \(1994\)](#) observed over 500 4-month-old infants who were exposed to visual, auditory, and olfactory stimuli. Only 10% of this group smiled at least three times during the 45-minute battery. Although three smiles may seem relatively low, that value represented the 90th percentile of the distribution. When these smiling infants were matched on gender and level of motor arousal and irritability with infants who did not smile, the former group had significantly lower sitting diastolic blood pressure when they were 2 years old. This fact suggests that the high smiling infants may also inherit low sympathetic tone in the arterial tree.

### Activity Level

Level of activity, which Thomas and Chess nominated as a temperament, has one meaning when it refers to the vigorous moving of limbs, but a different meaning when children have attained motor coordination after the first birthday. Because the form and vigor of limb activity change a great deal over the first 2 years, it is not surprising that investigators do not find preservation of variation in activity level ([Dunn and Kendrick, 1981](#); [Matheny, 1983](#)). Further, there is no correlation between activity level during the day and level at night ([Korner et al., 1985](#)) and twin studies reveal minimal preservation of activity between 7 months and 3 years ([Saudino and Eaton, 1995](#)). These data imply that a concept of general activity that does not stipulate the age of the child, the context in which activity is being measured, and the time of day may not be useful.

### Attention

The tendency to devote long (or short) bouts of attention to external stimuli might be a temperamental quality. Infants differ in the rapidity of orienting and duration of sustained attention to an object or sound. Neuroscientists posit several attentional networks. One involves the parietal cortex, thalamus, and colliculus, and is modulated by noradrenergic neurons originating in the locus ceruleus that modulate attention to outside events. An anterior attentional network, involving parts of the prefrontal cortex, anterior cingulate, and supplementary motor area, influenced by dopaminergic inputs from the ventral tegmental area and basal ganglia, is more involved in the inhibition of inappropriate responses to a distracting stimulus. A third network mediates maintenance of a vigilant, alert state over a long period of time. The right lateral midfrontal cortex is important for this state and, like the posterior attentional system, is influenced by noradrenergic projections from the locus ceruleus.

Although one study of children from 1 to 3½ years of age revealed stability of variation in attentiveness to varied events ([Ruff et al., 1990](#)), another study, which quantified duration of attention to human faces and forms, revealed no stability from 4 to 27 months and only modest stability from 13 to 27 months ([Kagan, 1971](#)). Because the bases for attentiveness change from prolonged attention to discrepant events during early infancy to the activation of cognitive structures in the second year, investigators should not expect robust preservation of any index of attentiveness from infancy to later childhood.

## PSYCHOPATHOLOGY AND TEMPERAMENT

Temperament makes a contribution to differences among children in their reaction to threatening or stressful events ([Werner, 1993](#)). That is why only a proportion of children develop psychological and/or biological signs of stress to threatening or traumatic events. It is not clear if the temperamental processes that protect against a serious psychological reaction to stress do so by blunting the limbic system's initial reaction to the stressor, shortening the duration of the stressful reaction, or acting directly through inhibitory processes to mute a consciously experienced feeling that is no less intense physiologically than it is in a majority of children.

A child with an inhibited temperament is most vulnerable to the development of social anxiety rather than other types of anxiety disorders. Adults diagnosed with social phobia or avoidant disorder report feeling embarrassed over meeting others they do not know because they fear that strangers will evaluate them as possessing characteristics that deviate from the profile the community regards as desirable. This phenomenology provides a clue to a more fundamental origin of the pathology. All adults know what traits are regarded as desirable by their community and appreciate that strangers and friends evaluate other people with respect to those characteristics. Therefore, we have to explain why a small proportion of the population feel anxious over the fact that others might evaluate them in an undesirable way.

One likely answer is that these patients are experiencing dysphoric body feedback and wish to understand the reason for this feeling. Many are apt to conclude that their behaviors and appearance deviate in an undesirable way from community standards. Social phobia is disproportionately more prevalent among less well-educated women. Hence, it is reasonable to suggest that the women who are vulnerable to dysphoric feelings will conclude that they have fallen short of the society's ideal and, as a result, anticipate embarrassment when they meet strangers who might have a condescending attitude toward them. This description of social phobia implies that a more correct description of the etiology is a vulnerability to dysphoric body tone combined with the belief that one is deviant from the community ideal. An accomplished, attractive individual possessing the same temperament will not feel threatened by meeting strangers and will not become socially phobic.

Most inhibited children will not develop any psychiatric disorder ([Weissman et al., 1999](#)). We estimate that about 15% of children are both high reactive at 4 months and behaviorally inhibited at 2 years. An estimate of the prevalence of an anxiety disorder in older children is about 5%. Thus, about two-thirds of high reactive, inhibited children will not develop a profile serious enough to be characterized by a psychiatrist as an anxiety disorder. Prediction of a shy, restrained introverted personality from early inhibition is the more likely outcome.

Children with parents who have panic disorder are at slightly greater risk than others for becoming inhibited. We gathered behavioral observations on 284 children from 2 to 6 years of age. One group of 129 children had a parent with panic disorder and major depression, 22 children had a parent with panic disorder without comorbid depression, 49 children had parents with major depression but without panic disorder, and 84 control children had parents with neither anxiety nor depressive diagnoses. Behavioral inhibition was most frequent among the children of parents with panic disorder and panic combined with major depression. Approximately 29% of the children of these patients were behaviorally inhibited compared with 12% of controls ([Rosenbaum et al., 2000](#)).

## TEMPERAMENT AND THE MORAL EMOTIONS

Variation in the intensity of the moral emotions of shame and guilt is likely to be influenced by temperamental factors. The experience of these emotions, as an accompaniment to the contemplation or commission of an act that violates personal moral standards, is a continual source of restraint on asocial actions. Even though parental behaviors are important influences on the development of conscience, temperament plays a modulating role. The intensity of the experience of shame and guilt, whose foundations originate in limbic sites and project to the prefrontal cortex, is relevant.

Afferent feedback from the body, which ascends to the medulla and amygdala and arrives at the prefrontal cortex, forms the foundation of a conscious perception of one's internal body tone. Inhibited children, who have a more reactive sympathetic nervous system, might be especially vulnerable to experiencing more salient feedback and therefore more frequent dysphoria. These children are likely to learn, with age, that unfamiliar, asocial, or challenging situations produce an



uncomfortable feeling and, as a result, they will avoid these contexts.

Adolescents will want to understand the reasons for these unexpected feelings of dysphoria. A frequent first guess is that they probably violated one of their ethical standards. The list of moral lapses is so long, few will have trouble finding an ethical flaw to explain the unwelcome feeling and, as a consequence, might become vulnerable to a moment of guilt. The body tone each person lives with is so completely hidden from observers and, at the moment, so far beyond measurement, it does not enter into most theorizing.

[Damasio \(1994\)](#) has described an adult man who lost the ventromedial surface of the prefrontal cortex, which receives information from the amygdala that originates in the heart, gut, and muscles. Without this neuronal surface, individuals cannot experience anticipatory feelings of anxiety. This patient who had been an intelligent and successful adult prior to the surgery began to make impulsive decisions after the surgery despite no change in his measured intelligence.

Low reactive infants, who become uninhibited toddlers, show much less fear to an examiner's criticism compared with those who are high reactive. Further, [Kochanska \(1991, 1993\)](#) has discovered that inhibited children raised by mothers who used reasoning in their socialization developed a relatively strict conscience. Neither the form of maternal socialization nor the child's shyness, considered alone, predicted variation in Kochanska's measure of conscience development.

It is important to appreciate that almost all children are capable of the moral emotions. Although some children inherit a temperament that favors an exaggerated guilt reaction, they need not show pathology in later life. Nor is it likely that most children with a temperament favoring a less intense moral response will become delinquents. Most parents impose appropriate socialization demands on their children. Nonetheless, if the environment is permissive of aggression, stealing, and lying, the child with a temperamental bias for a diluted fear reaction to violations of standards is probably at greater risk for asocial behavior than children with a different temperament growing up in the same circumstances.

A small group of low reactive boys with high vagal tone—about 15% of boys—are a special temperamental group. If these boys grow up in typical American middle-class homes with accepting parents who socialize control of aggression, they become popular and often group leaders; however, the same boys raised by parents who are less consistent in their socialization of aggression are at higher risk for delinquency. Antisocial adolescents who show minimal autonomic reactivity (low heart rate and less frequent skin conductance reactions) are more likely to continue a criminal career than equally asocial youth with normal autonomic tone ([Raine et al., 1990](#)). It is possible that the small group of criminals who commit violent crimes possess a special, but rare temperament. A longitudinal study revealed that violent, young, New Zealand adults were likely to have been rated at ages 3 and 5 as having low control of their behavior (Henry et al., personal communication). However, a minority of impulsive, minimally fearful 5-year-old boys become adolescent delinquents. Their teachers had rated only 28% of high delinquent boys 8 years earlier as asocial ([Tremblay et al., 1994](#)). It is likely that most groups of asocial adolescents contain only a small proportion born with a temperament that placed them at risk for this profile.

## ETHNICITY AND TEMPERAMENT

Differences in temperament among varied ethnic groups remain a delicate issue because of racial and ethnic strife in America and around the world. A team of scientists compared the frequencies of over 100 different alleles for physiologic markers in the world's geographically separate human populations and averaged the differences in frequencies to create an index of genetic distance between pairs of populations ([Cavalli-Sforza et al., 1994](#)). The index of genetic distance was largest when Asians, Africans, and European-Caucasians were compared with each other. But even within European samples, individuals from Scandinavia, England, and Northern Europe were genetically different from those living in Spain, Italy, and the Balkans. The greater the geographic and linguistic distance between any two populations and therefore the greater the reproductive isolation, the greater the genetic distance. It is reasonable to assume that some of the alleles measured in this analysis make a contribution to temperament.

The most consistent set of evidence on early infant temperaments involves comparisons of Asian and white infants. Over 30 years ago, [Freedman and Freedman \(1969\)](#) reported that newborn Asian-American infants, compared with European-Americans, were less labile and more easily consoled. A decade later, [Kagan and associates \(1978\)](#) found that Chinese-American infants living in Boston, Massachusetts, were less active, less vocal, less likely to smile to stimulation, and more inhibited during the first year, compared with European-American infants from the same city. [Kagan and colleagues \(1994\)](#) administered a battery of visual, auditory, and olfactory stimulation to 4-month-old infants living in Boston; Dublin, Ireland; and Beijing, China. The white infants from Dublin and Boston were more easily aroused and distressed than the Chinese infants born in Beijing. It is important that Asian-American adult psychiatric patients require a lower dose of psychotropic drugs than European-American patients ([Lin et al., 1986](#)), implying that Asian populations may be at a lower level of limbic arousal. It is likely that when the genome project is completed, researchers will find that reproductively isolated human populations differ on alleles that contribute to temperament.

## TEMPERAMENT AS CONSTRAINT

Although relations between variables are always contingent, the tightness of any relation varies over a broad range of probabilities, from the near perfect relation between the force with which a stone is thrown and the distance it travels to the far from perfect relation between an infant's Apgar score and grade point average in high school. When the probability of one event following a prior one is high (e.g., about 0.7), it is reasonable to conclude that the prior event influenced the latter one directly. But when the probability is low (perhaps less than 0.4), it is likely that the prior event affected the later one indirectly, only for extreme groups, or in combination with other factors. When this is the case, it is more correct to use the verb "constrain" rather than "determine" to describe the effect of a prior factor on a subsequent one. Only 18% of the children who were high reactive infants became fearful in the second year, very shy at 4½ years, and developed anxious symptoms at 7 years. However, not one high reactive infant developed a complementary profile. Because more than 70% of high reactive infants did not become consistently inhibited and anxious, it is misleading to argue that a high reactive temperament determines a consistently inhibited profile and more accurate to suggest that the high reactive temperament constrains the probability of becoming a consistently uninhibited child. The term "determine" implies a particular developmental course, whereas "constrain" implies a restriction on a particular set of future outcomes. The human genome does not determine any particular psychological outcome. For that reason, we should regard each temperamental bias as imposing a constraint on the probability of developing a particular family of profiles, rather than assume that each temperamental bias determines the development of particular traits.

## EPILOG

Because every individual possesses a unique biology, some might contend that every adult profile contains one or more temperamental contributions. However, it is more profitable in this early stage of understanding to limit the term "temperament" to demonstrated predictive relations between behavioral and physiologic profiles appearing early in life that are preserved, theoretically reasonable, and predictive of a feature in adolescent or adult personality. The adolescent's personality is not a temperament because it is the product of a dozen years of experience.

The evidence implies that high and low reactivity in infancy are probably valid temperamental categories. The data are adequate enough to suggest that irritability, activity, and frequency of smiling and laughter are also temperamental categories. An important task is to find the physiologic foundations of these temperaments.

Finally, although temperamental biases affect the development of psychiatric disorders, each current diagnostic category in *Diagnostic and Statistical Manual*, fourth edition (DSM-IV) is biologically heterogeneous. The assignment of patients to their proper diagnostic group will require the gathering of biological and behavioral data, in addition to the evidence gleaned from psychiatric interviews. Adoption of this strategy by clinicians and investigators will help psychiatry make the progress that the larger community needs and expects.

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# 15 TEMPERAMENT AND ITS CLINICAL APPLICATIONS

Stella Chess, M.D. and Alexander Thomas, M.D.

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The modern concept of temperament was introduced by Alexander Thomas and Stella Chess, in the 1950s, motivated to improve our psychiatric diagnostic and treatment skills. The theoretical and clinical relevance of temperament is now accepted in psychiatry, psychology, and pediatrics, as well as education and other child- and adolescent-related fields. In this chapter, we present the history of the New York Longitudinal Study (NYLS); discuss the applications of temperament, theoretical considerations, and controversies; and update recent work.

## BASIC FINDINGS, UNDERLYING CONCEPTS, AND IMPLICATIONS OF TEMPERAMENT

Our interest in the study of temperament arose primarily from our practice in the 1940s and early 1950s. The dominant theoretical formulations attributed exclusive etiologic importance for both healthy and pathologic psychological development to environmental forces. For most the individual differences in behavioral style, evident even in the neonate and young infant; with few exceptions, these were ignored, minimized, or categorized as secondary reactive effects of the caretakers' attitudes and practices.

As clinicians, however, we saw many cases in which this one-sided environmentalist approach could not explain adequately the child's, adolescent's, or adult's personality structure or developmental course. Something was missing. We hypothesized that children took an active as well as reactive role. Development then would reflect the continuous dynamic interaction between the individual and environment at all ages.

These clinical experiences made it imperative to initiate a systematic study of individual behavioral styles. The literature revealed many pertinent reports of specific aspects of behavioral individuality, but none that had attempted a systematic comprehensive study of this phenomenon or provided a methodology adequate for such an endeavor (Thomas et al., 1968). It was clear to us that a prospective longitudinal study was required, from which we could gather data of sufficient scope and pertinence to test our hypothesis; therefore, we launched the NYLS in 1956.

We conceptualized temperament as representing the *how*, or style of behavior, as contrasted to the *why*, or motivations and goals of behavior, and the *what*, or perceptions, abilities, and talents of the individual. Two children may dress themselves or ride a bicycle with the same dexterity and have the same motives for engaging in this behavior. Two adolescents may display similar learning ability and intellectual interests, and their academic roles may coincide. Two adults may show the same reason for devoting themselves to their jobs. These behaviors reflect their motivations and abilities. Yet these two children, adolescents, or adults may differ significantly with regard to the quickness with which they move; the ease with which they approach a new physical environment, social situation, or task; and the effort required by others to distract them when they are absorbed in an activity. These variations reflect differences in their temperament characteristics. Or, in other instances, temperaments may be similar, and abilities or motivations may differ. This analysis of behavior into the *how*, *why*, and *what* also had been suggested by several developmental psychologists, especially Cattell (1950) and Guilford (1959), and a number of important studies of motivations and abilities had been undertaken; however, investigations of the nature and significance of temperament and the relationship of temperament to motivations were still lacking.

### Methodology

Our research into temperament required an anterospective longitudinal study, gathering behavioral data on the same group of children at sequential age periods. Cross-sectional studies cannot trace the developmental course of individual subjects over time. Only a longitudinal study, which follows the developmental course of the same group of subjects over time, can hope to explore individual patterns of change and continuity and their meaning.

A prospective study, in which data are collected at the time or very close to the time of their occurrence, also is essential. Studies have revealed significant distortions in the accuracy of parents' retrospective reports on the early behavioral history of their children, even when development has been normal (Haggard et al., 1963; Robbins, 1963; Wenar, 1963). The problem is the same with adults' recall of their earlier life. Vaillant reports this vividly from his findings of the Harvard Grant Study:

How then may we obtain truth about the adult life cycle? Clearly, it must be studied prospectively. It is all too common for caterpillars to become butterflies and then to maintain that in their youth they had been little butterflies. Maturation makes liars of us all (Vaillant, 1977, p. 197).

### The Parent as a Source of Research Data

In starting the NYLS, we had to determine the nature and source of our raw data. It became evident that the primary child caregivers, usually the parents, were indeed a unique and comprehensive source of information on their infants' behavioral styles. The parents, with their continuous involvement with their infants, could observe and report to us on details of their babies' behavior within all the routines of daily life, the child's sequence of reactions to anything new and different—the first bath, a new food, a stranger, an illness, a move to a new home. An outside observer coming into the home for a few hours could catch only a slice of the overall behavioral repertoire of the child and that slice might not even be typical if some unusual situation and stimulus had just occurred.

To avoid parental bias, our interview protocol focused on detailed, factual, and descriptive items of behavior, especially new events. The description of the child's initial reaction to the new was followed by questions such as, "What did you (the parent) do when the baby responded so-and-so?" "How did the baby respond to what you did?" "What did you do next?" and so on until the whole sequence of the child's reaction to the new and the parent's handling of this reaction had been reported. In this way, we obtained richly detailed data not only on the child's behavior but also on the parents' patterns of childcare. Our initial interview with the parents was scheduled when the child was 2 or 3 months of age.

The sample comprised middle- and upper-middle-class families living in the New York area. A homogeneous sociocultural group minimized the influences that could be introduced by substantial sociocultural variability. Our study protocols did include such phenomena as trauma, unusual events, and idiosyncratic parental attitudes. The use of a relatively homogeneous sample provided a baseline for generalizing the findings to other populations of different economic, cultural, and racial status. We initiated a second longitudinal study of 95 children born in New York of unskilled and semiskilled working-class Puerto Rican families. This group was also followed longitudinally from early infancy through childhood, using the same approach to data collection and analysis as in the NYLS. The reports in the discussion that follows are from our major study, the NYLS.

### Data Collection

Our NYLS sample comprised 129 children from 80 families. There was a retention rate of 97% over this 25- to 30-year period with 129 subjects and their parents. Our semistructured parent interviews were conducted at 3-month intervals during the first 18 months of life, at 6-month intervals until 5 years of age, and yearly until 8 or 9 years of age. The subjects and their parents were interviewed separately in adolescence, and the same procedures were followed in the 18- and 22-year age period. In the follow-ups in the mid- and late twenties and again in the thirties, only the subjects were interviewed. Data were also collected yearly through interviews and observations from nursery school, kindergarten, and first grade. Standard psychometric testing was carried out at ages 3 and 6. A special structured interview to elicit information regarding parental practices and attitudes was held in the child's home with the mother and father, separately but simultaneously when each child was 3 years old.

### THE CLINICAL SAMPLE

A major goal of the NYLS was the determination of the functional significance of temperament for the origins and evolution of behavior disorders. Psychiatric consultation and advice were provided at any point in the child's development.

A full clinical evaluation was offered if the child's undesirable patterns of functioning persisted or became worse. Neurologic or psychological studies were arranged as needed. Teachers were reinterviewed for school problems, and additional observations were done in class. All diagnoses correspond to *Diagnostic and Statistical Manual*, third edition, revised (DSM-III-R). The child's anamnesis was reviewed following the diagnostic evaluation. The formulation of the dynamics of pathogenic child-environment interaction, and the ontogenesis of the behavior problem were derived from a composite of clinical and research information. A decision was made as to direct treatment of the child, parent guidance, or other patterns of therapeutic intervention, and then discussed with the parents. A yearly follow-up was discontinued only if the child showed a sustained recovery.

### Data Analysis

Our data collection was primarily qualitative: parent and teacher interviews, narrative description of the child's behavior at school and during an IQ test, and special interviews with the parents when each child was 3 years old. In other words, our primary data were not obtained through scores or other methods of quantitative ratings. We focused on meaningful subtleties in the developmental course of individual children. It also permitted the raw data to be reexamined.

Herbert Birch then conducted an inductive content analysis of the four parent interview protocols of each of our first 22 NYLS subjects for the first year of life. Blind to our postulated temperament traits, he defined nine categories of temperament, established scoring criteria for each category, and determined a method of rating each item of behavior in each interview. His unique contribution served to transform our narrative data into categories with precise definitions and criteria for the scoring of behavioral items that could then be rated quantitatively for each temperament category.

### TEMPERAMENT CATEGORIES

The nine categories and their definitions are as follows:

1. Activity level: The motor component present in a given child's functioning and the diurnal proportion of active and inactive periods.
2. Rhythmicity (regularity): The predictability and/or unpredictability in time of any function. It can be analyzed in relation to the sleep-wake cycle, hunger, feeding pattern, or elimination schedule.
3. Approach or withdrawal: The nature of the initial response to a new stimulus, be it a new food, toy, or person. Approach responses are positive, whether displayed by mood expression (smiling, verbalizations, and the like) or by motor activity (swallowing a new food, reaching for a new toy, active play, and so on). Withdrawal reactions are negative, whether displayed by mood expression (crying, fussing, grimacing, verbalizations, or the like) or by motor activity (moving away, spitting new food out, pushing new toy away, and so forth).
4. Adaptability: Responses to new or altered situations. One is not concerned with the nature of the initial responses but with the ease with which they are modified.
5. Threshold of responsiveness: The intensity level of stimulation necessary to evoke a discernible response, irrespective of the specific form that the response may take or the sensory modality affected. The behaviors utilized are those concerning reactions to sensory stimuli, environmental objects, and social contacts.
6. Intensity of reaction: The energy level of response, irrespective of its quality or direction.
7. Quality of mood: The amount of pleasant, joyful, and friendly behavior, as contrasted with unpleasant, crying, and unfriendly behavior.
8. Distractibility: The effectiveness of extraneous environmental stimuli in interfering with or altering the direction of the ongoing behavior.
9. Attention span and persistence: These two categories are related. Attention span concerns the length of time a particular activity is pursued by the child. Persistence refers to the continuation of an activity in the face of obstacles.

Qualitative analysis, supplemented by factor analyses, led us to formulate in addition three constellations of temperament made up of various combinations of the individual categories, which appeared to have functional significance. These are:

1. Easy temperament: Typically, this comprises the combination of biological regularity, approach tendencies to the new, quick adaptability to change, and a predominantly positive mood of mild or moderate intensity (approximately 40% of the study population).
2. Difficult temperament: This is the opposite of easy temperament, namely, biological irregularity, withdrawal tendencies to the new, slow adaptability to change, and frequent negative emotional expressions of high intensity (approximately 10% of the study population).
3. Slow-to-warm-up temperament: This category comprises withdrawal tendencies to the new, slow adaptability to change, and frequent negative emotional reactions of low intensity. Such individuals are often labeled "shy" (approximately 15% of the study population).

Temperamentally easy children typically adapt quickly and positively to new situations and demands. The temperamentally difficult child, by contrast, although normal, often finds adaptation to the new distressing and stressful. The slow-to-warm-up child may also present difficulties in management, but her or his negative reactions to new foods, places, or people are expressed mildly, rather than with the violent intensity of the difficult child. Caregivers and teachers can usually tolerate this slow-to-warm-up behavior and give the child time to make a gradual adaptation to the new.

As can be seen from the percentages given in the preceding, not all children fit into these three temperament groups. This results from the varying combinations of temperament traits manifested by individual children. Also, there is a wide range in degree of manifestation among those children who do fit one of these three patterns. Some are extremely easy children in practically all situations; others are relatively easy in some situations but vary in their reactions to others. A few children are extremely difficult with all new situations and demands; others show only some of these characteristics, and only relatively mildly. For some children, it is highly predictable that they will warm up slowly in any new situation; others warm up slowly with certain types of new stimuli or demands but warm up quickly with others.

It should be emphasized that the various temperament categories and constellations all represent variations within normal limits. Any child may be easy, difficult, or slow-to-warm-up temperamentally, have a high or low activity level, be distractible with low persistence or the opposite, or show any other relatively extreme rating score for a specific temperament attribute. Such amodal ratings are not criteria of psychopathology but rather an indication of the wide range of behavioral styles exhibited by normal children, adolescents, or adults.

We have been able to rate our NYLS subjects on the nine categories and three constellations at all age periods in childhood, adolescence, and adult life. As the patterns of behavior become increasingly complex at sequential age periods, the criteria for rating temperament reflect behavioral changes. Thus, behavioral items for scoring temperament in infancy focus on sleep and feeding schedules, the first reactions to the bath, adaptation to new foods and people, loudness and frequency of

crying and laughter, distractibility during feeding, and so on. During the toddler stage, data collection concentrates on peer reactions, play patterns, distractibility, and persistence when playing with a new game or toy. In the older child, the adaptation to school, parties, family, peers, play, and task-oriented activities are of interest. In the adolescent and adult, the identification of temperament is more complex because of the increasing individual variation in activities, such as athletics, hobbies and special interests, social life, school curriculum, and job experiences.

Temperament can be rated in other populations we have studied: children born in New York of unskilled and semiskilled working-class Puerto Rican parents; premature children with birth weights ranging from 1,000 to 1,750 grams; children with mildly retarded intellectual levels; and a large group of handicapped children with congenital rubella.

Having presented convincingly through these studies that temperamental individuality was both a reality and of importance in normal and problem development, pediatricians, nurses, psychologists, psychiatrists, educators, and child care workers evidenced interest in research and practical application. The NYLS method of detailed interviews, although necessary for the initial study, soon proved impractical for large cohorts and clinical situations. Carey and colleagues initiated a series of questionnaires. At this time, questionnaires, covering ages 1 month through 12 years, are available from Sean McDevitt (Behavioral Initiatives, 14634 N. 55 St., Scottsdale, AZ 85254) or Robin Hegvik ([Tempera Metrics, 243 Cohosset Lane, West Chester, PA 19380](#)). Cameron and associates, Rothbart and Bates, and others have developed alternative temperament questionnaires and videotapes.

### The Validity of the Parents' Reports

Controversy exists in the temperament research field as to the accuracy of parents' reports of their child's behavior, whether by interview or questionnaire. Some parents have subjective biases, which may distort their perceptions and reports. On the other hand, the mother and father are in the special position of being familiar with the child's behavior over long periods of time. This special knowledge is shared with teachers and pediatricians, who can compare it with the typical behavior of children under their care. Two independent trained observers, on separate days, observed 18 infants at home for 2 to 3 hours each, and within 2 weeks of a parent interview. These observations were scored for temperament, using the same definitions as for the parent interviews. A significant positive correlation was found between observer and parent reports ([Thomas et al., 1963](#)).

A number of reports from other centers ([Matheny et al., 1995](#); [Rothbart and Bates 1998](#); [Rutter, 1987](#)) have indicated a strong objective component in the mother's report consonant with the findings of an objective observer (Crockenberg and Acredolo, 1983; [Vaughn et al., 1981](#)). In our judgment, the parents' report still represents the best single source of information on the child's temperament, when the data supplied are concrete, descriptive reports of behavior. Studies of specific biological markers for several temperament traits are discussed in the following. Reports from teachers and pediatricians who have experienced repeated contact with children are another valuable source for temperament data. [Weisz and Sigman \(1993\)](#) have observed that "assessing parental reports may be a particularly useful first step because parents have a particularly comprehensive observational base (i.e., more exposure to the child across more settings than, say, teachers or trained observers)." Rothbart and Bates emphasize this issue: "Parents are likely to be in a good position to observe the child's behavior, especially infrequently occurring behavior that is nevertheless critical to defining a particular dimension of temperament" ([Rothbart and Bates, 1998](#)).

### Goodness or Poorness of Fit

Stella Chess did a clinical study for each subject who required evaluation. She added already existing temperament data. We identified expectations and demands on the child. From the evaluations that traced each child's healthy or pathologic psychological development, we formulated our conceptualization of "goodness of fit" or "poorness of fit." We postulated that goodness of fit results when the expectations and demands of the parents or others in the environment are in *consonance* with the child's temperament characteristics and capabilities. With such a goodness of fit, a child's optimal development in a progressive direction was enhanced. Conversely, poorness of fit involved discrepancies and *dissonances* between environmental opportunities and demands and the child's capacities and temperament characteristics. Distorted and maladaptive functioning was potentiated under such circumstances. Goodness or poorness of fit was always determined in terms of values and demands of a given culture or socioeconomic group; it was never an abstraction.

This does not imply advocating eliminating all stress and conflict—quite the contrary. They are inevitable concomitants of the developmental process, in which new expectations and demands for change and progressively higher levels of functioning occur continuously as the child grows older. Demands, stresses, and conflicts, when consonant with the child's developmental potentials and capacities for mastery, have constructive consequences and should not be considered causes of a behavior disorder. The issue involved in disturbed behavioral functioning is rather one of excessive stress resulting in poorness of fit.

Researchers and clinicians in a wealth of illustrations can easily see this concept of a dynamic integration between temperament and environment as conceptualized by goodness or poorness of fit ([Chess and Thomas, 1999](#)); for example, we can spell out the type of fit through the temperament category of motor activity level. The high-activity child whose outlets for exercising muscles are insufficient becomes restless, impulsive, and difficult to manage. Such a child is a nuisance at home, and a disciplinary problem at school if required to sit for long periods of time. All too often, a harassed teacher may criticize the child when this happens, intensifying the disturbance. What emerges is a derogatory self-image. This sequence is an outcome of significant poorness of fit; however, the story is different if thoughtful parents and teachers recognize the child's need for adequate motor activity as legitimate. The parents can seek programs that emphasize active play and not expect the child to sit uninterrupted through a long religious attendance or prolonged automobile ride. The teacher also can give the child class responsibilities with motor activity, such as inventing active errands when noticing that the youngster is particularly restless.

The importance of cultural expectations in the consequences for high-activity level children was dramatized in the contrasting findings in the middle-class NYLS children and the Puerto Rican working-class (PRWC) children living in the congested and underprivileged East Harlem section of New York City. Of the PRWC children with behavior disorders before the age of 9, half had high activity levels. They presented symptoms of excessive and uncontrolled motor activity. By contrast, only one NYLS youngster displayed these symptoms, and this was a brain-damaged child. The PRWC children were more likely to be cooped up at home because of more children and small apartments. Safe playgrounds and recreational resources were meagerly available for these families, and the many household responsibilities did not permit mothers to sit for long periods safeguarding their children's outdoor play. High-activity children were exposed to poorness of fit, excessive stress, and a high risk for behavior disorder development. By contrast, the NYLS families provided their highly active children with ample space and after-school programs. Consequently, clashes with caregivers on this score were few, and a goodness of fit with low risk for pathogenic excessive stress existed.

Any amodal child's temperament trait or constellation, such as the difficult or slow-to-warm-up patterns, may endow daily behavior episodes with much potential for confrontation. Some parents may label the youngster as a psychopathologic or "bad" child. If such a parent or other authority insists that the child conform speedily and fully to the culturally expected norm, excessive stress and a poorness of fit are created. Parents who understand the child's temperament characteristic accept the fact that the behavior is basically normal and healthy. Through patience and the type of helpfulness to the child that conforms both to temperamental necessities and minimum cultural expectations, excessive demands are not imposed, and a goodness of fit results. Goodness or poorness of fit at times can also involve issues other than temperament. Dissonances may emerge from inaccurate parental views of their child's intelligence, talents, motives, and so on.

### Consistency and Change in Temperament Over Time

Our search for categories and ratings of individual differences in temperament initially started with infants and young children. We assumed that specific individual temperament characteristics would remain constant throughout life. The boisterous baby would become the vigorous adult; the shy child would become the quiet grown-up; the youngster who moved toward most new activities would be the "joiner" rather than otherwise in middle childhood, adolescence, and maturity.

As our young children became older, however, we have found that such continuities are not always the case. And, in reflection, how could it be so? All other psychologic phenomena, such as intellectual competence, coping mechanisms, adaptive patterns, and value systems, can and do change over time, as well as other psychological factors, as would be inevitable from our fundamental commitment to an interactionist viewpoint, in which individual behavioral development is conceived as a constantly evolving and changing process of organism-environment interaction over time. Routine functioning may blur a child's earlier characteristic expression of temperament. Infants who showed marked withdrawal from the bath, new foods, and new people may later show positive responses because of repeated exposure and final adaptation. At subsequent age periods, if the youngster experiences few new situations, then his or her withdrawal reactions will not be evident. If child or adolescent is exposed to a number of unfamiliar situations simultaneously, then the withdrawal behavior reactions may be evident again.

When the youngest of the NYLS sample had reached 5 years old, we examined this concept of the consistency of temperament over time ([Chess and Thomas, 1996, 1999](#)). We found significant correlations from one year to another for almost all temperament categories, but the number of significant correlations decreased as the time span increased. Qualitatively we have found both consistency and change in temperament over the years, reflecting changing environment. A recent analysis



compared the temperament ratings of the NYLS sample in early childhood with the ratings of the early adult life follow-up period ( [Thomas and Chess, 1984](#)). The difficult–easy temperament category at age 3 related to early adult life disposition, whereas other temperament categories did not. Some individual subjects suggested significant and sometimes even dramatic consistency in one or another temperament category from early childhood to early adult life ( [Chess and Thomas, 1999](#)).

The subjects of the NYLS had now been followed into their mid- and late thirties. As of January 2000, Lerner had examined data for 20 of the subjects, 10 females and 10 male. Variables examined as factors influencing both change and consistency were: (a) self-awareness, (b) social cognition, (c) motivation, (d) self-esteem, (e) support network, and (f) fortuitous life events. Ages examined were 3 to 3½ years, adolescence, and early adulthood. Ninety-seven percent of the population of the 129 subjects was retained. Trends of data analysis to date show that self-awareness and social cognition increase with age. [Halverson and Deal \(2001\)](#) found stability at the group level but considerable change at the individual level between age 4 and 7.

[Clarke and Clarke \(2000\)](#) summed up their extensive studies and those of other researchers into predictability over time of such characteristics as cognitive development, attachment, and temperament. The data they report reaffirms their earlier thesis that

. . . there is little indication that any one point of development is more critical than another; all are important. And in the ongoing shaping and reshaping of the person's life path, it is to continuing influences that significance must be ascribed (p. 105).

They find continuities only if there are environmental continuities in both cognitive development of retarded children and the influence of early attachment to quality of later relationships.

### Biological Issues in Temperament Research

A number of findings led us to the concept of a biological origin for temperament. Characteristics in temperament are apparent within the first few months of life, become more evident by 1 year, and are dramatic by 2 years. No consistent qualities in parental practices and attitudes have been found to account for this variability in temperament ([Thomas and Chess, 1977](#)). We also compared the temperament scores of our NYLS infants with those in our working-class Puerto Rican families—two groups with marked culturally divergent approaches to child care—and found no dramatic differences in temperament distribution ( [Thomas and Chess, 1977](#)). A literature review by [Korner \(1973\)](#) indicated that parents treat girls dissimilarly from boys even in infancy. Our study and others found only very modest sex differences in temperament scores in boys versus girls, indicating that temperamental individuality in young children is little shaped by any variability in parental attitudes and practices based on gender ([Thomas and Chess, 1984](#)).

These studies challenge the idea that parental attitudes and practices determine infant temperament. Twin studies have demonstrated a genetic factor in shaping individual likeness and unlikeness in temperament, using the classic method whereby the intrapair differences in a group of monozygotic twins, who are 100% genetically similar, are compared with the intrapair differences in a group of same-sex but heterozygotic twins, who are 50% genetically similar on average.

In one study conducted in Bergen, Norway, a sample of 50 same-sex twin pairs was collected at birth ( [Torgersen and Kringlen, 1978](#)). On the basis of blood typing, 34 pairs were identified as monozygotic and 16 as dizygotic. At 2 months of age, there were statistically significant intrapair differences in three temperamental categories, whereas at 9 months, all nine categories showed statistical differences. In all categories, the monozygotic twin pairs were more similar to each other than were the dizygotic twins. The hypothesis was that the residual effects of the birth process had disappeared by 9 months, at which time the definitive expression of temperament was clearer. Buss and Plomin (1975) and [Plomin and associates \(1997\)](#) reported that in a sample of 139 pairs of same-sex twins the intrapair correlations were significantly higher for the monozygotic versus the dizygotic twins for these temperament categories with genetic origin. In addition, a long-term longitudinal study of twins, initiated in 1971 at the University of Louisville, has found that initial analysis, performed at age 12 months, showed monozygotic twins more concordant than dizygotic twins for individual temperaments. This suggested a genetic influence on developmental change itself ( [Wilson and Matheny, 1986](#)).

Other biological nongenetic facts, such as hormonal or other aspects of the prenatal environment or birth process, also may be important. Genetic influence in no way implies fixed predetermination and immutability of temperament. Phenotypic characteristics, as emphasized by modern geneticists, are always the final product of the continuously evolving interaction between genetic and environmental factors ( [Dobzhansky, 1962](#)).

Jerome [Kagan and associates \(1988\)](#) initiated a pioneering study of the relationship of temperament to various physiological and hormonal factors at Harvard University.

### Temperament and Personality

We do not advocate a temperament theory of personality ( [Thomas et al., 1968](#)). Temperament is one of the significant factors in development, but is not identical with personality. In the very young infant, temperament characteristics seem to be the whole personality; over time a host of other factors enter, all of which contribute to personality development. Judd Marmor, a leading student of personality theory, has enumerated 14 types of variables that must be considered. He concludes, “We begin to get a glimpse of how difficult it is to accurately trace the origins of specific personality patterns at all, let alone to try to derive them from just one or two variables” ([Marmor, 1983](#), p. 856).

### Practical Implications

The functional significance of temperament holds the promise for the prevention, management, and treatment of many behavior disorders of children. Parents, mental health professionals, teachers, pediatricians, and nurses can identify and recognize the behavioral individuality of children's temperament characteristics ( [Chess and Thomas, 1999](#)). For the parents, knowledge of the child's temperament may reshape guilt into effective responsibility so that they may strive to reach the optimal parenting style for each pattern of temperament. Clear-cut objective insight into the child's temperament and its implications is now substituted for speculative assumptions of complex psychodynamic mechanisms. Branding a youngster as “sick” or “disobedient” or other negative judgments distort the child's self-image, create inappropriate defenses, and lead toward a self-fulfilling prophecy. If parent or teacher respects the child's behavioral style, he or she can then learn self-confidence and gain mastery of his or her particular behavioral individuality.

The paradigm of goodness or poorness of fit provides a framework for the prevention of treatment of childhood behavior disorders through the valuable tool of parent guidance. The rationale of such a guidance procedure is first to collect the data and analyze the dynamics of goodness or poorness of fit. Specific areas are identified as the centers of unhealthy parent–child interaction, these are described to the parents, and alternative handling is outlined. Half of the parents of behavior problem children in the NYLS were eager and able to carry through the recommended program of behavioral change. Only several discussions were needed for its full implementation. The other parents rejected a temperament interpretation, insisting on a malevolent motivation, and pursued an unchanged approach. In these cases, the child continued to respond to the excessive stress and developed or retained a behavior disorder ( [Chess and Thomas, 1999](#)).

Inevitably, the guidance sessions revealed misconceptions, confusion, defensiveness, anxiety, or guilt in a number of cases. With additional clarifying discussions, parental attitudes and practices that interfered with the child's desirable development were clarified. Some parents responded positively to this new knowledge, but other parental attitudes were so rigidly fixed as to prevent changes in the daily handling of their child. In these cases we recommended direct psychotherapy for the child.

### Longitudinal Studies

There have been several large population prospective longitudinal temperament studies; some are ongoing. Because of their size and prospective nature, they are summarized in the following:

1. Quebec City, Canada. Michel [Maziade and associates \(1990a, 1990b\)](#) explored the clinical relevance of temperament. Their basic population was all the second-grade pupils (97%) in Quebec City with controls. Their 1990 report indicates that children with early extreme temperament in dysfunctional families displayed more clinical disorders at adolescence than the remainder of the 980 adolescents.
2. In the Helsinki Longitudinal Study, Matti Huttunen and colleagues, from the public health centers for the year 1974 to 1975, enrolled 6,401 children born to 6,332 mothers. All social classes were included. Data obtained were: (a) a pregnancy questionnaire at each prenatal visit on somatic and mental health, (b) temperament questionnaires at age 5, (c) an adjustment difficulty scale at age 12, and (d) a teacher behavior rating at age 12. The most recent behavioral data

([Teerikangas et al., 1998](#)), indicated that a fussy and demanding temperament in infancy protected the subjects from developing psychiatric symptoms in adolescence. This correlates with our NYLS experience that difficult children who recovered from early behavior disorder fared well. Roy Martin and colleagues found correlations between nausea during mid- and late pregnancy and temperament in infancy with lowered sensory threshold and heightened activity and emotional intensity. At age 5, the correlation of the pregnancy reports was with low task performance as reported by teachers ([Martin et al., 1999](#)). Martin and associates have also correlated maternal distress during pregnancy and child negative emotionality and behavioral inhibition at age 5. They provide data suggesting a relationship between season of birth (and hence of maternal viral infection during pregnancy) with potentially pathogenic consequences (Martin et al., in press).

3. The Australian Temperament Project promises to be the largest and most unified of multicultural studies on temperament. Initiated in 1983, a cohort of 2,443 infants aged 4 to 8 months was enrolled in the state of Victoria. The main investigators, Oberklaid and colleagues, are based at the Royal Children's Hospital in Melbourne, Australia. The major aims are to: (a) delineate, measure, and determine the stability of temperament; (b) examine the relationship between indices of temperament and concurrent and later behavioral adjustments; (c) examine the significance of social class, ethnicity, and family relationships; (d) examine sex differences in temperament and behavioral adjustment; and (e) identify "at-risk" groups in early childhood to follow through the early school years. The Thomas and Chess conceptualization was the basis of temperament data collection. The authors' (Sanson et al., 1989) summary report found moderate stability of temperament from infancy to preschool, greater stability at both the extreme easy quartile and extreme difficult quartile, indicating that the children with more moderate temperaments may be more influenced by environment. Using the same cohort of 2,443 infants, the authors ([Prior et al., 2000](#)) reported prediction from childhood shyness to adolescent clinical anxiety to be modest for the group. Most of the shy children did not develop anxiety disorders, and most of the adolescents with anxiety disorders had not been especially shy as younger children.
4. An impressive contribution to temperament research and its clinical application has been made by Savita Malhotra, in a series of studies starting with the devising of a culturally appropriate interview guide in Hindi in 1983, and presently ongoing, from the Department of Psychiatry at the Postgraduate Institute of Medical Education and Research (Chandigarh, India). The NYLS interview has been factored and standardized as modified by the Indian colleagues. Although the factors resemble some of those published by Western researchers, such as Bates and Rothbart, their contribution to risk factors is very different. For example, the risk of the constellation of the difficult child is low in the rural culture with a flexible time schedule with few novel situations requiring adaptations. High emotionality (negative mood and high persistence) placed a high risk for psychiatric problems. Temperament relatedness to psychiatric illness such as conduct and conversion disorders, children of mentally ill, and addicted parents were also explored.

### Application of Temperament in Education

[Keogh and colleagues \(1997\)](#) found that children with learning delays and difficult temperament elicited more attention and teacher interactions than those with easy temperament. The difficult children consequently developed better cognitive skills.

[Nelson and coworkers \(1999\)](#), [Rothbart and colleagues \(1998\)](#), [Teqlasi \(1998\)](#), and [Keenan and associates \(1998\)](#) all found correlation between early child temperament and school behavior. Increasing attention is now being given to the inclusion in educators' training of a basic understanding of the development of temperament and individual differences in children's emotional reactivity and attention self-regulation ([Rothbart and Jones, 1998](#)).

### Definitions and Conceptualization of Temperament

The studies of temperament in East European centers have been traditionally shaped by the Pavlovian biological concept of strength versus weakness of the central nervous system ([Strelau, 1983](#)). With his neo-Pavlovian view, [Strelau \(2000\)](#) emphasizes "reactivity as a primary feature of temperament," and the classification of reactivity is based on the concept of individual differences in both degree and intensity (magnitude or amplitude) of expression. Andrzej [Eliasz \(1990\)](#), who is now rector of the Warsaw School of Advanced Social Psychology, and in his "Transactional Model of Temperament" modified Strelau's theory, pointing out that the role of environment in shaping a person's temperamental characteristics is also salient in addition to the genetic roots of temperament. The very important element of his understanding of temperament and its impact on people's functioning is the concept of the goodness of fit. In that way, he bridges the tradition of studies launched by [Thomas and Chess \(1977, 1984\)](#). The outcomes he collected with his associates show that, depending on person-environment fit or misfit: (a) personality mechanisms (resulted mainly from influences of social environment) could be congruent versus incongruent with temperamentally shaped capacities of persons or (b) internal incongruence could lead to disturbances in social interactions and/or health ([Brandstaetter and Eliasz, 2000](#); [Eliasz and Ansletner, 2001](#)).

### Difficult Temperament (the Difficult Child)

We have called attention over the years to the special functional significance of the temperament cluster we have termed "the difficult child," starting with our first detailed report ([Thomas et al., 1968](#)). The constellation of irregularity, withdrawal tendency, predominantly negative mood, slow adaptability, and high intensity appeared in about 10% of the NYLS population, but represented about 24% of the behavior disorders that emerged up to age 9. By age 9, 70% had developed a behavior disorder and 30% had not ([Thomas et al., 1968](#)). This indicated to us that, in middle-class Western society, the temperament features of this group resulted in poor fit in meeting cultural demands for socialization and task mastery at home, in school, and/or with the peer group. We found that the 30% functioning well had been provided with opportunities to adapt at their own slow pace and had not been denigrated for their negative mood expression. With familiarization, negative mood changed to positive acceptance and "zestful" replaced "difficult" and "rotten." They achieved expected social habits, made friends, and demonstrated developmentally appropriate accomplishments.

Several child psychiatrists have questioned the concept of temperament risk factors. They suggested that these factors were in fact a behavior disorder ([Graham and Stevenson, 1987](#)). If correct, this would assume that the child's difficult behavior was primarily responsible for the parents' distressed and often confused behavior. Temperament was assessed by maternal compilation of the Rothbart Infant H Behavioral Questionnaire in a study of 604 3- to 16-month-old infant twins and their parents. Shared environment accounted for cotwin soothability; whereas additional genetic effects accounted for co-twin similarity for distress to limitations to novelty and to the activity level (Goldsmith et al., 1999). John Bates expressed similar doubts regarding difficult temperament ([1998](#)). Plomin suggests that frequent temper tantrums and high activity cause parental concern and sees little value in the difficult child cluster (1997a). In contrast, William Carey, a pediatrician has found important links with colic, night awakening, and other common pediatric complaints. He cautions against labeling a child "difficult" to parents for fear that the pediatrician cause problems that might not otherwise arise, or even create a self-fulfilling prophecy ([Carey, 1998b](#)).

### Attention Deficit Hyperactivity Disorder

Discussing attention deficit hyperactivity disorder is pertinent because there is controversy as to the dividing line between normal high activity level and organically caused hyperactivity. The National Institutes of Health Consensus Development Conference statement on the Diagnosis and Treatment of Attention Deficit Hyperactivity Disorder (ADHD), [2000](#) concludes:

Finally after years of clinical research and experience with ADHD, our knowledge about the cause or causes of ADHD remain speculations. Consequently, we have no strategies for the prevention of ADHD.

Carey, a member of the Conference, has made several specific criticisms over the years.

1. ADHD symptoms and temperament are not clearly distinguishable.
2. There is no clear evidence that ADHD symptoms are related to brain malfunction.
3. There is neglect of the role of the environment as a factor in etiology.
4. The diagnostic questionnaires for ADHD in use are highly subjective and impressionistic.

He concluded: "Is ADHD a valid disorder? What is now most described as ADHD appears often to result from the aversive interactions between biologically intact individuals and their incompatible environments." Carey thinks a recognizable group of approximately 1% to 2% of the child population has pervasive high activity and inattention. Finally, a number of studies have shown that difficult temperament concept is culture-bound as a high- or low-risk factor (deVries, 1984; [Korn and Gannon, 1983](#)).

### Qualitative (Clinical) and Quantitative Analysis

It is of interest that several studies have shown the difficult child factor to have a protective function. Mentally retarded children in special education settings showed a trend toward a lowering of cognitive functioning over time. For difficult children the decline in cognition was less than that of the group as a whole ([Keogh, 1997](#)). In



the Helsinki Longitudinal Study, infants with difficult temperament were protected from having behavior disorders in adolescence ( [Teerikangas, 1998](#)).

## NEW DIRECTION AND OVERVIEW

A number of the controversial issues discussed in the previous section have indicated new directions for temperament work. Beyond these, the expanding temperament literature has raised many new exciting ideas and challenges for current and future theoretical and practical activity. There are discussions in this chapter of stability and change in temperament and some of the steps being taken to explore the facts and their meanings. Such questions and others emphasize the need for greater accuracy and comprehensiveness in the collection of temperament data. A dozen questionnaires that previously described direct observational and laboratory techniques have been elaborated. Parental interviews and interview protocols for adolescents and young adults are available. Clinical observations by child-care professionals are all sources of temperament data. All these methods have received refinement, standardization, and analysis of their reliability and validity. A further important task is the challenge to integrate and combine these separate sources of information, giving full value to the differences in behavior in different situations.

In another study by [Nulman and colleagues \(1997\)](#) there was an examination of the neurodevelopment of children exposed in utero to antidepressant drugs: tricyclic (80 children), fluoxetine (55 children), and 84 children who had been exposed to no teratogenic agents. No significant differences were found in these three groups of children at 16 months and 86 months of the temperamental qualities of mood, arousability, activity level, or distractibility.

### Temperament Research in Nursing

Researches on the application of children's temperaments by nurses have been fruitful. One example is [McClowry \(1998\)](#), from the School of Education at New York University, who is conducting a 5-year clinical trial testing the effectiveness of INSIGHTS, a temperament based intervention. This program is directed at high-risk, minority first- and second-grade children, their parents, and teachers.

A study of Barbara [Medoff-Cooper](#) (personal communication) at the University of Pennsylvania has included 78 premature infants of 1 to 2 months of age. A comparison group is a standardized population of full-term infants of the same postconceptual age. These premature children were significantly more withdrawing, negative in mood, low in persistence, arrhythmical, low in intensity and activity in comparison with the standardized population. The Preventive Ounce ( [Cameron and Rice, 2000](#)) established an Internet program for nurses in collaboration with Nancy Melvin at the Arizona State University College of Nursing. The positive clinical experience at the pediatric units of Kaiser Permanent Temperament Program was the basis for this program. The analysis of the 2,000 pediatric Kaiser patients and controls for prevention of behavior problems and cost effectiveness is now under way. In addition, Cigna, a health maintenance organization, has utilized this temperament program for the past 10 years.

In Vancouver, Canada, [Andersen \(2000\)](#) published a detailed temperament guide for professionals and established a Website in conjunction with McDevitt's Behavioral Initiatives. They distribute the six temperament questionnaires, from infancy through adulthood. In addition Kate Anderson, on the Website (<http://www.b-di.com>) hosts a monthly column discussing temperament issues with "high maintenance" children. Together with Sheeher, they contributed a chapter reviewing existing Parent Training Programs on Temperament-based Intervention in *The Handbook of Parent Training* ([Anderson, 2000](#)).

### Temperament Education

A number of teaching programs involving temperament as an aspect of child development have been ongoing. At the University of San Francisco, Jan Kristal teaches toward a degree as temperament counselor to psychologists, educators, therapists, and child development workers at the University of San Francisco, College of San Marino and Santa Rosa Junior Colleges. As yet there are no formal criteria for training as a temperament counselor. This is now under consideration.

## FINAL OVERVIEW

We present our basic conceptual theme as a final overview: As we have considered the development course of our NYLS subjects, what has come to the fore has been the diversity of interactional processes and personality outcome, so clearly unique as these youngsters matured from infancy to childhood, through adolescence, and into early adult life. The capacity for flexibility, adaptability, and mastery in the face of all kinds of adverse and stressful life experiences has been equally striking. We have also been impressed that our preventative and therapeutic interventions have made differences at all age periods. There is no age at which closure for growth and change occurs.

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# 16 DEVELOPMENT OF ATTENTION, PERCEPTION, AND MEMORY

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To understand the development of attention, perception, and memory is to comprehend the development of the mind. A “consilience” ( [Wilson, 1998](#) ) of perspectives from separate branches of scientific inquiry has fostered a new interest in “mind.” This chapter is based on an interpersonal neurobiology ( [Siegel, 1999](#) ) of mental processes.

The principles of interpersonal neurobiology are as follows:

1. The mind is a process that regulates the flow of energy and information within and between individuals.
2. The mind is at the interface between neurophysiologic process and interpersonal experience.
3. The mind develops as the genetically programmed maturation of the nervous system is shaped by ongoing experience.

The energy consumption of the brain can be assessed via functional brain imaging techniques, whereas the flow of information can be examined via psychometric assessments. However, neurobiological research has tended to focus on the functioning of a single mind, whereas human beings are extremely social. Recent studies indicate that brain is hardwired to place great significance on social experience. For this reason, the second principle of interpersonal neurobiology conceptualizes the mind as being in the context of both brain activity and interpersonal experience.

There has been much debate on the issues of nature versus nurture and genetics versus experience. Recent research clearly demonstrates intricate transactions between genes and experience in the unfolding of brain structure and function. The third basic principle addresses this issue. The genes encode an architectural blueprint about how neurons migrate and how they should grow and prune their synaptic connections. Experience involves neural firing, which directly activates the genetic machinery. Thus, the genes are transcribed and proteins are produced and transported for the creation of new synapses. Debates that pit nature against nurture are simplistic and misleading.

In an interpersonal developmental neurobiology, the flow of energy and information within one brain is conceptualized as being shaped by the interpersonal experiences that occur as life unfolds. Experiences shape and are shaped by the genetically and experientially influenced maturation of the brain. Genes provide the blueprint for how neurons are to grow and connect; physical and interpersonal experiences fine-tune the connections.

## INFORMATION PROCESSING AND NEUROBIOLOGY

The tens of billions of neurons in the human brain are connected via synaptic junctions that average about 10,000 per neuron. In other words, each neuron influences or is directly influenced by about 10,000 other neurons. The on-off firing of the neurons generates a “neural net profile,” the pattern of neural firing at a given moment. It is unknown how neural firing patterns become mental processes. Somehow, neural net profiles yield mental representations experienced as images, sensations, or thoughts ( [Mesulam, 1998](#) ).

The brain region in which the neural net fires determines the modality of mental process (e.g., visual versus auditory). The pattern of firing within the region leads to the particular representation, such as the visual image of a dog or the Eiffel Tower. Thus, neural location (visual processing centers) and neural activation patterns (type of visual image) both contribute to mental processing.

As the infant grows, synapses are created, strengthened, or “pruned” (allowed to die back) in accordance with experience ( [Greenough and Black, 1992](#) ). For this reason, even identical twins do not have identical brains. Experience and chance determine neural firing, which, in turn, activates genes and leads to synaptic growth ( [Kandel, 1998](#) ). A poverty of experiential stimulation can lead to the excessive pruning of synapses or deficiency in the creation of new synaptic connections. Excessive stress can lead to the death of neurons, mediated in part by high levels of circulating cortisol ( [De Bellis et al., 1999a](#) ). Chronic posttraumatic stress disorder is associated with a diminished hippocampal volume ( [Bremner and Narayan, 1998](#) ). Early child abuse is associated with impairment in the growth of the corpus callosum, as well as overall decrease in the size of the brain ( [De Bellis et al., 1999b](#) ).

Memory is related to alterations in the synaptic strength of clusters of neurons. Thus, an individual who has seen a picture of the Eiffel Tower, for example, will be more likely to activate the visual representation of the tower in the future than someone who has never experienced such a stimulus. Memory shapes neuronal connections and is, thus, a fundamental part of brain growth and the development of the human mind.

## THE BASIC PROCESSES OF COGNITION

### Attention

Attention is the mental function that regulates the flow of information. Attention can be conscious or nonconscious. Conscious, focal attention is a linear, content-limited, energy-consuming process. The lateral prefrontal cortex is believed to be a mediator of focal attention or working memory. Working memory is limited by its capacity to focus only on “7 ± 2” items at a time. Nonconscious, nonfocal attention, in contrast, does not involve conscious awareness and is a nonlinear,



low-energy process not limited in capacity. Hearing one's name at a distant location in a loud party while consciously focusing on a close-by conversation is an example of nonfocal awareness.

## Sensation and Perception

Sensation involves the registration of input from the body, brain, or external world. Sensation may have no signal value. Signal value refers to input that has symbolic meaning, encoding information about something other than the thing itself. Feeling abdominal pain is a sensation. Being aware that the stomachache “means” that one is nervous is a perception. We sense the pain but perceive an anxious state of mind. Perceptions transform sensations by utilizing processes that generalize and cluster prior experiences, enabling the filtration of sensory input into perceptual categories. In this way, perceptions are a step removed from the thing itself and a manifestation of top-down processing.

## Representations and Modes of Processing

The mind creates representations of experience in a variety of modalities. One way of dividing representations is to examine sensory, perceptual, and linguistic groupings (Edelman, 1992). Sensation and perception are analogic. Analogic representations are mediated by a mode of cognitive processing that emanates primarily from the right hemisphere of the brain. The right mode of processing is holistic, analogic, and spatiotemporal. Events are represented as experienced, with little evaluation of their “rightness” or “wrongness.” In contrast, linguistic representations are “digital” in that they can be assessed as yes–no or right–wrong. Digital representations are mediated by a mode of processing emanating primarily from the functioning of the left side of the brain (Springer and Deutsch, 1993; Tucker et al., 1995). The left mode of processing is linear, digital, and linguistic. Its logic utilizes syllogistic reasoning in which cause–effect relationships are assessed and information is filtered through a right–wrong process of evaluation.

The asymmetry of subcortical structures in the embryo is the likely origin of the differences in processing between the two hemispheres (Trevarthen, 1996). The specialized function of each side of the brain may enable the mind to carry out functions that would not be possible without anatomic differentiation. Although differentiation between the left and right modes has survival value, integration of these processes may be essential for proper cognitive development. Early in life, the infant has a predominance of right hemisphere activity and development (Chiron et al., 1997). As the child develops, both hemispheres go through spurts of growth (Thatcher, 1997). Furthermore, the fibers that link the functions of each side grow for at least the first two decades of life (Trevarthen, 1990). Conditions such as posttraumatic stress disorder may be associated with impaired hemispheric integration (Siegel, 1999).

## Memory

Memory allows the mind to be influenced by experience. The most fundamental division of memory is between two forms that have been given a variety of names (McClelland, 1998; Siegel, 2001b; Squire, 1992). Early *implicit*, *procedural*, and *nondeclarative* memory are contrasted with later developing *explicit*, *semantic-episodic*, and *declarative* memory. In this chapter, the terms implicit and explicit are used.

Implicit memory operates early in life. It does not require focal attention for encoding, and when recalled, it does not convey the subjective sensation that “I am recalling something.” Implicit memory encompasses emotional, behavioral, perceptual, and possibly, somatic memory. *Mental models* or *schema* are also a part of implicit memory. Mental models enable the mind to abstract generalizations from many experiences, generating a schema for each type of event. *Priming*—the readying of the mind to respond in a certain fashion—is another aspect of implicit memory. Implicit memory does not require the hippocampus for mediation. It is thought to involve neural structures specific to each kind of implicit memory (e.g., the amygdala and orbitofrontal regions are involved in implicit emotional memory).

Explicit memory emerges later than implicit memory, requires focal, conscious attention for encoding, and conveys a sense of “I am recalling something.” Explicit memory has two forms: *semantic* and *episodic*. *Semantic* or *factual memory* is the ability to acquire knowledge, such as the definition of words. It is not associated with a sense of time or self. *Episodic* or *autobiographic memory* involves the sense of time and self as one recalls the events of one's life. The development of the hippocampus is required for the emergence of explicit memory when the child is about 1½ years old. Autobiographic memory also requires the maturation of the orbitofrontal cortex. Autobiographic narratives begin around this time because this region develops after the second birthday.

## Emotion

Emotion has been considered separately from cognition. Modern views of the mind consider this an erroneous division (Damasio, 1998, Damasio, 1999; LeDoux, 1996). Emotion is a mental function that integrates separate processes into a functional whole (Ciompi, 1991). Emotion combines the different elements of cognition into a state of mind that links sensation, perception, attention, memory, reasoning, and self-reflection. Emotion is both regulated and regulatory. Emotional regulation is necessary for self-regulation (Lewis, 1997).

The mind focuses its attention on a salient internal or external event as though to say, “This is important, pay attention!” Next, the event is appraised as either “good” or “bad,” activating the circuits that lead to either approach or withdrawal. These primary emotional processes occur constantly.

As the primary emotional states of the mind continually emerge, particular states may coalesce into a categorical emotion such as sadness, anger, fear, disgust, surprise, joy, or shame. These emotions have been found in all human cultures. They appear to represent the universal ways that the mind and body channel emotional states into visible affects. Mood is the enduring tone of a person's general emotional state.

Emotion encompasses a number of domains (e.g., the subjective, neural, somatic, and interpersonal) that develop during childhood and beyond (Sroufe, 1996). Each of these domains has a fundamental influence on cognition. For example, the regulation of attention is a central part of the primary emotional process. Moods directly influence memory, and perceptual interpretation is intimately shaped by the interpersonal context.

## Metacognition

An important component of mental functioning is the capacity to think about thinking, the mind's creation of mental representations about itself. At first, children learn to understand other people's states of mind. Later, they begin to understand their own minds. Metacognition begins after the third birthday, becoming more and more complex across the life span.

Metacognition encompasses the notion of the appearance–reality distinction: Things are not always as they appear. One aspect of this capacity is that of *representational diversity*: What one person thinks about an event may be different from what others think. *Representational change* refers to the observation that what a person thinks today may be different from what he or she thinks tomorrow (Flavell et al., 1993).

Children learn that emotions influence how people behave. (If a father is in a bad mood, don't ask for candy!) They learn that it is possible to have conflicting emotions about the same person at the same time. The understanding of how one's own memory works is a “metamemory” process that examines the nature of recall.

## Self-Knowledge

As each of these aspects of mental processing develops, it contributes to the child's growing sense of self. Interactions with caregivers have a profound influence on the child's emerging sense of an autobiographic self. Children with emotionally distant or rejecting parents have relatively underdeveloped life stories. The primary caregiver of such a child often has little recall of his or her own early life experiences (Hesse, 1999). Recent brain imaging findings point to the importance of the right hemisphere, prefrontal region, in autobiographic recall (Tulving et al., 1994; Wheeler et al., 1997).

The right hemisphere may be important in mediating a sense of self and a mental model of self with others (Schore, 1996, Schore, 1997). In addition to autobiographic memory, the right hemisphere appears to be predominant in the processing of a number of functions: an integrated representation of the body; the perception and expression of nonverbal signals; the regulation of emotional states; the registration and regulation of the sympathetic and parasympathetic branches of the autonomic nervous system; and the creation of representations of other people's minds (called “theory of mind” or “mindsight”). The right hemisphere dominates growth and activity during the first 3 years of the child's life (Chiron et al., 1997), suggesting that early attachment experiences may rely primarily on the exchange of



right hemisphere representational processes.

These findings suggest that the right mode of processing allows a better access to a patient's sense of self. Right mode communication involves the interchange of nonverbal signals and the attunement to the other person's primary emotional state. Children in particular require more attention to the nonlogical, nonverbal processes of the right hemisphere for communication and understanding.

### Integration and Differentiation

The mind moves toward increasingly complex differentiation of function on the one hand and integration of separate processes into a functional whole on the other (Siegel, 2001a). Chaos theory suggests that development is characterized by the movement of systems (individual, family, community) toward ever more complex functioning (Cicchetti and Rogosch, 1997; Fogel et al., 1997; Globus and Arpaia, 1993; Thelen, 1989). Nonlinear dynamics proposes that complexity is achieved by balancing differentiation and integration. Differentiation is defined as the functional specialization of the components of a system. Integration is the clustering of the differentiated elements of a system into functional wholes (Lewis, 1997).

## THE DEVELOPMENT OF ATTENTION

Clinicians often speak of attention as though it were unitary and distinct. On the contrary, attention is a multidimensional construct composed of such phenomena as strategic scanning, exclusion of irrelevant stimuli, sustained attention, divided attention, inhibition of impulsive action, and selection and monitoring of response. It is not possible to design a test that taps a "pure" attentional faculty. Furthermore, children are unlikely to attend unless they know why they should, they find the task interesting or rewarding, and they are able to distinguish figure from ground and signal from noise.

Under the age of 5 years, the salient features of a novel stimulus capture children's attention; early attention is stimulus bound. Between 5 and 7 years, a shift occurs: Attention comes under the control of inner processes, such as selective search strategies. As children mature, they become more systematic and flexible, and less egocentric. In essence, older children know when and how to attend. Poor learners, on the other hand, spend inadequate time "on task," lack cognitive strategies for analyzing task demands, and mobilize too little effort to succeed (Zelniker and Jeffrey, 1979). The metacognitive processes that facilitate attention overlap those relevant to perception and memory.

Given the frequent reference to defective attention in child and adolescent psychiatry, surprisingly little is known about its normal development. Reaction time, the capacity for vigilance and sustained attention, and the control of impulsive responses, improve up to 12 years. Thereafter, although reaction time stabilizes, accuracy continues to advance as more efficient search strategies are acquired (Salkind and Nelson, 1980). In one of the few studies that have provided age-normalized data, Levy (1979) found that measures of sustained attention, reaction time, and motor inhibition showed strong age and social class effects from 3 to 8 years, and that the three measures improved exponentially between 4 and 6 years of age. Multivariate analysis subsequently isolated a generalized "attention" factor on which reaction time, continuous performance, and motor activity loaded heavily and that was separable from motor inhibition. Subsequently, a study of clinic-referred children demonstrated that the clinical sample performed significantly more poorly than normal children of the same age on measures of attention, motor inhibition, and activity. However, when performance was related to age, the performance curves of the clinic sample paralleled those of the normal children. This study suggests that the diagnosis of attention deficit may be problematic in children younger than 5 years, since normal children show marked variation in activity level, the capacity for sustained attention, and motor inhibition between 4 and 5 years; furthermore, social class has a powerful effect on the capacity for sustained and selective attention in the transitional period between 4 and 6 years of age.

In an extensive review of research into the development of attention, Taylor (1980) concludes that little is known of the development of the capacity for divided attention, other than that it improves with age. It appears that older children are better able to resist distraction by stimuli irrelevant to the task at hand. Younger children find distractions harder to ignore, to the extent that the stimuli are salient, novel, or similar to the stimulus that is relevant to the task.

Barkley (1988) has discussed the importance of motivation in regard to attention. The capacity for sustained, selective attention and reflective, nonimpulsive responding is affected by both maturation and learning. These may be a part of the executive functions that allocate attentional resources. Executive functions regulate the flow of energy and information and direct both focal and nonfocal attentional processing.

Experiential factors may play an important role in disrupting attentional functioning. A child who has neither been expected to concentrate nor rewarded for doing so, is likely to be slow in acquiring the capacity for sustained, reflective attention. Children with disorganized attachment have difficulties with attention, emotional regulation, and social functioning (Carlson, 1998; Lyons-Ruth et al., 1993; Ogawa et al., 1997). This condition is also associated with a propensity to have dissociative symptoms. Disorganized attachment is believed to result from the child's repeated exposure to frightened, frightening, or alarming parental behavior (Main and Hesse, 1990).

If an inattentive child has previously had the benefit of secure attachment and consistent reinforcement and has not been exposed to traumatic events, it is more likely that the origin of the difficulties is in the constitutional neural substrate. Two causative hypotheses have been advanced: (a) the sensory analyzers mediating selective attention might be abnormal or (b) the neural links among memory, punishment, and reinforcement might be defective. (For further discussion of the development of inattention, see Chapter 28.)

Mirsky and associates (1991) propose that attention has the following components: *focus*, *sustain*, and *shift*. Focusing is the selection of target information for enhanced processing. Sustaining refers to the maintenance of focus and alertness over time. Shifting is the changing of focus in a flexible, adaptive manner. The validity of the three components of attention has been supported by multivariate studies of the attention performance scores of both adults and children. Sohlberg and Mateer (1989) hypothesize that attention is a multidimensional cognitive capacity with five levels: focused attention, sustained attention, selective attention, alternating attention, and divided attention. The higher levels of attention require controlled processing, whereas the lower levels are automatic.

## PERCEPTUAL DEVELOPMENT

### Auditory Perception

In the third trimester fetus, external sounds evoke movement, cardiac acceleration, and electrocortical responses (Bench, 1978), while the neonate reacts to loud or sudden sounds with a generalized startle response; however, an infant's intensity threshold is higher than that of an adult, especially for the lower and higher frequencies. Hearing at low and high frequencies continues to improve during the first 20 years of life (Trehub et al., 1989).

The rudiments of hemispheric specialization are apparent soon after birth: Lateral asymmetries in the auditory evoked potential can be produced by speech stimuli (Molfese and Molfese, 1979). The infant is especially receptive to sounds in the human frequency range (Hutt et al., 1968) and sounds with intonational patterns similar to those of speech (Eisenberg, 1979). Condon and Sander (1974) report that infants move in rhythm to the stress patterns of speech. Infants under 6 months of age have been found to discriminate among a number of speech consonants (Eimas, 1975). These important findings suggest that the infant has an inherent tendency to perceive those categorical discriminations that are fundamental to phonological development and that speech processing differs from that for other sounds. By the sixth month, infants can differentiate most of the acoustic contrasts required for phonological development (Lynch et al., 1992).

### Visual Perception

Although the neonate has some visual acuity, adult levels are not attained until 6 months (Lewis et al., 1978). Fantz (1958) was the first to show that neonates see patterns; it has subsequently been established that they tend to fixate on high contrast features within geometric forms (Salapatek, 1975). As the infant develops, increasingly more complex patterns are preferred (De Loache et al., 1978). The infant has begun to recognize the mother's face by 1 month, and moving eyes are of special interest by 2 months (Girton, 1979; Mauer and Salapatek, 1976). In the 2-month-old infant, complex patterns evoke electrocortical responses that may represent a shift from subcortical to cortical processing (Hoffmann, 1978). Field (1979) has shown that infants attempt to modulate the effect of highly arousing stimuli (e.g., animated faces) by averting their eyes. The perception of facial expressions appears to play a central role in the development of affect regulation during the first year of life (Schore, 1994).

As language reflects, adults perceive the continuous spectrum as though it were divided into different colors. Is color categorization an inherent propensity, or is it

molded developmentally by the arbitrary discriminations imposed by such words as “red,” “blue,” and “green?” In short, does the infant perceive categorically? [Bornstein and associates \(1976a\)](#), [Bornstein and associates \(1976b\)](#) found that 4-month-old babies discriminated green from blue but treated a second blue as similar to the first, even though the contrasted green and blue were equidistant in wavelength from the central blue. Like adults, babies tend to perceive the color spectrum in shades of red, yellow, green, and blue ([Bornstein, 1981](#)); however, form and color are not integrated as wholes until 6 months ([Cohen, 1979](#)).

The 3-month-old infant already has the rudiments of shape and size constancy and preferential orientation to the vertical ([Bornstein, 1984](#); [Bower, 1966a](#), [Bower, 1966b](#); [Caron et al., 1978](#); [Hayes and Watson, 1981](#)). Infants of 4 months are sensitive to symmetry ([Bornstein, 1992](#)); however, it is not until the second year of life that babies use landmarks to locate themselves in space, an ability probably facilitated by crawling ([Benson and Uzgiris, 1985](#)). [Coldren and Colombo \(1994\)](#) have found that 9-month-old infants can process compound stimuli in a dimensional manner and that they can solve discrimination-learning problems by the categorical selection and testing of perceptual features.

Visual accommodation, ocular convergence, and motion parallax are used as depth perception cues within the first 6 months. [Field \(1977\)](#) has demonstrated preferential reaching for near objects as early as 3 months. Head retraction to looming objects (which appear to move toward the infant on a collision course) has been noted in early infancy (Ball and Tronick, 1971); however, there has been controversy whether the head movement represents innate fear or interest. According to the index of heart rate change, infants as young as 2 months of age can perceive a visual cliff illusion ([Campos et al., 1970](#); [Gibson and Walk, 1960](#)).

### Taste and Smell

Neonates show their preference for sweetened water by smiling and sucking in longer bursts, with shorter pauses ([Crook, 1978](#); [Lipsitt et al., 1976](#)). Sour or bitter fluids, in contrast, evoke lip pursing or grimacing. Similar responses have been noted in anencephalic infants, suggesting that those responses are of subcortical origin ([Steiner, 1979](#)). Newborns are able to detect strong smells ([Engen and Lipsitt, 1965](#)), discriminate their mother's breast pads (McFarlane, 1975), and grimace at unpleasant odors ([Steiner and Finnegan, 1985](#)). The grimacing response, which is found in anencephalic infants, is probably a subcortical reflex. [Ruff \(1990\)](#) found that mouthing predominated as a perceptual tool during the first 6 months but that fingering displaced it during the second 6 months.

### Cutaneous Sensation

The mouth and genitals of the fetus become sensitive to touch during the first trimester and are followed by the palms, soles, and rest of the body. Thus, before birth, the structural and functional basis of skin sensation has already been established. The motor responses of the neonate to different cutaneous stimuli can be assessed by a number of neurologic tests (e.g., the rooting, sucking, and plantar reflexes). [Emde and colleagues \(1971\)](#) and [Anders and Chalemian \(1974\)](#) noted that neonates fuss and cry during and after circumcision, suggesting that they are capable of experiencing pain.

### Intermodal Perception

[Piaget \(1971\)](#) theorized that the coordination of the senses to form integrated perceptions could be achieved only after multiple parallel sensory experiences. In contrast, [Gibson \(1969\)](#), [Bruner and Koslowski \(1972\)](#), and [Bower \(1977\)](#) have hypothesized that early perception is diffuse and that perceptual specificity evolves from global sensory impressions.

[Mendelson and Haith \(1976\)](#) and [Muir and Field \(1979\)](#) have found that neonates will turn their heads and eyes in search of sustained sounds. [Spelke \(1979a\)](#), [Spelke \(1979b\)](#) has demonstrated that 4-month-old infants prefer to look at films in which sound is synchronized with action (compared with films that are out of synchrony). [Spelke and Owsley \(1979\)](#) have shown that 4-month-olds associate a parental voice with the visual image of that parent. [Meltzoff and Borton \(1979\)](#) have demonstrated mouth–eye crossmodal transfer as early as 1 month, and [Lewkowicz and Turkewitz \(1980\)](#) have found evidence that 3-week-old babies associate loudness and brightness. The global sensation theory appears to have been strongly supported, and there is evidence that premature babies are delayed in the capacity for cross-modal transfer ([Rose et al., 1978](#)).

Neonates can hear, see, smell, taste, and feel. The rudiments of hemispheric lateralization appear quite early, along with a particular sensitivity to speech and to the categorical distinctions associated with human phonologic development. As they grow, infants prefer increasingly complex visual patterns, show a special liking for animated faces, exhibit shape and size constancy, and perceive color categorically. Early sensation appears to be global, but perceptual specificity evolves rapidly during the first year. In brief, nativist theory has been strongly supported by recent research; however, there is clearly an intricate interaction between learning and postnatal neural maturation.

### Perceptual Development and Reading

Of all developmental theorists, [Gibson \(1969\)](#) puts the greatest emphasis on perception. To her, the child is an active perceiver, primed by species-specific genetic evolution to explore the environment and select what is required for adaptation. In contrast to Piaget, who postulates that children construct their own representational worlds, Gibson describes the child as extracting available information from the stimulus field. The information elicited can vary from concrete (e.g., perceiving an object) to abstract (e.g., the apperception of a melodic pattern). Perception grades imperceptibly into conceptualization as the child learns the distinctive features of particular objects (e.g., the mother's face), the common features of similar objects (e.g., the genus of dogs), and the deep structure of stimulus arrays (e.g., the syntax of a sentence). Perceptual processes would not be possible without search strategies, selective attention, and the exclusion of irrelevant input. According to Gibson, perceptual differentiation proceeds in linear fashion, not in stages.

[Gibson and coworkers \(1962\)](#) found that perceptual acuity for letter-like shapes increases from 4 to 8 years of age but that, at all ages, children are able to discriminate, in order of difficulty: (a) figural breaks (C versus O); (b) rotations and reversals (b versus P versus d); and (c) transformations (H versus N). [Bornstein and Stiles-Davis \(1984\)](#) found that, between 4 to 6 years, vertical symmetry begins to assume precedence over horizontal and oblique symmetries, as is the case with adults. Gibson's theory of perceptual development has implications for the acquisition of reading skills. For example, poor readers exhibit the following phenomena:

1. Literal decoding. Poor readers tend to read word for word, without extracting meaning ([Ryan, 1979](#)).
2. Poor comprehension. In that they tend to be oblivious to contradictions or ambiguities in written material, poor readers seem not to monitor what they read for meaning ([Paris and Myers, 1981](#)).
3. Poor strategies for the abstraction of meaning. Poor readers have difficulty abstracting the gist of a story ([Owings et al., 1980](#)). Young children and older poor readers may be unaware of the purpose of reading, and unable to apply such metacognitive strategies as scanning rapidly for meaning, and looking for syntactic and semantic cues. Metacognition transcends concrete perception and are considered further in the development of attention and memory. It has important implications for remedial reading ([Ryan, 1979](#)).

## DEVELOPMENT OF MEMORY

The term “memory” has connotations ranging from the act of recalling a personally experienced event (autobiographic memory) to the automatization of learned behavior (procedural memory) ([Milner, Squire, Kandel, 1998](#)). Memory is inextricably linked to other cognitive processes such as attention, perception, categorization, schematization, consciousness, and metamemory (which assesses the origin and accuracy of memory). The development of memory is not separable from that of cognition in general. Interpersonal experience, as well as neuronal maturation directly influence different forms of memory ([Siegel, 2001b](#)).

### Information Processing

Memory has been studied from the perspective of information processing. In this view, sensory input impacts sensory registers, producing a short-lived sensory memory (<250 msec). At this stage, attentional processes scan information and a small proportion placed in short-term (“working”) memory. Without active processing, short-term memory decays after about 30 seconds. After attention and processing (e.g., by classification or rehearsal), information in short-term memory may be deposited in long-term memory.

Encoding refers to the transfer of information from sensory registers to short-term memory and then to long-term memory. Storage refers to the capacity of the mind to represent information and have it potentially available for later access. Retrieval is the accessing of information from storage. Retrieval processes activate behavior,



emotion, and verbal recounting.

The recounting of autobiographic events often takes the form of a narrative and is related to the capacity to retrieve items from memory, to sequence them in story form, and to communicate them to a listener. Thus, recounting involves both cognition and discourse.

### Early and Late Memory

Cognitive psychologists have described two forms of retrieval: direct and indirect. Direct retrieval includes free recall (at which young children are poor) and recognition (at which they are much better). Indirect retrieval acts on processes and representations that convey a general pattern of action (e.g., long-term memory, sensory imaging) (Siegel, 1995a).

Retrieval can be conceptualized as the reactivation of a neural net configuration similar to that activated at the time of encoding. Memory is reconstructive, not reproductive. Organizing processes such as attention, perception, emotion, and schematization influence encoding, storage, and retrieval. From a developmental viewpoint, the brain's ability to store experience as neural representations with the potential for reactivation depends on the increasing sophistication of encoding, storage, and retrieval. Access to stored representations (i.e., the activation of latent neural net configurations) is dependent on retrieval. Thus, the inability of young children to recall aspects of an experience directly may mean that it was never encoded or that it was encoded and stored but that reactivation is impaired by immature retrieval strategies.

As discussed earlier in this chapter, given the distinction between early, nondeclarative memory and late, declarative memory, one must be clear about what kind of memory is being discussed. Emotional learning and behavioral learning occur early and do not involve an awareness of their origins in the past. Childhood amnesia, for example, refers to an impairment of access to declarative autobiographic memory, whereas nondeclarative (emotional and behavioral) memory may be intact.

Clinicians and researchers have focused on the development of declarative memory; however, early emotional and behavioral learning also influence child development. Attachment theory (Bowlby, 1969; Cassidy and Shaver, 1999; Main, 1995) derives its constructs from the idea that, after repeated patterns of relating, an infant constructs a mental model of attachment. This implicit mental model organizes the infant's behavioral and emotional responses and later influences declarative memory, self-reflection, and autobiographic memory.

Past maltreatment can affect implicit memory and the integration of implicit with explicit processing. The retrieval of early, learned emotional-behavioral responses may be triggered in adulthood by emotional states similar to those that applied in early childhood (Siegel, 2001a). This could explain the transgenerational transfer of child abuse, and the perpetrator's unawareness of its origin. Some of the symptoms of posttraumatic stress disorder (e.g., intrusive memories, avoidance, startle response, traumatic amnesia) could be explained on the basis of a combination of impaired explicit memory with intact implicit memory for traumatic experiences (Siegel, 1995b).

### Domains of Memory Development

#### STRATEGIES

Immature memory strategies limit the young child's ability to encode and store memory and retrieve it in declarative form. As memory strategies develop, the mind becomes better able to encode, store, and retrieve memories. Young children often require help with retrieval cues, whereas older children may be capable of both free recall and effective access to relevant memories.

Encoding and storage strategies are conscious activities that facilitate encoding and storage. They include looking, pointing (to direct attention), naming, and talking about things. Strategies become more complex, flexible, and specific with maturation. Rehearsal strategies help to move items from short-term memory to long-term memory. Organizational strategies, such as clustering, establish associational links that facilitate storage. Elaboration strategies enhance storage by adding meaning to presented items. Finally, the development of efficient strategies to allocate cognitive resources facilitates encoding. The evolution of these complex processes allows the child to search memory more intelligently, efficiently, flexibly, systematically, and exhaustively. As children develop, they tailor the strategy of retrieval to the task at hand.

Research suggests that some memory strategies can be taught; however, all children rely on techniques they have acquired spontaneously in the course of development.

#### KNOWLEDGE

Memory is not as simple as photography. What people already know influences what they learn and remember. Encoding and storage are constructive; retrieval is reconstructive. Memory is organized in part by schemata or concepts that are in flux between organization and reorganization. Neural net representations carry meaning that is fundamental to schematization. Thus, retrieval may involve memory reactivation and cognitive inference, both of which bridge the gaps that block retrieval.

As the child develops, new knowledge schemata and structures shape memory (Johnson-Laird, 1983). The constructive aspect of memory is exemplified by memory for events. General schemata influence event memory. For example, the memory of something occurring at school is influenced by the generic memory of a typical day at school. The predominance of general schemata in early childhood retrieval may be explained by the late maturation of the hippocampus and related brain regions.

*Priming* influences indirect measures of retrieval and involves behavioral learning independent of conscious awareness of prior experience (Schacter and Buckner, 1998). Priming readies the brain to respond in a particular fashion and affects the speed at which a task is learned. The indirect recall characteristic of infancy and early childhood is known as *early memory*, *procedural memory*, *nondeclarative memory*, or *implicit memory*, terms that are used synonymously in this chapter. Implicit memory is multifaceted: It embraces behavioral learning, emotional learning, mental models, and priming.

In contrast, direct recall involves another form of memory, *late memory*, *episodic memory*, *semantic memory*, *declarative memory*, or *explicit memory*. These terms overlap in meaning and refer to a general kind of recall known as "remembering." Remembering involves the recall of facts or concepts (*semantic memory*) or of personally experienced events (*episodic memory*). Semantic and episodic memory are usually explicit or declarative, that is, recountable in words or other symbols. (For further discussion of information processing, see Chapter 12.)

#### The Maturation of Memory

The mature brain contains over 20 billion neurons. At birth, the number is greater. Synaptic connections are created in accordance with both genetic information and experiential stimulation. As discussed earlier, synaptic connections are created, maintained, strengthened, or eliminated (pruned) as a result of the presence or absence of environmental stimulation. Experience induces neurons via gene activation to synthesize proteins that create and modify synapses. The neuronal pruning that occurs during infancy and early childhood is also thought to be both genetically and experientially determined. It is possible that much pruning occurs in adolescence, explaining in part the massive cognitive changes during this period of development.

Neurons interconnect in parallel distributions known as neural networks. Cognitive processes derive from the parallel processing that occurs as a function of the structural and functional properties of these neural nets. As discussed, a mental "representation" is thought to be created from a neural net activation pattern. A "process" can be considered as a neural activity that acts on representations, transforming them, creating new connections, or extracting common features from different representations. The term cognitive "structures" refers to complex functions that have repeated patterns of action, such as focal attention or long-term memory.

Cognition can represent cognition. This process, metacognition, develops during the preschool and elementary school years. One form of metacognition—metamemory—allows the child to understand memory itself. Metamemory conveys knowledge about how memory works as well as how to monitor and regulate it. Metamemory embraces the assessment of the current memory state, the selection of strategies, and the evaluation of progress toward cognitive goals.



An example of metamemory would be the subjective sensation of hunting for a memory and then recalling it.

The capacity to locate the origin of a memory is called source monitoring. One form of source metamemory, reality monitoring, is the process by which the mind determines if a memory is external or imaginary in origin. Reality monitoring becomes more efficient in later childhood ( [Johnson, 1991](#)).

### *Memory Capacity*

If the energy resources allocated to information processing are limited, then the operation of intentional, nonautomatic processes will limit the resources available for other cognitive processes. Thus, in a young child, immature storage and retrieval drains resources from and restricts other memory processes ( [Fivush and Hudson, 1990](#)).

Cognitive development enhances memory capacity by freeing resources for use in other mnemonic activities. For example, as strategies become automatized, storage capacity and retrieval efficiency increase. The automatization of metamemory together with the way that knowledge structures provide scaffolding for storage and retrieval contribute to the uniqueness of each child's memory development. The full development of memory, thus, depends on an interaction among strategies, knowledge, metamemory, and capacity ( [Flavell et al., 1993](#)). The next section illustrates this complex interplay.

### *Development of Autobiographic Memory*

**Memory, Self, and Time.** Autobiographic memory refers to memory of the self at a particular time in the past. Explicit memory, declarative memory, and late memory encompass episodic or autobiographic memory. The retrieval of a particular autobiographic memory conveys a profile of representations of sensations, emotions, facts, and self-in-the-past. In this manner, autobiographic memory can be thought of as an integrative process that involves many layers of mental processing, from mental models of the self to sensory images and cross-time representational processes. These capabilities relate to the concept of "mental time travel" ( [Tulving et al., 1994](#)) in which the mind connects elements of the past and present with imagined aspects of the future.

**Infantile Amnesia.** The maturation of autobiographic memory involves an interaction among language, social interaction, self-concept, and the sense of time. The earliest form of declarative memory is generic event memory. Children construct generalized patterns ("scripts") that represent familiar, oft-repeated events. New experiences are processed in accordance with generic memory. Encoding is biased by expectations based on preexisting structures, whereas accurate retrieval is biased insofar as retrieval scanning is influenced by generic memory. Generic event scripts give rise to preevent and postevent suggestibility ( [Ceci and Bruck, 1993](#)).

The immaturity of autobiographic memory in infancy and early childhood is associated with infantile and childhood amnesia. [Freud \(1963\)](#) proposed that infantile amnesia is owing to an active repression of early experience. The encoding deficit theory ( [Wetzler and Sweeney, 1986](#)) proposes a lack of attention to experiential detail, with impaired encoding and blocked retrieval. A third view ( [Pillemer and White, 1989](#)) hypothesizes a discrepancy between encoding state and retrieval state, in that prelinguistic encoding is incompatible with more mature language-based retrieval. Freud suggested that the repression barrier lifts at 8 years of age. The encoding-deficit and the encoding-retrieval-discrepancy theories propose 5 years of age for the termination of childhood amnesia. [Bauer \(1996\)](#) contends that explicit semantic memory emerges at around 1½ years old and that autobiographic memory may emerge after the second birthday.

As discussed in the following, explicit memory may require the maturation of the hippocampus. The later onset of autobiographic recall may depend on this hippocampal development and the additional maturation of the prefrontal region, especially the orbitofrontal cortex. As these regions mature, the child is able to recall personally experienced events with a sense of self and time. Interestingly, children around 5 years and older may have difficulty explicitly retrieving events that they could recall without difficulty earlier in their lives. This finding has not been explained as yet. Early, implicit memory, which is intact during "childhood amnesia," may influence play therapy and transference within psychotherapy ( [Lewis, 1995](#)).

**Narrative and Autobiographic Memory.** Children begin to tell the story of their experiences during their third year of life. In fact, autobiographic memory is often expressed in story form revealing the finding that autobiographic memory and narrative capacity intertwine. A narrative is a discourse in which the teller takes account of the listener's interests and expectations while recounting a temporal sequence of events, and incorporating the intentions of the characters in the story. One function of the narrative process is the facilitation of personal relationships. Some parents help their children construct narratives; a lack of exposure to coconstruction and memory talk may lead to an impairment of narrative memory. Children classified in infancy as avoidantly attached subsequently tend to exhibit a deficit in autobiographic memory that is correlated with similar deficiencies in their parents ( [Main, 1991](#)). Further research is required to determine whether this correlation is causal in nature.

As language and social competence mature, the child's sense of self and capacity to reflect on self enhance autobiographic memory. Theoretically self-reflection enables the elaboration of memory as "self" experience ( [Wolf, 1990](#)). Both encoding and retrieval may be organized by the self-concept. In other words, self-reflection, social interaction, and language intersect in the facilitation of autobiographic memory ( [Toth et al., 1997](#)). The capacity for sequencing or temporality is also involved. The child's sense of time and order develop throughout the preschool- and early school-age years. As temporality develops, the sense of self-in-time begins to shape autobiographic memory. A sense of personal continuity thus emerges, permitting new experiences to be processed from a temporal perspective.

### *Neurobiology of Attention and Memory*

**Attention.** [Margulies \(1985\)](#) proposes that the hippocampus mediates selective attention. Features or patterns abstracted from sensory data are conveyed to the hippocampus and matched against a cognitive "map" in order to determine whether a particular stimulus is noteworthy. Noteworthiness is determined in accordance with such criteria as novelty, salience, and emotional valence. The threshold and propensity of hippocampal selectivity may be modulated on a phasic basis (by norepinephrine), on a diurnal basis (by serotonin), and on a long-term basis (by corticosteroids). Once a stimulus is selected as noteworthy, efferents flow from the hippocampus to the medial septum (where spatial localization is determined) and the nucleus accumbens (which interrupts thought and allows the integration of the noteworthy stimulus into behavior). The organism, thus, can change the direction of attention toward the new stimulus.

[Hunt and associates \(1987\)](#) and [Zametkin and Rapoport \(1987\)](#) regard the noradrenergic theory of attention deficit disorder as the strongest current contender. According to this theory, attention deficit and hyperactivity are owing to an increase in noradrenergic transmission, possibly emanating from the locus ceruleus. Such an increase could be due to a variety of processes (e.g., heightened postsynaptic receptor sensitivity, or increased presynaptic production or release). It is unclear how the noradrenergic hypothesis of attention deficit fits the hippocampal theory of selective attention; perhaps a tonically high noradrenergic state causes a reduction of the hippocampal threshold to stimuli, leading to increased distractibility.

[Mirsky and colleagues \(1991\)](#) refer their three proposed components of attention—focus, sustain, and shift—to different brain systems. Focusing and execution are related to the superior temporal and inferior parietal cortices and to the corpus striatum. Sustained vigilance is related to the mesopontine reticular formation and to the midline and reticular thalamic nuclei. The capacity to shift from one salient aspect of the environment to another is associated with the prefrontal cortex.

Working memory is mediated by the lateral prefrontal cortex in conjunction with the executive functioning of the anterior cingulate regions. The complex interplay of focal attention, executive function, and encoding is likely to be a central focus of future research on attentional mechanisms. The prefrontal region, especially on the right side, may play an important role in these functions and has been found to be abnormal in numerous imaging studies of children with attentional difficulties ( [Arnsten, 2000](#); [Hendren et al., 2000](#)).

**Memory.** The components of implicit memory function independently of the medial temporal lobe memory system. Implicit memory is thought to be associated with the basal ganglia, amygdala, and possibly, the motor and somatosensory cortices. Recent research suggests that the medial temporal lobe is the key to declarative or explicit memory. Within the medial temporal lobe is the hippocampus, one of the few brain regions that undergo significant postnatal neurogenesis. In humans, hippocampal neurogenesis was believed to occur up to 2 years of age, and synaptogenesis to continue even longer. However, recent research suggests that new neurons continue to grow throughout the life span, especially in the hippocampal region that integrates the processing of disparate areas of the brain ( [Benes, 1998](#)). The relative immaturity of the hippocampus during the first year and a half of life may explain the delayed onset of explicit memory. The later development of the prefrontal regions following the second birthday may explain the onset of explicit autobiographic memory.

The hippocampus is thought to be associated with cognitive "maps" that structure time, space, and sense of self in such a way as to facilitate explicit memory. Autobiographic memory appears to be mediated via the prefrontal regions that draw on representational processes from posterior regions of the brain, especially in

the right hemisphere ([Wheeler et al., 1997](#)).

Clinical disorders caused by lesions to the hippocampus impair declarative memory, whereas implicit memory remains intact ([Squire, 1992](#)). Even in the absence of hippocampal processing, some learning is possible, but it lacks an explicit sense of time, self, or context. Infants exhibit implicit memory postnatally, develop generic event memory, and then, by 2 to 3 years of age, autobiographic memory.

Social interactions that facilitate the sense of self and time may both depend on hippocampal and prefrontal cortical maturation and also stimulate their further development. Individual differences in autobiographic memory capacity may thus reflect the interdependency of social interaction, cognition, and neural substrate ([Siegel, 1999](#)).

Recent research has illuminated a possible cellular substrate of explicit memory. Neurochemical explanations must account for the extraordinary associativity, durability, efficiency, capacity, and rapidity of memory. Some, at least, of those qualities are consistent with long-term potentiation of the hippocampus by brief bursts of high-frequency stimulation. Long-term potentiation is associated with the opening of calcium ion channels by glutamate, an excitatory neurotransmitter. Calcium ions enter the neuron and function as a second messenger system, activating intracellular kinases, proteases, and lipases. The consequent degradation of cytostructural proteins and membrane phospholipids is thought to promote an increase in the number of excitatory receptors, changes in the shape of dendritic spines, and a proliferation of dendritic synapses. These structural and functional alterations would be consistent with long-term potentiation. Furthermore, the coincident activation of multiple sensory synapses on the one association neuron (or set of neurons) could provide a neural basis for crossmodal perception.

It has recently been discovered that the serotonergic stimulation of neurons elevates intracellular levels of cyclic AMP. When a critical level of cyclic AMP is exceeded, the nuclear cyclic response element binding (CREB) gene is activated (by phosphorylation). CREB transcription proteins then activate other genes, which in turn activate memory storage (by an as-yet-unknown mechanism).

## THE DEVELOPMENTAL PSYCHOPATHOLOGY OF ATTENTION, PERCEPTION, AND MEMORY

Although neither attention, nor perception, nor memory is ever exhibited in isolation, in different forms of developmental psychopathology; it is possible to discern defect, hypertrophy, and deviance of each of these functions.

### Disorders of Attention

#### DEFICIENT ATTENTION

Attention deficit is thought to be the cardinal sign of attention deficit disorder, a condition characterized in part by distractibility, difficulty following instructions, difficulty sustaining attention on an imposed task, heedlessness, impulsiveness, and recklessness. [Douglas \(1983\)](#) has described how poor attention and impulsivity can impede the acquisition of concepts, learning strategies, and effectance motivation. However, despite the confidence with which attention deficit disorder is customarily diagnosed, there are many questions concerning the homogeneity and categorical distinctness of this disorder ([Taylor, 1988](#)). As described, since the capacity for sustained and selective attention increases markedly between 48 and 54 months ([Levy, 1979](#)), the diagnosis should be cautiously made in children younger than 4 years of age. It is unclear how inclusive or exclusive the diagnostic criteria for this condition should be; as a result, the estimated prevalence of the condition varies between Europe (where it is relatively low) and North America (where it is much higher). As [Rutter \(1983\)](#) points out, inattention and overactivity are encountered in many situations, from pain, fatigue, illness, and transient emotional upset, to psychopathological conditions such as posttraumatic stress disorder, dissociative disorders, mania, major depression, panic disorder, schizophrenia, intoxication, and confusional states. Furthermore, as previously discussed, attention is not a unitary phenomenon. Not surprisingly, it is unclear whether oppositional defiant disorder, conduct disorder, and attention deficit disorder are distinct, coterminous, or coincidental. [Douglas \(1983\)](#) notes that, although attentional problems are often associated with impulsivity, poor arousal modulation, and an inclination to seek immediate reinforcement, it has not been clearly established that defective attention is the basic dysfunction of attention deficit disorder. An impairment of executive functions that enhance self-regulation might be the central deficit in these conditions. Defective attention has also been found in children at high risk for developing schizophrenia ([Mednick et al., 1978](#)); however, the relationship between this phenomenon and later schizophrenia is unclear. (For further discussion of inattention, see [Chapter 28](#).)

#### HEIGHTENED ATTENTION

Hypervigilance and narrowing of attention occur in situations involving focused fear (e.g., watching a horror movie), night terrors, paranoid conditions (rare in childhood), and in some acute schizophrenic states. Attention fluctuates in delirium and is diminished particularly at night and in situations associated with reduced sensory stimulation. Heightened attention may be associated with perceptual illusions; for example, the anxious or phobic child who mistakes nocturnal rustlings for the approach of an intruder. Cocaine and stimulant abuse are associated with intense, fixed concentration.

### Disorders of Perception

[Ludwig \(1980\)](#) classifies perceptual abnormalities as follows:

1. False perceptions independent of reality
2. False perceptions dependent on reality
3. False perceptions that stem from sensory deficit or dysfunction

#### INDEPENDENT FALSE PERCEPTIONS

Independent false perceptions take the form of hallucinations or pseudohallucinations. A hallucination is a perception that has no basis in objective reality. Hallucinations vary in sensory modality; form (from amorphous to organized); intensity (e.g., loud or soft); clarity (vague or clear); spatial location (internal or external, near or far); and the degree of conviction or urgency they convey.

Epileptic auras and temporal lobe seizures may be associated with poorly formed auditory, olfactory, or visceral hallucinations. Panoramic visions may be described. Delirium is typically associated with poorly formed, unpleasant, visual, auditory, or cutaneous hallucinations. Cerebral space-occupying lesions may be associated with intense, compelling hallucinations, and with bizarre phenomena such as negative hallucinations, autoscopia, the perception of a Doppelgänger, distortions of the body image, depersonalization, and déjà vu experiences. The hallucinations that result from the ingestion of hallucinogens are associated with profound distortions of sensory perception and affective experience and subsequently with traumatic "flashbacks" of intrusive memories of the hallucinatory state.

Schizophrenia is typically associated with unpleasant auditory hallucinations that emerge from amorphous perceptual distortions and take the form of conversations, commands, commentaries on the subject's actions, or echoing of the subject's thoughts. In paranoid states, auditory hallucinations tend to be accusatory, degrading, controlling, or laudatory in nature and consistent with the patient's delusional beliefs.

Disorganized auditory or visual hallucinations are sometimes encountered in mania; whereas in melancholia, depressive hallucinations (relatively rare in adolescence) are consistent with the prevailing delusions of guilt and self-abasement.

[Cantor and associates \(1982\)](#) contend that preschool children can manifest schizophrenia, but this condition is exceptionally rare before 7 years of age and quite uncommon before adolescence. There has been uncertainty whether autistic children are prone to develop hallucinatory psychotic disintegration in late childhood or adolescence ([Petty et al., 1984](#)). The hallucinations in such conditions are said to be relatively unsystematized and perceived as alien, commanding, or threatening in nature.

Pseudohallucinations are experienced in the form of heightened internal thoughts or images. Pseudohallucinations convey less conviction than true hallucinations, and have a content directly related to the individual's psychic predicament. For example, a sexually traumatized adolescent may have recurrent vivid memories ("flashbacks") of being sexually attacked, with accompanying conversations and visual imagery; or a miserable, lonely child may have an image of a deceased relative speaking words of consolation. Pseudohallucinations are common in the overlapping conditions of hysterical psychosis, dissociative disorder, and posttraumatic



stress disorder. Care should be taken to distinguish these common conditions from schizophrenia and temporal lobe epilepsy, with which they are frequently confused. (For further discussion of the development of psychotic thinking, see [Chapter 27](#).)

#### *DEPENDENT FALSE PERCEPTIONS*

An illusion is sensation given a false interpretation, that is, the erroneous perception of a real experience. Auditory and visual illusions are characteristic of intense emotional states (e.g., religious conversion) and physical deprivation (e.g., hunger, thirst, or fatigue). They are also encountered in delirium, intoxication (especially with hallucinogens), epilepsy, hysteria, and acute psychotic conditions. The phenomena of depersonalization, derealization, déjà vu, body image distortion, and alteration of the sense of time are commonly associated with anxiety in adolescence but are also found in intoxication with hallucinogens and in epilepsy, hysteria, dissociative states, and psychosis.

#### *FALSE PERCEPTIONS ASSOCIATED WITH SENSORY DEFICIT OR DEPRIVATION*

Deafness and blindness may be associated with misperception, especially when the individual is delirious, deprived of sensory input, or affected by certain medications (e.g., salicylates).

#### *HEIGHTENED PERCEPTION*

Eidetic imagery has the clarity of real perception and is perceived as though external to the viewer. The propensity to generate accurate, vivid, perceptual memories of events may be a normal variant. It could conceivably interact with stress or metabolic instability to generate visual hallucinations.

#### *PERCEPTUAL IMPAIRMENT*

The association between perceptual impairment and reading retardation has been a topic of investigation and debate for many years. Is dyslexia fundamentally a perceptual disorder? Or is the perceptual immaturity associated with dyslexia secondary to higher-order language dysfunction? Or do children with perceptual disorder represent a subgroup of dyslexics? These issues are discussed in [Chapter 50](#).

### **Disorders of Memory**

Disturbances of memory in children can range from an inhibition of semantic recall and impairment of encoding in the aftermath of head trauma, to a blockage of the retrieval of autobiographic memory in posttraumatic stress disorder. The intricate relationships among attention, perception, schematization, and memory suggest that impairment in a variety of cognitive processes could affect memory. Thus, the clinical assessment of memory in a child requires a broad developmental profile including cognitive development, experiential history, family functioning, and school environment ( [Boyd, 1988](#)).

Memory dysfunction can be present in any of the domains of memory described in the preceding. Thus, clinicians encounter deficits in encoding, short-term memory, access to long-term memory, or retrieval of autobiographic or semantic memory. Cases have been described in which implicit memory is intact but explicit memory impaired. Hippocampal lesions, Korsakoff syndrome, hypnotic amnesia, the aftermath of surgical anesthesia, the side effect of benzodiazepines, and childhood amnesia are associated with this kind of mnemonic disassociation. Memory disturbances can involve a loss of previously attained memory, temporary abnormalities in established functions, or maturational delay. Because memory is embedded in cognition as a whole, specific learning disorder, mental retardation, language disorder, attention deficit disorder, and organic brain disorder are all associated with memory dysfunction.

Depending on the location and extent of brain pathology, neurologic disorders can present with a variety of memory abnormalities. Disorders leading to dementia may produce impairment of the encoding and retrieval of both short-term and long-term memory. Examples of such disorders are infections (e.g., subacute sclerosing panencephalitis, acquired immunodeficiency syndrome [AIDS]), metabolic disorder (e.g., Tay-Sachs disease, Wilson disease), neurocutaneous degenerative disorders (e.g., Sturge-Weber syndrome, neurofibromatosis, tuberous sclerosis), and toxic syndromes (e.g., lead encephalopathy).

Head injury can produce anterograde and retrograde amnesia. Retrograde amnesia is thought to be due to disruption of the cortical consolidation required to establish long-term memory. Seizure disorders can be associated with defects in memory. Generalized seizures impair the retrieval of recently experienced events. Partial complex seizure disorder (temporal lobe epilepsy) causes a disturbance in verbal recall and encoding. Preictal (aura) states are associated with dissociative symptoms (derealization, distortions in the sense of time, perceptual distortion). Ictal and postictal states can impair attention, language processing, and memory retrieval. Other neurologic disorders affecting memory include viral infection (e.g., herpes encephalitis), multiple sclerosis, and brain tumor. The location of the lesion determines the nature of the memory pathology.

Experiential factors affecting memory include attachment disruption and psychological trauma ( [Nelson and Carver, 1998](#)). Culture, family environment, and school all shape memory development. The family's encouragement of intellectual achievement, memory talk, and the shared construction of stories stimulate the domains of memory development: strategies, knowledge, metamemory, and memory capacity.

As described, attachment research suggests that two groups (avoidantly attached children, adults with dismissing states of mind with respect to attachment) exhibit a paucity of autobiographic narrative and diminished access to episodic memory ( [Hesse, 1999](#); [Main, 1991](#)). In the United States, at least 10% of the normal population has impaired autobiographic memory. The factors that mediate this impairment are unclear. It is possible that remote or rejecting parents fail to stimulate their children by telling, sharing, and listening to stories. Another possibility is that the association is genetic.

A failure to tell stories about an overwhelming event may aggravate the effects of psychological trauma. If parent and child cannot talk about the child's frightening experience—for example, if the parent is too misguided, preoccupied, oblivious, or compromised to do so—the child may be at risk of posttraumatic stress disorder. Posttraumatic amnesia is a controversial matter. The mental mechanisms known by clinicians as repression, dissociation, and distortion require empirical validation and clarification. Repression has been ascribed, hypothetically, to the automatization of conscious suppression: Encoding has occurred, but retrieval is blocked ( [Anderson and Green, 2001](#)). Dissociation is akin to the divided attention and altered consciousness observed in hypnotic states ( [Siegel, 1996](#)). The subject's mental state at the time of encoding can influence later accessibility to retrieval. Research using dichotic listening methodology has shown that implicit memory may be intact in the nonattending ear. If a child learns to adapt to trauma by means of autohypnotic division of attention and dissociation, explicit, narrative memory may be impaired whereas implicit memory is intact ( [Siegel, 1995b](#)).

Error and distortion are possible because memory is reconstructive. Both adults and children are subject to preevent biasing and postevent suggestion, for example, by the use of leading questions, persuasion, coercion, or group pressure. After repeated suggestive questioning, some children may begin to construct memories that are consistent but false. This is a serious problem in the investigation of allegations of abuse, particularly with regard to preschool children who have difficulty providing sequential narratives and who often require a certain amount of leading ( [Ceci and Bruck, 1993](#); [Goodman and Bottoms, 1993](#)).

### **CONCLUSION**

Both neurophysiologic processes and interpersonal relationships create the mind. As an individual experiences events, the brain responds by altering the connections among neurons. Thus, genetically encoded information and experience both lead to changes in brain connections. The growth and shaping of neural connections is the physical basis of how the mind remembers and the brain develops.

This interpersonal neurobiology of the developing mind incorporates an interdisciplinary approach to understanding mental processes. Attention, for example, is influenced by the genetically determined unfolding of brain structures that subsume working memory and executive control, as well as experiences that influence the maturation of these regions of the brain. Impairments to attention, perception, and memory may be primarily owing to genetic or experiential variables.

Advances in cognitive neuroscience and developmental psychology can be combined with psycholinguistics, artificial intelligence, anthropology, neurology, psychiatry, and systems theory to create an interdisciplinary view of mental processes. The human mind moves toward ever-increasing levels of complexity. Blockages to this self-organizational process may cause impairments to mental health. The balance of differentiation and integration enables the most complex, and hence, most adaptive states to be achieved. The enhancement of the balance between differentiation and integration may be at the heart of how therapeutic



interventions promote the development of attention, perception, and memory.

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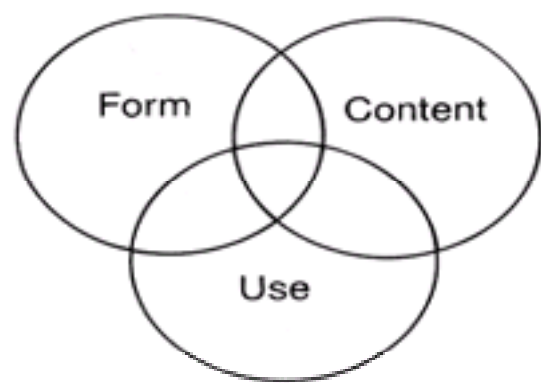
# 17 DEVELOPMENT OF COMMUNICATION

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This chapter provides a brief outline of the normal developmental processes involved in the acquisition of communicative skills. We examine the acquisition of these skills within several broad stages, looking first at prelinguistic communication during the first year of life, and then describing the acquisition of basic language skills during the preschool years. More advanced communicative abilities acquired during middle childhood and adolescence are outlined also. In each of these developmental periods, four major components of language are addressed: the processing or comprehension of language, acquisition of speech sounds, expression of words and sentences, and pragmatic or social and contextual aspects of communication.

Readers should be aware that this organizational scheme is not the only way to conceptualize language development. Because there is no consensus on the best model to describe language processing or acquisition, there is no universally accepted categorization method for analyzing the richly integrated system of communication in which we all take part. Bloom and Lahey's (1978) *content/form/use* model (Fig. 17.1) is often used. This divides language into aspects of *semantics* (content) or meaning; *syntax/phonology* (form), the generative rules that govern the formation of words and sentences; and *pragmatics* (use), the way language is used in context to accomplish social communication. This model is useful for understanding normal development, but it does not correspond as closely as the one employed here to the disorders of language learning typically seen in children. *Diagnostic and Statistical Manual, Fourth Edition* (DSM-IV) classifies disorders of language learning along the speech sound/production/comprehension/pragmatics dimensions, and using this categorical scheme will make it easier to discuss these disorders when we look at them in Chapter 49.



**Figure 17.1.** Bloom and Lahey's taxonomy of language. (From Bloom L, Lahey M: *Language Development and Language Disorders*. New York, Wiley, 1978.)

A second caveat readers should bear in mind is that, although the various components of speech and language are described in terms of disparate stages, there is often a good deal of overlap among the stages in reality. These stages of language development are not necessarily discontinuous steps marked by clear transitions or divisions. Nonetheless, it is usually possible to identify which broad stage of development any given child is in, and consequently, these stages represent a useful method of conceptualizing development for discussion.

Finally, readers should be aware that the normal range of language development is relatively wide, particularly during early childhood. The speech and language milestones that are presented here represent the ages during which the various stages of development generally occur. They are, of course, group averages, which to some extent ignore the possibility of specific (normal) individual differences. There are relatively large standard deviations, or normal variation, around these averages, which decrease somewhat with age. For example, the average vocabulary size for an 18-month-old child is 110 words with a standard deviation of 114. At this age, the normal range of variation is over 100% of the mean! By 30 months, though, the standard deviation in vocabulary size declines to 18% of the mean (Stoel-Gammon, 1991). We can see, then, that wide variation in language milestones is very common, especially in the early stages of acquisition. As children grow older, the normal range narrows considerably, which makes the determination of whether a difference represents a disorder much easier in an older child than in a younger one. Generally, the order of acquisition of particular structures, meanings, and uses is similar across children (with some exceptions), although the rate of acquisition can differ. Clinicians should keep in mind that a specific delay in one or several milestones is not necessarily indicative of disordered acquisition unless the delay persists beyond early childhood or that there is a general pattern of such delays across several areas of development.

## PRELINGUISTIC COMMUNICATION

The production of a "first word" is frequently considered by parents as the first step in communicative development; however, infants engage in a great deal of communication long before the first word is heard. From the moment of birth, this communication is an active process between the child and social environment. Far from being a passive recipient of adult speech, even the newborn infant engages in behaviors that rivet the adult's attention and elicit social interaction. Let us look at the areas of communicative development that were outlined in the preceding and examine how infant communication proceeds in each.

### The Infant's Perceptual Equipment for Processing Language

Although, of course, young infants do not understand the literal meaning of language addressed to them, it does appear that newborns begin life with attentional preferences for human, linguistic interaction and with a set of social behaviors that can elicit this stimulation. These abilities appear, because of their emergence so soon after birth, to be innately programmed, and, as such, provide a great deal of economy in the task of mastering language. Certain propensities present from the first days of life include a preference for sounds in the frequency range of the human voice (Hutt et al., 1968) and for speech over other rhythmic or musical sounds (Butterfield and Siperstein, 1974). Newborns look for the source of a voice they hear, register pleasure with facial expression when they identify the source, and remain quiet, inhibiting their movements, until the voice ceases (Owens, 2000). They do not show this kind of recognition when a nonhuman auditory stimulus is heard. Three-day-old infants are able to recognize their own mothers' voices, as opposed to the voices of other women (Hepper et al., 1993), probably as a result of prenatal experience with the mother's voice heard through the amniotic sac (Jusczyk, 1999b). Newborns are also attracted to and prefer to look at faces (Kagan and Lewis, 1965). Parents, conveniently, interpret this preference as a sign of willingness to interact. The newborn appears, then, to be biologically organized to attract

language input and attune to the linguistic environment.

There is evidence that speech perception begins at a very early age and may, in fact, be preprogrammed in human infants (Eimas, 1975). For example, infants only weeks old are able to discriminate among a variety of speech sounds, including /pa/ versus /ba/, /ta/ versus /da/, /ba/ versus /ga/, to name a few (Aslin and Smith, 1988; Eimas et al., 1971; Graham et al., 1983). Even more interesting, infants make these distinctions along the same categorical boundaries as adults do.

The fact that such distinctions can be made as early as 1 month of age could be interpreted to mean that the ability to make them is innate. Alternatively, it could suggest that this perceptual learning happens very quickly. Researchers have attempted to address this question by looking at sound discriminations that infants could not have learned from their environment because they never heard them made. For example, Werker and Tees (1984) found that infants in English-speaking environments were able to distinguish sounds that are not used in English but rather in Hindi. By 1 year of age, though, this ability to discriminate sounds not heard in the native language had all but disappeared. These findings suggest that the infant does have some “built-in” capacity to make discriminations among sounds that are important in speech, but that this ability is modified with experience. Rather than learning to make these distinctions from the language they hear, though, it seems that the infant comes to the task of language with some discriminations “preset.” Depending on what particular distinctions are used by the ambient language, some of these innately programmed distinctions are maintained by the child’s experience, whereas others are extinguished. The acceleration of this process toward the end of the first year might also signal a shift in focus for the language learner from sound discrimination to the mapping of sound to meaning (Werker and Tees, 1999).

Children also acquire a range of differentiated responses to the sounds of language throughout their first year. By 4 months of age, they respond to different tones of voice; by 6 months, they show evidence of selective listening (choosing to respond to some sounds and ignore others). Toward the last half of the first year, babies inhibit their behavior if told “no” in a loud, sharp voice, but they do the same if they hear “yes” spoken in the same tone (Spitz, 1957). Their response at this stage, then, is not to a specific lexical item, but to an emotional tone in the speech.

Young babies also appear to be able to coordinate acoustic information about speech with visual information about oral posture. Kuhl and Meltzoff (1988) showed that the infants looked for a significantly longer time at the picture of the face whose oral gesture corresponded to the vowel the babies were hearing at the time (retracted lips for /i/; open lips for /a/). This surprising finding suggests a very early ability to integrate visual and auditory cues in perceiving speech and would seem to provide children with an excellent foundation for learning the articulatory movements associated with the speech sounds they will eventually learn to produce.

Although children do not understand words per se until near the end of the first year, they appear to begin to develop the bases for the grammatical and semantic categories with which these words will be associated much earlier. Infants are able to use acoustic and phonologic cues to distinguish words based on grammatical class. Shi and colleagues (1999) showed that infants presented with lists of lexical (content) and grammatical (function) words could detect a switch to a word from a different category, although they were not sensitive to changes within the same grammatical category. Colombo and associates (1987) showed babies 6 to 7 months of age slides of various kinds of birds until the children habituated to these stimuli and reduced their visual fixation time. These researchers then showed two new slides simultaneously, one of a parakeet and one of a horse, for example. Babies reliably looked longer at the horse, suggesting that it was more novel to them than the parakeet, which they had also not seen before. Thus, it appears that the children included the parakeet in the category they had formed for the objects viewed previously (i.e., birds). These findings suggest that even at this early age, babies are able to organize their perceptions into conceptual categories that eventually can be mapped onto words.

Infants also appear to have specialized abilities that facilitate the task of segmenting words from the ongoing stream of speech. Saffran and colleagues (1996) showed that by 8 months of age, infants are able to recognize word-like sound units from an ongoing acoustic field, based on expectations they have developed for the likelihood that certain sounds will appear together. Moreover, they accomplish this task, using nonsense syllables presented by examiners, after only 2 minutes of exposure.

Jusczyk and coworkers (1999) also reported that infants at 7 to 10 months of age can use stress patterns in a similar way to recognize word-like elements within an acoustic stream. When presented, for example, with a series of syllables that have a strong-weak syllable pattern (hamster), they show preferences for listening to new combinations that have the same pattern and are more likely to react to strong-weak patterns as if they were words, than to weak-strong patterns (giraffe). As they develop, children acquire increasingly sophisticated means of locating word boundaries in fluent speech. By 11 months of age, children’s sensitivity to word boundaries depends on the multiple sources of information, including the order of sounds in the word, stress pattern in the word, and the way in which individual sounds are pronounced (Jusczyk, 1999a; Myers et al., 1996). This ability to use a variety of acoustic cues to segment words from a speech stream by relying on probabilities of sound structure abstracted from a relatively small amount of listening experience helps to explain how children learn to pick out words from the many sounds they hear in their environments.

Infants also appear to be sensitive to auditory information that is associated with syntactic boundaries. Hirsh-Pasek and associates (1987) showed that babies of this age preferred to look toward a speaker that played sentences containing pauses at clause or phrase boundaries (Cinderella lived in a great big house/ but it was sort of dark/ because she had a mean . . .), as opposed to sentences with pauses in the middle of a clause (Cinderella lived in a great big house but it was/ sort of dark because she had/ a mean . . .). These results suggest that babies as young as 7 months can detect syntactic boundaries, an ability that would greatly economize the amount of information they ultimately need to acquire in order to understand sentences.

Although parents have acted as if they believed their child understood speech almost from the first day of life, true lexical comprehension does not emerge until about 8 months of age. Around the last quarter of the first year, babies begin to respond to certain words that they hear in familiar routines and, for example, will clap hands when mother says, “Let’s play patty-cake.” This early comprehension is contextually bound, however. If the baby is used to playing patty-cake on the changing table but is told to clap hands in the bathtub, she or he will probably not comply.

Chapman (1978) points out that this contextually bound comprehension often leads parents to believe that babies at 8 to 12 months understand much more of language than they actually do. Chapman described a set of strategies for comprehension that are frequently used by infants of this age to comply with parental requests and that give the parent the impression the child is actually understanding language. These are summarized in Table 17.1. For example, babies frequently look at what mother looks at, and this can give the impression that the baby understands what the mother is saying. In fact, though, all the baby has to do is follow the mother’s line of regard.

Age	Comprehension skills	Comprehension strategy
8-12 months	Understands a few single words in routine contexts	Look at object mother looks at Act on object mother looks at Initiate ongoing action
12-18 months	Understands single words outside of routine but still requires some contextual support	Locate object mentioned See evidence of action Do what you usually do
18-24 months	Understands words to identify objects, some two-term comparisons	Act on objects in the way mentioned (pick up object)
24-36 months	Comprehension of three-term sentences, but context or past experience determines meaning; no understanding of word order	Probable location Probable event Apply meaning information

Adapted from Chapman, R. Comprehension strategies in children in: Hooper, J. (Eds.) *Speech and language in children*. Oxford and Chicago: Cambridge University Press, 1978, pp. 103-127.

**Table 17.1. Summary of Comprehension Abilities in Children Younger than 3 Years**

Babies also tend to do something to the objects they notice, and because their repertoire of actions is fairly limited at this stage, parents can, by judiciously choosing their instructions, have a very good chance that the child will comply. For example, a mother might say to the baby, “See the pretty ball!” The baby, following her line of gaze, looks at the ball. The baby is very likely, then, to move toward it. If the mother, at the same time says, “Go get the ball for Mommy,” the baby can appear to comply with the instruction when, in fact, that was what she or he was going to do anyway.

The function of these strategies is to help the baby participate successfully in an interaction. Parents are also trying to get the baby to participate and succeed. They unconsciously behave in such a way as to give the baby ample cues, both linguistic and nonlinguistic, as to their meaning. In this way, parents’ speech to infants can



be viewed as a form of “hyperspeech” that provides contextual support for comprehension on concrete, observable levels ( [Fernald, 2000](#)). When the baby responds appropriately, two things happen. First, babies experience the pleasure of a positive social interaction. Second, they get another example of the way in which language works to encode what they already know about the world. In this way infants move closer to linguistic comprehension.

### Infant Sound Production

Crying is the newborn's principal form of vocal behavior. Beginning in the first month after birth, the infant masters the ability to produce cries that differentiate among affective states. Pain versus hunger cries are differentiated within the first week of life ( [Graham et al., 1983](#)). Other noncry vocalizations also emerge early in life. Contentment vocalizations can be distinguished from distress sounds within the first month ( [Ricks, 1975](#)).

The quality of these infant vocalizations changes drastically throughout the first year of life. [Stark \(1979\)](#) has presented a framework for describing infant vocal behavior ([Table 17.2](#)). According to this framework, the infant from birth to 2 months of age produces primarily reflexive cries and other vegetative sounds. Although the newborn's cries have a profound effect on the adults who hear them, the infant is not using the crying in any intentional way to attract the adult's notice. Rather, the cry is an instinctual response to an internal state such as hunger, cold, or boredom. Vegetative sounds such as burps, coughs, and sneezes are also reflexive, but adults respond to these noises as if they were communicative. This willingness on the part of the adult to attribute intentionality to the infant's early reflexive sound production may be one of the ways in which infants are “taught” to use sound to communicate.

Stage	Age Range	Vocalization Types
I	0-2 months	Reflexive cries and vegetative sounds
II	2-5 months	Cooing and laughing
III	4-8 months	Vocal play and beginning babbling
IV	6-9 months	Reduplicated babbling
V	9-18 months	Jargon babbling

**Table 17.2. A Summary of [Stark's \(1979\)](#) Stages of Infant Vocalization**

As the baby grows, the head and neck anatomy changes, resulting in a greater diversity of sounds that can be produced and a more speech-like resonance associated with vocalization. Between 2 and 5 months of age, babies begin two behaviors that are important for the development of speech and communication. One is the pleasant, somewhat speech-like sound that babies produce primarily in response to social interactions, known as *cooing*, or “comfort” sounds. The name arises from the “oo”-like quality of most of the vowels heard during this type of vocalizing and from the fact that most of the consonants produced sound like /k/ and /g/. Again, the reasons for the “coo” quality of these vocalizations are anatomic. As the baby lies in a prone or semiprone position, gravity operates most strongly on the relatively large posterior portion of the tongue, pulling it back toward the roof of the mouth. This oral posture produces the consonants we recognize as /g/ and /k/, as well as the vowel we recognize as “oo.”

A second new vocal behavior in this stage is the infant's laugh, which emerges around the same time as cooing. Usually accompanied by a social smile, infant laughing is produced in response to an interaction the infant perceives as pleasurable, very often because it is a known routine whose components are predictable. Thus, babies may laugh when mother plays “peek-a-boo” for them or when she assumes a posture as if ready to tickle the baby. Crying becomes less frequent during this period of increasingly diverse and speech-like vocalization.

The next stage of vocal development begins at about 4 months of age and extends to about 8 months. Stark refers to this period as “vocal play.” In this phase the infant begins to pronounce what sound like single syllables with vowel-like and consonant-like components. Although not approximations of words and not meant to convey any referential meaning, these early forms of babbling continue the infant's progress toward increasingly speech-like sounds. The consonants produced tend to be made more toward the front of the mouth than were those used for cooing. The infant's productions at this stage appear to be largely a function of mandibular oscillations, or oral opening-closing alternations, that create a motoric “frame” for speech ( [Davis and MacNeilage, 1995](#); [MacNeilage and Davis, 2000](#)). This notion is supported by co-occurrence patterns between consonants and vowels; for example, there is evidence that labial consonants tend to be accompanied by central vowels in early babbling ( [Davis and MacNeilage, 1995](#); [Vihman, 1992](#)).

Vocal play, unlike cooing, does not appear principally in response to social interactions. Although infants do use vocal play as a means of responding to or initiating contact with adults, babies also engage in vocal play when alone. Vocal play may, then, function as a means for the infant to “practice” the new sound production abilities he or she is acquiring.

[Lenneberg \(1967\)](#) was the first to note that it is during this stage that the deaf baby's babbling begins to differ from that of the normal hearing infant, although prior to the vocal play stage, the babbling of deaf babies resembles that of their normal hearing counterparts. [Stoel-Gammon and Otomo \(1986\)](#) substantiated these observations, showing that hearing-impaired babies at this age level had smaller repertoires of consonants than normal hearing peers and that, in fact, the size of the hearing-impaired babies' inventories decreases between 4 and 18 months, whereas that of the normal hearing babies increases. Thus, the ability to derive auditory feedback from vocal play appears to be important in the development of babbling repertoires and their eventual transition to speech. There is also evidence that vocal learning begins to be shaped in the middle of the first year by imitation of sound patterns in the ambient language. [Kuhl and Meltzoff \(1996\)](#) presented audio and video recordings of three vowel sounds to infants at 12 and 20 weeks and analyzed their vocalizations. Twenty-week-old infants were able to imitate specific vowels with appropriate relative formant frequencies. By 1 year of age, infants' reliance on ambient language models for production becomes evident in spontaneous utterances ( [Kuhl, 2000](#); [Vihman and de Boysson-Bardies, 1994](#)).

[Stark \(1979\)](#) reported that a new form of vocal behavior, which she referred to as “reduplicated babbling,” appears in the second half of the first year of life. This type of vocalization includes consonant-vowel combinations, such as /bababa/ or /nanana/, in which the same syllable is repeated over and over. Consonants most likely to appear include /b/, /p/, /t/, /d/, /m/, /n/, and the glide *y*. Like vocal play, reduplicated babbling often occurs when the baby is alone. [Oller and associates \(1998\)](#) report that this form of vocalization, which they refer to as “canonical babble,” is an important milestone of communication development. Their research suggests that children who do not develop canonical babbling by 10 months of age are at risk for later language disorders.

Toward the end of the first year of life, many babies begin to use “vocalables” or phonetically consistent forms. These are productions that are unique to the child in that they do not closely resemble any adult word, but are used reliably in certain situations. For example, [Carter \(1979\)](#) reported that one child consistently used an /m/ sound along with reaching to indicate that he wanted something. These early consistent forms are sometimes referred to as “protowords.”

### Infant Interaction and Communication

Infants' early vocalizations appear to constitute protoconversations with parents ( [Bateson, 1975](#)). Other early forms of interaction include making eye contact with the parents (around 1 month of age), smiling and laughing in response to speech (at 3 months), and vocalizing in response to sounds (at 4 months). Babies may also begin to imitate some of the parent's intonation patterns ( [Trevarthen, 1979](#)) and by 3 months of age show more vocal responsiveness to their mothers than to other adults. As the baby gains better motor coordination, more formal interactions occur, such as patty-cake or peek-a-boo games, waving bye-bye, or “following” conversations by looking first at one person then another. However, there is evidence that, even during the prelinguistic stages, infants can detect deviant parental interactions and are disturbed by them ( [Cohn and Tronick, 1989](#); [Tronick, 1981](#)). [Tronick \(1981\)](#) found that infants responded to a lack of appropriate maternal reactions by unusual patterns of gaze and finally by self-comforting behaviors.

Babies use gaze extensively to regulate interactions. They look at the parent when they are interested in interacting, and avert their gaze when they become tired or overstimulated ( [Stern, 1977](#)). Likewise, babies are sensitive to changes in parental gaze during interactions. Infants as young as 14 weeks smile significantly more



when an adult's gaze is directed toward them than during periods of averted gaze ( [Hains and Muir, 1996](#)). It seems likely that, from very early on, babies are sensitive to gaze as a signal of initiation and termination of social interaction. Babies can also follow their parents' gaze to attend to an object they look and/or point at, and appear to do so reliably by 1 year of age ( [Corkum and Moore, 1998](#); [Morissette et al., 1995](#)). They also begin to direct the parents' attention by looking at objects themselves. Parents will follow the infant's line of regard and look at what the baby looks at. Frequently, parents will comment on this object of the baby's gaze. These interactions, in which the parent and child share focus on an object, have been called "joint attention routines" ( [Bruner, 1977](#)) and are thought to be very important in laying the foundation for the basic topic-comment structure of language in which one speaker directs the other's attention to a focus of interest, on which the conversation then elaborates.

At about 8 months of age, babies begin to develop the representational and intentional skills that allow them to hold goals in mind long enough to pursue them through action. Cognitive development at this time also supports the ability to understand actions as a means to an end, and babies begin to use communication as a means to the outcomes—in parental attention or the acquisition of objects—that they desire. Such communicative acts usually become manifest at about the same time as other forms of intentional behavior emerge—about 8 to 10 months of age.

Communication at this stage is expressed primarily with gestures, such as holding an object up for the mother to view, or pointing. [Acredolo and Goodwyn \(1988\)](#) reported that the use of idiosyncratic symbolic gestures to communicate is quite common during the last half of the first year of life. They found that some children as young as 11 months of age used relatively stable gestures that resembled manual signs to stand for objects or actions for the purpose of communication. For example, a child might bounce up and down to indicate "rabbit" or press on the eyes to indicate "sun." Often these gestures are accompanied by a look at the parent to see that she is attending.

[Bates \(1976\)](#) categorized these early intentional behaviors into two broad types based on their communicative function: protoimperatives and protodeclaratives. Protoimperative speech acts are those in which the child attempts to get the listener to do something for him or to stop doing something, and would appear to evolve into linguistic imperatives or commands. Protoimperative speech acts include requests for objects, which the child can indicate by pointing or reaching. Requests for actions, which the child can convey by miming some part of a familiar ritual, also fall into this category. For example, the child might climb on the mother's lap and touch his nose to indicate that he wants his mother to play the "body-part-naming game" that is frequently a part of interactive routines at this stage. Protests or rejections also occur, in which the child communicates, by pushing away or turning away, the desire to turn down some object or activity the mother is attempting to offer.

Protodeclarative speech acts direct the mother's attention to an object of the child's interest by pointing to it, holding it up, showing, or giving it. These acts are thought to lay the basis for the later use of referential language. Protodeclarative speech acts are thought to evolve out of the earlier established joint attentional routines that appear to be very important for later language development. Several studies of language-impaired individuals ( [Baron-Cohen et al., 1996](#); [Paul and Shiffer, 1991](#); [Wetherby et al., 1989](#)) suggest that children who have various types of language disorders use protodeclarative speech acts with restricted frequency.

As the babies begin to evidence this intentional communication, parents "up the ante" ( [Bruner, 1978](#)), requiring a more sophisticated form of response in order for it to "count" as the child's contribution. Whereas we saw earlier that mothers accepted any child behavior, such as a burp or a cough, as a communicative response, when true intentionality does develop, the mother requires the child to do something more communicative in order to fulfill a turn. The child is now expected to imitate the mother, produce a conventional gesture, and eventually vocalize. In this way, the baby's communication is "shaped" into language.

## LEARNING THE LANGUAGE BASICS

Children's first words usually appear around the first birthday. From this time, until they reach the end of the preschool period, their communication develops at an enormous rate. Vocabulary grows from two or three words to over 5,000, sentence lengths grow from one word to five or more, and the sophistication of their pragmatic skills often amaze and astound the adults around them. We examine the four areas of language development that were outlined in the preceding, to get a sense of how these changes proceed.

### Understanding Language

Comprehension skills begin with the understanding of single words. Before children say their first word, they typically understand the meanings of several ( [Benedict, 1979](#)). This gap between receptive and expressive vocabulary size continues to exist throughout development. Even adults can often recognize meanings for words that they never use in their own speech. The meanings that children attach to their first words are not identical to the meanings of those words in the adult lexicon. Studies of vocabulary acquisition suggest that children use a "fast mapping" strategy ( [Carey, 1978](#)) to acquire an incomplete notion of the meaning of a word that allows them to use the word and refine its definition through subsequent feedback. This kind of strategy could help to explain the exponential rate at which words are acquired during this second year ( [Kay-Raining Bird and Chapman, 1998](#)). Fast mapping of novel vocabulary appears to be greatly influenced by maternal labeling practices as well as the child's orientation and focus. Mothers tend to combine labeling of novel items with physical designation of the items, a behavior that increases the likelihood that the child will make an accurate connection between label and object ( [Masur, 1997](#)). In the same way, children are more likely to correctly map novel vocabulary when focusing on the referent ( [Dunham et al., 1993](#)). For children who have difficulty following the speaker's locus of attention and associating novel words with the object on which they are focusing, as is often the case in children with autism, attempts to teach novel vocabulary can be less successful ( [Baron-Cohen et al., 1997](#); [Chapman, 2000](#)).

At first, as we saw, comprehension is context-bound, and children understand words only within familiar routines. Comprehension begins to be freed from the context around 18 months of age, when children are first able to respond to words for objects that are not immediately visible. When asked to "go get a diaper," at this stage, for example, they can fetch one from another room.

Certain classes of words tend to be acquired in comprehension during this period. By the age of 15 to 16 months, the child can point to his or her own nose, eyes, mouth, and other body parts on request, and by around 20 months, the child can point to these body parts on a doll or on another person. Names of animals the child has toys to represent or sees in picture books, and names of family members tend also to be acquired in receptive vocabulary at this time. By around 2 years of age, children understand the meanings of several prepositions (e.g., in, on, under) ( [Wiig and Semel, 1980](#)) and action verbs (e.g., run, hit, jump), although comprehension of verbs begins slightly later than the comprehension of nouns.

During the 12- to 24-month period, children's production of sentences is limited to one or two words. Are they able to understand much longer ones? Many parents believe so and often claim that children as young as 12 months "understand everything" said to them. Linguistic comprehension is still quite limited in this period, however ( [Chapman, 1978](#); [Paul, 2000](#)). Children manage to convince adults that they understand more than they do by employing a series of strategies or shortcuts for responding to the language that they hear, as they did at the end of the first year. Strategies in the second year, however, integrate emerging linguistic knowledge with the understanding of contexts and interactions.

[Chapman \(1978\)](#) characterizes the 12- to 18-month-old child's comprehension as demonstrating the use of "lexical guides to context-determined responses." Here, the child pairs his or her newly acquired knowledge of the meaning of single words with knowledge about what usually happens in interactive situations. This integration allows the child to act on objects the mother mentions by name, even if she does not look at them. Knowledge of how objects are conventionally used gives the child access to a "do what you usually do" strategy. This results in the child's using the mentioned object for its intended purpose, without having to understand the full linguistic force of an instruction. Thus, if told to "Brush your hair," the toddler is able to comply without fully understanding the instruction, simply by recognizing the word *brush* and knowing for what brushes are used.

In the latter half of the second year, as the child begins to combine two words in his or her own speech, the ability to understand two-word combinations also emerges, but comprehension is probably limited to not much more than two words per sentence. Understanding two words in a sentence allows the child to respond to apparently complex instructions without having to process their full linguistic form. For example, a child in this stage might successfully comply with the request, "Why don't you go and close that door for me?" not because the child understands the whole question, but because she or he was able to pick out the words *close* and *door* and knows that adults usually ask children to do things ( [Shatz, 1978](#)). There is also evidence that children at this age continue to make use of prosodic information, sentence length cues, and key word position cues in sentence comprehension ( [Shady and Gerken, 1999](#)).

Around the age of 3 years, there is a considerable expansion in the number of vocabulary and linguistic items (e.g., plurals, present verb tense, adjective comparison forms) that are understood. During the period of rapid growth in expressive language abilities, there is a corresponding rapid growth in the ability to understand language. The 3-year-old child understands much of what is said to him or her, but there are still some sentence types that cause problems, and children continue to

use comprehension strategies in responding to these difficult utterances. For example, the probable event strategy is used to respond to sentences that contain prepositions the child does not fully understand. Young 2-year-old children, for example, if asked to put a spoon *under* a cup, would be most likely to put the spoon *in* the cup, because they know that containers like cups are usually used to put things *in*.

By the age of 4 years, the young child has learned many of the basic grammatical rules of the language. But preschool children often have difficulty understanding complex sentences in which the order of mention of clauses does not correspond to the order of events, such as, "Before you brush your teeth, turn off the water." And they have a hard time understanding sentences with conjunctions such as *unless* and *although*, which involve negative hypothetical propositions (Owens, 2000). Preschoolers and young school-aged children also typically misunderstand a few other specific sentence forms that violate the regular rules for sentence interpretation (Chomsky, 1969). Although the preschooler sounds much like an adult speaker in terms of the production of grammatical sentences, it is possible to trigger misinterpretations of sentences with forms that violate the general rules of the language.

## Speech Sound Production

Typically, children pronounce their first understandable word around 12 months; however, the range of 8 to 18 months is considered normal (Morley, 1965). Although children begin using real words around their first birthday, babbling and nonmeaningful sound play continue to coexist with speech for some time. In both speech and babbling, the same sounds tend to be used. Stark (1979) calls the sound play heard during the second year of life "nonreduplicative babbling" or "expressive jargon." This babbling is more varied than the earlier, reduplicated form. It involves new types of consonants, particularly those known as *fricatives*, that involve constriction rather than complete obstruction of the airway. The /s/ sound is one common early fricative. In addition, more than one consonant may appear within an utterance. So instead of /bababa/, a child may produce /pata/. Syllable structures that are produced also become more complex. Reduplicated babbling primarily contains consonant-vowel sequences (/ba/). But jargon babbling may, in addition, contain vowel-consonant-vowel sequences (/aba/), as well as consonant-vowel-consonant productions (/bap/). Still missing from this form of babbling are the liquid sounds (/l,r/), and any combination of consonants, such as the *pi* in *play*. Sounds made in the front of the mouth, such as /b/, /p/, and /m/ are more frequent than those made in the back, such as /k/ or /g/. Jargon babble begins to take on the intonation contours of the ambient language at this time, so that the child's vocalizations sound as if the child is speaking, but the listener is unable to understand the words.

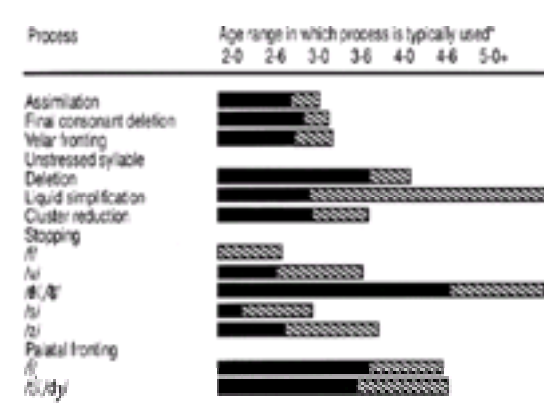
Regardless of the language being acquired, the first words children say are generated from a limited set of consonants that are similar to those used in jargon babble. This phenomenon is referred to as *phonologic selectivity*, or *phonologic bootstrapping* (Chapman, 2000). These include sounds produced toward the front of the mouth (e.g., /b/, /p/, /m/, /n/, /t/, /d/) (Jakobson, 1968). Although there is some individual variation in the precise order of acquisition of speech sounds, the acquisition of speech sounds does follow certain general patterns across all children (Bernthal and Bankson, 1998). The vowel inventory is mastered quickly (by the age of 18 months), whereas acquisition of the full inventory of consonant sounds continues for several more years. The first consonant sounds to be consistently articulated correctly are nasals (/m/, /n/, /ŋ/), stops (/p/, /b/, /t/, /d/, /k/, /g/), and glides (/h/, /w/, /y/). Subsequently, most fricatives (/s/, /ʃ/, /v/, /f/) are articulated correctly. Liquids (/l/, /r/), affricates (/tʃ/, /dʒ/), some fricatives (*th*; voiced [thy] and voiceless [thigh]), and consonant clusters (*bl*, *st*, *tr*, *str*) are among the last types of sounds to be consistently articulated correctly.

The first words children say usually have simple syllable shapes, such as consonant-vowel combinations (CV), or reduplicated syllables (CVCV): "ma," "mama." Between 18 and 24 months of age, however, children's repertoire both of sounds and syllable shapes expands with his or her expanding vocabulary. Stoel-Gammon (1987) found that by 24 months of age normal children produce nine to 10 different sounds and at least a few words with consonant clusters, like the *pi* in *play*. Paul and Jennings (1992) report that by 24 months normal children produce many syllables that contain two different consonants as well as some multisyllabic words. Stoel-Gammon (1987) also showed that 70% of the consonants produced by normal 24-month-old children were correct according to the adult target word.

Although 2-year-old children are accurate in their production of speech sounds most of the time, they do tend to make some characteristic changes in their pronunciation that serve to simplify the task of articulation. They may leave off the last sounds in words (*ca* for *cat*), delete unstressed syllables in long words (*nana* for *banana*), make the sounds in a word more alike (*doddie* for *doggie*), change certain sounds that are hard to pronounce (*wabbi* for *rabbit*; *sair* for *chair*), or leave out one of the consonants in a consonant blend (*pay* for *play*). These types of errors are very typical of children's speech in the 24- to 36-month period (Grunwell, 1982; Shriberg and Kwiatkowski, 1980; Vihman, 1998).

As we have seen, there is considerable individual variation in normal communicative development. Among the individual variations that have been reported with regard to the production of speech sounds are the avoidance of certain sounds or preference for certain sounds (Farwell, 1977; Vihman, 1981), a tendency toward consistency versus inconsistency of pronunciations (Vihman, 1981), reduplicating the first syllable of a word instead of producing a completely different second syllable (Ferguson, 1978; Schwartz et al., 1980), and using particular modifications of specific sound sequences (Ingram, 1974).

Between ages 2 and 4, though, most of these sound changes, both the typical and the idiosyncratic ones, drop out. Figure 17.2 shows the general order in which these errors, often referred to as *phonologic processes*, are resolved. Children older than 4 years who retain use of a significant number of these processes, so that the intelligibility of their speech is affected, would be considered to have an articulation, or phonologic, disorder.



**Figure 17.2.** A sequence of phonologic processes based on Grunwell (1987). *Language Disorders from Infancy through Adolescence: Assessment and Intervention*. St. Louis, Mosby, 2001.

By age 4 the normal child is fully intelligible, producing nearly 100% of speech sounds correctly. If errors remain, they are likely to occur on the last sounds to develop: *l*, *r*, *th*, and possibly *s*, as well as on the production of several consonants that occur together within a word, as in *scrape*. Normal preschoolers also continue to have difficulty with multisyllabic words, such as *spaghetti* or *aluminum*. About 5% of children enter school with phonologic deficits (Shriberg et al., 1997), but most of these involve residual errors on one or two sounds.

During the stage of systematic acquisition of speech sounds, speech dysfluency or stuttering may appear. It appears that approximately 75% to 85% of the young children who stutter during this period of time recover spontaneously within a few months' time (Homzie and Lindsay, 1984; Yairi and Ambrose, 1999). Consequently, intervention is generally not recommended at this age, unless there is evidence of distress, fear of talking, or avoidance of speech.

## Producing Words and Sentences

### EXPRESSIVE VOCABULARY DEVELOPMENT

The first words of children learning to speak any language share some properties in common. They tend to be words for objects and activities with which the child has direct contact. So, *shoe*, which a child can put on and take off by himself or herself, is more likely to be one of the first words than *shirt*. First words also tend to be those used very frequently in social interactive routines, such as *hi* and *bye-bye*. Although most first words are nouns, or names of things, not all are. *More*, *up*, and *no* are frequent entries in lists of early words.

Children, like adults, understand many more words than they say. What determines which of these comprehended words will be produced? One characteristic that



seems to be a factor is the sound structure of the word. Children are more likely to produce words that have at least a beginning sound that is already in their repertoire than they are to produce a word with sounds that are not under their control ( [Leonard et al., 1978](#)). This active process of selection and avoidance of words based on their sound structure is typical of children in this very early stage of language acquisition ( [Bernthal and Bankson, 1998](#); [Ferguson and Farwell, 1975](#)).

Children's use of words during this period also may not always conform to adult usage. Two common types of errors seen are *overextension* and *underextension*. Overextension is using a word to mean more than its true meaning (e.g., using "doggie" to mean "animal"), whereas underextension is using a word to mean only part of its true meaning (e.g., using "car" to mean "a car that is moving"). Overextension errors are most often based on shape (e.g., using "moon" to mean "sliced grapefruit") and taxonomic relatedness (e.g., using "banana" to mean "apple") ( [Gelman et al., 1998](#); [Graham and Poulin-DuBois, 1999](#)). It is important to note, too, that words are overextended in production much more often than in comprehension. [Rescorla \(1976\)](#) reports that children who label both a truck and a plane with the word *car* can very often point to pictures of the correct items when *truck* and *plane* are named for them. [Gelman and colleagues \(1998\)](#) examined overextensions in children of three age groups and found that although children in the two younger age groups (2:0 and 2:6) made overextension errors more frequently on production than comprehension tasks; this effect was not significant in the older group of children (4:5).

During the period of the first 50 words, children's "sentences" consist of one word at a time. These early one-word sentences are sometimes called "holophrases" because they seem to function as sentences and may convey meanings that are more complex than mere labels. For example, a baby saying "Mama" may actually be trying to convey a more complex message, such as "Mama, come here." First sentences tend to express the same protoimperative and protodeclarative intentions as children expressed during their first year with gestures and vocalizations.

Average expressive vocabulary size at 12 months is three words; at 15 months it is ten words ( [Templin, 1957](#)). By 18 months, most children are producing more than 50 words ( [Nelson, 1973](#)), and average number of different words produced is about 100 ( [Fenson et al., 1990](#)). By 20 months, average expressive vocabulary size is about 150 words ( [Dale et al., 1989](#)). The average 24-month-old child says 300 different words ( [Dale, 1991](#)). There is a great deal of individual variation in expressive vocabulary size, but recent studies suggest that children of 24 months of age who produce fewer than 50 words can be considered to be performing below the normal range of expressive language and are at risk for chronic linguistic handicap ( [Paul, 1996](#); [Rescorla, 1989](#)).

The rate of growth of vocabulary during the second year of life is not linear. Typically, there is slow acquisition of the first 50 words, with points at which new acquisition appears to halt for a time. Some words may temporarily drop out of the vocabulary. But about the time, the child acquires the fiftieth word or so, around 18 months, a spurt in vocabulary acquisition is seen, and vocabulary size increases suddenly and sharply ( [Fenson et al., 1994](#); [Hamilton et al., 2000](#)). The failure of a child to undergo this spurt in vocabulary growth during the second year of life may be another warning signal of risk for chronic delay.

Both expressive and receptive vocabulary size continue to grow rapidly during the third year. [Smith \(1926\)](#) estimated expressive vocabulary size at 36 months to be about 900 words, a threefold increase over this size at 24 months. As children increase their vocabulary, they develop mastery over a range of semantic categories. Spatial terms including *in*, *on*, *under*, *beside*, *next to*, *on*, *off*, *out*, and *over* ( [Boehm, 1969](#)) are generally mastered by 3 years of age. *Big* and *little* are the first pair of dimensional adjectives to be acquired, with *long* and *short* following soon after, both generally learned by 3 years. 2-year-old children use color terms occasionally. By 36 months, most typical children name two to three colors correctly. Children also learn to produce and understand question words *what* and *where*, and occasionally use *who* and *how* by age 3 ( [Tyack and Ingram, 1977](#)). Pronouns including *I*, *me*, *my*, *mine*, and *it* are usually used by age 3. *You*, *your*, *she*, *them*, *he*, *yours*, *we*, and *her* are frequently mastered by age 3 as well (Haas and Owens, 1985). The child's ability to use first-, second-, and third-person pronouns correctly appears to be strongly linked to his or her capacity for perspective-taking ( [Loveland, 1984](#); [Ricard et al., 1999](#)).

The average spoken vocabulary size at age 4 is about 1,500 words, and about 2,100 words at age 5 ( [Smith, 1926](#)). Receptive vocabulary size continues to exceed these levels at each age. Children learn to use and understand a great many classes of words during the preschool period. They master the use of most pronouns, with the exception of the reflexives (*himself*, *herself*, *ourselves*, etc.), which are not learned until school age. Kinship terms in addition to *mother* and *father* or their diminutives, which are part of the child's earliest vocabulary, enter the lexicon. Depending on the child's experience, most terms for extended family members are at least partially understood by age 5 ( [Haviland and Clark, 1974](#)), but full comprehension is not achieved until age 10 or so. The preschooler, for example, may know that he has a brother, but may not realize that he is a brother to someone.

Temporal terms enter the vocabulary at this time, with *before*, *after*, *since*, and *until* coming in earliest. These are used first as prepositions ("I'll go *after* school."), and only later as subordinating conjunctions ("I'll go *after* I get home from school.") Question words *why*, *how*, and *when* are used with greater frequency during this period, and questions containing these words are answered correctly more often, although the context of the question has an influence on correct responding ( [James, 1990](#)). Children are more likely to answer most types of wh- questions correctly when they can see the person, object, or event to which the question refers.

Children learn to refine their use of adjectives, adding more precise terms to their vocabulary during the preschool years. Although a 3-year-old child may know only *big* and *little*, by 5 years children generally learn *large/small*, *tall/short*, *long/short*, *high/low*, *thick/thin*, *wide/narrow*, and *deep/shallow* ( [Owens, 2000](#)). The positive member of the pair is usually learned first. *More* and *less* and *same* and *different* are other pairs of dimensional adjectives usually learned during the preschool years. *More* and *less* may be used initially interchangeably. By 5, most children use these terms correctly.

## DEVELOPMENT OF SENTENCES

Another important language milestone appears at about the time the child produces the fiftieth word, usually around 18 months of age. The child begins to combine words into "telegraphic" sentences ( [Brown, 1973](#)). Structurally, these two-word utterances are not random combinations of words, but follow a "minigrammar," with the positions in which certain words may occur being governed by specific rules ( [Bloom et al., 1975](#); [Braine, 1963](#)). Two-word utterances may express a variety of different types of meanings ( [Clark and Clark, 1977](#)), including an agent who performs an action (e.g., "baby cry"), an action that affects an object (e.g., "hit baby"), an entity given a location (e.g., "Daddy home"), or an object or person with a description (e.g., "nice Mommy"). These utterances resemble telegrams in that they include the most important words of the adult sentence, while leaving out the function words and word endings. Consistent production of telegraphic utterances is typically observed in children who have achieved vocabularies of between 100 and 200 words ( [Fenson et al., 1994](#)).

By the end of the second year of life, most children are beginning to produce some three- and four-word sentences. These early longer forms tend to include the most meaningful elements that were left out of the telegraphic sentences. So, although the telegraphic child might say "Daddy ball," to indicate that dad should toss the ball, the slightly older child may say, "Daddy throw ball." Many small function words and word endings are still omitted ( [Owens, 2000](#)).

An enormous amount of growth takes place in the child's production of syntax between 2 and 5 years of age. And, as is true for vocabulary development, the child's acquisition of syntactic skills depends on his or her ability to share attentional focus with those who are providing language models ( [Rollins and Snow, 1998](#)). During this period, children learn to elaborate telegraphic utterances through the use of *grammatical morphemes*, the units of meaning that are expressed as inflections on nouns and verbs, or as functor words such as articles (*a*, *an*, and *the*) or *be* verbs. [Cazden \(1968\)](#) studied the acquisition of 14 of these morphemes that seemed to undergo substantial change in children's usage. Her studies and others that followed found that children learning English acquired the morphemes in a relatively consistent order. Between 2 and 3 years, children were found to learn to produce the *-ing* ending, plural */s/* and possessive (*'s*), and the prepositions *in* and *on* in nearly all the contexts in which they were required by the adult grammar. Children learning other languages, though, do not acquire these morphemes in the same order as do English-speaking children ( [Kvaal et al., 1988](#)). The differences found across languages in the order of acquisition of grammatical morphemes are thought to reflect the fact that some languages have more difficult or complicated ways of expressing the meaning encoded by these morphemes. For example, in English, articles are learned relatively late, but in Spanish, children produce articles as one of their earliest morphemes. The relative ease of acquisition of articles in Spanish is thought to be a result of the fact that English articles are somewhat harder to hear in running conversation, whereas the Spanish forms, *el*, *la*, *los*, *las*, and so on, are more salient acoustically.

Between 3 and 5 years, children learning English acquire mastery of many grammatical morphemes studied by [Cazden \(1968\)](#), including use of *be* verbs, articles, and third-person singular *s* marking on verbs (I run, you run, he run *s*). Children also master the use of past tense markers during this period. Typically, children start out by marking irregularly formed past tense verbs, such as *come/came*, *see/saw*, and *go/went*, correctly, before they begin using the regular past tense marker *-ed*. Between 3 and 5 years, the regular marker is acquired and, when it is, the child very frequently overgeneralizes it, marking incorrectly those irregular forms that were produced right earlier. This overgeneralization of past tense markers results in productions such as, "I falled down and got hurted." This replacement of an early correct form by an error that is the result of a process of overgeneralization is often taken as evidence that the child is actually inducing rules from the input language and not merely memorizing the forms heard because, presumably, adults do not produce these overgeneralized forms. The use of overgeneralized past tense forms may persist to age 5 or 6. Some overgeneralized plural forms (foots, mouses) may also appear during this period. Other grammatical morphemes that are mastered during the preschool period include the comparative (small/small *er*) and superlative (small *est*), as well as the agentive *er* marker, used to denote one who performs an action (teach/teacher) ( [Carrow, 1999](#)). As children gain knowledge about syntactic elements such as morphologic markers, they use this information to "bootstrap"



their way into learning of new forms. For example, children can discern the form class (e.g., noun) of a novel word by analyzing co-occurrence patterns of function words (e.g., articles *a*, *an*) and bound morphemes (e.g., plural *-s*) (Behrend et al., 1995; Chapman, 2000; Naigles and Hoff-Ginsberg, 1995).

A second aspect of syntactic development includes the expansion of the two basic units of simple sentences: the noun phrase (usually the subject or object of a verb) and the verb phrase, or sentence predicate (Miller, 1981). During the telegraphic period, the noun phrase and verb phrase segments of the sentence generally contain only one word. During the third year, the child learns to elaborate these elements by adding additional words to each. Noun phrase elaboration starts with the addition of a single element, usually an article or a modifier. Modifiers frequently used by 2-year-old children include *this*, *that*, *these*, *those*, *a*, *the*, *some*, *a lot*, *more*, *two*, *my*, and *your* (Miller, 1981). A child just beginning noun phrase elaboration, for example, would be likely to say, "My doggy!" but not "My black doggy." By the end of the third year, children are embedding elaborated noun phrases within sentences. When this occurs, the elaboration is most likely to occur on the object noun phrase at the end of the sentence, rather than on the subject at the beginning (Miller, 1981). So the 3-year-old child is apt to say, "Kitty has black spots," but not, "My new kitty has spots."

Verb phrase development begins with the production of single verbs that are unmarked for person, tense, or number. The child may say, "He throw ball" to mean, "Daddy threw the ball to me." *Can* and *will* are the earliest auxiliary verb forms to be used. Forms of the auxiliary verb *be* may also emerge at this time, but will not be used consistently or with correct marking (Miller, 1981). So the child may say, "You am going," or "You going home." Some use of irregular past tense forms (*came*, *saw*, *went*, *felt*) may appear by age 3, although regular past tense forms, which are marked by *-ed*, may continue to be produced without any marker (Tranham and Pedersen, 1976). The child may use both, "I came home," and, "I help Daddy" to refer to events in the past.

Children continue to increase both the elaboration of basic noun and verb phrases, as well as the complexity of sentence types produced during the preschool years. Young 3-year-old children consistently include a subject noun phrase in sentences, no longer omitting it as 2-year-old children do. Noun phrase elaboration becomes richer and more flexible throughout the preschool years by means of the ability to mark either the subject or object noun phrase with a broader range and greater number of modifiers. Toward the end of the preschool period, children begin using clauses rather than single words or phrases to modify nouns. These relative clauses, again, usually are used first to modify nouns in object position ("They're boys *that I know*"). Modification of subject nouns with relative clauses ("The boys *that I know* are big.") is seen in children's speech very infrequently until school age.

Verb phrase elaboration during the preschool period consists of the mastery of the verb inflections for past and third singular marking, as we saw. In addition, new auxiliary verbs are acquired, including *could*, *would*, *should*, *must*, *might*, and *shall* and past tense forms of the verb *to be*, that is, *was* and *were*. Auxiliary verbs *have*, *had*, and *has* are used very infrequently during the preschool period (Miller, 1981). When preschoolers do use auxiliary verbs, they use only one at a time. They rarely produce sentences with multiple auxiliaries, such as, "I *could have* helped you."

A third area in which syntactic development is seen is the production of the various sentence types available in the language. Two major sentence variants have been found to show significant development between ages 2 and 3 years in children acquiring English: negative sentences and questions. Negative intentions appear very early, as we saw; one of the first preverbal intentions children express is the desire to reject, protest, or deny their interlocutor's utterance. As we saw earlier, *no* is one of the first words almost every child says. Children in the telegraphic period can produce some simple sentence variations. Negative sentences, for example, are produced by putting a *no* or *not* at the beginning or end of a two-word phrase (Klima and Bellugi, 1966). Therefore, the child may say, "No Daddy go," meaning, "Don't leave, Daddy," or, "Go bed no," meaning, "I don't want to go to bed."

Between 2 and 3 years of age, the child learns some new negative markers. These include *can't* and *don't*. It is interesting to note that these early negative forms appear before their positive correlates *can* and *do* are used (Brown, 1973). When *can't* and *don't* are added to the repertoire of negative markers, the child also modifies the rule for placing them in sentences. Although negative markers were first placed at the beginning of the sentence, between 2 and 3 years of age, the child begins to move them inside, generally putting them between the noun and the verb. So the 2-year-old may say, "Daddy, no eat my candy" or "I can't find it."

Negative sentences are generally produced correctly by age 3. Forms such as *isn't*, *aren't*, *doesn't*, and *won't* are added to the repertoire. By age 4, children are also using past tense negative markers such as *wasn't*, *wouldn't*, *couldn't*, and *shouldn't*. By 4½, children use indefinite negative markers such as *nobody*, *no one*, *none*, and *nothing* but may err by producing these forms in double negatives ("Nobody didn't come.") (Miller, 1981).

Children also ask questions early in their language development, although these tend to be routines that are limited to a few memorized forms such as, "Whazzat?" and "Where (X) going?" A rising intonation contour is used to express questions that require a yes/no answer ("Have cookie?"), as it sometimes is by adults (Klima and Bellugi, 1966). Between 2 and 3 years, the child moves beyond the first routine questions to produce *what* and *where* questions that are novel, and not the result of a learned formula. They can now, for example, not only ask, "Whazzat?" but also, "What you eating?" Similarly, they can not only ask, "Where daddy going?" but also, "Where my glass of milk?" Still, these questions usually leave out auxiliary and *be* verbs. Yes/no questions continue to be marked only by rising intonation in this period (Klima and Bellugi, 1966).

Auxiliary verbs begin to appear in both the yes/no and *wh-* questions of young 3-year-old children. Yes/no questions are produced with auxiliaries that are appropriately inverted, or placed before the subject, at this time ("We *can* go."/"*Can* we go?"). Rising intonation also remains an option for asking this type of question. Shortly after they begin using and inverting auxiliaries in yes/no questions, children begin to use auxiliaries in *wh-* questions as well. At first, these questions are often produced without inverting the auxiliary (Tyack and Ingram, 1977). Instead of asking, "Why *can't* I have a cookie?" preschoolers may say, "Why I *can't* have a cookie?" They leave the auxiliary between the subject and the verb, as it would be placed in a declarative sentence, rather than moving it before the subject, as the rule for forming questions in English requires. Not all preschoolers make this mistake (Klee, 1985), although it is quite common. By age 5, most children produce all questions and negatives with auxiliary verbs appropriately placed.

A major addition to the child's repertoire of sentence forms appears at about age 3: Children begin producing complex sentences (Limber, 1973). These forms involve either joining two sentences together by means of a conjunction such as *and*, *if*, or *because* ("I like ice cream because it tastes good."), or embedding one sentence within another ("I think *I'll have an ice cream cone*"). The sentences are important not only because they allow the child to make his or her utterances longer, but also because they allow the combination of ideas within an utterance to produce a more condensed, elaborated statement. At 3 years, 5% to 10% of the typical child's sentences contain these conjoined or embedded clauses. This proportion increases to 20% to 30% by age 6 (Paul, 1981). The proportion of complex sentences continues to increase throughout the school years, although the most dramatic increases are seen not in children's speech, but in their writing (Loban, 1976).

## Pragmatic Development

### LEARNING TO CONVERSE

By the time the first word is uttered, the child has already learned to express affect vocally and attend to adults (Dore, 1975). Indeed, as children expand the purposes for which they are communicating, their vocalizations become increasingly differentiated and closer in form to conventional, "readable," word productions (Chapman, 2000; Messer, 1994). Still, conversations with 1-year-old children are very erratic and disjointed; the child typically uses sequences of utterances that are not directed toward the listener. Throughout the one- and two-word utterance stages, egocentric language is mixed with language being used to ask for needs and make observations about the environment. It is not abnormal during this period for the child to talk perseveratively about a single topic or indiscriminately echo utterances (Fay and Anderson, 1981; Tranham and Pedersen, 1976).

Although young toddlers can respond to adult speech with the communicative means they have available—gestures, vocalizations, or words—they are not very reliable about doing so and often need coaxing to speak when spoken to. Chapman (1981) reports that children begin to answer questions more reliably at 18 to 24 months, at first responding to primarily routine questions, such as, "What does a doggy say?" and, "Where is your nose?" In addition, toddlers show their compliance with the conversational obligation to speak when spoken to by acknowledging their partner's comments, often by imitating a portion of the partner's utterance.

A new communicative function emerges in the second half of the second year. Halliday (1975) refers to this as the "mathetic" or heuristic function of language. Although children at 12 to 18 months of age generally talk about the here and now, making comments on objects and events that are obvious in the immediate context and add little new information to the listener's knowledge base, the older toddler begins to use language both to learn about the world and to provide the listener with new information. One of the first heuristic uses of language is the asking of questions. "Whazzat?" is a frequent early question with which the toddler requests the names of objects. The use of this function is a manifestation of the child's developing understanding that language can be used to learn about the world.

The child's ability to stick to a topic improves in the third year, although by 36 months still only 50% of child utterances continue the topic of the previous utterance

([Bloom et al., 1976](#)). Children of this age have difficulty sustaining a topic for more than one or two turns, particularly if the topic is one they did not initiate ( [Owens, 1999](#)).

Two-year-old children also begin to learn how to deal with conversational breakdowns. If an adult signals such a breakdown, by asking, "What?" the 2-year-old child typically responds by either repeating the utterance, usually with some phonetic change (C: I kit ball. M: What? C: I kick ball.) or by deleting an element (C: I kick big ball. M: What? C: I kick ball.). Children of this age rarely request another speaker to clarify an utterance, however.

Even at age 2, children realize that we talk differently to different people. A 2-year-old child frequently talks differently to his or her mother than to the father, for example, often using more polite language to the father. Two-year-olds frequently speak to babies in a high-pitched voice, as adults do. Another determiner of how we choose to say things is the level of knowledge we believe the listener possesses. Two-year-old children are not very good at making such judgments. They frequently make the assumption that adults know everything and fail to provide necessary information. This tendency can lead, for example, to a 2-year-old child telling his grandmother over the phone, "See, I got this for my birthday!"

Two-year-old children are still in the process of learning the obligation to say things, particularly to make requests, politely. Frequent parent reminders help facilitate this development, and politeness is one of the few aspects of language development that is explicitly taught. Two-year-old children have little flexibility in their range of politeness markers, and if told to "ask more nicely" will either add *please* to their request or produce it with a whining intonation ( [Bates, 1976](#)). They may also produce problem or need statements, such as "I'm hungry," or "I can't reach it," as forms of requests. Two-year-old children do understand some of the social parameters of polite requesting and are more likely to include *please* in their request if the listener is bigger, less familiar, or in possession of something they want ( [Ervin-Tripp and Gordon, 1986](#)).

Another way in which the child's communicative skill advances in the third year of life is through the expansion of purposes to which language can be put. Children learn to use language to convey new information, talk about past events, and imagine or pretend ( [Chapman, 1981](#)), as well as combine more than one intention within an utterance. Similarly, 2-year-old children learn to use a variety of forms to express their intentions. They may say either "I want cookie" or "I hungry" for example.

By the age of 3, pragmatic development becomes more sophisticated and interactional. Egocentric speech and echolalia gradually disappear, whereas the range of language functions increases. The child's language is now used to announce intentions and describe ongoing events. There is a growing awareness of the conversational function of language, with the child being more likely to await responses and less likely to ignore interruptions ( [Garvey and Berninger, 1981](#)).

There is rapid development of pragmatic skills between the ages of 3 and 5 years. Language is still used to announce intentions and describe ongoing events, but there is also increasing use of language to describe events and incidents from the past and relate incidents from the past to present events. The child's conversations show increasing sensitivity to the listener ( [Van Kleeck, 1984](#)) (in allowing turn-taking, responding appropriately to the other person, self-correcting speech, recognizing taboo words, and providing clarification when requested). Language play occurs in the form of rhymes, jokes, and exaggerations.

In the preschool period, language begins to be put to use for the purpose of reasoning, solving problems, monitoring thought and actions, relating events, and constructing complex imaginative play ( [Tough, 1977](#)). Language now becomes an instrument of thought, rather than just a system for mapping what one sees and does into words, as toddlers primarily use it. This new function of language is, of course, made possible by increasing cognitive development. But it also, in and of itself, contributes to the child's intellectual skill, so that the growth of language and cognition become more closely entwined during the preschool years.

Preschoolers learn a broader range of devices for making requests politely during this time. Rather than merely adding *please*, they begin to use permission requests ("Can I . . .?"), question requests with modals ("Would you . . .?" "Could you . . .?"), and indirect requests such as "Why don't you . . .?" and "Don't forget to . . ." ( [Garvey, 1975](#)). By 5 years, children are using hints and other forms in which the real object of the request is never mentioned explicitly at all. So they might request a snack by saying, "Gee, Mom, those cookies you made sure smell good. You make the best cookies in the world." In addition, in playing with peers, preschoolers demonstrate knowledge that requests must fulfill certain conditions, such as that the speaker must have some reason for making the request rather than doing the action himself or herself. [Garvey \(1975\)](#) shows that preschoolers often make adjuncts in their requests to each other, in order to establish that these conditions have been met. They might say, for example, "Get that hammer for me. *I can't reach it.*" Despite this demonstration of the forms and conditions necessary for successful requests, preschool children still have much to learn about using verbal means to regulate others' behavior. They are not very efficient at getting others' attention when it is not already directed to them or in persuading others to do something about which the listener is reluctant. During the preschool years, children learn to increase the degree to which they can elaborate on a single topic of conversation. By age 5, children spend an average of five utterances on a single topic ( [Brinton and Fujiki, 1984](#)), increasing the degree to which each utterance maintains the topic and adds new information.

Around 3 or 4 years of age, children begin to use linguistic devices to provide links between the ideas they talk about in the conversations. One of these *cohesion devices* is the use of pronouns to link referents, as in "I have a *friend* at school. *He* builds blocks with me." Use of definite/indefinite articles (a, the) is another cohesive device that emerges during this time. The indefinite article *a* is used to signal that a referent is being introduced into the conversation for the first time ("Alice saw a large cake."). When the same referent is mentioned again, the definite article *the* is used to signal the listener to retrieve the original referent from memory ("Alice ate *the* cake."). The ability to build these cohesive units or *texts* is one of the ways the child's discourse skills advance during the preschool years.

Children between 3 and 5 years also become more adept at repairing conversational breakdowns. They are able to respond to requests for clarification of specific information, such as "You went *where* for dinner?" and can reply with just the piece being requested ( [Gallagher, 1981](#)). Still, they rarely request clarification themselves when they misunderstand and cannot reformulate their message or provide missing background information when the listener looks confused.

With increasing linguistic competence, the child is able to take advantage of many more communicative situations during the preschool years than she or he was as a toddler. Not only can the child engage in a carefully scaffolded conversation with mother, but now the preschooler can talk also with peers, older children, and adults outside the family. Less conversational management is needed for the child's talk to succeed. But parent-child conversations at this time still play a very important role in structuring the child's social world, and much social learning goes on through the medium of language ( [Cook-Gumperz, 1979](#)). Parents tell preschoolers what to do in different situations, how to behave, and what to expect, and interpret situations verbally for the child, providing reasons and explanations. Language itself becomes part of the experience of social relations, and the preschool child is socialized linguistically, as well as behaviorally, into the community in which he or she will be expected to operate. Talk with peers, too, particularly in the context of cooperative play, teaches the child how language can be used to initiate and construct social relations. Much preschool interactive play is done primarily through the medium of language, and the child learns to exploit its flexibility by using language in this interactive fashion.

#### NARRATIVE DEVELOPMENT

Although conversation is one form of discourse acquired during the preschool period, narrative is another. Narratives are extended monologs that involve the reporting of the actions of people or animals or objects that take on human characteristics. Stories have particular structures that dictate their form and content; these are often referred to as "story grammars" ( [Stein and Glenn, 1979](#)). Unlike conversation, narratives almost always involve the use of *decontextualized* language, or language that refers to events outside of the immediate context. Because the use of decontextualized language is associated with acquisition of literacy skills, narrative development is encouraged from very early on ( [Peterson et al., 1999](#); [Snow, 1983](#)). Children as young as 2 can tell stories, although their narratives do not adhere to the typical story grammar structure. They are usually collections of tangentially related events without any discernible plot. Their narrations consist of elements "heaped" together and not organized in causal or thematic ways ( [Applebee, 1978](#)). As children progress through the preschool years, their stories gradually move closer to the conventional form used by adults in their culture. By age 6, most children can relate a more or less coherent narrative that follows a simple story grammar format. As children continue to develop their narrative abilities, they use increasingly sophisticated means of creating narrative cohesion. They not only maintain clarity about what happened to whom by making unambiguous reference to specific characters ( [Wigglesworth, 1997](#)), but they also are able to use inferential and perspective-taking skills to provide evaluative information regarding causes of events and characters' feelings, beliefs, and motivations ( [Eaton et al., 1999](#)).

#### METALINGUISTIC DEVELOPMENT

One new area of language use that arises during the preschool years is the ability to use language to talk about language itself. This capacity is called *metalinguistic awareness*. One way in which preschoolers manifest this ability is in their play with language. Children begin to use words just for the fun of it, in interchanges such as this one described by [Garvey \(1975\)](#):



Child 1: It's snaky because it has snakes.

Child 2: And it's hatty because it has hats.

Interest in rhyme is another manifestation of metalinguistic awareness. Here children show that they are attending not only to what a word means, but also to other properties, independent of meaning, such as the word's sound and its similarity to the sounds of other words.

Children begin to understand words with multiple meanings (such as *run*) when: (a) they have developed a sufficiently broad base of vocabulary knowledge, and (b) they have gained the awareness that one word can be "mapped" in different ways. Multiple meaning words require children to think about what is represented (the word) and how it is represented (the meaning). Use of language in role-playing also demonstrates the child's metalinguistic skills. For example, preschoolers often correct each other's usage in role-play, commenting on its appropriateness. A child might tell another, "You can't call me 'Willy.' You're the baby. The baby has to call the father 'Daddy!'" These emerging metalinguistic abilities help to lay the foundation for the acquisition of literacy during the school years.

## LANGUAGE DEVELOPMENT AFTER AGE 5

### Comprehension

The comprehension abilities that are acquired after the age of 5 years include the ability to decode syntactic structures that constitute exceptions to the usual rules of the language. By age 7 or 8, most children have gone beyond the use of comprehension strategies in processing these sentences and can decipher the intended meaning of sentences such as "The doll is easy to see" (versus "The doll can easily see"), "John asked Mary where to go" (deciphering who it is that is confused about where to go), and "John promised Bill to leave" (versus "John promised that Bill would leave") ( [Chomsky, 1969](#)). In addition, the child's growing ability to recognize the deep structures of language permits the recognition of different levels of meaning and the understanding that ambiguous sentences have more than one meaning.

Similarly, after the age of 5, the child begins to understand discrepancies between meaning and form in utterances ( [Ortony et al., 1978](#)). For example, the child gradually learns to move away from literal interpretations (e.g., of metaphoric language, proverbs) to understand connotations, to "read between the lines," and make inferences ( [Livingston, 1982](#); [Nippold, 1998](#); [Resnick, 1982](#)). This is a slow process; although the basic comprehension of figurative language is present at the age of 9 years, major improvements in the understanding of idioms, metaphors, and proverbs continue after this age (Nippold, 1998a). It is not until the teen years that discrepancies between voice and message (e.g., the sarcastic tone of voice) are fully understood.

In addition, a wider understanding develops of the concepts of temporal relations, spatial relations, number markers, emotional expressions, and abstract ideas. This development involves more than simply adding new vocabulary items; it includes refining the child's entire stored lexical network of interrelated meanings. These refinements in vocabulary comprehension are also a slow process that continues until college age ( [Nippold, 1998](#)).

### Speech Sound Production

Most children can produce all sounds of their language correctly by age 7. Sound errors that persist past this age are no longer developmental; instead they represent a disorder of speech sound development. During the years from 6 to 16, it appears that children add subtle skills to their knowledge of their sound system. These skills may include full use of appropriate morphologic derivative forms (e.g., vowel changes in the pronunciation of differing forms, such as *divide/division*) ( [Moskowitz, 1973](#)), the use of contrastive stress (e.g., to distinguish between *greenhouse* and *green house*) ( [Myers and Myers, 1983](#)), intonational contrasts, such as *pre'sent* versus *present* ( [Cruttenden, 1985](#)), use of dialectal variants (Grunwell, 1986), and, ultimately, representations of the sound system in the form of spelling skills.

### Producing Words and Sentences

The child's basic knowledge of the rules of his or her language is complete by age 5, and the child's expressive development takes the form of more subtle refinements to this essential knowledge. Grammatical errors become less common, although they may still occur in noun-verb agreement (e.g., "He haves the toy"), mass-count distinction (e.g., "I want more macaronis"), pronoun case forms (e.g., "Him and her went"), and irregular verb formations ("The candy had been ater"). Much learning about other morphologic markers, particularly about prefixes and suffixes, which augment the meaning or modify the part of speech of a word, goes on during the school years ( [Wiig and Semel, 1984](#)). Vocabulary growth continues throughout the school years, at an estimated nine words per day ( [Carey, 1978](#)). Both these developments, as well as the acquisition of more formal, literary types of syntactic structures, depend heavily on exposure to literate language forms used in written expression. Children who do not acquire good reading skills will be limited in their access to these more mature forms of expression, and their language development will begin to lag behind that of their peers.

Grammatical differences between the child's language and adult language are not apparent in general conversations by the age of 6 years. The formations and concepts that have still not been learned at this stage consist not so much of new structures, but of the ability to combine a greater number of complex structures within one utterance to produce language that is denser. In addition, children exhibit more frequent usage of complex structures more common to literary forms of language than to everyday conversation, but these changes are more likely to be seen in writing than speech ( [Barrie-Blackley, 1973](#); [Chomsky, 1969](#); [Scott, 1988](#)). The increase in the use of these low-frequency literary forms and the propositional density of utterances in written expression continues throughout adolescence.

### Pragmatic Development

Pragmatic development continues through the school years, with language being used in a more diverse range of social interactions ( [Barnes and Todd, 1978](#); [Stephens, 1988](#)), including telling stories, engaging in holiday rituals, sharing ideas, providing examples, taking perspectives, discussing problems, and appraising alternatives. The child's knowledge of rules for language usage expands to include "code-switching" (how to adapt language forms to specific environments) ( [Gleason, 1973](#)), politeness markers (e.g., "please," "sorry") ( [Leonard et al., 1978](#)), how to make indirect requests, and how to anticipate and repair breakdowns in conversations.

Gradually, the child's ability to narrate stories becomes more sophisticated, moving from a temporal chain approach toward the ability to link elements logically and develop a central theme ( [Applebee, 1978](#)). Narrative skills continue improving until college age ( [Johnson, 1983](#)), and there is an increasing ability to embed multiple episodes within stories, understand and express the motivations of characters, and imply and infer information in narratives.

Metalinguistic abilities continue to improve throughout the school years, serving as both a cause and consequence of the acquisition of literacy. One particularly important metalinguistic skill that typically emerges at the beginning of school is the ability to break words down into units of sound and associate these sounds with letters. This skill, referred to as phonologic awareness, has been shown in a large body of research to be highly related to later reading ability ( [Snow et al., 1998](#)). Moreover, it has been shown that children who do not come to school with phonologic awareness who receive instruction in counting sounds in words and associating sounds with letters do better in reading than children who do not receive this instruction ( [Blachman, 1999](#)). Phonologic awareness skills, then, have an especially strong link to literacy development.

Children also learn what a "word" is, that it can be thought of as apart from the concept it stands for, and that it can be identified as a printed symbol. They learn that words can be broken down into smaller units of sounds, and these sounds can be represented as letters that can be decoded into spoken words. Another metalinguistic skill acquired during the school years involves the ability to define words using synonymous or categorical terms instead of by simply stating a function or by giving personal association. The ability to discuss the structure of language is also included in metalinguistic skill. School-age children learn to identify parts of words—such as syllables, first and last sounds—and parts of speech. They learn to discuss similarities and differences in meaning among words and sentences and talk about ambiguities in meaning and figurative uses of language. Unlike the other areas of language acquisition we have been discussing, though, the acquisition of these metalinguistic skills is highly dependent on instruction. They are not part of the "natural" progression of language, and children not "taught" these skills, through exposure or direct instruction, do not necessarily learn them on their own.

## SUMMARY



The developmental pathway from infancy to young adulthood is characterized by increasing complexity in decoding and comprehension of words and structures, speech sound production, encoding and production of words and structures, and the use of language in interactions. Although there is evidence of certain individual differences, speech and language acquisition follows general patterns in which more and more complex structures are acquired, permitting more and more subtle distinctions in meaning and greater flexibility in form.

The enormity of the speech and language acquisition task and the speed with which it is normally accomplished helps us to understand the relatively high frequency of delays and disorders in this area of development. An intact linguistic system is crucial not only for fluid communication, but also for the acquisition of literacy skills that are so necessary to academic success. For this reason, children who demonstrate delays in the development of language are at risk not only for communicative disorders, but also for learning difficulties in school. These disabilities, in turn, may result in problems with behavior, social development, and self-esteem that can accompany learning disorders. For these reasons, early identification and remediation of language learning problems is especially important.

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## 18 COGNITIVE AND AFFECTIVE IMPLICATIONS OF IMAGINATIVE PLAY IN CHILDHOOD

Jerome L. Singer, Ph.D.

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*I dwell in Possibility  
A fairer house than Prose  
More numerous of windows, —  
Superior of doors.  
Emily Dickinson*

Why do children play? This issue has intrigued poets, as well as behavioral scientists from the days when Friedrich Schiller first proposed his theory of surplus energy, which explained that the boisterous play behavior of boys was a necessary preparation for later work activities. Adopting a more clearly evolutionary orientation, the first major researcher on play, Karl [Groos \(1901\)](#), proposed that play emerges out of natural selection as a form of necessary practice on the part of animals or children. The playful fighting of animals or the rough-and-tumble play of children and many of the playful courtship behaviors of animals and children are essentially the practice of skills that will later aid their survival. This adaptive viewpoint is further amplified in the conception of the Viennese psychologist [Karl Buhler \(1930\)](#), who pointed to the inherently pleasurable nature of play activity as a feature of children's growing skills and abilities in his concept of *Funktionlust*.

The psychoanalytic conceptions of play originated in Freud's proposals that play itself emerged under conditions of deprivation of initial gratification in the child, which caused it to "hallucinate" the image of a satisfying object such as the mother's breast. This hallucinated image later could become a source of at least partial drive satisfaction and permit the child to delay or control random, restless movements and cries of distress. The brief interval of delay becomes a central foundation in psychoanalytic theory for the later formation of the ego ([Freud, 1908/1958](#); [Singer, 1955](#)).

Later [Freud \(1920/1962\)](#) modified the theory from its initial purely sensual, instinctual aspect to include the concept of mastery, which he exemplified in a much-cited example of his grandson's playing a kind of hide-and-seek game. Even here Freud continued to emphasize that the game involved an attempt on the part of the child to deal with a conflict over maternal attachment and resentment of the mother's leaving through repetitive efforts at mastery. Psychoanalytic theories of play continued to emphasize the libidinal and drive satisfaction features of play, but there was increasing emphasis in work such as that of [Peller \(1959\)](#) and [Waelder \(1932\)](#) on the mastery elements, which in some respects formed the groundwork for a more cognitive, as well as drive-oriented approach. With the important observations of [Erikson \(1963\)](#), this early notion of play was broadened considerably. Erikson suggested that a psychoanalytic theory of play move beyond the satisfaction of early childhood psychosexuality toward incorporating broader features of social development that were "modeled" within the play format. Play thus becomes not just a form of vicarious gratification but provides a way in which complex difficulties and problems experienced by the child can be addressed and even possibly "healed" through the creation of a miniature and manageable world.

Perhaps the most thoroughly worked out theory of the origins of play, one with a much stronger cognitive focus, has been that proposed by [Jean Piaget \(1962\)](#), who believed that play emerges in the context of children's enactment of the two fundamental characteristics of their mode of experience and development, accommodation and assimilation. The former provides an opportunity to imitate and interact physically with the environment, whereas the latter represents the attempt to integrate externally derived percepts or motor actions into a relatively limited number of schemas or differentiated motor and cognitive skills already available to the child at this age. Physical mastery games, with their repetitious and practice qualities, are more associated with the accommodation feature, whereas the symbolic play of the child is associated more with assimilation. Piaget used the two notions to argue that play therefore is critical in early childhood for the eventual emergence of concrete operational thought in the child; he tended to minimize the role of play for children in the older age groups of 7 and up.

For Piaget, play is at least analogous to the fantasy life of the adult, but he does not devote much time in his extensive writings to elaborating this notion. By contrast, [Brian Sutton-Smith \(1966\)](#), although accepting the importance of Piaget's emphasis on early childhood development, has proposed that play continues through adolescence as a means by which preschool and older children can create miniature environments in which they gain a sense of power through elaborating and reconstructing those complexities, emotional and cognitive, that they confront in the adult world that surrounds them.

It is important at the outset to be clear about a definition of play. The term has been widely extended to range from children's activity to more complex forms of adult ritual and competition. For the latter, see [Caillois \(1958\)](#), [Geertz \(1976\)](#), and [Huizinga \(1950\)](#). Indeed, the emphasis on all forms of play and their role in learning elaborated by [Bruner and his coworkers \(1976\)](#) has moved the notion perhaps too far afield from the focus of this chapter. The definition we entertain was proposed by [Klinger \(1971\)](#): Play is behavior that is neither consummatory, instrumental, nor competitive; it involves behavior that is neither socially prescribed, as in ritual, nor constrained in some way by social expectations and conventions. More concretely, then, play is behavior that is not obviously associated with direct satisfaction of biological needs such as eating, drinking, overt sexual gratification, or the overcoming of immediate obstacles in one's life situation. It is also relatively free of any effort to meet standards set up by society. At least the standards of play, one might say, are established by the players themselves within a context that may not be the same as those ordinarily observable in society.

Piaget confronted this issue more clearly in his organization of play into games that involve simple sensory pleasure and exploration, games that involve mastery and expression of physical skills, symbolic play (with which this chapter deals), and games with rules that are manifest in activities, such as the structured play of athletic contests or board or card games. The focus of this chapter is specifically on the nature of symbolic play as exemplified by games of pretend and make-believe. Such play seems of special importance not only for the child's mental development but also for the gradual emergence of the capacity for fantasy and imagination in the older child and adult. The theories we mentioned in many ways have gradually been integrated into a cognitive-affective approach that serves as the organizing focus of the present chapter.

It is becoming increasingly clear to those of us who study both adult and children's imagination that the make-believe or pretending of early childhood is fundamental for the development of all competent adult cognition and emotional functioning. [Piaget \(1962\)](#) emphasized the role of symbolic play as a precursor for those processes of logic and orderly sequence whose epistemology was his main concern. Increasingly, we can also recognize that all mature human thought and information processing is not limited only to scientific or mathematical sequences that are sequential in nature and characterized strongly by verbal expression and grammatical structure. The seminal analyses of [Jerome Bruner \(1986\)](#) have made it evident that effective thought takes on a narrative or subjunctive form as well. In our processing of information, we must not only organize it into logical structures but also examine the alternative and future possibilities or even consider the darker alternatives that appear in any new human experience. Indeed, we store information, as is increasingly clear in memory research, by organized verbal schemas, on the one hand, and through narrative episodes or possibilities in the form of fantasies and daydreams, on the other ([Singer, 1985](#); [Singer and Salovey, 1991](#)). Narrative thought and subjunctive structure reflect the human capacity for what the great neurologist [Kurt Goldstein \(1940\)](#) called "taking an attitude toward the possible." He believed this capacity reflected the optimal functioning of both a healthy and intact brain. Through this orientation to the possible, one becomes capable of exploring a range of potential futures or, in effect, traveling through time and space to a different or better childhood or maturity ([Singer and Singer, 1990](#)). [Sigmund](#)

[Freud \(1911/1962\)](#) too used the term *trial or experimental action* to characterize thought and the emergence of his concept of the ego.

This capacity for reconstructing one's past or for planning for one's daily activities through mental rehearsal or simply daydreaming about future vacations, sexual opportunities, or fantastic space adventures also serves a broader function. Our imagination liberates us from the tyranny of *this* place, of *these* particular duties and obligations, of *these* particular people in our social milieu. We accomplish this restructuring not only through the abstraction of high-level logical or mathematical processes but also through our capacity for creating narrative and for using our skills at imagery to provide us with alternative temporary environments that we can manipulate for self-help and ultimately put into the service of orderly living or simply sustaining hope and effort ( [Singer, 1974](#); [Singer and Bonanno, 1990](#); [Taylor, 1989](#)). As Bruner has proposed, the object of narrative thought is not "truth" but verisimilitude or "life-likeness." He writes: "Efforts to reduce one mode [the narrative] to the other [the paradigmatic or logical] or to ignore one at the expense of the other inevitably fail to capture the rich diversity of thought" ( [Bruner, 1986](#), p. 11).

Viewed from this perspective, the simple make-believe play of the child (pretending early on that a stick is an airplane, that a soft toy can talk or respond to nurturance or admonition, that a few blocks or dolls can become the basis of an imagined city in which a relatively lengthy story unfolds) can be regarded as a fundamental precursor of the full-blown adult imagination. This chapter proposes that we accord the imaginative play of childhood its full weight as a fundamental and significant development of competency in the child. Without an adequate development of this narrative, subjunctive, or imaginative dimension, the child is subsequently handicapped both in cognitive and emotional development. Indeed, without sufficient practice in the skill of generating fantasy for self-regulation, the child not only will experience difficulties in school adjustment but also, if psychotherapy is required at some point, may not move ahead effectively into the process. Play therapists actually find that some encouragement and even training efforts are necessary to aid young children to engage in symbolic play as part of an ongoing treatment process ([Singer, 1993](#); [Singer and Singer, 1990](#)).

## A COGNITIVE-AFFECTIVE PERSPECTIVE ON PRETENDING

### Information Processing and Emotion

In the so-called cognitive revolution that has dominated psychology since about 1960, we perceive a new model of the human being. We now look on babies and children as information-seeking organisms striving to organize and integrate novelty and complexity, curious and exploratory but also more likely to feel comfortable and smile (as the research by Papousek on infants has shown) once they can experience control over novelty and assimilate new information into prior concepts and scripts about the sequence of events. The "smile of predictive pleasure" in babies, described by [Papousek \(1987\)](#) and his collaborators, and other signs of positive emotion (smiling and laughter associated with familiarity in adults) suggest that although cognition and emotion may be different systems in terms of bodily structure, they are closely related in actual human response ( [Izard, 1977](#); [Kreitler and Kreitler, 1976](#); [Mandler, 1984](#); [Tomkins, 1962, 1963](#)).

Theoretical analyses initiated by Silvan Tomkins have contributed greatly to the paradigm shift toward the cognitive-affective view of the human organism. Tomkins' work has succeeded in putting emotions back at the center of active research in personality and social psychology. He led the way in demonstrating that a cognitive perspective that involved an emphasis on the fact that human beings are continuously assigning meanings and organizing their experience in schemas and scripts does not preclude a significant motivational role for affect or emotion. With the support of increasing empirical research carried out on both children and adults by investigators such as [Carroll Izard \(1977\)](#) and [Paul Ekman \(1973; Ekman et al., 1982\)](#) and many others, we can now regard human beings as showing differentiated emotional response patterns that are closely intertwined with responses to the novelty, complexity, and other structural properties of information confronted from moment to moment ([Demos, 1995](#); [Shapiro and Emde, 1992](#); [Singer and Singer, 1990](#)).

This cognitive-affective perspective broadens our conception of human motivation considerably. Rather than reducing all human motivation to some symbolic reflection of infantile sexuality or aggression, one can propose that the basic emotions that have now been shown to exist across human species in the research of Ekman and Izard are motivating human beings in dozens of different situations independent of presumed drive pressures. Human beings seek, as Tomkins has proposed, to reconstruct in life or in thought situations that evoke the positive emotions of interest-excitement or joy; they seek to avoid in action or thought those situations that have evoked specific negative emotions of anger, fear-terror, sadness-distress (weeping), or the complex of shame-humiliation-guilt. Human beings are further "wired up" to express emotions as fully as possible and finally to control emotional expression where social experience suggests such control is necessary, either for safety or to avoid humiliation. Situations that permit experience and expression of positive emotions or that allow for appropriate control of negative emotions are intrinsically positively reinforcing. Those situations that are more likely to evoke negative emotions, such as fear, anger, distress, or shame, or that have blocked the expression of socially adaptive control of emotions may be experienced as inherently punishing or negatively reinforcing ( [Singer, 1974, 1984](#); [Tomkins, 1962, 1981](#)).

Memory and anticipation become central features related to emotional experience. Identifying, labeling, and gradually organizing new information into mental representations that are technically labeled as schemas accomplish this. These structures include schemas about persons or physical objects, schemas about self and others, and also scripts about action sequences or prototypes that become means for encapsulating a variety of common features of situations and persons into one fuzzy concept ([Mandler, 1984, 1988](#); [Singer and Kolligian, 1987](#); [Singer and Salovey, 1991](#)).

### The Child's Task in Information Processing

The problem for the child becomes, in effect, one of making sense of a complex world through the gradual formation of schemas and scripts and through the assimilation of new situations into established organized mental structures or, as language increases, into lexical categories as well. A child may respond with fear or terror when confronted with extreme novelty that cannot at once be easily assimilated into established structures. The child may respond with a smile of pleasure once a match can be made between new information and some well-known schema, and the novelty or ambiguity of the environmental situation can be assimilated. When the new situation is only moderately complex and some overriding schema is still available, the child may move to explore the moderate amount of novelty in the situation, and this evokes the positive affects of interest and excitement. Children and adults live in a situation of a perennially delicate balance between the potential for fear or anxiety evoked by new situations and the excitement of exploring such situations. By such exploration, one can assimilate incongruity into established schemas, enrich such schemas, or start to form new ones. The persistence over time of large amounts of unexpected or ambiguous information evokes the negative effects of anger or distress and sadness ([Singer and Singer, 1990](#); [Tomkins, 1962](#)). We all learn to bring sets of expectations of what may occur to each new situation. We practice such expectations through brief anticipatory fantasies, some more realistic than others depending on our maturity, the complexity of our schema structure, and our social development. Our task in each new situation is then to examine new information and determine whether it confirms or disconfirms some of our anticipations. [George Mandler \(1984\)](#) has particularly developed the implications of interrupted sequences of action and thought and of confirmations and disconfirmations of anticipations as the basis for emotional response.

If identifying and organizing new information becomes the fundamental, overarching demand placed on the child, then we can begin to understand the evolutionary function of imaginative play and the thought processes that seem to grow out of such play. As Piaget has shown, the efforts of children to master the environment and their own motor activities involve to some extent an accommodation to the physical characteristics of the environment and also to the speech, gestures, and other physical actions of adults or older children. In these efforts, children succeed through some form of successive approximation, but at the same time, they need to be able to assimilate such new actions into organized mental structures. This assimilation process is at first expressed by children through repetitive actions and talking aloud.

What seems like the intrinsic unmotivated character of play to an adult represents the child's continuing effort to create new meaning structures and to provide itself with a sense of control and power by reducing large-scale settings, persons, or social interactions to meaningful structures that can be assimilated into the as yet limited number of schemas the child has at its command. The startle responses or terror evoked in a toddler by the size and noise of a huge passing truck may be gradually transformed into curiosity and interest as the child attempts to reproduce the noises and movements of the truck through creating its own sound effects and manipulating blocks or toy trucks. Imaginative play may thus be understood as a means by which the uncontrollable qualities and complexity of one's physical and social environment can be gradually miniaturized and manipulated. In effect, we can see that much of adult human thought involves a similar effort to create at least temporarily a world one can control through replaying memories or anticipation and fantasy. Indeed, it can be argued that the very act of rehearsal and anticipation or even of elaborating possible future events into somewhat more bizarre fantasies may gradually approximate possible situations we do encounter. Such mental rehearsal may leave us better prepared to handle these or, at least, be less frightened by them when they do occur ( [Singer and Singer, 1990](#)).

### Attachment and Individuation: A Persistent Human Dilemma

Beyond the cognitive demand for meaning assignment and organization and its link to the arousal of emotion, human beings also must confront a persisting dialectic tension in their general motivation throughout their life span. This tension becomes evident in some of the very earliest months of childhood. On the one hand, there is



the need to feel close to others, be attached to or encompassed by parents, older siblings, or other caregivers and, later, by friends, or by a group by means of some symbolic group participation (e.g., a religious, ethnic, or nationalistic association). On the other hand, we all experience to varying degrees the need to preserve some areas of personal autonomy, some sense of privacy, personal competence, and individualized skill development. We must move through life in effect struggling to preserve a balance between affiliations with others or groups, thereby gaining a sense of community, while we also strive to maintain a sense of individuality and personal power ([Anqyal, 1965](#); [Bakan, 1966](#); [Jung, 1971](#); [Rank, 1945](#); [Schachtel, 1959](#)).

More recently in the framework of an object relations psychoanalytic analysis, [Sidney Blatt \(1990\)](#) has sought to show how the early childhood struggle between attachment and individuation may, if one pole or the other is overemphasized, become a focal area of conflict and eventuate in particular forms of psychopathology. [Bonanno and Singer \(1990\)](#) have further extended this polarity to identify a series of personality dimensions that recur in the literature along with particular affective tendencies, defensive patterns, variations in physical illness proneness, and emotional disorders that had already been identified along such dimensions by Blatt.

This conceptualization, although still largely speculative, is built around some available research evidence that suggests that optimal personality functioning and a hardy physical health status necessitate a reasonable balance between one's needs or strivings for affiliation and one's abilities to experience autonomy or individuation ([Bonanno and Singer, 1990](#)).

Within this conceptual framework, the child's make-believe world is one that represents a continuous working out of the dialectical tension between the need for closeness and affiliation and the need for privacy with its concomitant experience of personal power and individuality. Indeed, the very act of beginning to form individualized images, memories, and anticipatory fantasies becomes, in our crowded and sensory-bombarded world, the last refuge for an experience of individuality and personal privacy. For the developing child seeking on the one hand to sustain relationships with parents and others and feel the warmth of what [Schachtel \(1959\)](#) called embeddedness-affect, the ability on the other hand to create private games and to engage in floor play or sustain a relationship with a personally possessed stuffed animal or invisible playmate provides that experience of individuation that also seems so necessary in our human condition.

In view of this persistent human attachment-individuation tension, the emergence of an increasingly complex imaginative dimension subject to reasonable control (a kind of cognitive skill in itself) sustains the need for self-definition, for a sense of uniqueness and private power. The child must learn gradually to establish priorities in the direction of attention, whether toward the environment or material recurring from memory or forming itself into fantasies. Our practical survival may well demand that we assign a somewhat higher priority to the processing of externally generated stimulation, what [David Rapaport \(1960\)](#) wrote of as the "permanent gradient toward the external attention-cathexis." As we will see, our affective development also must involve reflection, introspection, the capacity to enjoy private experiences, to gradually shape and direct them, plan, and also create stories and mentally manipulate the range of future possibilities. For young children with limited motor and linguistic capacities and a smaller and less differentiated range of stored schemas and scripts, the balancing of such priorities may be reflected in the varying amounts of physical, rule-oriented, or social and imaginative play in which they engage.

Of course this very dilemma of attachment versus individuation is not only reflected structurally in the degree of priorities assigned by children to social interaction or imaginative experience. It is also the basis for the content of the play behavior. The themes of play in the child often reflect the continuing tension between the desire for closeness and for assertions of personal power, importance, or the need for privacy. Such manifestations are readily identifiable in play therapy observations. For the purposes of this chapter, however, we focus more on the structural features of imaginative play and how make-believe and the gradual internalization of such make-believe into childhood fantasy serve the cognitive and affective interests of the developing organism.

## THE EMERGENCE OF IMAGINATIVE PLAY

### Forms of Play

The generally accepted definition of play has focused on its intrinsic, nonmotivated nature. [Eric Klinger \(1971\)](#) has perhaps expressed this most clearly by defining play as behavior free of consummatory, competitive, religious, or social motives or constraints.

In his review of the available literature, [Philip Smith \(1982\)](#) raises the issue of whether play in animals serves any intrinsic evolutionary function. He argues that there is little evidence of the benefits of play for later social and cognitive skills, except in a few instances for the most humanoid primates. However, he does make it clear that with respect to human children, where language, imagery, or symbolic functions are involved, play, especially imaginative games, may turn out to have more broadly useful roles.

One need not argue that pretend play serves ultimate survival or reproductive functions from an evolutionary standpoint, however. Most careful observers of childhood and adult play behavior, such as [Brian Vandenberg \(1986\)](#), [Brian Sutton-Smith \(1982\)](#), [Michael Lewis \(1982\)](#), and [Mihalyi Csikszentmihalyi \(1982\)](#), point out that play is just an enjoyable activity in its own right. From the position already presented in the preceding, one can argue that most forms of play involve situations of moderate challenge, novelty, or incongruity. The playful interactions between self and others or between self and objects or, in the case of pure fantasy play, between self and symbolic others usually result in a further reduction of novelty or incongruity, thereby invoking the emotions of joy and the smiling response. What follows from such activities may be a further learned recognition (conscious or increasingly automatic) that within the defined structure of such play one can experience moderate challenge and also some reduction of incongruity in the future. I would propose that such a conception seems more specific than [Freud's \(1920/1962\)](#) proposal of a "repetition-compulsion" to explain why children repeat games again and again. As [Ernst Schachtel \(1959\)](#) has also pointed out, the repetitive nature of play in children may reflect not so much a compulsion as a continuing discovery of novelty in what for adults may seem commonplace but for children still provides puzzling material that must be assimilated into a previously limited range of schemas. The repetition thus is characterized by a sustained affect of interest in the elements that are still novel in even a simple story like "The Three Bears."

[Jean Piaget \(1962\)](#) has proposed that play follows a particular sequence in its emergence and the service of the development of more concrete operational thought. The child begins with more purely sensorimotor play, gradually evolves the capacity for symbolic or pretend play, and then goes on to more structured and orderly games with rules. These rule games range from simple examples such as "Ring Around the Rosy" (with hand holding, circling, singing, and the dominant rule, dropping down on the signal "all fall down!") and evolve into more complex games often played outdoors, such as "Red Light," or into parlor games such as "Statues" or "Charades" and board games such as *Chutes and Ladders* or checkers. Such games are critical for mastery of orderly thought, moral judgment, and other phases of concrete operations for mature thought in Piaget's scheme.

It is important to note, however, that for Piaget the functions of play were largely delineated in relation to their role in the emergence of orderly, formal logical thought processes. Other researchers, such as [Inge Bretherton \(1984\)](#) and [Brian Sutton-Smith \(1966\)](#) have criticized Piaget because they feel he tended to devalue the importance of play as a source of mature adult imagery and as the foundation for the playfulness that characterizes all human thought. Bretherton has also pointed out that Piaget tended to think of imaginative play as declining after the age of 4 or 5 rather than recognizing the importance of play as a forerunner for Bruner's narrative thought.

The research of the author and his colleagues with large numbers of children from diverse backgrounds who were observed during spontaneous play has made it clear that pretend play goes on well into the early school years and continues either "underground" in private thought or in the more sanctioned group forms of make-believe that are observable through the life cycle ([Singer and Singer, 1981, 1990](#)). Even games with rules, which are certainly an important step in the child's development of orderly and regulatory processing and self-control, often include elements of pretending or evoke private or shared fantasies well into adult life. If one watches adults and children playing relatively structured games such as *Monopoly*, one can frequently observe the tendency for the players to introduce make-believe components, sometimes even taking on particular make-believe roles for themselves as they participate in this otherwise rule-organized game. The reordering of the "given" realities is just as much a fundamental feature of human thought as is the attempt at faithful accommodation to the environment through coherent schemas, which Piaget emphasized in his epistemology. Indeed it was only in his posthumously published work that he finally addressed the question of the origins of possibility ([Piaget, 1980](#)).

The balance of this chapter examines some of the potential advantages for cognitive development and emotional expressiveness and control of imaginative play. To assert that imaginative play has a variety of short- and long-term benefits may reflect some cultural bias, as Brian Sutton-Smith has argued in some of his papers ([Sutton-Smith and Kelley-Byrne, 1984](#)). But even the subversive quality of *Mad Magazine* cartoon humor, of cards such as the *Garbage Pail Kids*, or of the punning and teasing of adults by children may serve an important balancing function for the developing child in a world in which children are powerless. In the face of life tragedies, parental neglect or abuse, parental poverty or humiliation, abandonment, illness, or death, the ability to step back from the situation and create playful narrative offers at least some solace if not a complete way out of the distress. Those children who do not experience encouragement through storytelling or pretending that fosters the development of a symbolic dimension or through repetitive sensory or physical play or games with rules may find themselves condemned either to

impetuous instrumental activity or to the apathy of a sense of isolation or extreme dependence on conventional rituals ( [Singer, 1993](#); [Singer and Singer, 1990](#)).

### Beginnings of Imaginative Play

It is rare for adults to observe any clear evidence of what might be called pretending or make-believe play before at least the age of 2½ in children. One can observe that such play gradually emerges out of parent-child interactions, the beginnings of games of peek-a-boo.

Make-believe play or early pretending takes on two general forms in the preschool child. One involves the overt floor play or monolog of the child, often using limited props and evolving eventually into very elaborate play with a mixture of structured toys, such as Barbie dolls or robot soldiers mingled with less clearly defined objects such as blocks or cloths and bits of string, that become employed in the more general service of storytelling. Another form of imaginative play emerges from the so-called transitional object of [Winnicott \(1953\)](#), the soft cloth or furry teddy bear that becomes either a subject of feigned conversation between child and teddy bear, or eventually, an invisible playmate assigned a name and personality. I will first deal with the role of floor play and turn a little later to the issue of imaginary playmates and their role in the child's development.

The following criteria, proposed by [Greta Fein \(1981\)](#), represent symbolic or pretend play:

1. Familiar activities may be performed even in the absence of necessary material or social context (e.g., a child pretends to drink from an empty cup or puts a doll to bed on the floor rather than in a doll bed or even on the couch).
2. Activities may not be carried out to their logical outcomes (e.g., a toy presumably imprisoned in a dollhouse escapes quite magically through the roof).
3. A child may treat an inanimate object as animate (e.g., a furry animal is offered food or a cup of tea).
4. An object or gesture may be substituted for another (e.g., a block is transformed into a spaceship).
5. A child may carry out an activity usually performed by another person or especially by an adult (e.g., pretending to be a doctor, pilot, or a teacher).

Of special importance is the conception of transformation, or the emergence through play of what [Alan Leslie \(1987\)](#) in a fine paper has called a metarepresentational mode of thought. [Leslie \(1987\)](#) has proposed the challenging question,

[How] is it possible that young children can disregard or distort reality (as in using a banana as a telephone). . . . Why does pretending not undermine their representational system and bring it crashing down? (p. 412)

Leslie's argument is that a major step in development involves the "decoupling" of the direct representations that we sustain of objects, persons, or situations from their perceptual images into a new set of metarepresentations that are symbolic or mental representations of the same original set of objects but now treated as part of an entire system of thought that one can modify, manipulate, analogize, or transform to metaphor. With the help to some extent of adults but also on the basis of an inherent capacity in the child, the ability emerges to create a frame in which otherwise very stable objects can be transformed into representations that bear only a tenuous link with their original shapes. When we walk into a room and perhaps unexpectedly see an elongated object on the floor, we may jump because we think it is a snake until we recognize with relief that it is a telephone extension cord. This is an "error" because we had no reason initially to treat the objects of the room as other than percepts. When on another occasion we say to a child playing "Explorer" with us in the same room, "Look! There's a dangerous snake!" the telephone cord is already being treated in this metarepresentational mode, as cued by our remark. Its only casual resemblance to a snake suffices to permit an abuse of what ordinarily might be a fixed representational and semantic structure.

Leslie's conception of the theory of mind implies that human beings have available a domain of metarepresentations that they can manipulate to make inferences about causes, predictions about future events, recognize the consequences of ignorance, distinguish reality from fantasy, acquire a language of words and phrases depicting mental experiences or states, and infer motivations. Such a development begins perhaps in the middle of the second year but does not really reach its peak until the third and fourth years, although some children may use words such as, *know*, *remember*, *pretend*, and *dream* by the end of the second year. As Leslie puts it, "Pretend play is, thus, one of the earliest manifestations of the ability to characterize and manipulate one's own and others' cognitive relations to information" ( [Leslie, 1987](#), p. 422).

Pretend play and the beginnings of the use of this metarepresentational system can be identified by the end of the second year. [Greta Fein \(1981\)](#) in her research has shown that mothers who interact with their daughters foster the likelihood of an earlier emergence of transformation, such as the child's pretending to drink from an empty cup or at the next stage pretending to feed milk from the empty cup to a toy plastic horse, which is itself treated not as a horse but as a baby. Even with an early manifestation of pretending, such as in the case of a 2-year-old with only a limited vocabulary of perhaps a half-dozen words pretending to read a book by babbling along and pointing at pictures, turning pages and only occasionally saying a clear word, the more advanced forms of make-believe require a higher level of cognitive development ([Singer and Singer, 1990](#)). The emergence of an ability to move beyond what Piaget called egocentricity to the ability to decenter experience and identify that others may think differently from oneself is necessary for more complex symbolic play.

This is exemplified best in the so-called false belief experiment. A child (the target participant in the study) watches as another person hides a piece of candy in a box (A) and then leaves the room. Someone else then moves the candy from box A to another box (B). The child is then asked where the candy is now and correctly points to box B. The original person who had hidden the candy now returns to the room. At this point, the child is asked where this person will look for the candy. Research evidence clearly indicates that only by age 4 do children regularly begin predicting that the deceived original hider of the candy will look in A rather than B ([Wimmer and Perner, 1983](#)).

Leslie and others have actually demonstrated that this fundamental ability to penetrate the "mind" of another is demonstrable in older children who are quite severely retarded but rarely is manifest in autistic children who may actually show higher IQs than the retarded participants. The autistic children, as Leslie argues, seem to have a special deficit in such perspective taking and perhaps more generally in their ability to develop the metarepresentational world decoupled from primary representations. Indeed, one of the special difficulties observed in our own and in other studies has been that autistic children show very little capacity for spontaneous or encouraged make-believe play ([Leslie, 1987](#); [Nahme-Huang et al., 1977](#)). Recent observations in babies and toddlers born of crack cocaine-addicted mothers seem also to indicate findings comparable to those for autistic children. These children, despite apparently average general intelligence, seem deficient in play skills ([Blakeslee, 1989](#)).

It may well be the case that preschool children practicing imaginative play may also be more likely to move naturally into adopting a metarepresentational orientation. They may show an ability to demonstrate a "theory of mind," that is, an ability to be aware of their own thoughts as distinct from others ( [Harris and Kavanaugh, 1993](#); [Wellman, 1990](#); [Wimmer and Perner, 1983](#)). In a series of studies under my direction ([Rosen et al., 1997](#); [Schwebel et al., 1999](#)), it has been demonstrated that preschool children who play more imaginatively (on observation by raters on several occasions) may actually perform better on the reality-fantasy and false-belief measures used as estimates of theory of mind. As a matter of fact, even when age and other factors are partialled out in multiple regression analyses the scores on make-believe play still predict "theory of mind" results.

What I am proposing, then, is that imaginative play in childhood emerges almost necessarily as the child's cognitive capacities unfold through heightened brain capacity and inevitable social experiences. At the same time, this metarepresentational mode makes it possible for increasing complexity of play to occur, and such play provides pleasure for the children by allowing them to miniaturize complex events and objects, as well as to gain power over the objects and people around them through manipulating them in original story lines.

This practice of narrative and make-believe play further crystallizes and defines the metarepresentational ability of the child. The child reduces extreme incongruity or the initial shock of unexpectedness to more controllable dimensions. These miniaturized forms still may have many elements of sufficient novelty to excite and interest the child until gradually by play and replay they become assimilated into previous schemas or integrated into new organized schemas about a variety of social situations. The schemas formed may be "erroneous" if examined by an adult; depending on what further opportunities the child has to reshape the schemas and scripts formed in this fashion and to what extent experiences of humiliation around play or fear and terror in connection with some social setting preclude their exposure, they may persist as "neurotic beliefs." What [Sullivan \(1953\)](#) might call parataxic distortions or the transference phenomena that are manifest in the psychoanalytic situation may have been formed in the schemas of children that have not been played out openly enough to be corrected by other children or adults ([Singer, 1985](#)).

The compensatory or psychologically adaptive role of imaginative play and the adoption of a metarepresentational schema system are exemplified by an example provided by [Piaget \(1962\)](#). His daughter, just a little less than 4 years old, was told that she could not go into the kitchen because pails of hot water were being



prepared for her bath. The little girl then said, "I'll go into the pretend kitchen then. I once saw a little boy who went into the kitchen, and when Odette went past with the hot water, he got out of the way."

There is good evidence that play between parents and children is a universal experience that has a mutually reinforcing impact ( [Singer and Singer, 1990](#)). In a study by [Van Hoorn \(1987\)](#), observations of a diverse mixture of Chinese, Filipino, Mexican, and North American mothers of European descent were made during play interactions with their infants. Despite some cultural variations in games played, there was considerable evidence of mutual enjoyment. The range of interactive behaviors that promoted cooperation and successive attainments characterized games. The positive emotions of joy and surprise and laughter were consistently in evidence.

### **Imaginative Play in the 3- to 5-Year-Old Child: Research Approaches**

In studies conducted from our research center at Yale, we followed children from eight nursery schools or day care centers over a year's time. Pairs of independently recording raters who watched a given child for a period of time on two successive weeks and then returned a few months later to repeat this pattern made observations. Altogether there were eight observations spread across the year's time. In this sample of 141 3- and 4-year-olds the children were rated for variables such as specific positive emotions (e.g., liveliness, excitement, elation and joy, or sadness, anger, etc.) as well as behaviors such as imaginativeness, cooperation with peers, and overt aggression. The data clearly indicated that positive emotions such as joy and liveliness were consistently positively correlated with imaginative play. The children who showed various make-believe transformations during play emerged across the year's time as those who engaged in a good deal of smiling and laughter and who were motorically active. Imaginativeness was negatively correlated with evidence of fearfulness, sadness, or fatigue. These relationships could not be attributed solely to language usage, for when there was statistical control for numbers of words employed, the relationship between imaginative play and positive affect still remained statistically reliable ( [Singer and Singer, 1980, 1981](#)).

As [Greta Fein \(1981\)](#) has stated, "pretense provides an unusual opportunity for children to control their own emotional arousal and to maintain a level that is both comfortable and stimulating." In our own research, as well as in reports by Fein and others, it is rare that we have observed pretend play among children who are very angry, anxious, in conflict, or who are hyperactive. We did see children who used their toys as mock weapons or who played fantasy games that involved simulated aggressive acts, but the children were generally in control and used metacommunicative statements such as, "This is my bad guy . . . make believe he is chasing your good guy." Other studies supporting this generally positive nature of imaginative play as occurring in spontaneous settings confirm this general finding ( [Stoneman et al., 1984](#)).

In two separate studies, one carried out with a more clearly middle-class sample and one with children from a blue-collar background; we observed 3- and 4-year-old children over a year's time. Samples of play behavior were obtained. From the larger sample of children, 40 children who scored well above or well below the group means over the year on both imaginativeness of play and frequency of television viewing were identified. Four groups were delineated: high imaginative/high television viewers, high imaginative/low television viewers, low imaginative/high television viewers, and low imaginative/low television viewers. Each group was composed of 6 boys and 4 girls. Observers who recorded all of the children's actions for a 10-minute period and who were "blind" to the television-viewing patterns of the children recorded in detail both the verbalizations of the children and their actions during play. The data were then scored for categories such as where the play took place, the social structure of the play, the major themes (e.g., adventure, families, school, doctor), the roles assumed (e.g., mother, father, baby, super hero, victim), the type of play (e.g., sensorimotor, mastery, rules, pretending or symbolic play), evidence of psychodynamic themes (e.g., orality, anality, cleanliness, separation, rejection, sibling rivalry, etc.), and finally the references made to television in play. A play protocol analysis chart was developed, listing all of the kinds of play and the scoring procedure. Our data yielded seven statistical factors of play. These included: (a) adventurous fantasy play, (b) domestic fantasy play, (c) gross physical activity (running, jumping) versus artistic play, (d) a more general emphasis on make-believe pervading all forms of play, (e) involvement with ritual games or dancing and singing, (f) play with pet animals, and (g) games with rules.

Those children who were highly and consistently imaginative also engaged in more social play; they also participated more in ritual games and singing and were less likely to play alone. The imaginative children tended to be those who initiated games and were rarely solitary, withdrawn, or defensive. They could occasionally play alone, but they did not carve out a limited territory in their course of activities nor did they play in more bounded space. Over the year, we observed that the children tended to decrease their larger motor activity and increase their art, ritual, or singing and dancing play and in general showed increased social interaction. There was, interestingly, an increasing role of themes of danger, disaster, and physical mutilation. It may well be that these developing preschoolers had become increasingly aware of the real-life dangers and could now express these fears more verbally and work them into their play. It is, of course, also possible that the children chose such plot lines because these themes seemed to be interesting in themselves and more varied than the standard "putting the baby to sleep" and "going to the doctor" games that they had by now assimilated into organized schemas ( [Singer and Singer, 1980, 1981, 1990](#)).

The role of television viewing in play was evident in some of the content of themes. The children who were heavy television viewers and who showed in general low fantasy play tendencies were more likely simply to mention television super heroes or heroines and then, in imitation, bound around the room in a rather destructive fashion, knocking over others' toys or occasionally hitting other children. Although television may well provide a rich source of thematic content for imaginative play, it is evident from our own and others' research that heavy-viewing children are often less spontaneously imaginative in their play. Symbolic play practice may be displaced by the ease of flicking on a television set to provide entertainment. Moreover, the heavy doses of violent content and the rapid pacing and much-interrupted format of commercial television may generate influences on the heavier viewer that work against imaginative and reflective thought. In one of the few longitudinal studies of imagination in children, we found that early heavy television viewing predicted less imagination in children several years later, even when family life factors were also accounted for ( [Singer et al., 1984; Singer and Singer, 1990](#)).

### **Transitional Objects and Imaginary Playmates**

A special form of imaginative play essentially reflecting the same tendency toward the development of a metarepresentational symbol system can be found in the development of an imaginary playmate. [Winnicott \(1953\)](#) identified an early stage in such a development. From about 1 year of age many children show a tendency to carry a soft cloth about with them and often to take it to bed. Often the soft cloth may be a blanket from the crib; it may also be a spare diaper or some other item among the child's assortment of play materials to which the child has become especially attached. In its earliest forms children, especially when sleepy or hungry, may be seen sucking their thumbs and fingering a soft cloth. In later stages of early childhood, the soft cloth may simply become a kind of fetish that is sustained even as it becomes more and more ragged; eventually it may simply end up as a bit of cloth, to be tucked under one's pillow or into a purse.

Winnicott's perception was that these cloths serve the purpose of providing a concrete reminder in the absence of the mother's warmth and physical presence. Such a tendency can also be carried over to soft toys such as teddy bears. The term *transitional object* implies that the child is gradually giving up the physical clinging to the parent but sustaining some concrete and palpable feature of that experience.

The function of this soft toy is not only one of reminiscence but also one of possession. That is, although the child cannot possess indefinitely the warmth and closeness of the parent, he or she can possess the soft cloth or toy. Indeed, one can even argue that the emerging sense of individuality and personal entitlement that characterizes all of us even to the point of adults, insisting on our rights for pensions and medical care or "respect," are traceable to these humble beginnings. The child clings to his or her teddy bear, fights to retain possession, and begins to delineate a sense of self through being able to assert, "This is mine."

[Tolpin \(1971\)](#), in an extensive analysis of the phenomenon of the transitional object, has gone beyond Winnicott in demonstrating that the transition also involves the child's awareness that thought and fantasy can substitute for the palpable presence of the object. When such blankets or cloths are destroyed, accidentally thrown out by parents, or simply pulled to pieces, children gradually learn they can sustain the experience through their increasing capacity for imagery. Indeed, [Metcalf and Spitz \(1978\)](#) proposed that such transitional objects are sustained during the period when the child's cognitive capacities primarily involve recognition memory and can be gradually given up as the child develops increasing skill of voluntary recall or mental retrieval of desired objects. The extensive research of [Litt \(1986\)](#) has indicated that after 18 months and between the ages of 2 and 5 almost three-quarters of American children sampled showed evidence of play with a transitional object. Although data from other countries reveal somewhat lower levels of such play, cultural differences may in part reflect differences in forms of inquiry, availability of soft toys, and so forth. Of special relevance is the fact that the child introduces elements of make-believe with the soft toy and begins to treat this teddy as a companion, who can be talked to, admonished, and can also serve as a potential helper.

Litt's research also indicated that transitional objects serve a functional role and are not in themselves indications of emotional distress, poor attachment, or neurotic tendencies. On the contrary, the evidence seems clear from her data that children who have transitional objects report fewer sleep disturbances and are more attractable, self-confident, independent, and affectionate. Such children did not show any evidence of disturbance on personality test measures and if anything tended



to be less impulsive, aggressive, or antisocial than children who had not played with transitional objects.

Studies with children who report invisible imaginary friends indicate that the phenomenon is relatively common ( [Manosevitz et al., 1973](#); [Masih, 1978](#); [Somers and Yawkey, 1984](#); [Taylor, 1999](#)). Generally, the data indicate that at least a third of various child samples report the phenomenon. In one study comparing blind and sighted children, all of the blind children recorded a sighted imaginary friend ( [Singer and Streiner, 1966](#)).

In some of the research carried out by the author and his colleagues, with 111 3- to 5-year-old children, the parents of 55% reported that the children had shown indications to some degree of an imaginary playmate—an invisible child or animal for whom a place had to be set at the table on occasion. The children were rated for the degree of frequency of imaginary playmates and the extent of such reported play. The score generated by these data proved to be predictive of the children's spontaneous play over a year's time in day care centers and nurseries. Children who had more imaginary playmate experience were rated by “blind” observers as more imaginative in free play, showing especially more positive emotionality, more cooperation with adults, and a more extended language usage. When the imaginary playmate variable was entered as a predictor in multiple regression analyses to predict overt behavior, it proved an especially powerful variable in predicting not only imaginative play or positive affect but also greater persistence in play, less aggression and less anxiety and fearfulness. There was also considerable evidence that boys who had no imaginary playmates showed more television watching and less vivid language.

A recent impressive examination of almost every aspect of the imaginary playmate phenomenon by [Marjorie Taylor \(1999\)](#) has reviewed the available research literature on this phenomenon and provided further data on its prevalence, origins, and mental health implications. Taylor's findings in general support the research results cited in the preceding. Her studies demonstrate that, even in cultural settings where parents or other adults disapprove of children playing with or talking about such fantasy friends, careful inquiry reveals that a significant number of children are likely to reveal evidence of the phenomenon.

In effect the imaginary companion may be understood as a form of imaginative play that emerges as the child gradually acquires the capacity for heightened mental imagery. It has the adaptive function of allowing the child a means of resolving the tension described early in this chapter between the need for affiliation and for a sense of privacy and individuality. The phenomenon is somewhat greater among children without siblings and as such reflects, as in the study of the blind children mentioned in the preceding, a means of peopling one's world, maintaining some sense of control, and at the same time sustaining the experience of personal possession and privacy, because most children are well aware of the inherent unreality of an invisible imaginary friend ( [Singer and Singer, 1990](#); [Taylor, 1999](#)).

## THE ADAPTIVE ROLE OF IMAGINATIVE PLAY

### Specific Functional Correlates of Imaginative Play

#### GENERAL POSITIVE EMOTIONALITY

A number of studies consistently point to the fact that the use of make-believe in the nursery school or a variety of other settings or in the form of imaginary playmates is associated with more positive affective states in children. This consistent finding, as well as the tendency for there to be an inverse relationship between imaginative play skills on the part of the child and overt manifestations of anger and aggression, suggests the value of the use of play in a variety of therapeutic efforts with disturbed children. Thus, play therapists confronted with children who show limitations in their ability to engage in make-believe may have to find ways of subtly fostering these skills in the child, perhaps by example or gradual training exercises. Without some experience of enjoyment in the psychotherapy sessions, children are unlikely to want to sustain a treatment relationship ( [Singer, 1993](#); [Singer and Singer, 1990](#)).

#### ENHANCED LANGUAGE SKILL

One feature of imaginary play is that the children are verbalizing aloud descriptions of complex situations. Although some of their statements may reflect misunderstandings of adult remarks (as in the case of a boy lining up his toy soldiers to rescue Daddy, who was “all tied up at work”), such verbalization provides feedback to the child and may also evoke correcting responses from overhearing adults or peers. In addition, the inherent nature of make-believe play involves the development of plot sequences. This requires increased use of adjectives, future verbs, and extended utterance lengths ( [Singer and Singer, 1981, 1990](#)).

#### PERSISTENCE

Imaginative play, because it requires in effect a story line, tends to provide the child with focus and direction and then sustains concentration for longer periods. Children who have shown no tendency for imaginative play often are captives of the momentary changes of objects or toys in their environment. They seize at new things and often are embroiled in struggles for possession with other children or flit from group to group. The child engaged in symbolic play has by virtue of entering into this domain developed a sense of sequence, is moving in a privately defined direction, needs less sustenance from concrete external objects.

#### DISTINGUISHING REALITY FROM FANTASY

There is at least some research evidence to suggest that children who have experience in make-believe games are better able to discriminate real from unreal situations and have learned to identify within their own thoughts that metarepresentational realm described by [Leslie \(1987\)](#). For example, in one study with somewhat older children, those who had scored higher on indications of imaginativeness were better able to recall details of a story and then could discriminate real instances from those that were pure fantasy ( [Tucker, 1975](#)). It may well be that even those rare instances of multiple personality, the affected adults may have grown up without a clear sense of the extent to which fantasized alternative selves are natural occurrences as part of a general dimension of make-believe or metarepresentation. Because there is some evidence of early child abuse as a forerunner for such multiple personality developments, as well as for certain extremely hypnotizable or fantasy-prone individuals, it could well be that the same brutalizing family atmosphere that necessitated the child's escape to an alternative self also precluded a natural unfolding of make-believe play ( [Lynn and Rhue, 1988](#)). The abused child has had less opportunity to engage in many different sequences of fantasy play, and thus, fails to perceive the escapist alternative self as a natural part of a range of make-believe activities. Instead, dissociations may occur that are outside the control of the child.

#### EMPATHY

One of the consequences of solitary and group make-believe play is that the child often learns to take on different roles. One can be the doctor examining the real or imaginary doll, and then, one can reverse roles and pretend that the doll or another child is the doctor. Often in the make-believe play of two or three children, one observes brief struggles over who will be the “good guy” or the “bad guy,” the hero or victim. In such instances, these disputes are often resolved by reversing roles either later in the game or on other days. Gradually, the child engaged in such play learns what it is to be a victim and to experience others' dilemmas or pain. Although we do not have extensive data on this issue, careful clinical studies have supported this finding ( [Gould, 1972](#)).

#### COOPERATION

Observational studies of children at play such as those cited in the preceding consistently demonstrate that children prone to symbolic play are more likely to prove cooperative both with adults and peers. Indeed, the very necessity of negotiating roles and plots with other children in order to sustain make-believe provides useful practice in this important social skill. One can observe children who show very little spontaneous imaginative play or who have been heavy television viewers and have not been practicing any make-believe at home dashing madly about the room, disrupting others' play and in general bringing down on themselves the wrath of the adults and children ( [Singer and Singer, 1981](#)).

#### LEADERSHIP

Although studies have generally not focused on early leadership tendencies at the preschool and early school ages, there are some data to suggest that children who are active in make-believe often are the first to initiate new games. The very practice of creating a series of plots may foster the ability to introduce a new group of children into some game. The experienced make-believe player thus gets “first crack” at assigning roles and plot lines. As imaginative play becomes more internalized and children move into reading and storytelling, there are clinical indications that children who have such storytelling capacities may win the respect of others in a play group ( [Singer and Singer, 1980, 1990](#)).

## TOLERATION OF PLAY

Of extreme importance for the developing child is the ability to defer immediate gratification in the interest of a longer-term goal, or simply to tolerate naturally occurring delays. Make-believe play, whether it is sustained by the use of just few primitive toys or as it gradually becomes internalized in the form of imagery, has been shown in quite a number of studies to be associated with the ability to deal with delay ( [Singer and Singer, 1976](#)). For example, in a study with somewhat older children, those who had shown patterns of imaginary play and other signs of imaginativeness were able to sit still longer when asked to do so or could tolerate waiting in an interview situation until the reviewer was ready to speak with them ( [Singer, 1961](#); [Singer and Singer, 1990](#)). The importance of this capacity cannot be minimized. Indeed, one might argue that the entire conception of the ego in psychoanalysis is built around the notion of deferred gratification and the capacity for delay and self-control in response. Imaginative play may be an important contributor to the development of the experience of self-regulation in this sense ( [Singer, 1955](#)).

## NEW IMAGERY COMBINATIONS

One of the important features of imaginary play is that it provides a child with a general opportunity to practice imagery, that is, to try out mental representations of concrete objects or of transforming such concrete objects. I have already suggested this in connection with [Leslie's \(1987\)](#) more general notion of the metarepresentational system and of the decoupling. But skill in producing images is something that develops in its own right and can be advanced by the ongoing imagined play sequence ( [Lewis, 1973](#); [Singer, 1973](#); [Tower, 1983](#)).

## DIVERGENT THINKING

Beyond new imagery elaboration pretend play may be a basis for divergent thinking and creativity in both its verbal and imaginal forms ( [Russ, 1996](#)). A recent longitudinal study has shown that children who play more imaginatively in make-believe games not only show continuity in such tendencies over a 4-year period from ages 6 and 7 to ages 10 and 11 but also that such earlier imaginative play predicts later performance on measures of divergent thinking, a possible forerunner of creativity ( [Russ et al., 1999](#)).

## TAKING TURNS

The careful observations of [Catherine Garvey \(1974\)](#) demonstrate that one of the important features of pretend play in children is the manner in which it is associated with turn taking and a form of social interaction that has long-term socialization potential. Observations of children in various kinds of make-believe indicate that exigencies of the plot in any initially agreed on make-believe activity impose forms of self-control on the child. As in the case of cooperation more generally, turn taking provides a valuable delineation of orderliness and sequence that has special meaning for the child.

## TRYING OUT DIFFERENT ROLES

A special feature of make-believe that goes beyond empathy is the opportunity for children to try different roles and gradually identify the special qualities of the different kinds of individuals in their environment. When children play "Bus Trip," one of the children becomes a driver, others become passengers, and others become traffic policemen. Each game reduces to miniature size a variety of adults in the child's real world who are often not well understood. In their efforts to formulate a plot, children again and again try out these roles and can gradually assimilate them into structured schemas that will serve them better as they move on in life and encounter such individuals. The notion of role taking is also a critical one for the development of awareness of gender and sex role differences. There are indications from research that children who are more experienced in make-believe may also be able to develop a more androgynous orientation and are freer of very traditional and often limiting sex role attitudes or expectations for the future ( [Repetti, 1984](#)). In the psychotherapeutic situation, one important function of play therapy may be to have the child begin to try out new kinds of roles and interactions, beginning with the therapist but extending to imagined persons with whom the child may come into contact later on ( [Singer and Singer, 1990](#)).

## ORDERING AND SEQUENCE

I have already emphasized the important role that the inherent plot line of a make-believe game plays in encouraging persistence over time by the children and also in encouraging cooperation or turn taking. The very nature of make-believe play calls for a certain logic, a sense of beginning, middle, and end that gradually becomes the basis for the formation of differentiated scripts that are important forms of action schemas in the child's memory system. Extensive research in cognitive science has pointed to the importance of the ability to sustain a variety of systematic scripts about hundreds of social situations that individuals encounter, from birthday parties to restaurants to attendance at sporting events and, ultimately, to marriage or specific vocational activities. The make-believe play sequence for children provides them with opportunities to learn the necessity for sustaining and organizing miniature scripts and allows them to be free of purely impulsive associative responding to some degree ( [Bretherton, 1984](#); [Nelson and Seidman, 1984](#)).

## ANTICIPATING CONSEQUENCES

A further extension of the nature of plotting and the emergence of narrative sequences as part of thought fostered by pretend play is the awareness that actions have specific consequences. Thus the beginnings of social cause-and-effect thinking in the child are developed in the course of imaginative play ( [Bretherton, 1984](#)).

## Factors Conducive to Imaginative Play

If pretending and make-believe are so useful in the development of the child, what features of babies or toddlers' milieux generate, sustain, and enhance the predisposition to such play? Space does not permit an extensive review of this topic, but there are clear indications from research that particular parental or caregiver activities are critical. An early optimal attachment to at least one parent or consistent adult caregiver predicts later emergence of imaginative play. Parental willingness to initiate games of fantasy, to suggest plots or the use of toys but then to step back from them so that the children experience the games as their own is also an important correlate of children's pretending capacities. Parental storytelling or bedtime story reading are also regularly found to be associated with evidence of greater resort to imaginative play in children. A family atmosphere of tolerance for floor play, a physical setting that allows children to find at least some private areas for uninterrupted pretend games, and adult attitudes of acceptance rather than humiliation of the preschoolers' talking aloud or role-playing all contribute. An overall sense of playfulness in the home setting opens the way for the imagination, humor, and even that sense of illusory control that are so crucial for normal coping capacities ( [Singer and Singer, 1990](#)).

## Imaginative Play, Psychopathology, and Psychotherapy

A fuller exploration of the implications of children's make-believe play for psychopathology and psychotherapy would take us far beyond the scope of this chapter. The general thrust of this presentation suggests that pretend play, the capacity to create miniature, possible worlds, is a critical feature of the healthy development of a child. The available evidence as reviewed by Dorothy Singer and myself elsewhere has suggested that emotional disturbance and cognitive and affective difficulties are often associated with an inability to sustain imaginative play in middle and early childhood ( [Singer, 1973](#); [Singer and Singer, 1976, 1990](#)). Some classic cases in which children's play revealed elaborate, unrealistic fantasies reflecting their troubled life experiences demonstrate that those children who could sustain such play had better prognoses once play therapy was initiated ( [Singer, 1973, 1993](#); [Singer and Singer, 1976](#)).

A critical role for the psychotherapist with children is to help them to find ways of miniaturizing their private fears, brutal experiences of abuse, exposure to parental quarrels, or neglect into manageable chunks. The therapist's encouragement of play by the provision of an appropriate setting of toys or (for middle childhood) games conducive to symbolic representations of children's troubles casts the therapist in the role of the needed mediating parental figure. Delineating in play form key life issues, significant adults or siblings, and conflicts helps the child to develop new schemas and scripts and new knowledge structures that can later reduce the ambiguity, confusion, or affects of fear and terror that new situations may present.

I believe that a key feature of the play therapist's role is to provide a model for the child of a zestful, curious, lively approach to one's experience. Although the therapist must often avoid moralizing about the child's hatreds or about presumed parental or sibling cruelties or neglects reflected in play, this does not preclude an emotional response. The therapist's own professional curiosity and willingness to share thoughts and play possibilities, conveyed in a spirit of liveliness and humor, can open the troubled child to the joys of the world of introspection and fantasy. By such an approach one can change the fearful, despairing, or angry child, trapped

in a mental world of limited constructs, schemas, or scripts, into one who can savor the excitement of reshaping what had seemed like the given world of recent experiences into a domain of innumerable possibilities. By replaying and, thus, reshaping schemas, the child can gain at least some sense of control and power within a small region of a vast, seemingly impenetrable universe. The skilled play therapist, whether psychoanalytic or cognitive-behavioral in orientation, not only provides the child an opportunity to identify and work through specific problems but also enhances the child's development of the capacity for play, a powerful cognitive tool for further enhancement of a sense of self and individuation, as well as a continuing private theater for developing new scripts of affiliation and attachment. The medium of make-believe in childhood, internalized by middle childhood into a richly elaborated fantasy capacity, becomes eventually a major functional system through which all of us can entertain opportunities and possibilities for warmth, closeness, and communion with others while sustaining our sense of individuality and privacy.

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# 19 MORAL DEVELOPMENT

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As we have no immediate experience of what other men feel, we can form no idea of the manner in which they are affected, but by conceiving what we ourselves should feel in the like situation. Though our brother is upon the rack, as long as we ourselves are at our ease, our senses will never inform us of what he suffers . . . it is by the imagination only that we can form any conception of what are his sensations. We endeavor to examine our own conduct as we imagine any other fair and impartial spectator would examine it. . . . Our continual observations upon the conduct of others, insensibly leads us to form to ourselves certain general rules concerning what is fit and proper. . . . ([Adam Smith, 1759/1976](#))

Philosophers have grappled with the uniquely human quality of morality for centuries ([Blum, 1996](#); [Kagan, 1998](#)); however, research interest in moral development is much more recent. In 1984, Martin Hoffman could still write that guilt was a relatively neglected research topic. Morality shares many features with attachment ([Bowlby, 1982](#)). Both are systems of behavioral interactions, cognitions, and emotions, present in everyone unless pathology or environmental adversity intervene; they are biologically adaptive, promoting individual and group survival; their development forms a regular sequence, deviation from which can have serious consequences.

Much progress has been made in the exploration of the development of empathy, shame, and guilt, of prosocial behavior and moral judgment. The context within which young children become socialized and acquire moral standards and beliefs and the vicissitudes of this developmental process have become clearer, a start has been made in elucidating its psychopathology, and the stage is set for a more rational approach to the prevention and treatment of antisocial conduct.

This chapter focuses, in turn, on the classical contributions to our understanding of moral development of psychoanalysis and the cognitive-developmental studies of Piaget and Kohlberg. It deals with Hoffman's work on empathy, Kagan's observations of the development of self-appraisal and inner standards, Turiel's documentation of children's ideas about morality and convention, and with work on the emotions related to morality. Some aspects of the psychopathology of moral development are described: social learning and role modeling, cognitive attributions and expectancies, and the effect of maternal depression. Finally, the context within which children develop moral behavior is discussed, including Kochanska's imaginative studies relating parenting styles and child temperament to early precursors of conscience.

## PSYCHOANALYSIS AND THE MORAL SELF

### Freud

[Freud \(1930/1961a, 1923/1961b\)](#) saw the origins of ethical tendencies in the emotional attachments of young children to their caregivers and thought the long period of helpless dependency of the human baby was the biological basis for the development of conscience. Dependency on parents fosters fear that their love may be lost. Threat of such loss induces fear, the main emotion associated with moral development in this first stage, equivalent to social anxiety in adult life: a fear of being found out. Only in the next stage, when parental authority is internalized with the formation of a superego, do conscience and a sense of guilt arise, whereas the fear of being found out wanes. Freud stressed the lack of distinction during this stage between doing something bad and wishing to do it, between the act and the impulse, because the superego, being a part of oneself, is as aware of bad thoughts as bad deeds.

Whereas in the first stage of moral development, parental prohibitions (the "don'ts") ([Emde et al., 1987](#)) instill fear that parents will withdraw their affection if disobeyed, in the next stage the objects of attachment become internalized, and children now judge themselves critically. To recover from a sense of a flawed self, they develop an ego-ideal and strive toward its attainment. Failure to meet the standards of this fantasized ideal self (the "do's") results in a sense of guilt. James Baldwin, an extraordinary developmental psychologist, first described the functions of the ideal self in the development of moral consciousness (1897). He saw the ideal self as submissive to the exhortations of adults, whom the child perceives as superior and to be both obeyed and imitated.

Freud's most original and controversial contribution in this field has been his theory of the Oedipus complex and its attendant anxieties, which he held to be a prerequisite for identification with parents and the formation of an ego-ideal. Children identify with both parents, but become erotically most attached to the parent of the opposite sex. The rivalries, jealousies, and ambivalences toward the same-sex parent engender fears of retribution: castration anxiety, with resulting defensive repression of erotic urges and strengthening of parent identifications, especially with the same-sex parent. Freud emphasized that the introjection of the father's authority in particular lays the foundation for the superego or conscience, the basis for guilt, when one falls short of the demands of one's ideal self. He thought castration anxiety was more severe in boys than girls. The threat for girls was less because the damage seemed already to have been done. Boys, in contrast, tend to develop a harsher conscience, and paradoxically, this puts them at greater risk of antisocial conduct. An overharsh conscience, he held, instead of protecting the individual from antisocial acts, at times fosters "criminality from a sense of guilt." He also developed the contrary notion that the more virtuous a person is, the more severe his or her superego—that saints reproach themselves the most for being sinners ([Freud, 1930/1961a](#)). Thus, an exceptionally punitive superego is to be found both among some criminals and excessively good people.

The importance of Oedipus conflict resolution for conscience development has probably been overstated ([Emde et al., 1987](#)). Researchers at Anna Freud's Hampstead Clinic documented significant pre-Oedipal features of morality and found many latency children with unresolved Oedipal struggles but strong superego development.

### Anna Freud

[Anna Freud \(1966\)](#) presciently emphasized the importance of language for conscience formation and ego control and stressed the role of empathy in the development



of children's ideas, for example, about the property rights of others. She too saw the superego as an incorporation of parental strictures—"identification with the aggressor"—but also held that the acceptance of one's own culpability played an essential part. Repression and sublimation, or the displacement of instinctual aims in conformity to social values, are part of this developmental process ( [Freud, 1961](#) ). Although the defensive processes of projection, introjection, and regression develop very early in life—as soon as children can distinguish between themselves and the outside world—the defense mechanisms of repression, sublimation, and reaction formation, essential for subduing childish greed and hostility, develop later.

### Melanie Klein

[Klein \(1927/1948a;1933/1948b\)](#) placed the Oedipus complex between the first and second years and superego formation in the second year. Her timing does not tally with what we now know about children's cognitive capacities, but her descriptions of the harshness of the infantile conscience, much harsher than the real parents had ever been, have been illuminating.

### Erik Erikson

[Like Freud \(1961a\)](#), Erikson, in his scheme of personality development ( [Erikson, 1968](#); [Wolff, 1989](#) ), divides the growth of the moral self into two phases: from about 18 months to 2½ years, the “anal” stage of primary socialization, and from 2½ to 5 years, the “genital” stage of primary identifications. During the “anal” stage, toilet training and the acquisition of sphincter control are symbolic of the parents' first attempts to teach the child the basic rules of the society in which they live and, on the child's part, of the earliest efforts at self-discipline for the sake of approval. The toddler, on his feet, with arms free, is ready for action and, although still with poor impulse control, he has a choice: to throw, grab, defecate, or (in response to parental prohibition) desist. He needs the parents' presence to back his efforts to succeed but he also needs them to step in when disaster threatens. For the first time, the child gets pleasure from cooperation and pride from achievements, but also a sense of shame at failure. In contrast to the concerns of previous psychoanalytic writers with the young child's fantasies, Erikson gave due weight to actual life experiences at this stage as determinants of future self-esteem, in particular a sense of autonomy and self-confidence in relation to cooperative enterprises, or of shame and self-doubt.

Many parents, especially the socioeconomically deprived with limited occupational autonomy and little sense of control over their lives, find this stage of child rearing difficult. Although offering good nurturing care to the baby, they have unrealistic expectations of their toddler's capacity for self-restraint. Instead of physically taking over control when necessary, they exhort and admonish verbally and from a distance and, when accidents occur, they reprimand, threaten, and punish, sometimes aggressively. The anxiety engendered by such child rearing reinforces the toddlers' fears of what could happen if they were to lose control and, in line with the idea of “criminality through guilt,” induces them to act ever more outrageously. The actual consequences are never as bad as those feared; anxiety is temporarily assuaged, only to return as the cycle of interactions repeats itself.

In the second Eriksonian stage of moral development, the “genital” stage, language transforms children's social interactions and inner life, and a sense of the future is born. It is the “play age” of curiosity and make-believe, of storytelling and identification with role models, especially parents. The earliest triadic and group relationships now occur and new emotions such as rivalry and jealousy have to be mastered. The child now has more self-control and can compensate for present frustrations in play, imagination, and hopes for the future.

Erikson thought the development of girls was the more hazardous because their erotic longings for their father engender jealousy of the mother, which is the more frightening because she becomes a potentially hostile rival and is lost as a haven of security. Boys, in contrast, although now attached to their mother in a new way, still have her as a safe source of comfort. On the other hand, the role models parents provide for their children at this stage are more secure for girls than boys. Far fewer mothers than fathers are lost from the family, and the status of mothers within the home is universally high, whereas that of fathers, especially in families living in poverty, tends to be low.

As children identify with and incorporate parental standards of conduct, a powerful new source of anxiety—guilt or the pangs of conscience—arises together with a division between the self in action and the moral self (ego and superego). In Erikson's view, actual life experiences at this stage determine whether the person will emerge with strengthened capacities for initiative and intimate relationships or instead become constrained and inhibited.

Children's cognitive development during these years fosters misinterpretations of their experiences and magnifies their anxieties. The potential harshness of conscience in early childhood is understandable when we appreciate the nature of children's logic at this time of life.

Developmental psychologists now take note of children's inner worlds, their cognitions and emotions, and psychoanalysts are prepared to amend their theories in the light of observations. [Buchsbbaum and Emde \(1990\)](#) have questioned the idea that moral development depends on the resolution of the Oedipus conflict at the end of the genital stage, that is the fourth or fifth years of life. Using a play narrative technique, they found children as young as 36 months capable of articulating coherent stories about rules, reciprocity, empathy, and internalized prohibitions. Even such young children could deal with alternative outcomes to resolve a moral dilemma. In this important paper, the authors stress the dual origins of moral development—biological preparedness and early caregiving relationships.

## THE CONTRIBUTION OF JEAN PIAGET

The impetus for the objective study of moral development in childhood came from Piaget's systematic observations ( [Piaget, 1932](#); [Piaget and Inhelder, 1969](#); [Wolff, 1989](#) ). Before summarizing his studies of moral judgment, it needs to be said that his general discoveries about children's immature logic and reasoning between the ages of about 2 and 7 years have shed much light on early childhood fears and some of the psychoanalytic postulates about early emotional experiences. During the precausal, animistic stage, children cannot yet detach thoughts from the events and objects thought about, nor identify themselves as thinkers about the world. They cannot, for example, explain how it is that they are both in their bed at night and yet also in the dream they have while sleeping. Their explanations of events and circumstances tend to be psychological rather than rational, and the notion of chance events is not yet grasped. Although aware of their own viewpoint, they find it hard to conceive that this is not also uppermost in the minds of others. Piaget's ideas about a child's early egocentrism have been criticized by researchers who found children more capable of logical inferences when problems are embedded in a child centered context ( [Donaldson 1978, 1992](#) ). However, children spend much of their lives in an adult world, and the distortions and misperceptions that Piaget's work so vividly revealed are common experiences for everyone in touch with young children.

Like psychoanalytic writers in relation to emotions and behavior, Piaget held to a stage theory of cognitive development. Each stage differs qualitatively from the next, and teaching of logical operations is ineffective until the child has reached the necessary level of understanding. However, cognitive development is now held to be more gradual than Piaget thought: Not all children's thinking changes at the same time at each stage ( [Pine, 1999](#) ).

### The Rules of Games

Piaget concerned himself with children's perceptions of the rules of games and with their views about morality. He did not focus particularly on the distinction between convention and morality, as later workers did ( [Turiel, 1983](#) ), being concerned rather with identifying changing but parallel developmental sequences in how children perceive and respond to rules in both spheres.

The game of marbles is played the world over. Piaget watched how children of different ages played and questioned them systematically about their understanding of the rules and how these had originated. He found that under about 7 years, in the precausal, animistic stage of development, children grasp rules imperfectly, make errors in their application, and are content to play alongside others. Yet they think of rules as obligatory, inviolate, and inherent in the game. When asked who made them, they said the rules had always been there, and if pressed, they resorted to “God.” Older children, in contrast, played competently, knew what the rules were, but viewed them as conventions devised by “children long ago.” If everyone agrees, the rules can be changed and the game played differently. Piaget saw this shift at between 6 and 8 years of age as a move from an authoritarian to a democratic viewpoint, from a position of unilateral respect for sacred laws and duties to one of mutual respect and reciprocity.

### Ethical Beliefs

[Eckemberger \(1999\)](#) defined morality as comprising customs and traditions; ethics as justifications for these: ideally, how the world should be. Piaget systematically questioned children about their ethical beliefs. And here too, he observed a shift from an authoritarian morality in which obedience is equated with goodness and all adults are seen to be right, to a morality where concepts of justice and a more detached view of adults and ethical standards prevail. Piaget explored children's ideas

about culpability at different ages by giving them pairs of stories to evaluate; for example, one in which a child's clumsiness led to a big accidental breakage paired with one where a lesser mishap followed the pursuit of a forbidden goal (taking jam off a high shelf); stealing to help a poor and hungry child contrasted with stealing for oneself alone; inaccurate reporting of a frightening event paired with telling a deliberate but lesser untruth. Again he found a shift of moral attitude. Despite considerable overlap, younger children tended to judge actions according to their material consequences, older children, according to their underlying intentions. Children's views of naughtiness and punishment also changed with age. Younger children had notions of immanent justice—that the crime begets the punishment and that, if you are punished, you must have done something bad to deserve it. They also believed in retributive justice (the *jus talionis*)—that the punishment must fit the crime irrespective of circumstances and motivation. Moral realism is the label Piaget gave to these immature ideas, which he linked to adult constraints on young children and to the children's sense of unilateral respect or duty. Older children in contrast had different ethical beliefs based on autonomous judgments, reciprocity and cooperation, the internalization of rules, and ideas of justice and mutual service.

Piaget saw in the children he studied the development of a morality of good, deriving from affection, and a morality of rights and duties (ideas later taken up by [Turiel \[1983\]](#) in his distinction between morality and convention). Some people he thought operated only with the latter.

Although accused of ignoring the influence of the social environment in promoting development ([Haste, 1987](#)), Piaget had in fact anticipated the now acknowledged role of the peer group in socialization. He stressed the importance of interactions with other children for the transformation of an authoritarian to a democratic view of morality in middle childhood, from a position in which obedience to adults and avoidance of punishments are primary to one in which conformity to social rules and, later still, participation in rule making for the sake of universal, ethical principles predominate. Nor did he ignore the importance of emotions ([Eckenberger, 1999](#)).

Unlike Vigotsky ([Rogoff, 1990](#)), however, he paid little regard to the role of adults in children's learning. Cognitive development is facilitated in interaction with a more skilled partner, and the child is able to engage in skills beyond those he is capable of on his own. Development builds on the internalization of what was carried out in collaboration, that is, of shared cognitive processes to extend the child's existing knowledge and skills.

[Butterworth \(1987\)](#) suggested that children's cognitive and social immaturity and gullibility are biologically adaptive, that unquestioning acceptance of adult rules has to precede the doubting and self-judging phase. The culture must make indelible impressions on the very young for it to be maintained. This view parallels the notion that small stature and physical immaturity too contribute to the acculturation of the young in many species, ensuring that they are docile and noncompetitive during early socialization ([Tanner, 1984](#)).

### An Important Proviso

We must be clear that Piaget and even more so Kohlberg, as we shall see, was concerned with moral reasoning, that is, with what children and older people say about their ideas and beliefs about moral dilemmas, often concerning not themselves but other people. This requires considerable cognitive, linguistic, and social skills and is different from the way in which young children grapple with moral issues in their everyday lives, when they themselves are the central characters. As [Margaret Donaldson \(1978, 1992\)](#) made clear (see also, [Buchsbbaum and Emde, 1990](#); [Dunn, 1988](#)), when moral dilemmas are embedded in real-life or age-appropriate, child-centered play settings, tied to the here-and-now or to concrete events in the past or future, moral sophistication is seen at a much younger age. Moreover, as Vigotsky indicated, "scaffolding" by adults helps children toward greater cognitive attainments ([Pine, 1999](#)).

## KOHLBERG'S STUDIES

Lawrence Kohlberg and his colleagues ([Colby and Kohlberg, 1987](#); [Colby et al., 1983, 1987](#); [Eckenberger, 1999](#)), some 30 years ago, embarked on a long-term longitudinal study of the development of moral judgment. They constructed two sets of reliable interviews, with standard questions about stories involving people faced with moral conflicts. A detailed scoring manual was devised ([Colby et al., 1987](#)) in which the liveliness of the stories and their sensible scoring are evident. One story is about Heinz, whose dying wife needs a drug for which the pharmacist asks an exorbitant price that Heinz cannot afford. Would it be right for him to steal the drug, and if so, why? Others concern a boy who has saved his pocket money for camp when his father demands the money instead, a doctor faced with a mercy killing, and a mother who had promised to let her daughter go to a rock concert and then changed her mind. Piaget's work and theories were the starting point for these studies, but unlike Piaget, Kohlberg and his colleagues did not study very young children.

Eighty-four boys aged 10, 13, and 16 years at the outset, equally distributed by social class, IQ, and sociometric status, were individually interviewed, 58 at least twice, out of six planned occasions over the course of 20 years. The interviews and their scoring are complex, and the standard stories tap nine hypothetical but true-to-life moral dilemmas about which a series of questions are asked relating to the values and reasons underlying the subjects' judgments.

Kohlberg described six stages of moral judgment. Longitudinal stage reversals occurred less often than test-retest unreliability; that is, the sequence was always forward. No subject reached a stage without passing through the previous one; that is, the sequence was invariable. Most youngsters scored within a single or two adjacent stages at any one time, only a few in three stages, so that there was internal consistency in moral judgments. No differences were found between the three age cohorts. Relative developmental delays were modestly associated with low socioeconomic status at 10 years and with intelligence and, especially, educational level at 24 and 23 years, when both tended to have reached their ceiling. Upper levels of moral development were generally reached in the mid-20s or -30s, but not even then in a sizable proportion of individuals.

Here is an abbreviated summary of Kohlberg's six stages, grouped into three levels ([Colby et al., 1983](#)):

Level I—Premoral: The child is responsive to cultural rules but sees these in terms of pleasant or unpleasant consequences of behavior and relates them to the power of authority figures. He follows the rules for the sake of self-interest.

Stage 1: The child is oriented to obedience and punishment, with egocentric deference to authority figures and avoidance of unpleasantness for himself or herself.

Stage 2: There is a naively egoistic orientation, with concern for one's own needs but with some awareness of the needs of others, a wish for egalitarianism, and an orientation toward exchange and reciprocity.

Level II—Conventional Role Conformity: The child is now oriented toward maintaining the expectations of others close to him or her, as a value in its own right.

Stage 3: The orientation is to be a good person in the eyes of others and in one's own eyes, with a wish to please and help. Intentions of behavior are taken note of and the moral perspective includes interactions with others.

Stage 4: The orientation is toward the social order and its maintenance for its own sake and to fulfilling agreed duties in conformity to authority.

Level III—Self-Accepted Moral Principles: The individual is now concerned with defining moral values and principles apart from the supporting authority.

Stage 5: The orientation is contractual and legalistic, with a sense of obligation to the law, but also an acceptance that people can have a variety of different values and that their individual rights take precedence over the social contract.

Stage 6: The orientation has become a sociomoral one, with the recognition of valid universal ethical principles to which the person can choose to commit himself or herself.

A study in which attempts were made to induce stage skipping in 11- to 13-year-old children, by having them listen to adults reasoning at different levels about the solutions to moral dilemma stories of the Kohlberg type, found all changes to be to the next higher stage to the subject's own ([Walker, 1982](#)).

Kohlberg's interview methods, devised in the United States, have been used in other parts of the world with similar results. [Eckenberger \(1999\)](#), summarizing cross-cultural studies, concludes that it is not "Westernization" but industrialization with increasing complexity of a culture that fosters the attainment of Kohlberg's higher stages. High socioeconomic status and participation in social systems are positively associated with moral development, life in fundamentalist or small tribal societies with mainly face-to-face interactions are negatively associated with this development.



## What Causes Variations in Moral Judgments?

Cultural transmission plays a part, as do the different developmental stages outlined by Kohlberg including different domains of social judgment—moral or conventional (see the following)—and the need to judge multifaceted situations. Life experience and accommodation to the environment are also important. Individual moral decisions and actions take place within a social context, and are influenced by peer group norms.

A study of moral judgments of high school students found significant differences between children in a school that fostered democratic decision making compared to children in a conventionally organized school. Most students in the “democratic” school thought they and their peers would chose to act prosocially, whereas in the “regular” school most made the prosocial choice for themselves but thought their peers would not do the same ( [Higgins et al., 1984](#) ).

Another important factor determining moral choice is the information available. [Wainryb \(1991\)](#) studied children's moral judgments of events before and after hearing information that challenged their initial opinions. All children thought it was wrong for an irritable adult to spank a child who had not erred. But some held it was right to spank a repeatedly misbehaving child because they thought this taught children to behave better. When the responders were told spanking was ineffective in socializing children, most of them changed their mind. In this study, information profoundly affected the moral judgments of young people aged 11 to 21 years. This has implications for education but also for the information presented to the general public by the media.

## Two Aspects of Morality: Justice and Benevolence

Kohlberg's major study involved only boys. His colleague, [Carol Gilligan \(1982\)](#), developed the idea of fundamental sex differences in moral orientation, with men giving priority to justice and rights, women to care and responsibility in human relationships. She also held that hypothetical and real-life moral dilemmas elicit different types of responses. Her arguments are based on interviews concerning their experiences and thoughts about moral conflicts of three groups of people: 25 male and female college students taking a course on moral and political choice; 25 women undergoing counseling before a possible abortion; and a small number of male and female subjects, ages 6 to 60. No account of systematic analyses of the data is given.

A replication of Kohlberg's and Gilligan's work, with a bigger sample of male and female subjects between 5 and 63 years old, confirmed that there are few violations of stage sequence over time. In contrast to Gilligan's findings, no significant sex differences emerged but the types of responses to real-life and hypothetical dilemmas did differ, although not systematically, in the direction of care and responsibility for real-life problems, justice, and rights for hypothetical issues ( [Walker, 1989](#) ).

Although Gilligan's work led to the important rediscovery of benevolence, and its relationship to justice, as an aspect of morality ( [Eckenberger, 1999](#) ), the limitations of her sampling and the lack of clarity of the analyses of her findings have cast doubt on her proposition that morally men and women speak in a “different voice” ( [Okin, 1996](#) ). Yet sex differences in early socialization and early prosocial behavior do occur ( [Hay, 1994](#); [Lewis et al., 1992](#) ). In early childhood, girls tend to be more compliant ( [Kochanska et al., 1995](#) ) and share more toys, especially with other girls ( [Hay et al., 1999](#) ).

## THE ROLE OF EMPATHY IN THE DEVELOPMENT OF PROSOCIAL BEHAVIOR

Both Anna Freud and Piaget referred to the importance of empathy in moral development; but, [Hoffman \(1975, 1984, 2000\)](#) was the first see this as an intrinsic, internally based moral motive, with guilt and other emotional aspects of morality deriving from the capacity for empathy with the suffering of others.

The experience of empathy varies with cognitive level. Emotional responses to the feelings of others are seen even in the newborn: When one baby in a nursery cries, the others follow suit. This “reflexive emotional resonance” ( [Campos et al., 1983](#) ) manifests before infants can distinguish between themselves and others, between their own emotions and those of other people. Only at around 9 months are babies thought to be able to locate feelings in others. In their first year, children respond to distress in others as if it were happening to themselves; however, in the second year, they are capable of sympathy, that is, of offering comfort to others. Toddlers, for example, whose mothers suffer from depression, make active attempts to offer solace when their mothers weep ( [Radke-Yarrow et al., 1985](#) ). Such activity may be inappropriate at first. When one toddler cries, another will call on her own mother for help, even if the crying child's mother is present, or she will offer her own comforter rather than that of the toddler in distress. By the third year, however, when role-taking occurs in play and children begin to model themselves on other people, they can realize that the feelings of others are different from their own, and in the fourth year, empathic distress prompts the offering of help geared to the needs of the other person.

[Hoffman \(1975, 2000\)](#) saw empathic responsiveness together with a cognitive sense of the other person as the basis for altruism, a biological, prosocial motive to offer help, independent of self-interest and the prospects of reward. People help others in need even in the absence of approving witnesses. Moreover, popular children, emotionally secure, loved at home, and hence less in need of approval from others, are more inclined to help than children who receive little affection. Hoffman speculated that children exposed to frequent, severe distress and frustration are likely to develop egoistic self-absorption, which interferes with their sensitivity and openness to the needs of others.

[Hoffman \(1984\)](#) also suggested that empathic responses are further differentiated in middle childhood. Children withdraw when the distress of others is overwhelming. They empathize more with others like themselves, offering less help to outsiders. They now concern themselves with the more enduring sufferings of other people, even when far away or unknown, such as the victims of war or famine in other countries.

In summary, four main stages of empathic development have been described ( [Hoffman, 2000](#) ): global empathy in the first year; “egocentric” empathy in the second year, when differentiation of self from others and object permanence have been attained; veridical empathy based on awareness that others too have thoughts, feelings, and wants, and depending on role and perspective-taking capacities, at between 2 and 3 years of age; and, in later childhood, empathy for the experiences of others beyond the immediate situation, associated with the development of a sense of personal identity.

If the observer was the cause of another's misery, self-blame transforms empathic distress into guilt ( [Hoffman, 1984](#) ), which eventually becomes dissociated from empathy. Both are quintessential moral motives. Hoffman holds that a comprehensive moral theory requires both motives and principles of justice and fairness, as outlined by Kohlberg. He believes that children are best socialized to act in accordance with an inner principle of altruism if, once capable of empathic understanding of others, parents avoid power assertive methods and use an inductive style of child rearing, consistently pointing out the possibly harmful effects on others of antisocial conduct. His important work stimulated much subsequent research.

[Zahn-Waxler and Radke-Yarrow \(1982\)](#) trained 24 middle-class mothers to record observations of their young children in response to emotional distress in others (natural and simulated, caused and not caused by the child) over a 9-month period. Somewhere between 12 and 18 months, most children's behavior changed from a few interactive and merely diffuse affective responses to positive forms of reaching out toward and caring for another person in need. By 18 to 24 months, verbal expressions of sympathy accompanied such behaviors. The findings go against psychoanalytic ideas of egocentric narcissism because at this early age the responses could not have been learned. Innate, empathic distress and later observation learning, the authors hold, promote prosocial development. In their own samples this was linked to maternal disciplinary techniques, in particular to “inductive reasoning,” that is, the mothers' use of affective and moralistic explanations of the harmful effects on others of the children's hurtful actions.

Further insights into children's understanding of feelings, especially in relation to conflict, and into the earliest teaching and understanding of rules have come from [Judy Dunn's \(1987\)](#) observations of siblings and their mothers. Even at 24 months, in pretend play between siblings, social rules were explored and feeling states simulated. Some 18-month-old children with an affectionate older sibling could already take part in a shared fantasy involving the feelings of others. These very young children were clearly practicing, in play, how to conform to rules and engage in prosocial behavior.

## THE ORIGINS OF EVALUATIVE BEHAVIOR AND SELF-AWARENESS

A critical study of early moral development, supporting a biological basis for the acquisition of inner standards and self-evaluation, and for readiness to learn the morality and conventions of the culture, is [Kagan's \(1981\)](#) short-term longitudinal investigation of American and Fijian children in the course of their second year. Although reluctant to support psychoanalytic theories about the development of a sense of shame in the second year and conscience and guilt in the third, Kagan's observations led him to identify a stage in the maturation of children in which the capacity for the development of inner standards first appears.



He observed children together with their mothers at monthly intervals in a variety of standard play settings. In one the child was presented with 10 unflawed toys, 10 damaged toys (such as a doll with a missing eye or a car with chipped body work), and two undamaged meaningless objects. No 14-month-old child discriminated between damaged and undamaged toys, but thereafter, and increasingly as the second birthday drew near, children began to show unambiguous concern about the damaged objects, commenting "broke," "fix it," "yucky," or taking the broken toy to the mother. In another experiment, an adult demonstrated skilled play with toys. All 13- and 14-month-old children watched calmly or seemed bored. But from 15 months onward more and more began to fret, protest, wish to leave the room, or indicate "It's mommy's turn to play." These older children seemed to feel obliged to copy what the adult did, realized it was beyond their capacity, and became distressed. These changes occurred just before the use of "evaluative language." Between 19 and 26 months, the first references to "dirty," "can't," and "hard do" appeared, and at 20 months, most children used words like "good," "bad," "dirty," and "nice."

Kagan concluded that the acquisition of these new functions centered on children's sensitivity to adult standards and their own capacity to meet these, that is, on an awareness of what they can and cannot do. Because American and Fijian children responded similarly, this development in the second half of the second year constituted a maturational shift rather than a cultural effect. The standards now acquired relate to cleanliness, integrity of property, toileting behavior, and harm to others.

Work on this important early milestone of self-evaluation was extended by the experimental studies of [Stipek and associates \(1992\)](#). They identified three stages in its development: (a) in the first half of the second year, pleasure at mastery and joy in causality, with anger and distress but no shame at failure and no anticipation of the reactions of others to their performance; (b) from 21 months onward, pride in accomplishment, that is, an anticipation of the reactions of adults, and active seeking of positive responses to success and avoidance of negative reactions to failure; and (c) a gradual internalization of these external reactions with children beginning to evaluate their own performance and reacting emotionally to success and failure independently of their expectations of adult responses. The authors stress that they investigated only the domain of achievement, not morality, and they point out that praise is always more common than disapproval except in the moral domain.

These workers made one further observation: Winning or losing in a competitive task was not understood by children younger than 33 months, but thereafter, pleasure in completing a task was increased by winning. The timing of this development, we must note, is in line with psychoanalytic ideas about the "genital" stage, when competition was thought to enter the child's range of experiences for the first time.

## MORALITY AND CONVENTION

Even young children distinguish between morality and convention. Conventions are behavioral uniformities that serve to harmonize social interactions and form, and are themselves determined by the specific social system. Morality concerns prescriptive judgments of justice, rights, welfare, and the avoidance of harm to others and is inherent in interpersonal relationships, not tied to the social context. It is a convention that in most schools children address teachers by their second, not first, name. Such rules are arbitrary and could be changed by consensus. [Turiel \(1983\)](#) asked children from 6 to 17 years if it would be wrong to call teachers by their first names if there were no rule about that. Even quite young children thought not. He also questioned children about the rights and wrongs of a playground situation in which all swings are occupied, and a little girl eager for a turn comes along and pushes another child off a swing. All agreed that this was wrong and would be wrong even if there were no rule about it.

Most children thought that, if everyone agreed, the rules of games and social conventions could be changed, but not such inherent moral dictates as that it is wrong to hurt other people and to steal. Younger children regarded rules as more fixed, and older ones had a better understanding of their purpose. But all children, from 6 upward, distinguished between conventional rules and morality, and in an earlier study, even preschool children could do so ([Nucci and Turiel, 1978](#)). However, very young children respond more often to moral rather than social conventional events; and even in middle childhood, children are more responsive to moral events and moral injustice than conventional transgressions, which only acquire salience with increasing age ([Nucci and Nucci, 1982](#)).

It has been argued that moral ideologies too are social constructs, depending on different conceptions of the self, society, and nature; that Indian cultures, for example, are duty-based, US cultures rights-based. [Turiel and colleagues \(1987\)](#) argue that despite discretionary divergence, all cultures share a common, mandatory morality. A study of Korean children bears out their similarity to American children in their views of morality and its distinction from conventions ([Song et al., 1987](#)).

[Blair \(1995\)](#) postulated that moral behavior and the capacity for distinguishing between morality and convention are fostered by what he called a "violence inhibiting mechanism," the suppression of aggression, presumably through the activation of empathic responses, in the presence of distress cues emitted by a victim. Even in some animal species aggression is inhibited when a conspecific gives out submissive cues.

## IMPLICIT KNOWLEDGE PRECEDES EXPLICIT UNDERSTANDING

[Stipek and coworkers \(1992\)](#) found children's behavior following success or failure to be much more mature than their expressed ideas, although the sequence, from the pleasure of success, to pleasure from approval of others, to satisfaction from meeting inner standards, was the same in both spheres. Implicit knowledge long precedes explicit understanding. This is strikingly revealed in [Judy Dunn's \(1988\)](#) studies of the origins of social and moral understanding in early childhood. Detailed home observations were made of children in their second and third years in the company of their mothers and younger siblings. Dunn takes issue with the seriousness with which moral development tends to be discussed and the "bleak emphasis" on anxiety, guilt, and fear. This is what impresses clinicians in their practice with disturbed children. In the natural setting of normal children's own homes Dunn found that, even before they use many words, children are amused by rules, roles, and the relationships in their world and share jokes about these with others. Just as they play with the rules of language, so they joke about what is expected, allowed, or punishable. They find it funny to distort or exaggerate rules, make scatological jokes, teasingly misname things, and make false assertions.

From 18 months, children understand how to hurt and comfort others, what the consequences of their own hurtful actions are, what is allowed or forbidden in their own family, and how adults are likely to react to their misdeeds and to those of others.

Dunn stresses four features:

1. Understanding the feelings of others develops in the first 2 years: ". . . the foundations for the moral virtues of caring, considerateness, and kindness are well laid by three years" (p. 170).
2. Understanding the goals of others begins in the second year, as shown by children's behavior during conflicts and cooperative endeavors, and in the third year in pretend games, narratives, and questions about other people.
3. Understanding social rules is clear from children's discussions and jokes about breaking and the differential application of rules. By 2½ to 3 years, children have a practical knowledge of the idea of responsibility and of excuses on the basis of intent and incapacity. They know that rules can apply differently to different family members, rules can be questioned, and transgressions justified. They understand about authority relationships within their family and comment on the behavior of others in moral terms.
4. Understanding other minds, which also starts in the third year, is yet another new way of social understanding. Children now talk about mental states such as remembering and forgetting. Although capable of feeling embarrassment, guilt, and shame, they are probably not yet able to recognize these more sophisticated emotions in themselves or others.

Dunn describes how parents' disciplinary strategies are affected by children's growing cognitive and communicative skills. She recognizes cultural differences in parents' reactions to children's anger and aggression. Parents also respond differently to sons and daughters. Three-year-old girls, for example, show more shame than boys when they fail at experimental tasks ([Lewis et al., 1992](#)).

Emotions are very evident when moral and social rules are discussed. Children in their third year are most likely to reason during disputes, which earlier had caused them most distress. But they also learn from calm reflective discourse. Dunn holds that the child's part in the power relationships within the family, rather than an urge to conform or assuage guilt, is the main motive to learn about social and moral rules. She believes moral behavior is fostered not so much by socialization pressures, but by the child's need to assert his or her place, for example in relation to a sibling; the active enjoyment of rule breaking, teasing, and exerting power within the family; and also happy, cooperative play.

## THE MORAL EMOTIONS

Accurate descriptions of the emotions of self-assessment—pride, shame, and guilt—are a basis for the exploration of the moral emotions. According to the philosopher [Gabriele Taylor \(1985\)](#), shame and embarrassment depend on the awareness of being observed and relate to all wrongdoing and one's standing in society; however, guilt, which appears later in development, relates to law breaking and, specifically, to moral rather than conventional wrongdoing. Awareness of an authority to be obeyed plays the major role in conscience, whereas a watchful audience is the major factor in shame. Guilt and punishment relate to a bad deed or omission and are linked to a sense of responsibility for one's own actions, but shame concerns the kind of person one is. In Taylor's view, neither shame nor guilt is related to empathic feelings for others, but remorse, the “emotion of salvation,” is. Remorse is also the precursor of self-forgiveness, essential if the destructive forces of guilt are to be assuaged. Guilt, shame, and pride are reflexive emotions, guardians of societal norms, elicited when one compares one's own behavior or achievements with those of others ([Terwogt and Stegge, 1999](#)).

[Harris \(1989\)](#) explored the development of children's emotional understanding by testing 3- to 5-year-olds on a perspective-taking task. Those who excelled at this were also more attentive to their younger siblings when distressed by a temporary separation from their mother. The mothers, in turn, more often asked these more empathically competent older children to “look after your brother.” Whether the maternal responses were causes or consequences of the older child's empathic skills is not known.

Children's recognition of emotions and their understanding of what brings them about develop in a similar age sequence in different cultures. Children are born with a capacity to experience the basic emotions of happiness, sadness, anger, and fear, associated as they are with facial expressions. They come to understand that other people have them too. They learn to differentiate between morality and convention on the emotional basis of the distress that moral, but not social, rule infringements induce in the victim. This, Harris believes, is learned from other children rather than adults.

Children under about 8 years have difficulty identifying pride, shame, and guilt in others because these emotions are not accompanied by distinctive facial expressions. Understanding these more complex emotions depends on the ability to imagine the mental state of another person that gives rise to these emotions. It depends also on the availability of concepts of personal responsibility and normative standards of morality and propriety. Children experience a shift of awareness from believing that people's feelings about their behavior depend on its consequences, to attributing them to the approval or disapproval to be expected from other people in the light of social norms. Only at about 8 years do children feel proud or ashamed “of themselves” without reference to an external audience. The developmental sequence [Harris \(1989\)](#) detects is congruent with both Turiel's and Kohlberg's findings. Even 3- to 4-year-old children know that harming others causes distress and is therefore wrong. Four- to 5-year-old children know that people are happy if they get and do what they want. By 7 to 8, children know that people are happy if others approve of them and they exert themselves to gain approval. Only after the age of 8 can children work out that they themselves and other people get satisfaction from doing what they think is right.

There is further evidence that the comprehension of emotional accompaniments of immorality, as the development of empathy itself, changes with age and begins to be accurate only at about 7 or 8 years ([Arsenio, 1988](#)). In an illuminating experiment ([Nunner-Winkler and Sodian, 1988](#)), 4- to 8-year-old children were told a series of stories of moral transgressions and had to judge how the wrongdoer would feel. Most 4-year-old children thought that the story person who violated a moral injunction, got away with it, and succeeded would be happy, whereas 8-year-old children attributed negative emotions to this person because they would feel bad about what they had done. There was also a shift between 4 and 8 years in the attribution of emotions to a story character who had resisted temptation. Very young children judged according to the material outcome; older children judged according to the character's sense of virtue. When asked to judge the degree of badness of story figures, 6-year-old children already thought that happy wrongdoers were worse than those who felt sorry for what they had done.

When we compare these findings about children's expressed ideas about their understanding, with observational studies of children's behavior in response to success and failure ([Stipek et al., 1992](#)), we see again the large age gap between the attainment of behavioral accomplishments on the one hand and children's expressed ideas on the other. Yet there is a similar sequence: from the satisfaction of success, to pleasure from approval of others, to satisfaction from meeting inner standards.

An experimental study of 7- to 12-year-old children's understanding of the “self-conscious” emotions of guilt and shame ([Ferguson et al., 1991](#)) showed that shame resulted both from moral transgressions and social blunders and led younger children to feel embarrassed and older ones to feel they could not do things right. Guilt, in contrast, was aroused by moral norm violations and involved a conflict between approach toward and withdrawal from the victim as well as self-criticism, remorse, a desire to make amends, and fear of punishment.

Masking of emotions starts in the early school years, but even 3-year old children can hide their feelings, for example, of disappointment at a gift ([Terwogt and Stegge, 1999](#)).

[Harris \(1994\)](#) draws an important distinction, quite insufficiently acknowledged by legal experts and the general public, between knowing what is right and wrong and abiding by moral rules. The impulse to tease or hurt often overrides the moral code: Knowledge of right and wrong is not closely related to action.

[Harris \(1994\)](#) also believes that children's emotional experiences influence age-related cognitive changes, as well as behavior. When parents reason with children about the possible effects of their misdeeds on others instead of punishing, they induce anticipatory guilt and increase children's understanding of social emotions. When children witness parental quarrels, they become more physically aggressive with their peers. This may not owe merely to modeling, because verbal quarrels between parents without physical violence have the same effect. It is more likely that the children's state of arousal caused by the quarrels they witness leads them to attribute more malign intent to their peers and react accordingly ([Dodge et al., 1990](#)).

## MORAL DEVELOPMENT AND PSYCHOPATHOLOGY

The psychopathology of moral development is likely to be as important for child psychiatry as that of attachment behavior, and is of great relevance in understanding the origins of the most worrying child psychiatric conditions: the conduct disorders, with their high risk of adult antisocial personality disorder, but also of affective illnesses in girls. Although we know a great deal about the affectional needs of young children and the hazards of early separations from parents and deprivation of continuous care, explorations of the ingredients necessary in early childhood for the development of empathic feelings for others, inner standards of self-appraisal and morality, the capacity to feel shame and guilt, impulse control, and sympathetic prosocial behavior are less advanced.

Studies of the moral development of deviant children and children exposed to different kinds of adversity are now beginning ([Campbell, 1995](#)).

### Social Learning and Role Modeling

Learning theorists ([Chapter 10](#)) have contributed to our understanding of the genesis of both prosocial and antisocial behavior in childhood, although they have not generally taken a developmental viewpoint, although the early stimulus-response, or reward and punishment, theories of behavioral change have been too simplistic. [Bandura's approach \(1977; Bandura and Walters, 1963\)](#) was more sophisticated and included the cognitive accompaniments of behavior. His experimental studies of how and under what conditions children copy the behavior of others have been illuminating for understanding social development and in the treatment of childhood behavior disorders. We now know that the capacity for role modeling starts toward the end of the second and the beginning of the third year ([Meltzoff, 1988](#)) and that young children, after watching films of other children playing aggressively, are susceptible, especially when frustrated, to develop similar play patterns.

In what remains a classical study, [Patterson and his colleagues \(1967\)](#) found that in the setting of an unstructured nursery, with little active intervention from their teachers, 4-year-old middle-class boys learn to become ever-more aggressive in imitation of other assertive children when they see assertiveness rewarded with success (as it always is without adult intervention). Only passive children with low rates of interaction with others escape this development.

That parental punitiveness and aggression, usually directed against boys more than girls, and often associated with substance abuse, induces aggression in children has been documented repeatedly ([Chapter 29](#) and [Chapter 37](#)).

### Coercive Child Rearing

[Patterson's later work \(1982, 1986\)](#) has been unique in analyzing the mutually reinforcing processes by which coercive, punitive parents evoke counter-aggression from their children with escalating domestic violence. Contributing factors are the parents' own past experiences of coercive rearing, their failure to monitor their children's behavior, the aggressive role models they provide, maternal depression, and, on the children's part, a difficult temperament, including impulsivity. Social



and educational failure at school and low self-esteem often follow such domestic interactions, with further reinforcement of the children's antisocial conduct. Patterson also regards stealing as a form of noncompliance to unskilled parents. He found the parents of children who steal more often themselves to be delinquent and, unlike the parents of aggressive children, inadequately attached to their offspring. A recent study found that mothers of 3-year old children with behavior problems used more reactive and fewer preemptive strategies than mothers of well-adjusted children. Moreover, when behavior problems at 3 were controlled for, reactive strategies at this age predicted conduct problem at 5 years ([Gardner et al., 1999](#)).

The work of [Dodge \(1986; Dodge et al., 1990\)](#) documented mutually reinforcing processes that engender aggression between peers. He reminds us that aggressive behavior correlates with deficits in a variety of cognitive skills: self-rehearsal of responses, inhibition of premature social impulses, interpersonal problem solving, referential communication, and role taking. Socially and verbally unskilled people have limited means for handling discord and become physically aggressive with very little provocation.

### Social Attributions

In his studies of children's cognitive attributions, Dodge showed that aggressive boys tend to interpret the motives of their peers as hostile, even when they are not, and to react aggressively, thereby stimulating dislike and hostility in previously nonhostile peers. This then reinforces the aggressive boys' view of other children's hostile intent. A vicious circle of hostile attributions and aggressive behavior with peers results, similar to the coercive interactions between parents and children described by [Patterson \(1982, 1986\)](#). The long-term outcomes are academic failure, school dropout, and delinquency. Furthermore, aggressive boys tend to overestimate their peer's aggression and underestimate their own; whereas nonaggressive boys base their perceptions more on actual behavior ([Lochman and Dodge, 1998](#)).

Dodge also found that children who have been abused, especially boys, are predisposed to develop hostile attributions and aggressive behavior ([Dodge et al., 1990](#)). The effects of harsh child rearing are in part mediated by such maladaptive social information processing, induced by these child-rearing methods ([Weiss et al., 1992](#)). Such developments are more common in families living in poverty and marital conflict, but these factors alone do not account for the findings.

### Atypical Socioemotional Expectancies

Other workers have focused on different psychological predispositions to conduct disorders: impaired understanding of the emotional consequences of sociomoral events; and the role of atypical emotional expectancies in perpetuating antisocial behavior. [Fisher and Blair \(1998\)](#) studied boys in a special school for disturbed children, comparing those with high and low scores on a somewhat unfortunately named "psychopathy screening device," which identified both callous, unemotional attitudes, and impulsivity and conduct problems. High scorers on both factors were significantly poorer on a test requiring the distinction to be made between stories about moral transgressions resulting in harm to another person, and conventional stories; those with high scores on impulsivity and conduct disorders alone, also did worse on a task measuring response-set modulation. [Arsenio and Fleiss \(1996\)](#), in a more comprehensive study, compared 7- and 10-year-old behaviorally disruptive children with nondisturbed controls. Four sociomoral rule systems were investigated with case vignettes: inhibitory morality, involving victimization; conventions; prosocial morality, involving altruistic acts; and personal restrictions. Emotional expectancies varied with the domain tested, and disturbed children differed from controls, especially for inhibitory and prosocial morality. They minimized the fear associated with victimization and emphasized the victimizers' emotions related to material gains and psychological consequences, while referring less to the loss, harm, and unfairness they had inflicted. They also tended to stress prosocial actors' emotions relating to the harm, loss, and unfairness that had been avoided, rather than those of the recipients of the benevolent acts. In addition, the disruptive children more often mentioned sadness as an emotional outcome than the controls.

These studies were of children from middle childhood onward. But the capacity for empathy, developing inner standards, and learning prosocial behavior from adult role models begins much earlier—in the second and third years—as we saw. What goes on in families then is likely to have important consequences for later life.

### The Impact of Maternal Depression

[Dowdney and colleagues \(1985\)](#) observed differences in the disciplinary behavior in interaction with their toddlers of mothers who were either depressed or had, as children, been in care (i.e., in children's homes), compared with a control group. [Zahn-Waxler and coworkers \(1990\)](#) documented the relationship between maternal depression in children's toddler years and their later distorted and exaggerated expressions of guilt. Young children, highly aroused, overinvolved, and causally implicated in their mothers' distress, before they are mature enough to cope with this, are at risk of later excessive guilt and distortions of their capacity for empathy and concern for others. Such overinvolved young children are also often deprived of normal opportunities for play with peers, vital for the development of social competence and morality. Boys are more at risk when mothers are depressed, less able to adapt their behavior in a compliant and conforming way than girls ([Zahn-Waxler, 1993](#)). This sex difference is supported by the finding that daughters and sons of healthy mothers did not differ in their advocacy of aggressive solutions to conflict, whereas sons of depressed mothers, despite being more prosocial and empathic than other boys, were the most likely to advocate aggression. Daughters of depressed mothers, in contrast, who also had high rates of empathy and prosocial behavior, almost never did, even in defense of their own property, wishing at all costs to avoid conflict ([Hay, 1994](#)).

## THE EFFECTS OF EARLY PARENTING STYLES AND CHILDREN'S TEMPERAMENT

[Dienstbier's \(1984\)](#) experimental studies focused on both cognition and affect and suggest that when children are coerced by means of parental punishments, they attribute their resistance to temptation to external forces. They feel bad if they succumb because of the punishment that follows and, if they believe their transgressions will not be discovered, they capitulate. Noncoerced children, in contrast, attribute resistance to temptation to their own choice and their inner judgment that one course of action is better than another. If they succumb to temptation, they feel bad because of the transgression itself rather than its consequences, and are less likely to repeat it even if unobserved. Dienstbier's findings agree with those of Kochanska (see the following), that children with high levels of emotional tension learn better to make internal attributions about transgression and feel appropriate guilt. Children with low levels of emotional tension learn poorly, evoke ever more coercion from parents, and have their external attributions reinforced every time they transgress and "get away with it" without feeling bad. This is in line with the view that children are best socialized when parents use inductive, that is, reasoning methods, but do so with an emotional charge.

The effects of differences in mothers' early socializing styles on children's later development of guilt and conscience have been meticulously investigated by [Kochanska \(1991\)](#) in her early longitudinal study of children of depressed and well mothers ([Radke-Yarrow et al., 1985](#)). She also studied the influence on these processes of the child's temperament. Self-reported and observed coercive/noncoercive parental rearing styles when children were 18 to 42 months old, were compared with children's contemporaneously observed compliance to maternal demands and with their affective/moral orientation at the age of 8 to 10 years. The latter was assessed by means of narrative responses to semiprojective stories. The main finding was that noncoercive child rearing was associated with evidence for internalized conscience in later years, but only for children prone to fearful arousal. Early compliance to maternal demands predicted later internalized conscience. The author concluded that anxious children are well socialized by a noncoercive style of child rearing and that conscience development may be interfered with by coercive methods. How less anxious children, less responsive to child rearing style, acquire their conscience was not then clear.

In a review of moral development, [Kochanska \(1993\)](#) proposed a new framework for the study of early conscience formation that integrates parental socialization methods with the temperamental characteristics of their children. She suggests two components of conscience contributing to moral conduct: a child's affective discomfort owing to actual or potential wrongdoing and the capacity for behavioral self-control. The first was salient in psychoanalytic models, the second in social learning and cognitive models of moral development. Both have temperamental underpinnings, which may account for the differential effects of parental socialization methods. Fearfulness and social inhibition interact with high levels of moral motivation to reduce immoral behavior in situations of temptation ([Asendorpf and Nunner-Winkler, 1992](#)). Impulsivity versus behavioral control, Kochanska suggests, is a more complex temperamental dimension, and precisely how it interacts with parents' socialization efforts is less well established. Reasoning may be less effective in encouraging the internalization of standards for impulsive children. [Shaw and Bell \(1993\)](#) presciently suggested that irritable, hyperactive, demanding infants increase maternal unresponsiveness and that the resulting anxious, avoidant attachment leads to later noncompliance and hostility. Securely attached infants, by contrast, become compliant in later years, eager to please their parents.

The most impressive researches illuminating our understanding of compliance, self-control, and conscience development in young children, are Kochanska's more recent observational and experimental studies of two large cohorts of children, the first at 2 to 3½ years, and again at 3½ to 4½ years; the second at 8 to 10 and 13 to 15 months ([Kochanska, 1995; Kochanska and Askan, 1995; Kochanska et al., 1995, 1998](#)). In the first, time sampled observations were made at home and in the laboratory during a toy clearing up task (the "do" context), and with toys their mothers had forbidden them to touch (the "don't" context). Children were observed in these two situations both with their mothers and alone. "Internalization" was judged to be present when children, even in the absence of their mothers, adhered to her requests and prohibitions. Mothers were additionally questioned about their children's temperament and behavior, especially about evidence for internalization



(latency to transgression, extent of transgression and rule-compatible conduct), and about their own disciplinary methods. A distinction was drawn between children's "compliance" (when they did what was required but only passively or protesting or so long as the mother was present) and "committed compliance," when they joyfully endorsed their mother's agenda, and their verbal comments indicated that they had identified with her goals, apparently with a sense of inner obligation. In the younger children, this was greater for "don'ts" than "do's," was higher in girls than boys, and was related to shared positive affect between mother and child. Internalization at the later age was predicted by earlier committed compliance and shared positive affect.

In addition, children's temperamental fearfulness and anxiety proneness as well as their security of attachment were measured at the early age and related to later evidence for internalization. Two pathways toward internalization were discovered: gentle maternal discipline, deemphasizing power, predicted this for relatively fearful or anxious children; for relatively fearless children, the salient factor was security of attachment with positive cooperation between mother and child.

P>In a later study of even younger children, self-restraint at 8 to 10 months (after mothers had prevented their babies from touching an attractive toy) was found to be associated with committed compliance and internalization of maternal prohibitions at 13 to 15 months, and this early self-restraint was associated with sustained, or "focused," attention. A confirmatory study by [Stifter and associates \(1999\)](#) also found that infants who display regulatory skills in response to frustration are more compliant in their third year.

Kochanska proposed that committed compliance in early childhood reflects the origin of internalization and is an important antecedent to conscience formation. We need to be clear, however, that her more recent work focused not on person-related, but object-related behavior, which may yet turn out to be allied to the adherence to convention rather than morality. [Zahn-Waxler and Radke-Yarrow \(1982\)](#), as we saw, had found inductive reasoning to be important for the development of empathic concern for others. Although it is very likely that in early childhood noncoercive child rearing with shared positive affect between mother and child, as well as security of attachment, are as important for the development of interpersonal morality as for committed compliance in object related tasks, further evidence for this is needed.

## IS THERE A BIOLOGICAL SUBSTRATE FOR MORAL DEVELOPMENT?

[Hoffman \(2000\)](#) sees the capacity for empathic morality as innate, but fragile and vulnerable to power assertive child rearing. Kagan argued for a biological propensity for the acquisition of inner standards. There is evidence for a genetic basis for individual differences in empathy and other forms of prosocial behavior ( [Hay, 1994](#); [Hoffman, 2000](#)); and [Brothers \(1989\)](#), discussing the evolutionary basis for empathy, suggested that primate survival depends on the capacity for rapid and accurate evaluation of the motivations of others. He instanced the absence of empathy in some pathologic states such as autism, and speculated about its neurologic substrate. Constitutionally based lack of empathy (or theory of mind) characterizes people with other autism spectrum disorders too and is likely to contribute in an important way to failure of early socialization and the genesis of some forms of conduct disorder and sociopathy in later life ( [Wolff, 1992](#)).

The most dramatic validation of these ideas comes from the investigations in early adult life of two patients, from stable, middle-class families, who had sustained prefrontal cortex lesions before the age of 16 months, and who, despite normal intelligence, developed severe and persistent antisocial behavior, including stealing and violence ( [Anderson, 1999](#)). In contrast to six patients who had sustained prefrontal cortex lesions in adult life and developed antisocial behavior, were insensitive to the consequences of their actions, and failed to respond to behavioral treatments, the patients damaged in early childhood were additionally defective in executive functions and social and moral reasoning. The two patients with early injuries, unlike patients injured in adult life, functioned at only a pre-conventional stage of moral reasoning on Kohlberg-type tests (Kohlberg's stage 1) ( [Colby et al., 1983](#)). Adult onset patients showed guilt and remorse; not so those with an early onset, who were also more violent. Magnetic resonance imaging revealed a bilateral lesion of the polar and ventromedial prefrontal cortex in one early onset patient, and a unilateral, right prefrontal lesion in the other.

The authors speculate that adult-onset patients fail in social interactions because of disruption of the system holding overt and covert emotionally related knowledge of social situations, so that recall of socially relevant facts and their associated emotions is insufficient to ensure adequate real-life social behavior, whereas reasoning about social interactions in the abstract remains intact. When lesions are sustained very early, the authors suggest, socially relevant factual and emotional knowledge is never acquired, and even factual knowledge about accepted standards of moral behavior ( [Dolan, 1999](#)) is deficient.

Further light on these findings has been shed by Damasio's "somatic marker hypothesis." He found that patients who had sustained prefrontal damage in adulthood did not have the usual autonomic nervous system responses to seeing fleeting pictures of cruelty and disasters interspersed with neutral displays, although they could remember what they saw. Retrospectively, they indicated that the feelings they would have had in response to the pictures prior to their injury were now lacking. [Damasio \(1994\)](#) speculates that we are born with a neural propensity to generate somatic states, accompanied by pleasant or unpleasant emotions, in response to stimuli concerning personal and social behavior. In the process of childhood socialization, specific classes of stimuli are paired with specific somatic states and emotions, so that in later life, certain social situations and moral dilemmas are associated with anticipatory somatic and emotional responses profoundly affecting how we choose to act, for example, altruistically. The somatic marker hypothesis is compatible with the notion that adaptive personal and social behavior requires an adequate capacity to form theories of mind, and that cognitive and emotional processes are intimately linked. Moreover, anticipation of a good future outcome helps us to sustain immediate and transient unpleasantness. Patients with frontal lobe damage are myopic for the future. The necessary substrate for adaptive sociomoral development is an intact prefrontal cortex and effective early socialization.

## SUMMARY

The last 20 years have seen much progress in the exploration of the development of morality and prosocial behavior. Emotions and cognitions are intimately connected in all aspects of development ( [Donaldson, 1992](#)), including moral development, throughout early and middle childhood. There is a biological basis for regular developmental changes with age both of reasoning and emotional experiences. Biological factors are important also in determining temperamental differences between children, for example, proneness to anxiety or impulsive disinhibition, which profoundly affect their responsiveness to socialization. Experiences of child rearing and peer relationships, in interaction with temperamental differences, then determine the development of moral behavior and beliefs.

Prosocial impulses arise in the first year of life with manifestations of empathic responsiveness and the capacity for self-restraint. In the second and third years, children become aware of norms of behavior and their own achievements and shortcomings. Within the context of family life, even 2- to 3-year-old children display surprising knowledge about moral and conventional rules and do so with enjoyment and humor. It is at this stage that parental behavior and parents' beliefs and expectations about their children's moral development and their own contributions to this are likely to be crucial. Parents' behavior is influenced by many factors: their socioeconomic and cultural milieu, marriage, health, and children's gender and temperamental responsiveness in family interactions.

Children can reason about moral issues only at 7 or 8 years. Concepts of guilt and shame are now articulated. Children develop notions of morality independent of external approval and begin to be able to reason about principles not tied to concrete events. Abstract ideas of justice and welfare arise later still. Children's cognitions, especially their attributions of hostile intent, peer relationships, and prosocial behavior, remain crucially influenced by their experiences, now both within their families and at school.

Although psychoanalysts were right to stress the importance of attachment to parents as a basis for conscience development, and were also correct in identifying the emotions as the vital mediators in socialization and the process of identification with parental standards as crucial, they erred in their overemphasis of anxiety and threat as promoting moral behavior in childhood. Dunn's observations and Kochanska's experimental studies show that very young children best acquire moral ideas in an affectively positive atmosphere, when they joyfully comply with their mother's requests and strictures, making them their own. The importance of pleasure at doing right has long been underestimated.

The discoveries of how parental disability and different methods of child rearing interact with children's individual temperamental styles in the development of conscience and prosocial behavior, is likely to contribute in a major way to the more effective treatment of antisocial conduct in childhood and its prevention in later life.

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## 20 CHILDHOOD SEXUALITY

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Genetic and environmental forces interact to shape sexual development before birth and throughout life. Genetic factors establish a level of sexual responsiveness that remains fairly constant across the life span relative to other people of the same age and sex ( [Kinsey et al., 1948, 1953](#)). Genetic factors operate by determining the levels of neurotransmitters and hormones that then influence the sexual response. Dopamine and serotonin act reciprocally to enhance sexual responsiveness and to promote satiety ( [Hull et al., 1999](#)). Overall responsiveness is positively affected by norepinephrine and testosterone but negatively affected by g-aminobutyric acid and prolactin. If the person is successful in coupling, this affects the external and internal environment, enhancing levels of dopamine and interest in further sexual pursuits. Although prepubescent children have lower levels of gonadal hormones than adults, the same mechanisms apply, making some youngsters more sexual and more erotically attuned than others.

In general, men, with their higher testosterone levels, are more active and interested in sex than women. Women, with their lower testosterone levels, are relatively more influenced by psychological and social factors ( [McConaghy, 1993](#)). This difference is readily displayed in courtship, where women typically express desire through body movement, presentation, and gesture, whereas men pursue a more direct approach ( [Perper, 1985](#)). The manner of expression is interpreted through the character structure, largely genetically determined. The robust youngster who enjoys taking a chance is less likely to be influenced by sanctions against sexual activity than is the shy, inhibited child. Environmental forces that affect sexual development are many and varied and include such “nonsexual” elements as the presence of parents who are able to spend time with the child and who relate with understanding and warmth.

This chapter begins with an overview of the biological and genetic aspects of human sexual development and continues with a description of environmental influences: historical, sociocultural, and family. This is followed by a detailed description of erotic development through infancy, preschool, school age, and adolescence. The chapter concludes with some thoughts about the future and a possible end to the split between what children observe and what they are told about sexuality.

The use of the terms *sexual* and *erotic* may be confusing to the reader. In general, *sexual* refers to the objective and instrumental aspects of possessing and using sexual equipment, such as the genitals, whereas *erotic* refers to fantasies, perceptions, and feelings, such as desire. Thus a person may respond erotically to a pornographic video but sexually when prescribed a medication for impotence. Clearly the two terms overlap, and it is unusual for one to come into play without the other.

### BIOLOGICAL INFLUENCES

Researchers are just beginning to address the effect of early experience on the mapping of the sensory cortex. It is possible that early genital stimulation, be it diaper change or sexual molestation, permanently affects sexual responsiveness through recruitment of neurons and enhancement of neuronal function. In primates, peripheral stimulation can expand the corresponding areas of the somatosensory cortex, and the process includes reorganization of subcortical structures, including the brain stem and thalamus ( [Jones, 2000](#)). Stimulation evokes strong neuronal activity, producing long-lasting changes in synaptic function, learning, and memory ( [Malinow et al., 2000](#)). Therefore, child sex play could protect the sexual response against later adversity such as rejection by a lover or loss of a job. Too much stimulation could eroticize the child, leading to punishment or repeated rejection by adults such as foster parents. Based on this, it is important to understand the scope and effect of normative sexual experiences.

#### Hormonal Influences

Androgen is produced by the embryonic testes. Androgen differentiates the male external genitalia from the primordial female apparatus ( [Wilson et al., 1981](#)). If the embryonic testes are removed before the eighth week of gestation, the embryo develops as a female ( [Jost, 1953](#)). In addition, testosterone alters the migration of neurons in a manner that reorganizes the hypothalamus, preoptic area, corpus callosum, planum temporale, cerebellum, and amygdala. These changes to the brain occur between the 6th and the 12th week of gestation and are permanent, affecting emotional, behavioral, and cognitive function across the span of life ( [Rubinow and Schmidt, 1996](#)).

Testosterone-enhanced male brains become larger and heavier and have greater dendritic arborization. In boys, a nucleus of the preoptic area of the hypothalamus grows two and a half times larger, with twice the number of neurons than in girls. This nucleus is likely involved in sexual orientation and gender identity formation. The influence of estrogen on the fetal brain is poorly understood but likely includes protecting neurons, stimulating nerve growth, and enhancing neural connections ( [Seeman, 1997](#)). Although estrogen is a female hormone, a relatively small quantity may be essential for male brain development in areas such as those responsible for the coordination of male sexual behavior. In addition, estrogen functions as a neural growth factor with important influences on the development, survival, plasticity, and regeneration of the brain ( [Toran-Allerand et al., 1999](#)).

Right hemisphere development is slower in the male fetus, and this promotes greater left hemispheric specialization ( [Geschwind, 1983](#)). A number of sex-specific changes in function are the result. These include (a) language organized on the left side of the brain in men and on both sides of the brain in women; (b) increased verbal and affective communication skills in women; (c) greater logical, analytic, and visual-spatial ability in men; and (d) sex-specific repertoires of sexual and nonsexual behaviors ( [Tiefer and Krings, 1995](#)). Direct effects of testosterone in men include greater activity and aggression, with more intense erotic thoughts, feelings, and sexual behaviors. In the past, high levels of testosterone were thought to promote impulsive aggression and social maladjustment in men. More recent work indicates an association instead with social dominance and success ( [Schaal et al., 1996](#)).

At birth, testosterone levels are elevated in boys, and estradiol levels are elevated in both boys and girls. The robust erotic interest found in 2- to 4-year-old children may be due to the relatively high levels of hormones that persist during that period. From 5 years of age until prepuberty, hormone levels are relatively low, which may explain the diminished, although still significant, erotic interest observed during those years.

#### PUBERTY

Normal puberty begins between 8 and 14 years of age in girls and between 9 and 14 years of age in boys. The first sign of approaching pubescence is the adrenarche at approximately 8 years of age that generates an elevation of adrenal androgens, estradiol, thyrotropin, and cortisol. Adrenal androgens increase in girls, but to levels that are lower than those achieved by boys. Hormonal changes influence the brain, causing an increase in the size of the amygdala in boys and an increase in hippocampal volume in girls ([Giedd et al., 1997](#)). When the adrenarche commences as early as 2 to 6 years of age, as it sometimes does, it is associated with increased anxiety, depression, and behavior problems ([Dorn et al., 1999](#); [Rieder and Coupey, 1999](#)).

Girls enter puberty approximately 2 years earlier than boys, and they take 3 or 4 years to complete the process. Boys enter puberty later than girls, and they complete it in 4 or 5 years. Boys attain a testosterone level that is eight times higher than that reached by girls ([Udry et al., 1986](#)). This provokes an avid interest in sex and hair-trigger penile erections ([Udry et al., 1985](#); [Udry and Billy, 1987](#)). Girls experience a dramatic increase in gonadotropins, growth hormone, and estrogen, and a lesser increase in testosterone ([Fitschen and Clayton, 1965](#); [McCandless, 1960](#)). Girls experience a lag in progesterone secretion in the first 2 years after menarche and often have irregular periods during this time. Hormones exert a weak effect on sexual behavior in girls, but a stronger effect on motivation ([Smith et al., 1985](#)).

Leptin is a recently discovered hormone that resides in fat cells and has an important role in controlling body fat by regulating appetite, body energy, metabolic rate, and the autonomic nervous system. When the prepubescent child puts on weight, leptin increases, and the increase signals the brain that the body is ready to mature. Leptin continues to increase in girls in the postpubescent years, paralleling a continued increase in body fat. In adolescent boys, leptin levels off and then decreases. This is because boys accumulate more muscle and less fat compared with girls but also because testosterone directly suppresses leptin synthesis ([Kiess et al., 1999](#)).

## Identity, Role, and Orientation

*Gender identity* is the primary identification a person establishes with one sex. *Gender role*, on the other hand, describes culturally underwritten behavior that differentiates men from women, such as game and work preferences, social aggression, and courtship and sexual behavior. *Sexual orientation* describes the individual's erotic preference for a particular sex. Individuals are not necessarily the same in all three domains. Variation from "all maleness" or "all femaleness" is not uncommon and does not necessarily connote a pathologic process.

### GENDER IDENTITY

The human infant is born with a bias toward a certain gender identity, one that usually, but not always, matches the anatomic sex. By 2 to 3 years of age, children learn to identify themselves as boys or girls on the basis of things like hair or clothing. Gender identity develops before most children appreciate sex differences in genitals ([Bem, 1989](#); [Goldman and Goldman, 1982](#); [Rutter, 1980](#)). Girls learn about genitals and genital differences sooner than boys but do not understand the association with sexual function ([Volbert, 2000](#)).

In the past, gender identity was thought to be determined by the assignment of sex and the parents' habit of treating boys like boys and girls like girls ([Money and Tucker, 1975](#)). Although gender identity was viewed as somewhat malleable during the first 2 years of life, serious psychological disturbances were thought to result if sex reassignment was attempted after 3 years of age. Under the rubric "the earlier the better," assignment often was made in early infancy, often followed by several years of corrective surgical procedures. These assumptions are now being questioned, since [Diamond \(1996\)](#) and others described cases of early sex reassignment in which the assigned identity never seemed to fit. One patient, originally a boy, was reared as a girl and was never told of his biological sex. After years of secret discomfort, he finally sought reassignment as a male, saying that he had never felt like a female ([Diamond et al., 1996](#)). The best-fit explanation would be that fetal testosterone had caused his brain to develop as a male. After surgical removal of the testes in infancy, the male brain continued to determine his gender identity.

What are physicians to do when faced with ambiguous genitalia? Although the critical studies have not yet been done, physicians should seriously consider waiting until the child is old enough to (a) demonstrate definitive role modeling and (b) state a preference for being male or female. Children's preferences seem to be reliable indicators of the maleness or femaleness of the brain, but preferences may not be reliably expressed until 4 to 6 years of age. Therefore, infants with compromised or ambiguous genitals should be allowed to be "neither or both" until at least the preschool years. There is no evidence that holding the door open damages children, although strong parental reactions can be harmful. An additional consideration is the difficulty in constructing a functional phallus compared with a functional vagina.

An additional issue is the immutability of gender identity. Gender identity usually is constant throughout life, but there are instances in which people have successfully changed their gender identity, with or without genital modification ([Sagarin, 1975](#)). Precipitants have included change in status such as trauma, divorce, an appearance more like that of the other sex, the appearance of other-sex characteristics at puberty, and the perception of the other sex as better off or more powerful. Changes in gender identity may occur more frequently in the future as the culture comes to tolerate, or value, sexual diversity.

### GENDER ROLE

Gender role refers to a collection of attitudes and behaviors that are typically male or typically female. These include play and work preferences, friendships, extracurricular interests, and courting patterns. Gender role is consistent across cultures in that the superior strength of men makes them the more successful hunters and guardians of the territory, whereas women are more patient and proficient at repetitive activities such as planting, gathering, and nurturing the family ([Ford and Beach, 1951](#)). Men almost always are dominant and are more likely to be jealous and possessive, whereas women are less aggressive and more likely to accept male dominance for the protection it affords ([Harlow and Harlow, 1962](#); [Piacente, 1986](#); [Symons, 1979](#)).

Gender roles can be set as early as age 3 years, but it can also remain malleable until age 5, especially in girls ([Levy et al., 1995](#)). However, by the time children enter kindergarten, virtually all have adopted a set of gender-specific rules and expectations. Boys gravitate toward action, whereas girls choose social and nurturing activities. When angry, girls are more likely to attack verbally, whereas boys are more likely to lash out physically. Gender roles are enforced by peers who tease and ostracize those who do not fit the stereotype. Teasing is most severe for effeminate boys and less severe for girls who act like boys. Teasing continues and intensifies in adolescence.

Prenatal hormones influence children's choice of toys and pattern of play. Girls with congenital adrenal hyperplasia are exposed to increased androgens before and after birth. During childhood, they are not interested in doll play but prefer roughhouse play outdoors. They report few fantasies of marriage, pregnancy, or motherhood ([Ehrhardt and Baker, 1974](#)). Boys exposed to prenatal antiandrogens display lower energy levels, less aggression, and less heterosexual activity in adolescence, whereas girls exposed to antiandrogens appear more traditionally feminine than other girls ([Ehrhardt and Meyer-Bahlberg, 1981](#)).

### SEXUAL ORIENTATION

Sexual orientation refers to a person's overall sexual responsiveness to men or women ([Meyer-Bahlberg, 1993](#)). Sexual orientation has four components: (a) imagery (e.g., daydreams, masturbation fantasies); (b) use of erotica, such as magazines; (c) erotic attraction; and (d) actual partner experience. Homosexuality is not a disorder of gender identity. Homosexuality is an orientation that, once adopted, commonly is constant throughout life. Approximately 4% of men and 1% to 2% of women are homosexual ([Bell et al., 1981](#)). A smaller percentage describes themselves as bisexuals. Other cultures reveal a similar percentage of homosexuals. There are societies where men are at first exclusively homosexual and then convert to become primarily heterosexual ([Herdt, 1981](#)). Gay (homosexual) men may or may not be effeminate. As a group, they differ from the rest of the population only in being better educated and somewhat less devoutly religious ([Bell et al., 1981](#)).

Adult gay men describe themselves as having felt "different" since early childhood. Homosexual youth usually experience homoerotic fantasies in early adolescence, and this can precipitate identity conflict ([Cass, 1984](#); [Yates, 1983](#)). Heterosexual fantasies also are experienced, and this generates bewilderment ([Bell et al., 1981](#)). During early adolescence, most engage in activities such as manual stimulation with the same and with the other sex. Girls become sexually active at an older age than boys and are more likely to engage in concurrent or sequential heterosexual intercourse ([Rosario et al., 1996](#)). Vacillation, confusion, and emotional upset often persist until a clear homosexual identity is established in early adult life. Gay youth account for almost a third of all adolescent suicides ([Remafedi, 1987](#)).

Bisexual men tend to be more conflicted, alienated, and depressed than gay men. This could be related to greater biological discordance or to greater difficulty in defining an identity in a society that specifies people as only male or only female. Conversely, those individuals who are conflicted may be less able to define themselves as male or female ([Lock and Steiner, 1999](#); [Yates, 2000](#)).

Lesbian women report feeling less feminine and less beautiful during childhood. They also report having imagined that they were male, preferring to play boys' games, and being called a tomboy. However, many women who are not lesbian also recall these experiences ([Bell et al., 1981](#); [Whitam and Zent, 1984](#)). Compared



with gay men, lesbian women are more flexible and less conflicted about adopting a homosexual role and identity. Lesbian adolescents are less likely to be rejected by peers and they have more social support available. It is not uncommon for girls to experiment with female peers and for women who were heterosexual during marriage to enter a homosexual relationship after divorce. More women than men claim to be bisexual.

Cross-cultural studies support the importance of biological forces in the genesis of homosexuality. Behavioral prodromata of male homosexuality (e.g., interest in girls' toys, cross-dressing) exist in other cultures studied regardless of how the culture views homosexuality. These prodromata first occur at approximately the same age and are remarkably persistent thereafter ([Green, 1978](#); [Pillard and Bailey, 1995](#)). Primates and domestic animals often engage in same-sex sexual activity ([Adler, 1997](#)). Homosexual relationships sometimes help the animal survive. In some species, females that engage in sexual activity together are more willing to share food, to forage together, and to groom each other.

Prenatal exposure to increases in estrogenic or androgenic hormones can exert a life-long influence on attitudes, thoughts, and behaviors ([Money, 1975](#)). Women with congenital adrenal hyperplasia not treated before 8 years of age present a feminine gender identity in consonance with the sex of assignment but a masculine pattern of arousal in that they quickly respond to visual and narrative erotic stimuli, experiencing a strong sexual appetite that is localized in the genitals and that leads to masturbation or the pursuit of intercourse with a casual partner ([Ehrhardt et al., 1968](#); [Money, 1965](#)). Forty-eight percent have homosexual dreams or fantasies, and 18% have had homosexual experiences. Women who were treated earlier in childhood demonstrate an increased likelihood of becoming bisexual ([Money and Schwartz, 1977](#)).

There is strong evidence for a genetic component to homosexuality. The concordance for homosexuality is 52% among monozygotic twins, compared with 22% among dizygotic twins ([Bailey and Pillard, 1991](#)). Monozygotic female twins demonstrate a 48% concordance, whereas dizygotic twins demonstrate only a 16% concordance for homosexuality ([Bailey et al., 1993](#)). A family pedigree study by [Hamer et al. \(1993\)](#) demonstrated that nonsibling increases in homosexual orientation occur mainly in male relatives on the mother's side of the family. This suggests transmission through the sex chromosome. Indeed, researchers have correlated homosexuality with the inheritance of five polymorphous markers at the tip of the long arm of the X chromosome, in the Xq28 region ([Hamer et al., 1993](#); [Turner, 1995](#)). However, more than one gene is likely to be involved because a significant number of monozygotic twins are discordant for homosexuality ([Bancroft, 1994](#); [Meyer-Bahlburg, 1993](#)). The genetic basis for homosexuality is an important reason why children adopted by gay couples are no more likely than the rest of the population to become gay.

Genes could influence sexuality by changing the organization and structure of the brain or by affecting the production of hormones and transmitters. Minute changes in hormone balance could alter structure and subtly recast attitudes, thoughts, emotions, and behaviors. For instance, testosterone increases and ovarian hormones decrease the development of the corpus callosum, which is larger in men than in women ([Fitch and Denenberg, 1998](#)). This could differentially affect information processing, emotional ambivalence, and anxiety ([Leavengood and Weekes, 2000](#)). Two interrelated, sexually dimorphic nuclei, the stria terminalis and the medial amygdaloid nucleus, are larger and have denser projections in men. This system influences sexually dimorphic behaviors such as aggression as well as nonsexually dimorphic functions such as social recognition memory ([de Vries and Miller, 1998](#)). The recently discovered human vomeronasal system responds in sex-specific fashion to smell by changing behaviors, adjusting the autonomic nervous system response, and even instigating the release of gonadotropins from the pituitary gland ([Monti-Bloch et al., 1998](#)). Clearly, brain dimorphism is enormously complex and has far-reaching effects on cognition, emotion, and behavior.

A boy with a female-configured brain would possess a strong innate bias toward homosexuality. Whether that person would become gay would depend on the strength of the bias (degree of femaleness in the brain), the boy's internal discomfort with becoming a homosexual, and his experience with homophobia in the environment.

Steroid hormones have been found in localized areas of the brain. These hormones are called *neurosteroids* because they are produced by neurons in the same way that the gonads and the adrenals generate steroids, but independently of the gonads and the adrenals ([Roselli, 1995](#)). Neurosteroids exert their effects locally; they could organize certain areas of the fetal brain toward maleness or femaleness or could affect postnatal behavior directly ([Kabbadj et al., 1993](#)). The existence of this third, independent hormonal pathway could explain why some very masculine-appearing and -acting men are gay ([Yates, 2000](#)).

Sexual orientation in men is not correlated with androgen levels in adolescence or adulthood ([Green, 1978](#)). However, approximately one-third of lesbians show elevated levels of testosterone ([Gartrell et al., 1977](#)), although the levels remain well below the range of normal for men ([Meyer-Bahlberg, 1982](#)). [Dorner et al. \(1991\)](#) suggest that these findings are due to prenatal stress and a congenital deficiency of enzymes involved in steroid synthesis. Adrenocorticotropic hormone administration significantly increases production of the cortisol precursor, 21-deoxycortisol, in gay men and lesbian women compared with nonhomosexual subjects. A similar increase occurs in the mothers of gay men.

Homosexual men are far more likely than other men to be born later on in the birth order and to have more older male siblings ([Blanchard and Bogaert, 1996](#)). Conversely, parents who produce many sons are more likely to produce extremely effeminate or homosexual sons ([Pillard and Weinrich, 1986](#)). Several explanations are possible, one of which is based on immunology. Long before a mother gives birth to a boy, fetal cells are circulating in her bloodstream. These persist as long as 27 years after the birth of the child ([Bianchi et al., 1996](#)). The presence of male cells could provoke an immune reaction against Y chromosomal material. Theoretically, this could lower prenatal brain testosterone in later-born boys, thus affecting the structure and function of the brain. Later-born male infants and those with many older male siblings are known to have lower levels of testosterone than first-born male children. Siblings born close together have lower testosterone levels than those born 4 or more years apart. The longer birth interval would allow the number of fetal cells in the maternal circulation to diminish.

## HISTORICAL INFLUENCES

Before the 16th century, families in Britain and Western Europe did not enjoy privacy as we know it today ([Jackson, 1993](#)). Family members, employees, and servants worked, ate, and slept under one roof, usually in the same room. Children were seen as small, socially inferior adults. They were expected to contribute to the family economy and were held legally responsible for their actions. They freely observed the sexual acts of adults and heard open discussion of licit and illicit acts. They engaged in masturbation and sex play with other youngsters. Sexual crimes involving children did occur, but the effect on children's emotional well-being was not an issue. Sexual development was not regarded as problematic.

From the 16th to the 19th century, families began to live apart from the workplace. The single great room had been divided by walls, so that sleeping rooms were apart from eating and work areas. Sexual relations occurred behind closed doors. Servants had their own quarters. With the advent of Puritanism, sexuality was identified with sinfulness, except in the narrow context of procreation. Sexual relations became more and more secretive. Conversation was sanitized and sexual matters were discussed only in private. Parents tried to raise modest, notably asexual children. Children were told to cover the body at all times and not to look at or touch other potentially sexual objects ([Elias, 1978](#)). Children became ashamed of the body and of such common behaviors as blowing the nose without a handkerchief, burping, and toileting ([Whitehurst, 1971](#)). Children could not refer to "legs" or to "bellies" but were to use the correct terms, which were *nether limbs* and *lower portion*. The dinner table stood on "limbs" rather than "legs," and children could never ask for slices from the "breast" of the chicken. Girls and women were told to avoid strong foods such as corned beef and blood pudding, but boys and men could eat anything they wished.

In the 19th century, Victorian parents were tormented by the fear that their children would masturbate because "self-abuse" was thought to cause insanity, lethargy, tuberculosis, syphilis, eventual impotence or sterility, deformed children, and epilepsy. Spike-lined rings that fit around the penis, steel and leather jackets that enveloped the entire torso, electric shocks, and tight bandages were used to prevent children from pleasuring themselves. Some parents would tie their children's extremities to each post of a four-poster bed at night. Children who masturbated in spite of these measures might have the nerves to the penis severed or the clitoris cauterized with a white-hot iron. Clitorectomy by scalpel or by scissor was a common treatment for recalcitrant masturbation in young girls ([Schwartz, 1973](#)). Among the many civilizations that practice clitorectomy, ours is the only one that has used the excision of the clitoris as a cure for masturbation ([Huelsman, 1976](#)).

In the mid-19th century, under the influence of literary romanticism, attitudes toward children and sexuality started to soften. Liberal, well-educated parents began to regard children as innocent, asexual, and in need of protection ([Jackson, 1993](#)). This idea slowly spread throughout society, although it has never completely replaced the concept of the demonic child among fundamentalist groups. Because children were vulnerable, the family's responsibility was to protect them against danger, especially threats to their "natural" state of innocence.

### 20th Century

The decades from 1930 to 1950 witnessed a marked decline in the severity of the sanctions against sex play and masturbation in childhood ([Wolfenstein, 1953](#)). Parents began to caution, dissuade, or distract rather than punish or scold youngsters for sexual activities or interests ([Finkelhor, 1980a](#)). In the 1950s, a new theme



emerged: Parents were told not to worry about children's sexual interests. It was perfectly normal for children to explore the body, and once they learned about the body, they would no longer be curious. Some parents could accept sex play as "probably not abnormal," but it remained a source of anxiety. In spite of greater comfort, parents were unable to talk to children about topics such as masturbation and intercourse. Parents in upper socioeconomic classes began to name the boy's penis but continued to avoid naming the girl's genitals. The trend toward increasing acceptance of childhood sexuality continued in the 1960s and 1970s with the sexual revolution.

The tendency toward acceptance abruptly reversed in the late 1970s when concern about child sexual abuse surfaced and evolved into a social movement. The need to protect children from sexual abuse soon extended to protecting any child from any sexual experience. Sex abuse prevention programs espousing "stranger danger" and "bad touch" were presented in grade schools across the nation without prior needs assessment, outcome studies, or data on the effect on normal development. Research studies throughout the 1980s and 1990s were unable to show that the programs prevented abuse, but a recent retrospective study does suggest less sexual abuse among adults who recall exposure to a prevention program while in school. The study did not find adverse effects on erotic development in terms of diminished sexual satisfaction or avoidance of sex in adult female students who recalled exposure to prevention programs ([Gibson and Leitenberg, 2000](#)). A prospective study may or may not confirm these findings.

During the last three decades of the 20th century, while concern about sexual abuse was at a peak, the amount of erotic material in the media increased dramatically. While watching prime time television, children viewed bedroom scenes and learned about the president's preference for oral sex. MTV (music television) aired "booty-bumping" videos of stars singing about "banging on the bathroom floor." Children could read articles on how to achieve a better orgasm or the latest technique to please a partner in popular magazines about the house. Not uncommonly, a child in a single-parent household would wake up to find an extra person at breakfast or in the parent's bed. Needless to say, these events have continued to occur in the first part of the 21st century.

### Dissonance

Of most concern is the fact that children largely have been left to decipher contradictory messages in the media, at home, and at school on their own. No single standard is underwritten as correct ([Benedict, 1950](#)), but the standard for children clearly differs from the standard for adults. In Victorian times, the task was simpler: "Good" (asexual) girls grew to be "good" women who tolerated intercourse to bear children. "Good" little boys grew to become "good" men who engaged in sex because it was necessary to make babies.

Over the past two decades, it has become easier for children to acquire extensive sexual knowledge ([Volbert, 2000](#)) but more difficult for them to integrate sexual material into an understanding of self and other. Integration is compromised because most parents avoid talking to children about their concerns, are embarrassed by their questions, or categorize sex as bad, dangerous, or a reason for not being liked ([Gagnon and Simon, 1973](#)). Exposure to sexual material in the media, in conjunction with parental reluctance to discuss it could create in children the sense that sexuality exists in a compartmentalized domain apart from life experience ([Fisher, 1989](#); [Yates, 1993](#)). Unless integration is achieved later on, individuals might come to prefer visually and emotionally charged, but impersonal, sexual practices such as Web sex, lap dancing, or masturbation while viewing pornographic videos.

## SOCIOCULTURAL INFLUENCES

Child-rearing practices that are not directly related to sex can influence sexual development. In the early 20th century, especially in rural areas, children were valued because of the work they did and the security they would provide as the parents aged. By mid-century, more families had moved to towns and cities, making children less useful and more of a financial burden. Social Security allowed aging parents to be less dependent on children. The invention of the latex condom and the diaphragm enabled women to bear fewer children. Families shrank as women joined the workforce. The rationale for having children changed: Children were valued for the emotional warmth and sense of purpose they gave the family. They were no longer expected to work or to do gainful chores to support the family economy. In the affluent family, children became the centerpiece: cuddled, protected, indulged, and closely monitored instead of being allowed to roam freely in the neighborhood. However, as they grew older, they were expected to readjust, making their mark in school and eventually in a business or profession. The effect of these changes in child rearing is not known, but ample early nurturance would be expected to change sexual development for the better but make separation/individuation more difficult owing to the highly gratifying early relationship to the parents.

In the last decades of the 20th century, children were treasured as never before but also were expected to adapt to frequent placement in day care apart from the parents as mothers moved into the workforce. This increased the pressure on children to become independent at the same time that being dependent was encouraged at home. There have been no studies that examine the long-range effect of these practices, but the strong emphasis on self-sufficiency could be expected to engender anxiety about dependence and ambivalence about intimate relationships ([Yates, 1991a](#)). Although this might not affect the sexual response, it might diminish the ability to sustain an intimate relationship over time. Current high rates of sequential cohabitation and divorce and the increased number of adults who choose to live alone may, in part, be related.

### Family Influences

In Western culture, parents tend to assume that youngsters will be overstimulated if they are allowed to see parents and others nude. Cross-cultural research does not support this concept ([Ford and Beach, 1951](#)). Indeed, a recent longitudinal study from the University of California, Los Angeles found no harmful effects from exposure to nudity or the chance observation of adult sexual activity ([Okami et al., 1998](#)). Young children enjoy being naked and watching parents and siblings go without clothes. When nudity is allowed in the home, children display higher self-esteem, and less discomfort with body contact ([Lewis and Janda, 1988](#)). However, family nudity also increases children's normative sexual interests and activities ([Friedrich et al., 1991](#)), and this, in itself, may be enough to increase parents' anxiety.

In Western society, parents are concerned about letting the child, especially boys ([Roberts et al., 1978](#)), have too much body contact, and they also worry about body contact between siblings. From the time children are 3 or 4 years of age, parents restrict how, where, and when children can touch them ([Blackman, 1980](#); [Lewis, 1965](#); [Martinson, 1991](#)). Asian societies are not so concerned; children are allowed access to the body, the bedroom, and often the bed ([Barry and Paxman, 1971](#); [Frayser, 1994](#)). In Western society, body concerns are translated into a vast, elaborate set of containers for infants and toddlers such as baby carriers, car seats, prams, and playpens. While we do not know how encapsulation affects children's erotic development, we do know that when parents enjoy touching and bathing children, children become more comfortable with their bodies and with talking about sex ([Rosenfeld et al., 1987](#)).

Parents' anxiety about sexuality is mirrored in their need to restrict infants from sharing the bed with them, even though bed sharing facilitates suckling and allows the mother more time and better quality of sleep ([Mosko et al., 1996](#)). Parents worry that bed sharing will inhibit cognitive and emotional development, overstimulate through excessive body contact, and keep children from becoming independent ([Colarusso, 1994](#)). The research that exists indicates positive, rather than negative, effects of bed sharing, including better adjustment, greater self-esteem, and more affection and greater comfort with sexuality in adult life ([Lewis and Janda, 1988](#); [McKenna, 1997](#)).

### Research

In the last quarter of the 20th century, research on normal sexual development was seriously impeded by problems in funding or in receiving approval from Human Subjects Committees. Approval was not granted for studies that included asking youngsters about sexual interests and activities. Relatively little research has been published, making students and researchers dependent on studies completed decades ago. Improvements in research are not likely to occur unless the culture becomes less concerned with negative consequences and more concerned about normal development. The books and articles on which this chapter is based often are dated or imperfect, descriptive rather than sample based, and derived from sources other than peer-reviewed journals, or are journal articles that suffer from methodologic flaws. Materials include (a) child analytic case studies and journal articles published between 1930 and 1980; (b) a few well-designed older studies, such as those by Spitz in the 1930s; (c) longitudinal studies of normal development published in the 1970s and 1980s ([Chess and Fernandez, 1981](#); [Galenson and Roiphe, 1979](#)); (d) direct child interview studies by Goldman and Goldman in [1982](#) and by [Martinson in 1994](#); (e) parent or teacher descriptions of sexual behavior in groups of children; and (f) articles published since 1990 as indexed in medical and psychological databases. Reports that are heavily biased toward pathology are not included.

Human sexuality is surely as old as the human race. Although sexual activity is based on instinct, the sexual act is influenced by assumptions about sex handed down through generations, depictions of sexuality currently in the media, fear of venereal disease (especially the acquired immunodeficiency syndrome), the changing status and role of women, and medical achievements such as effective birth control and *in vitro* fertilization.

## EROTIC DEVELOPMENT

In children who are not unduly inhibited, there appears to be a developmental progression, commencing with the highly pleasurable sensations generated by the infant's transaction with the parents and continuing through (a) the differentiation and appreciation of the genitals; (b) the incorporation of the sexual parts in the concept of the body; (c) exhibitionism to "test" adult reactions; (d) mastery of a variety of self-elicited sensations; (e) expansion of erotic interest to include parents, siblings, and peers; and (f) the final integration of the genitals and genital function into the concept of the self. Erotic development is separate from, but related to, that of psychosexual development, which includes object relatedness. The concept that eroticism is separate from psychosexual development provides a better model to explain the heightened eroticism of sexually abused children ([Johnson, 1993](#); [Yates, 1982, 1991b](#)) and the well-developed object relationships of many sexually dysfunctional adults. Some of the factors that influence the process of eroticization are (a) hormones and neurotransmitters; (b) appearance and self-esteem; (c) resolution of early conflicts; (d) presence or absence of severe social and cultural sanctions; and (e) experiences such as skin-to-skin contact with parents, sex play, and observations of sexual interest and activity in others.

Erotic behavior in infants and children has been well documented ([Bakwin, 1973](#); [Borneman, 1990](#); [Kinsey et al., 1948, 1953](#); [Martinson, 1981](#)). An absolute lack of eroticism in infancy is remarkably rare and usually is secondary to a catastrophe such as severe birth injury or debilitating disease. However, a relative decrease in eroticism usually follows severe neglect, parental rejection, or frequent changes in placement.

When feeding time approaches and the infant senses the closeness of the breast, his or her activities suddenly become organized, predictable, and goal directed. The infant inhibits crying, closes the fists, opens the mouth, searches and thrusts toward the nipple, seizes it, and sucks vigorously. The infant's face is red, and his or her mood might best be described as eager and anxious. When the milk comes down, the fists relax, the eyes turn up and close, and the infant's body melts to conform to the curves of the mother. Within a few moments, the infant slides into a peaceful, pleasurable state, continuing to suck even when satiated, repetitively touching the nipple with the tongue although apparently asleep. The mother's scent, warmth, and closeness are part of this first, highly erotic experience.

At approximately the third month of life, some infants derive comfort and pleasure from sucking the thumb, but the relationship to the thumb lacks the erotic intensity of the relationship to the parent. Because thumb sucking does not occur in cultures where the breast is readily available to the infant, it would seem that this early diversion of erotic interest from the nipple to the thumb is related to the relative unavailability of the breast ([Sarlin, 1975](#)). Thumb sucking functions as an early step in the separation individuation process and is not associated with psychopathology.

By 3 or 4 months, infants clearly are intrigued and delighted by the washing and drying of the genitals. By 6 months, infants may eagerly grasp the genitals in anticipation of being diapered. Most boys begin to play with the penis at approximately 6 or 7 months, and girls begin to stimulate themselves at 10 or 11 months ([Bakwin, 1973](#)). Genital play differs from masturbation in that there is no intentioned progress toward orgasm. Parents tend to view early genital play as part of children's need to explore ([Rutter, 1971](#)).

Genital play at an early age is associated with health and good relationship to the parents rather than any pathologic process. Conversely, when the relationship with the parents is difficult or neglectful, genital play diminishes or disappears and the child is less likely to be healthy. [Spitz and Wolf \(1949\)](#) studied foundling home infants, mother–infant pairs in a prison nursery, and normal mother–infant dyads. The well-nurtured infants, without exception, enjoyed touching themselves, whereas the poorly nurtured infants, almost without exception, did not. More recent research supports this early observation ([Borneman, 1990](#); [Galenson and Roiphe, 1979](#); [Martinson, 1981](#)). However, self-stimulation is not a prerequisite for healthy development because there are healthy infants who have never stimulated themselves ([Kleeman, 1975](#)) and many others who began to do so but were discouraged by the parents.

When infants intentionally bring themselves to orgasm, this is called *masturbation*. Genital play should not be confused with masturbation. The change from genital play to masturbation is a gradual, discontinuous process that extends into the second or third year of life in many, but not all children ([Galenson and Roiphe, 1979](#)). Masturbating infants appear singularly intent and difficult to distract. They breathe rapidly, flush, grunt, and make rhythmic thrusts or, in the case of female infants, may grasp and rub a pillow or doll between the legs. When infants first begin to masturbate, they try to maintain body contact with the parent, but most parents discourage this. Contact seeking is soon replaced by the familiar averted eyes and inward gaze ([Galenson, 1993](#)). Once children have established a certain mode of masturbation, the format persists and is resistant to change ([Perper, 1985](#)). Masturbation is something the child does autonomously, an activity that usually is effective and pleasurable. The ability to do something that is independent of the parents can foster the process of separation and individuation ([Sarlin, 1975](#)).

Masturbation is less conflicted for infants than it is for parents in Western culture. Parents who had been able to accept genital play react strongly to masturbation, although they may not understand why they are so upset. [Martinson \(1991\)](#) analyzed studies from 1957 to 1985 on parent attitudes about children's sexuality and concluded that although many parents said that they thought masturbation was healthy, they experienced disgust when they witnessed their son or daughter masturbating.

The format for masturbation established in the second year of life tends to remain constant among boys, although it undergoes a further evolution among girls ([Galenson, 1974](#); [Galenson and Roiphe, 1976](#)). Girls often learn to use indirect techniques, using legs, thighs, toes, or rocking horses. Girls may cease masturbation completely in the second half of the second year or they may continue to masturbate without pleasure. Accompanying reactions include frustration, regression, fearfulness, loss of enthusiasm, sadness, and the displacement of activity to other parts of the body and to inanimate objects such as dolls and fuzzy toys. Some girls become erotically interested in the father, whereas others show an increase in hostile dependence on the mother. Frustration reactions seem to enhance girls' symbolic thought and internal complexity. However, the girls most severely affected constrict their imagination and retreat emotionally ([Roiphe and Galenson, 1973](#)). Boys show far less overt disturbance, often displacing their anxiety into physical activity.

### Anatomy

Anatomic differences have far-reaching import for psychosexual development ([Gadpaille, 1976](#); [Kestenberg, 1968](#)). Boys can easily identify a tangible, predictably pleasurable focus in the erect penis. The penis is visible, often has a name, and has a readily observable function in urination. Clothing provides a special aperture for the penis. Parents approve when boys use the hand to steady the penis during urination. Because of this, it seems easier for little boys to integrate their genitals into the body concept. Testosterone's influence on the fetal brain augments active, acquisitive striving, task orientation, pragmatism, goal directedness, and logical thought, all of which contribute to the sense of maleness.

In homes where nudity and body contact are limited, girls experience the genitals as something hidden that needs to be kept clean and covered. Direct contact between hand and genital is limited and usually involves toilet paper or a washcloth. Girls may confuse the genitals with the anus and assume that they are dirty or bad. Girls find it more difficult to integrate the genitals into the body concept when they are restricted from viewing the mother or adult women naked ([Schuhrke, 2000](#)). In the absence of easy, external, male genital eroticism, girls become more inwardly attentive and aware, more intuitive, expressive, and patient, but with less of the "male" pragmatism and goal directedness ([Galenson, 1974](#); [Kestenberg, 1968](#)).

Curiosity about genitals is quite common at approximately 2 years of age, when children have the opportunity to observe others nude. Interest in personal urination seems to be a prerequisite for developing interest in the bodies of parents, siblings, and peers ([Schuhrke, 2000](#)). Interest in parents' genitals surfaces before interest in siblings' genitals. This is likely due to the larger size and stranger appearance of adult genitals. Children often want to touch or explore parents' genitals, and most parents feel threatened by this. Curiosity about parental bodies persists until at least 6 years of age, although boys' curiosity about fathers' genitals diminishes and is displaced to some extent to the genitals of male peers.

### Preschool Eroticism

As youngsters grow older, peer relationships become more important. The focus shifts from genital play and masturbation to sex play. In an Irish community sample, one-half of all 3-year-old children were observed to masturbate or play with their genitals ([Fitzpatrick et al., 1995](#)). Genital play and masturbation help to define the sexual self, whereas sex play defines the relationship between the sexual self and others.

Infant observation studies ([Galenson, 1993](#); [Galenson and Roiphe, 1976](#); [Kleeman, 1975](#)) indicate that toward the beginning of the second year infants become interested in toilets, in observing others defecate, and in sensing their own bowel movements. This is called *anal eroticism*. At the same time, youngsters become more opinionated, stubborn, and negativistic. Continuing in the second and third year of life, toddlers enjoy watching themselves urinate and they may try to play with the urinary stream ([Galenson, 1993](#); [Rosenfeld et al., 1987](#)). They are curious about how others urinate, and they enjoy faucets and squirting devices. Children 2 to 4



years of age spontaneously engage in bathroom games. They may fondle each other's genitals as they bathe together or playfully attack each other with rubber bath toys. Some briefly experiment with smearing feces. At this age, boys and girls are especially fascinated with the genitals of boys ( [Gunderson et al., 1981](#) ), perhaps because they are larger, more accessible, and can change size. They are fascinated by father's genitals and the genitals of dogs ( [Martinson, 1981](#); [Spiro, 1958](#) ). Boys may rub the penis, so they can show off an erection or may ask a parent to rub the penis because it feels good. Girls are more likely to entice the parent indirectly by using the body as a whole: strutting, lifting the skirt, dancing provocatively, and giggling. They enjoy the genital contact involved in "playing horsie" on the parent's knee.

Parents commonly observe preschool children walking around nude, scratching the crotch, trying to look at others undressing or on the toilet, kissing nonfamily adults, showing sex parts to adults, touching breasts, or sitting with crotch exposed ( [Friedrich et al., 1991](#); [Kleeman, 1975](#); [Johnson, 1993](#) ). A reporting bias is likely because parents who report more child sexual behaviors tend to be better educated, earn more money, attend fewer religious services, and have more liberal attitudes about sex ( [Shafan, 1995](#) ).

Some 2- to 3-year-old girls insist that they have a penis and try to urinate standing up, or they hold sticks or toys protruding from the genital area. ( [Galenson and Roiphe, 1976](#) ). This represents interest in, and anxiety about, the penis rather a gender identity disorder. By the same token, boys may express the wish to grow breasts or to have babies ( [Edgcumbe, 1976](#) ). These behaviors tend to be evanescent.

Adults ordinarily cannot recall sexual and other happenings that occurred before 4 years of age, and many can remember nothing before 12 years of age, yet 20% of adults remember being sexually aroused before 6 years of age ( [Ryan, 2000](#) ). Sex play among preschool children usually is collaborative and accompanied by caressing, cuddling, kissing, hugging, sitting close together, feeling sorrow when separated, and giving each other gifts. Commonly noted formats involve looking at, touching, or rubbing the genitals. The play is noncoercive, spontaneous, and invigorating. When boys get together they may engage in games of "competitive pissing" ( [Borneman, 1990](#) ).

From approximately 4 years of age, children use themes, props, and role assignment in games such as "Doctor" and "Make a Baby." Play almost always is spontaneous, although it can also occur in response to seeing the parents having sex. Sexual activity regularly increases after children observe the erotic activities of adults, other children, and animals. This suggests that the play is part of a learning process that helps youngsters understand sexual events. After age 4 or 5 years, youngsters hide sexual activity and become less open to talking about sex to an adult. They have learned that sex is "dirty," and even if they continue active sex play, they will say that the genitals and the play are bad or nasty ( [Ames, 1966](#); [Pitcher and Prelinger, 1963](#); [Schuhrke, 2000](#) ). What causes this change in perception is not known, although parents' anxiety and inability to talk about sex likely contribute.

A common adult concern is that if children engage in sex play, this will lead to more and more inappropriate kinds of sexual activity. Yet in the sexually permissive kibbutzim, sex play follows a predictable course. Masturbation and peer play begins in infancy, becomes very intense in early childhood, but becomes less intense in the early school years ( [Shepher, 1971](#) ). Sexual shame appears at approximately 9 or 10 years of age, and at that time, the relations between sexes become strained. This tension disappears at ages 13 to 14 years, when a warm and friendly, but completely asexual, relationship ensues. People from the same peer group do not become sexually active with one another in adolescence, and they do not marry one another later on. This avoidance is completely voluntary.

### Naming the Genitals

Parents rarely say anything positive about children's genitals ( [Rosenfeld and Wasserman, 1993](#); [Yates, 1978](#) ), and they usually treat them as nonexistent or unimportant ( [Gadpaille, 1978](#) ). Many parents do not name the genitals at all, and this conveys a message. Others apply colloquial terms and a few teach children the correct names for the genitals ( [Fisher, 1989](#); [Fraleigh et al., 1991](#) ). Parents who do name the genitals are likely to be better educated, practice nudity in the home, and allow children to sleep in their bed.

Names assigned to boys' genitals are most often diminutive, such as "pee-pee," "dinky," "dickie," "peetie," "dood-dad," "froggie," "button," "twinkie," "flipper," "dippy-bird," "dingaling," "pickle," "tee-tee," "wienie," and "wormy-Willy" ( [Fraleigh et al., 1991](#); [Sanders and Robinson, 1979](#) ). Such inconsequential terms may defuse parental anxiety about children's sexuality. Rarely, parents assign a term that connotes worth or power, such as "family jewels" or "tally-whacker." How this affects children's self-perception is not known, but positive names are likely to have a positive effect on genital esteem ( [Yates, 1978](#) ).

Parents virtually never name girls' external genitals ( [Gagnon and Simon, 1973](#) ), although they may use a nonspecific term such as *vagina* for the entire area. Names assigned to girls' genitals are singularly nonspecific, such as "down there," "your privates," and "your bottom." When the term *vagina* is used, it usually refers to the entire external apparatus, including the anus. Even the occasional picturesque terms such as "bucket," "coochie-cooch," "garage," "snatch," "twat," "little-popo," "toto," and "tushie" ( [Fraleigh et al., 1991](#) ) are applied in nonspecific fashion. The lack of differentiation is reflected in the fact that most girls do not know what exists "down there" until at least 12 years of age. This lack of differentiation could affect sexual function later on ( [Yates, 1978](#) ).

The years from 3 to 6 have been called the *oedipal period* because erotic interests often are focused on the parent of the opposite sex. Some youngsters ask many questions about the body, intercourse, and reproduction ( [Robinson et al., 1991](#) ), whereas others seem unconcerned. Despite apparent sophistication, children's concepts of sexuality remain based on earlier constructs; many believe that the infant is born by cutting the mother's stomach open or that the infant exits through the mother's anus ( [Goldman and Goldman, 1982](#) ). Boys may express an overt wish to displace the father and marry or sleep with the mother. Girls become highly erotic in the context of the relationship to the father. However, they are less direct than boys and instead may attempt to achieve a sense of completion and specialness through the relationship ( [Roiphe and Galenson, 1973](#) ).

In a large study based on parents' observations of sexual activity in their children, [Friedrich et al. \(1991, 1992\)](#) associated older children's sexual activity with behavioral problems. This is likely the case in that older children who openly express their sexuality care little about rules or the feelings of others, symptoms that suggest a behavior disorder. Children who are open about their activities are, of course, the ones in whom sexual activity is observed.

### Father/s Role

When both parents cooperate in rearing children, children perceive the parents in a more balanced and realistic manner. However, in many homes, fathers are considerably less involved than mothers ( [Hochschild, 1989](#) ). Yet fathers are more likely than mothers to play with young children ( [Flerx et al., 1976](#) ), and the play is far more stimulating and unconventional than mothers play ( [Ablin, 1971](#); [Lamb, 1980](#) ).

Father involvement greatly facilitates formation of gender identity and role. Fathers who are firm, warm, and actively involved are most likely to produce masculine sons and feminine daughters ( [Spieler, 1984](#) ). When fathers are absent, sons appear less masculine ( [Mead and Rekers, 1979](#) ) and form a less successful heterosexual adjustment in adult life ( [Cinch, 1949](#) ). In general, father absence has a more profoundly debilitating effect on boys when it occurs early and lasts for a long time ( [Hetherington, 1971](#) ).

Girls learn to be feminine through a positive relationship with a masculine father ( [Spieler, 1984](#) ). Fathers who validate their daughters' sexual attractiveness enable them to accept themselves as both feminine and sexual. Conversely, girls reared without fathers have difficulty learning the feminine role; they become sexually active earlier and tend to have more partners ( [Hetherington, 1971, 1972](#) ). Yet, overall, father absence appears less damaging to the psychosexual development of girls than boys.

### School-Aged Children

A number of retrospective studies have asked university students about their recall of sex play during childhood. In one study, 85% of the women recalled sex games and 44% recalled sex games involving boys ( [Lamb and Coakley, 1993](#) ). Most remembered feeling sexually aroused or excited at the time. More than half the women indicated that no adult was aware of the sex play, and it was not subsequently reported. Most of the play involved exposing or touching the genitals. Insertion of objects in the vagina and oral contact were distinctly unusual. Three other studies ( [Bauserman and Davis, 1996](#); [Haugaard and Tilly, 1988](#); [Reynolds, 1997](#) ) confirmed that most young adult students can recall early sex play and that they view the play in a positive light as pleasurable and exciting. Negative responses are associated with feeling coerced, playing with a person of the same sex or with someone other than a friend, and with being discovered by an adult ( [Reynolds, 1997](#) ).

Modesty becomes an issue for girls between 4 and 6 years of age and boys between 5 and 8 years ( [Rosenfeld et al., 1984](#); [Rosenfeld and Wasserman, 1993](#) ). Children with older siblings become modest sooner, perhaps because of role modeling or because they are more likely to be teased for being naked. Soon after they



enter school, boys and girls learn to toilet separately and dress apart. By fourth or fifth grade, children are extremely embarrassed if their underwear shows at all. Parents also become more modest as their children grow older. It is uncommon for mothers to bathe with sons who are older than 8 years of age or for fathers to bathe or shower with daughters older than 9 years of age ([Rosenfeld et al., 1987](#)).

Modesty or shame may affect other dimensions of children's lives. A study of 8- and 9-year-old boys and girls ([Rademakers et al., 2000](#)) documented the hunger for greater physical intimacy such as cuddling with the parents. This simply did not happen for many children, and others managed to cuddle only with toys or animals. Parents seemed unaware of children's need for greater physical contact.

When children start regular school, sexual activity drops sharply, and the activity that does occur is quite secretive. This may be due to greater structure; more attention focused on schoolwork; fear of apprehension; feelings of shame, guilt, and internalization of the process ([Tanqney and Fisher, 1995](#)). Boys who have been sexually active tend to remain more active than other boys but less active than they were earlier. In part, this is because very few girls are willing to participate—one to every seven boys ([Broderick and Fowler, 1961](#); [Kinsey et al., 1953](#)).

School-aged children continue to be interested in sex but may displace the interest to “doodling” breasts or genitals on human figures, sneaking *Cosmopolitan* or *Playboy* to read, telling dirty jokes, or going to great lengths to watch animals breed ([Johnson, 1993](#)). The concept of “sex is dirty” is joined by “sex feels good.” By 9 years of age, one-third of boys (but not girls) see enjoyment as a reason why adults engage in sexual activity ([Conn, 1948](#); [Goldman and Goldman, 1982](#)).

Sex games are likely based on group consensus: “Spin the bottle” and “strip poker” are examples. Others are modeled after television contests like *Wheel of Fortune*. In “truth, dare, and consequences,” the person who is “it” is asked an ambiguous question such as “Who is the smartest boy in class?” Because no one really knows the right answer and group members are certain to disagree, the child who is “it” must pay the consequences, which may mean stripping, spreading the legs, or touching another child's genitals. Games are sexually exciting; they provide a forum where the body is accepted and sexual feelings are validated.

When children are discovered while engaging in sex play, they can suffer intense personal shame and public humiliation ([Reynolds, 1997](#)). They can be ostracized by neighbors and teased mercilessly by peers. Boys, especially, may be viewed as perpetrators, such as 6-year-old Jonathan Prevette, who kissed a classmate on the cheek and was subsequently suspended from school and charged with sexual harassment ([Nossiter, 1996](#)). Some youngsters are devastated, and others rapidly adjust. This is symptomatic of society's confusion about what constitutes sexual abuse. Children who play sex games together, parents who go nude in front of children, and fathers who kiss older children can be judged abusive ([Haugaard and Reppucci, 1988](#)).

Teasing with sexual themes is ubiquitous in the schoolyard and hallway. Taunts of “fag,” “les,” or “teenie wienie” take an emotional toll. On the playground, kissing is most often an aggressive act calculated to embarrass or drive away the other person. The fact that virtually all children get teased in one way or another has a protective effect, and learning to deal with teasing can strengthen defenses. Yet vulnerable children suffer greatly, and some are never able to contend with taunts. In the year or two before puberty, hormones rise, sexual and aggressive impulses surface, and peer groups become sharply divided according to gender. Ridicule and taunting become more frequent and brutal. Certain children become scapegoats. They can be systematically degraded or humiliated in front of others by pulling pants down to expose the genitals or ripping the shirt open to reveal the breasts ([Ryan, 2000](#)).

In spite of the taunts, romanticism is alive and well because almost all school-aged children expect and want to get married. Between 10 and 12 years of age, most children say they have a sweetheart, and by 12 years, five of six children claim to have been kissed by a peer ([Broderick and Fowler, 1961](#)).

## Sex Education

Parents often avoid or choose not to sex educate. In one retrospective study, 36% of the respondents said that their parents had never discussed sex, 17% that they had discussed it once, and 41% that it was discussed “very little but intermittently” ([Ryan, 2000](#)). Topics such as masturbation, homosexuality, and sexual pleasure virtually are never brought up, but these are the topics that youngsters are most concerned about and least likely to receive information on at school.

Sex abuse prevention programs often begin in kindergarten, whereas sex education programs rarely begin before the fifth grade. The sex education curriculum almost always omits controversial topics. Parents fear that a more liberal curriculum will be harmful or overstimulating for children. Therefore, the curriculum focuses on “safe” topics such as the dangers of sexually transmitted disease. In grade school, analogies may be used to make the material more palatable. Analogies can confuse children who come to think that eggs are brittle, encased objects produced by hens, geese, and ducks that seeds will sprout into plants growing in soil attached to the wall of the mother's stomach, watered occasionally by the father's semen ([Goldman and Goldman, 1982](#)).

[Campbell \(1986\)](#) reviewed more than 400 sex advice books for young people, published over the past 100 years. These volumes uniformly emphasized the danger of sex, the consequences of unintended pregnancy, the ravages of sexually transmitted disease, and problematic aspects of sexual deviance and dysfunction. Discussion of sensual pleasure was conspicuously absent. In the past decade, sex advice books have been joined by sex abuse prevention books and pamphlets that generally focus on the threat of sexual abuse without normalizing masturbation, body exploration, or sex play. Fortunately, a few, more positive sex education programs are now available ([Krivacska, 1990](#); [SIECUS, 1991](#)), but there is some question as to how widely they will be used, given school administrators' concern about parent protest.

## Puberty

Boys and girls follow a somewhat different developmental path in adolescence. Girls are more likely to become eating disordered or depressed, and boys are more likely to drink or be delinquent. Girls are thought to have a more difficult process of separation and individuation because of blurring of boundaries between mothers and daughters or because mothers may project their hopes and dreams on daughters ([Blos, 1983](#)). As adolescent girls attempt to resist the profound regressive need for the mother, they may turn passionately to the other sex.

Early maturing girls may be depressed, ashamed of their breasts, and anxious about gaining weight ([Brooks-Gunn and Warren, 1989](#)). Late-maturing girls appear less concerned and more competent in responding to developmental demands (Schwab-Stone et al., 1985). Menarche itself brings an increase in self-esteem and awareness of the body to most girls. Girls who learn how to insert a tampon may understand the difference between clitoris, urethra, and vagina for the first time. Girls who successfully insert the tampon feel excited and proud ([Shopper, 1979](#); [Whisnant et al., 1979](#)).

In contrast to girls' menarche, boys' semenarche or first ejaculation has been accorded little attention. In rural Ukraine, it is a memorable event traditionally celebrated by the boy digging a hole and contributing his semen to Mother Earth (L. Hrbeck, personal communication). In our society, boys are poorly prepared for the event by health classes or by parents ([Shopper, 1979](#); [Whisnant et al., 1979](#)), and it is often experienced as a frightening occurrence that is neither revealed nor celebrated. [Stein and Reiser \(1994\)](#) asked white, Jewish camp counselors, on average 18 years of age, to describe how it had been for them. They described strong feelings, including pleasure, but many felt unprepared and unsupported. Most did not reveal the event to others, and some thought they had wet the bed and felt confused, frightened, disgusted, and embarrassed.

Reactions to semenarche reflect a negative value attached to sexuality and the penis. Adolescent boys do not seriously discuss the penis even in an all-male peer group. Instead, they refer to the penis as “rod,” “unit,” or “stick,” terms that are oblique and inferential ([Sanders and Robinson, 1979](#); Schwartz, 1985). Characteristics of the penis such as relative size and appearance remain obscure, in part because boys avoid looking directly at a peer's penis for fear of appearing gay. Yet adolescent boys are concerned about penis size and often assume that their penis is too small. Pornographic films may reinforce this sense of inferiority when the protagonist is heroically endowed.

## BOYS' SELF-CONCEPT

In the past, boys' sexual self-concept was in general positive, certainly more positive than that of girls. However, men today are far more dissatisfied with the body and more concerned with appearance than they were 20 years ago. This was shown in a recent study of ours comparing male university students (average age, 24 years) with male members of a jury pool (average age, 43 years) ([Yates et al., 1998](#)). The younger men were significantly more dissatisfied with body and self. Younger men today are more likely to have eating disorders and body dysmorphic disorder, worry about appearance, engage in weight loss programs, work out, and seek plastic surgery ([Mishkind et al., 1986](#); [Moore, 1993](#); [Pope et al., 2000](#); [Striegel-Moore and Kearney-Cooke, 1994](#); [Sykora et al., 1993](#); [Zerbe, 1996](#)). These changes may be related to (a) increased status of women, with more men attempting to model after women; (b) often unsuccessful competition for the (now more independent) women; or (c) negative perceptions of male sexuality leading to greater body dissatisfaction. Negative values attached to male sexuality are common in abuse prevention

literature, media accounts of date rape and sexual assault, and talk show episodes entitled “men who have sex with their wives' best friends.”

### HOMOSEXUAL EXPERIMENTATION

The biological changes that occur with puberty are rapid and, especially in boys, intense. Incestuous and bisexual conflicts are revived and sexual orientation becomes a central issue. Many boys and girls engage in homosexual activities even though they are heterosexually oriented. This is especially so during early adolescence, when contacts are almost exclusively with the same sex. Homosexual activities are even more common at boarding schools and at camp. Boys often experiment in groups, beginning by exhibiting the erect penis and continuing on to parallel or mutual masturbation, fellatio, and interfemoral and anal intercourse. Girls tend to experiment with a best friend rather than a group of friends, exploring the body, fondling each other's genitals, and making believe they are having intercourse. Sexual experiments engender intense excitement: boys quickly establish orgasm as a goal, whereas girls may experience orgasm but rarely seek it as a primary goal, preferring to kiss, hug, and snuggle for a sense of closeness ([Kinsey et al., 1948, 1953](#); [Langfeldt, 1990](#); [Ramsey, 1943](#); [Sorensen, 1973](#)).

### ADOLESCENT MASTURBATION

Twenty-four percent of teenage girls say they masturbate, and two-thirds feel guilty about doing so ([Coles and Stokes, 1985](#)). Twice as many boys as girls masturbate, and boys do so three times more frequently than the girls ([Leitenberg et al., 1993](#)). Most commence between 10 and 14 years of age ([Martinson, 1994](#)). Those engaged in solitary masturbation often think they are the only ones who have ever done it and that it must be harmful, sinful, dirty, or a sign of weakness. They usually are trying to stop.

Early studies indicated that almost all boys have masturbated by the end of adolescence, but the figures now are much lower than in the 1940s or the 1970s ([Capes, 1972](#); [Chilman, 1983](#); [Kinsey et al., 1948](#); [Martinson, 1994](#); [Ramsey, 1943](#); [Sorensen, 1973](#)). Those who masturbate often are reluctant to report it, even on anonymous questionnaires ([Clark and Tiff, 1966](#)). The reason for the decline has not been determined. Guilt may play a role, although youngsters who masturbated in the 1940s and 1970s also reported feeling guilty about the practice. Adolescents usually assume that their parents dislike or are disgusted by masturbation. However, parental attitudes have not changed much over time and therefore are not likely to explain the sharp decrease in male adolescent masturbation.

### INTERCOURSE

When adolescent boys become sexually active, they quickly focus on foreplay and intercourse, trying to “score” as soon as possible. Adolescent girls, on the other hand, construe sexual activity in a romantic light, and they try to get to know the partner better but try not to “give in.” This discrepancy paves the way for adolescent boys to have few partners and to be abstinent for approximately 6 months of 12 ([Sonnenstein et al., 1991](#)). After initiating intercourse, girls commonly avoid coitus for an extended period. Many teens have sex once and then not again for several years. Most first sexual liaisons are spur-of-the-moment events that take place at the home of the girl or her partner. The two usually have known each other for some time and are “going steady.” Girls are serious about the relationship, whereas boys tend to see it as casual ([Whatley et al., 1989](#)). When girls do have sex, they tend to think that it was wrong, and they wish that they had remained a virgin until they were married ([Coles and Stokes, 1985](#)).

Adolescents who feel close to their parents and who share the parents' values are less likely to have sex at an early age ([Fox, 1981](#); [Jessor and Jessor, 1975](#); [Shah and Zelnik, 1981](#)). When parents control dating, adolescents are less likely to initiate intercourse or become pregnant ([Hogan and Kitigawa, 1985](#)). This reflects the fact that early and frequent dating commonly precedes the first sexual experience ([Thorton, 1990](#)). Adolescents who view their parents as “moderately strict” are less likely to be sexually experienced than those who view their parents as “permissive” ([Miller et al., 1986](#)).

Sociopolitical forces in the United States focus on how to diminish “high-risk” behaviors. The stated goal is abstinence for all youth, although few expect that this can happen. Negative statistics figure prominently in the media, but current data indicate that although the number of sexually active teens increased in the 1970s and early 1980s, teen pregnancy rates actually decreased in the 1990s, falling 15% from the 1991 high of 116.5 per 1,000 women aged 15 to 19 years to 98.7 in 1996. Among the factors accounting for this decline are decreased sexual activity, increased condom use, and the availability of depot and parenteral contraceptives ([Hofferth, 1990](#); [Ventura et al., 2000](#); [Wyatt, 1990](#)).

In the past, the assessment of whether teens were sexually active was based on a single question: “Have you had sex?” The current trend is to ask for more information such as past and most recent frequency of sexual relations and number of partners. Still absent are questions about sexual enjoyment, erotic fulfillment, or relationship to self-worth. Reliability and validity of data are hampered because the definition of “having sex” changes over time and varies from place to place and from group to group. Only 40% of students at one university thought that oral sex was “having sex,” whereas 80% thought that peno-anal sex did qualify. Genital touching was considered “having sex” by one in eight women and one in six men ([Sanders and Reinisch, 1999](#)).

### CHOICES

Adolescent contraceptive use has improved since the early 1980s, but many youth never or rarely use it ([Santelli and Beilenson, 1992](#)). In the United States, adolescents initiate intercourse at approximately the same time as teens in Western Europe and Canada, but they are more likely to become pregnant ([Jones et al., 1985](#)). Compared with Sweden, Australia, and England, North American 15-year-olds, especially girls, acquire their contraceptive knowledge late ([Goldman and Goldman, 1982](#)). There are many reasons why adolescents do not choose contraception: for boys, this is commonly related to the drive to prove themselves or to “score” quickly when the opportunity presents. In the heat of passion, contraceptive measures seem irrelevant. Boys often view contraception as the girl's responsibility. Girls may not think about contraceptive use because they do not plan to have sex in the first place. Having sex “just happens” to them because they are overtaken or persuaded by the boy or because they are in love and powerless to resist. Many teenage girls think that planning to have sex would prove that they are immoral. The net result is that adolescent girls are very unlikely to use contraception at first intercourse ([Zelnik and Shah, 1983](#)).

One-third of all abortions are performed on adolescents ([Santelli and Beilenson, 1992](#)). Girls are more likely to opt for an abortion if they are younger, doing well in school, planning for the future, have better-educated parents, have little religious involvement, and have friends with negative attitudes toward pregnancy ([Hofferth, 1987](#)). When an adolescent girl becomes pregnant and does not have an abortion, this may be related to her need to remain close to her own mother, rebellion against the parents, self-destructive or self-defeating tendencies, guilt and religious convictions, the need to be loved by an infant, or the fear of entering dating, vocational, or educational arenas. Unfortunately, the culture tends to view the unmarried pregnant teen as “getting what she deserved” for having had sex.

A common assumption is that adolescents who would feel guilty about having sex are less likely to go ahead and have sex. [Davidson and Moore \(1994\)](#) compared adolescent girls who experienced much guilt over sexuality with those who reported relatively little guilt. Those with considerable guilt felt uncomfortable with their sexuality and were unlikely to be sexually satisfied. There was a strong correlation between guilt, high-risk behavior, and low self-esteem, suggesting that guilt strongly inhibits sexual development but not sexual activity. Those who report sexual guilt react to erotic stimuli with a major increase in negative affect ([Wiebe et al., 1994](#)). We do not know if this is so for boys, but we do know that boys experience as much guilt over masturbation as girls do ([Coles and Stokes, 1985](#)).

Vocational opportunity in a tight labor market that allows teenagers to be gainfully employed is a major reason why today's motivated adolescents are less likely to have sex early or to become parents of an out-of-wedlock child than adolescents in the 1970s or 1980s. Conversely, those adolescents who have less ability and motivation, low achievement, and poor grades are more likely to have sex and to become pregnant at an early age ([Abrahamse et al., 1988](#); [Hofferth, 1987](#); [Robbins et al., 1985](#)). Recent decreases in illegitimate births may be entirely due to improved economic circumstance. Motivation and job availability also affect the sexual activity of adolescent boys. Boys who forego sex in high school are less likely to drop out of high school and more apt to go on to college ([Kinsey et al., 1948](#); [Sonnenstein et al., 1991](#)).

### SEXUAL DYSFUNCTION

Adolescents who attend college have intercourse less often and with fewer partners than they did in 1979, and they are more ambivalent about it ([Darling and Davidson, 1986](#); [Yates, 1993](#)). Female students are more anxious about the social consequences of having sex, whereas male students are more worried about disease. Fifty-five percent of male college students describe themselves as afraid to engage in sexual intercourse; 44% doubt if there is such a thing as safe sex; 11% think that they have an abnormal fear of sex; and 10% want help for a sexual problem ([Katz et al., 1989](#)). Eleven percent of male students experience erectile dysfunction and 20% have premature ejaculation ([Miller and Cirone, 1978](#)). Only 28% of sexually active female students say they are satisfied. Students are too embarrassed to talk about problems, and they rarely seek help.



A number of authors ([Kaplan, 1974](#); [Masters and Johnson, 1966](#); [Martinson, 1994](#); [Money, 1989](#); [Schaefer, 1973](#); [Yates, 1978](#)) build a reasonable case that adult sexual function depends in part on a positive process of eroticization during the early years. Parents who accept normative sexual activity and who provide constructive messages about the body are thought to enhance erotic development. Studies that address these issues are few and often based on small samples and retrospective data ([Reynolds, 1997](#)). However, [Bauserman and Davis \(1996\)](#) found that when college students recalled early sexual experiences in a positive light, they reported greater sexual satisfaction and were more accepting of sexual issues. Subjects reporting only negative early experiences were similar on all outcome measures to those reporting no early experiences. [Finkelhor \(1980b\)](#) noted that girls who recalled sexual experiences with siblings after 9 years of age reported greater sexual self-esteem as young adults. [Reynolds \(1997\)](#) found that young women who reported more frequent sex play demonstrated a sexual self-concept characterized by low sexual conflict/anxiety, high arousability, and high sexual self-esteem. Frequency of recalled sex play was associated with being involved in a sexual relationship, enjoying it more, having sex more frequently, and engaging in a greater variety of sexual behaviors. On the other hand, [Leitenberg et al. \(1993\)](#) noted that the simple occurrence or nonoccurrence of sexual activity in preadolescence or early adolescence did not seem to be related to the sexual adjustment of young adults. Although the data overall support a relationship between early positive perceptions and adult function, the likelihood of retrospective distortion is high. Only a well-designed prospective study of normal sexual development could settle the issue. Study design would need to include direct interviews of children concerning their sexual thoughts, fantasies, and activities.

## THE FUTURE

When historians describe President Clinton's legacy, they may suggest that he opened up more than world trade. His term in office was marked by a significant event: The first time that any sitting president was publicly humiliated for having an affair. The media endlessly repeated every last detail, including the semen stains on the blue dress and the specifics about oral sex. President Clinton responded first with denial and embarrassment, and was visibly shaken. But in the months that followed, he acknowledged the event, asked forgiveness, tried to make amends, was eventually able to talk openly about it, and even wrote a book. Far from being exiled, he enjoyed greater success in office and his family remained intact and supportive. The First Lady said in essence: "He's a good person who does bad things." By these tokens, sex became more understandable, a part of the human condition. The children were watching.

Critical research is on hold because researchers are not allowed to question children about sexual attitudes and events. In the distant past and briefly during the 20th century, adults were able to ask such questions. It can happen again—but not without a major shift in social policy. Perhaps when this generation of youngsters grows up, they can change social policy. They will have had access to web eroticism and to "how to" articles in popular magazines, and they will know more than their parents ever did about sex. They will remember their parents looking at, and laughing at, erotic scenes in prime-time sitcoms, and becoming so embarrassed that they could not discuss the material. They will remember in school the carefully screened and sanitized materials on the dangers of sex. When these children mature, they may decide that enough is enough. The adults are being hypocritical. What better crusade for a new generation?

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## 21 FAMILY DEVELOPMENT AND THE ROLES OF MOTHERS AND FATHERS IN CHILD REARING

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[Coupling and Family Formation](#)  
[First Conception, Birth, and Nurture](#)  
[Toddlerhood and Individuation Within the Family](#)  
[The Preschooler and the Oedipal Constellation](#)  
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Families render humans human. Genetic endowment and maturational forces strongly predispose to the relationships and intimacies that draw the infant into the human race, one interaction at a time. But it is the family in all its permutations that ultimately embraces that child's maturational promise and, through powerful reciprocal, interactive forces, converts tissue, synapse and instinct into human development. In this chapter, we examine the unfolding of that process through the exploration of the basic unit of all emotional development, the family.

Although families vary enormously, they tend to share certain processes while moving through the life cycle of birth and burial. At first glance, it would appear that family processes would be easy to delineate because they seem linear (i.e., birth to growth to death). Family process itself, however, is more cyclical than linear. Family content is determined by the whole-family emotional system, including at least three generations. Those three generations, which have periodically lived together in large numbers in our culture, are accommodating to their own unique life cycle agendas simultaneously. At every moment in its life, the family exists at the intersection of two distinctly different linear axes. One axis is the trail of generational myths, expectations, attributes, memories, and secrets—the family's "givens." This is the family's narrative about itself, so articulately described by [Pincus and Dare in \*Secrets in the Family\* \(1978\)](#) and [Vangelisti \(1994\)](#). The narrative evolves like folk songs in the oral tradition passed on through children being taught who and what their family is now, has been, and is hoped (if not expected) to be.

The other axis is the family's forward progression through the dimension of time in its own life cycle. This encompasses the usual stresses and opportunities of the family's children's developmental requirements and the intrusion of "fateful events." Whether a family gets into trouble is determined in part by whether these two axes intersect at strong or weak points because they perpetually cross and recross each other. Real trouble seems most likely when a vulnerable stretch on the transgenerational axis intersects with an equally vulnerable stretch on the developmental axis. For example: The family narrative trail carries forward a myth/expectation that "Jones boys always marry wild women" at a time when, on the developmental track, the Jones' first-born son is beginning his adolescence by easing up on his academic discipline and starting to push against behavioral boundaries. He tells his parents less and less about his life and whereabouts (a developmentally appropriate change), especially where girls are concerned, and his and his family's fantasies about "wild women" collide with his own unique developmental course.

Family development as a phenomenon is hard to embrace in all its complexity, precisely because the clinician tends to encounter families, normal or not, at just one nodal point in time, depriving him or her of critical longitudinal perspective. Research hints that we have significantly underestimated the life cycle connections between early loss, trauma, or disturbance and later interpersonal dysfunction ([Ainsworth and Eichberg, 1991](#); [Borkowski and Ramey, 2001](#); [Bowen, 1978](#)). At the same time, we often are uncertain about which direction the causal links are moving (i.e., a vulnerable child who destabilizes the family, or vice versa?) ([Wamboldt and Wamboldt, 2000](#))

Cycles of family life are undergoing accelerating change. Lower birth rates, increasing divorce and remarriage rates, and longer life expectancies have reduced childbearing from being the major occupation of parents to what is now less than 50% of parents' lifelong commitment.

Historians urge caution in referring to "unprecedented change." Through careful research, [Demos \(1970\)](#) and [Laslette and Wall \(1972\)](#) have largely dispelled the myth of the ideal three-generational family holding sway in preindustrial family life. A process was begun then, however, that did affect major change in overall family functioning, and it is coming to fruition now. [Hareven \(1982\)](#) summarizes: "Through a process of differentiation, the family gradually surrendered functions previously concentrated within it to other social institutions. During the pre-Industrial period, the family not only reared children, but also served as a workshop, a school, a church and an asylum" (p. 460). The difficulties faced by contemporary families are rooted in this diminished capacity of the family to adapt and cope (partly because of smaller size) and the further decline in the variety of the family's socioeconomic functions.

The declining maternal and child death rates of the 1950s combined with a higher marriage rate and longer life span to create a higher percentage of children growing up in stable, two-parent families than had ever occurred in America. Beginning with the next decade, however, trends began to erode both the ideal and real traditional nuclear family. The sexual revolution uncoupled the association of sexual and reproductive behavior. From 1971 to 1998, the percentage of unmarried American girls 15 to 19 years of age who engaged in sexual intercourse rose from 28% to 56%. Curiously, figures are unavailable for boys, as though only female sexual activity is related to family formation. Second, the movement into the paid workforce of married women with children saw an increase from the 1960 level of 19% of married women with children younger than 6 years of age in the labor force to a real figure of 64% in 1999. Historian Robert [Griswold \(1993\)](#) noted that these forces, combined with attitudinal changes toward coparenting, have brought increasing numbers of fathers into the nurturing domain, regardless of whether they want to be there.

Fertility and fecundity also declined in the United States beginning in the 1960s. We are now at levels lower than those necessary for the replacement of the population, having moved from an average of 3.7 children per woman in 1960 to 1.79. This may well reflect a dissatisfaction with parenthood, in association with a significant decline in the stigma associated with being childless.

Finally, the divorce rate in America brought us past a landmark in 1974, when for the first time in our history, more marriages concluded in divorce than in the death of a spouse. Nine divorces per 1,000 marriages in 1960 became 26 per 1,000 in 1998. The percentages of single-parent families, including single by choice, unmarried and same-sex couples, and serial and stepfamilies have all increased, whereas nuclear unit percentages continue to decrease.

It is the opinion of many clinicians and researchers that the quality of life for children in the past 25 years has not improved at the same rate as it has for adults. Also, rates of distress seem to be on the rise. [Achenbach and Howell \(1993\)](#) studied the changes over 13 years in the prevalence of children with behavioral/emotional problems. He found more untreated children who needed psychological intervention in the 1989 sample than in the 1976 sample. Every child needs and longs for two biological parents in and for his or her life. Simply stated, child rearing is more successful when it involves both biological parents who are committed emotionally to the job in all its complexity. This is not to say that this is the only way to bring child development to fruition. However, it is to say that, as a group, nurturing forms other than the biological family are likely to struggle with more risk factors. In practice, however, we see repeatedly that, regardless of form, families, as opposed to institutions, promote development most efficiently by buffering, cushioning, and protecting young children while they are learning new ways to cope with the world about them. [Patterson \(1983\)](#) strongly suggests that the relation between stress and developmental outcome for isolated individuals is different from that for children who are part of a social group, the former being at more risk.

Some of these statistics illustrate an important, irreducible fact about change in the American family. Women are in the labor force to stay. Families have changed, yet the institutions the families rely on most heavily, schools and the workplace, have been much slower not only to change but to respond to the changes in the family system ([Galinsky, 1986](#)). The family structure that is most influential in the child's development, however, is the one perceived by the child as *his* or *her* family, not the one perceived by the Census Bureau.

Each child who enters the family changes it permanently and irreversibly, rendering the child's perception of the family unique to his or her own experience. [Sameroff and Fiese \(2000\)](#) have helped us move away from the restrictions of the linear, interactional model of child development, and toward one that better encompasses the progressive, dynamic, reciprocal forces that have helped children change families and vice versa. Their "transactional model" emphasizes the need for incorporating social and economic as well as biological forces. Proposing a "continuum of caretaking causality," such increased emphasis on the qualitative aspects of the nurturing

domain has encouraged clinicians and researchers to think anew about who in the family is doing what with the children, and not simply how long they are doing it.

The Berkeley Adult Attachment Interview ([George et al., 1985](#)) in its application to family development ([Main et al., 1985](#)) is an example of growing interest and skill in our capacity to assess the adult's state of mind (and not simply his or her behavior) with regard to attachment to his or her children, and vice versa. The interview for the Adult Attachment Classification System is done separately with mother and father. This is an example of how we are returning to the exploration of the overriding significance of the quality and intent of the nurturing interaction, and not merely the biological predispositions of the interactors.

Mothers, fathers, grandparents, siblings—all form unique attachments to children that, in formative settings, are welcomed and easily integrated by the child into a mosaic of consistent, predictable, comforting internalizations of the nurturing experience. Therefore, internalization of the nurturing experience, be it positive or negative, is not merely the result of a single adult attachment. Much clinical literature, however, falls short in its attempts to clarify distinct maternal and paternal antecedents to psychological syndromes. [Bezirgianian et al. \(1993\)](#) found that maternal overinvolvement paired with maternal inappropriateness combined to form pathogenic predispositions toward borderline personality disorder in children. Although paternal means were included, they were not commented on in the discussion.

Optimal child development is fostered by optimal family development. This occurs by translating the recognizable maturational stages of child development into the transactional modalities and developmental dynamics of the family. Optimal family development, as perceived by the child, begins with a secure individual attachment, which the infant must make with its primary caregiver, typically the mother, although fathers as primary caregivers certainly have the capacity to rear their children without placing them at developmental risk ([Pruett, 1983, 1987, 2000](#)). In fact, [Radin and Harold-Goldsmith \(1989\)](#) cite the advantages to young children of paternal involvement, almost regardless of the reasons for the father's presence. The other optimal phenomenon for promoting development in the family network consists of the capacity of mothers and fathers to form reciprocal, empathic relationships with the child, aided by a broad and complete range of affective expression. The parent must be ready to accept developmental progression and change because it comes rapidly, particularly in the first year. It is best if the children are appreciated for their unique and idiosyncratic traits, temperaments, skills, and weaknesses.

Despite the dramatic increase in the number of publications on fathering since the mid-1980s ([Lamb, 1997](#)), fathers continue to be so underrepresented in the literature compared with mothers that one sees a staggering deficit. A major prospective, longitudinal study on parental psychopathology and parenting styles as they relate to the risk of social phobia in children failed to include *any* data on fathers ([Lieb et al., 2000](#)). [Phares and Compas \(1992\)](#) reviewed research papers in the major journals dealing with clinical child development published from 1984 to 1991 and found that nearly half of all studies involved mothers only. Nearly one-fourth of the remaining studies did include father-related material, but did not bother to differentiate its effects. The final one-fourth did measure father-child effects and found them consistently present. So when researchers bother to look for father effects, they always find them. The authors suggested that the overreliance on mothers as research participants has fostered not only an incomplete data set with regard to child development, but also one that is heavily gender biased because "relations cannot be found among variables that are not investigated" (p. 406).

Before beginning a chronologic view of family development across the life cycle, clarification of certain phenomena that guide family function is in order. In understanding the family as a system, we are accustomed to conceptualizing change in one segment of the family as resonating throughout that system, and any negative or positive change in one segment of the system as either promoting or discouraging development in others. Homeostasis is the way that families conserve their energy, socialize their young, and balance risk and protective factors ([Werner, 2000](#)). Yet the demands of rapidly evolving maturational forces in the child offer continual perturbations to that homeostasis. Therefore, it is important for us to understand how to view a family's potential success in preparing its children for adulthood.

[Skinner et al. \(1983\)](#) created the Family Assessment Measure, consisting of a general and dyadic scale to distinguish reliably between normal and problem families. [Mrizek et al. \(1995\)](#) developed the Parenting Risk Scale, which uses a semistructured interview to rate difficulties and concerns regarding parental commitment, knowledge base, control, psychiatric disturbance, and emotional availability. [Fleck \(1989\)](#) has offered an efficient, five-factor method for assessing the system's capacity to support the development of its children, consisting of (a) leadership, (b) boundaries, (c) emotional climate, (d) communication, and (e) the establishment and accomplishment of goals and tasks throughout the life cycle. As we begin to examine the family's development across the life cycle, it is critical to remain conscious of these five factors.

*Leadership* is defined as the decision-making, facilitating source of power and discipline used by the parents who lead the family unit. It is shaped by the presence or absence of mutual support and esteem and by the effectiveness of the communication between the leaders of the family unit.

*Family boundaries* refer to (a) boundaries within the individual that define the self, (b) boundaries between generations, and (c) boundaries between the family and the community. It is important that these boundaries be semipermeable, permitting contact and discourse with others outside the family boundary. Self- and generational boundaries remain relatively stable throughout the life cycle, whereas family/community boundaries must become increasingly permeable as children cross them more frequently to participate in the community about them.

The *emotional climate* or affectivity of the family unit is the connective tissue that binds the family together into a functioning body. It sustains, or erodes, the family's capacity to care for and support one another, especially since the family has ceased to be such an essential economic unit. Chronic scapegoating of a family member, child abuse, and neglect are classic signs of failure in the family's emotional climate.

*Communication* within families is verbal and nonverbal. Communicative language itself and its uses for deepening relationships are learned best within the family. Experiences and feelings are shared through the medium of language, whereas values and culture are differentiated and reinforced by consistency of communication within the family.

Finally, the expectation that families will *nurture and socialize* their young, so that they develop into contributory members of society is the burden placed on the aforementioned functions. The success or failure of this expectation is determined by the way the family achieves its goals for individual members and sets the members' tasks toward reaching those goals. Goals and tasks throughout the life cycle change and evolve in complex ways and, unlike communication or boundaries, seem not to diminish in significance over time.

Cultural influences powerfully shape the tasks of the family across the life span. As child-rearing domains become increasingly complex, it is essential that clinicians and researchers remain vigilant for the normative cultural and functional forces that frame a particular family's expectations, strengths, and vulnerabilities in *their* context ([Johnson-Powell and Yamamoto, 1997](#); [Pruett, 1999, 2000](#)). Values about dependability, family loyalty, intimacy, autonomy, and extended family access vary widely and normatively ([Coll and Magnuson, 2000](#); [Rey et al., 2000](#)).

Appreciating values in context also is critical for the growing number of children raised in families of same-sex parents. Social and cultural isolation and conflict in certain communities often stem from the failure to appreciate such a family's contextual needs and competencies ([D'Augelli and Patterson, 1995](#)), rendering the children at greater risk in certain developmental stages, especially the transition to adolescence ([Lock and Steiner, 1999](#)).

Family development itself usually is arbitrarily divided into stages for better understanding of its predictable developmental phenomena. This approach can mask the enormous complexity of the relationship systems implied therein, which expand, contract, and realign to support the entrance, development, and exit of family members in profoundly meaningful ways emotionally, culturally, and historically. We tour these stages with an eye to the contributions made by the nurturers and the nurturants, examining the unique and differing roles of mothers and fathers as participants and facilitators of normal development across the life cycle.

## COUPLING AND FAMILY FORMATION

Family formation renews every time heterosexual gametes join. The human pair responsible for nurturing the newborn brings conscious and unconscious motivations, both libidinal and aggressive, to their coalition. The choice of mate is governed by a web of intrinsic and experiential factors, tempered by the degree of separation from the family of origin. Contemporary family formation frequently is characterized by a period of joint cohabitation. Making a deliberate, conscious decision about family formation strongly predisposes toward health, just as an unconscious, nonjoint decision predisposes to risk. One of the strongest unconscious motivations toward coupling is the wish to acquire in one's mate a longed-for or incomplete aspect of oneself. This can strongly predispose toward stability in the marriage. There is evidence to suggest that this may have an ameliorating effect on eventual family formation despite previous negative experiences, particularly on the part of the mother in her own childhood nurturing interactions. [Eichberg \(1987\)](#) has found in her research using the Adult Attachment Inventory that the father's role is positively ameliorative of a mother's ([Main, 1992](#)) negative experience with her own parents.



When the coupling results in marriage, it is a joining of two complex historical, interpersonal, emotional, and financial systems. Couples are marrying later and postponing birthing their children: The average age of marriage for women in 1998 was 22.8 and for men 24.7 years, and the birth of their first child came on average 1 year and 10 months later. This implies there is a relatively short time given to adjusting to this phase in the life cycle. Sociologists show us there does seem to be a relatively narrow window for the timing of this phase. Women are twice as likely to divorce if they marry before the age of 20 years than if they marry during their 20s. They are half again as likely to divorce if they marry after 30 years of age than if they marry during their 20s ( [Glick and Norton, 1978](#)). A variety of other factors also can contribute to difficulty at this life cycle transition: (a) the couple resides at either great distance or close proximity to either family of origin; (b) the couple meets or marries in close proximity to a significant loss; (c) the couple marries after knowing each other for fewer than 6 months or an engagement lasting over 3 years; (d) the wedding is performed without family or friends; and (e) the wife becomes pregnant before or within the first year of marriage ( [Bacon, 1974](#); [Becker, 1987](#)). It does seem that the rise in women's status is correlated with marital instability and with the increasing, although not absolute, marital dissatisfaction of their husbands. We are clearly in a successful transition toward more egalitarian relationships, and the educational and occupational equality of the sexes is a perturbation to couple formation as we currently know it ( [Burke and Weir, 1976](#)).

## FIRST CONCEPTION, BIRTH, AND NURTURANCE

This era begins with conception and ends at the end of the child's first year. There is much psychological work to be done by both mother and father in preparing for and dealing with conception. There also is enormous variability in the amount of conscious deliberation devoted to the decision to conceive a child. Once conception does take place, planned or unplanned, complex psychological responses follow in both wife and husband. The mother struggles with profound changes occurring in her body during pregnancy and after delivery.

Meanwhile, much of the psychological work is fueled by a conscious reassessment of the couple's own family experience. A new identity begins to come to fruition, that of *being* a parent, not just having one. What makes this riveting is that both maternal and paternal identifications are deeply rooted in each individual. The mother prepares herself psychologically for the coming attachment to her infant by drawing her attention to her own inner experience and her growing fetus, as her preoccupation with the outside world decreases.

Fathers are involved in psychological work of a different sort, albeit active and equally important in terms of preparation. Food cravings, somatic preoccupations such as vague gastrointestinal disorders, and nutritional changes are widely reported. Concern about his adequacy as a provider and protector may erode an expectant father's self-esteem. Mood changes, frequently expected in mothers, also occur in fathers. "Even before the birth of his child, the father's life, his body, and his mind are busy making ready in ways of which he may only have a passing awareness" ( [Pruett, 1987](#), p. 28). Birth preparation classes may be helpful and supportive to both mothers and fathers in promoting a sense of mutual commitment and in explaining the universal pleasures and fears during the pregnancy and birth phase. Both mothers and fathers have complex mental images of their children long before the child sees the light of day.

But nothing is more powerful than the parental experience of the birth in terms of its long-term impact. First impressions are enormously powerful for both mothers and fathers. Attachment and bonding research have clearly articulated for us the importance of the mother–infant haptic and tactile involvement in the hours after birth. Fathers who are present at the birth are more verbal about their babies, more accurate in describing them, and more intimately attached to them at follow-up.

The newborn's job is just as complex as that of his or her parents. The neonate must first stabilize and regulate his or her internal life to the point where he or she is able to perceive and respond to events in the external world by processing sensory, vestibular, and human interactional input ( [Ramey and Ramey, 1999](#)). Next, the newborn must use his or her repertoire of skills and intrinsic reflexes to elicit pleasurable experiences from the human world, which will make it possible for the newborn to fall in love and establish an intimate attachment to the main caregiver.

Finally, the infant must make use of pleasurable experiences to communicate with the important objects in the world around in a meaningful and personable way in order to be entertained, stimulated, fed, and have his or her bodily functions attended to. Both mothers and fathers with or without experience learn, by on-the-job training, to read, as well as anticipate their infant's signals. Empathic connections and the capacity to comfort, soothe, woo, and entertain are tasks common to both mothering and fathering. Qualitative differences are present, however, in the idiosyncratic ways in which mothers and fathers respond. Mothers tend to respond to their babies on a more intimate scale, facilitating fine motor development and affective differentiation. Fathers tend to be more active and gross-motor involved. Nevertheless, as shown by [Parke and Sawin \(1975\)](#), fathers were able to feed their babies as effectively and efficiently, although somewhat differently stylistically, as their spouses.

Infants appear quite interested in and responsive to the differences between paternal and maternal interactive styles. [Yogman \(1982\)](#) noted that by the time infants were 8 weeks old, they were responding differently to their fathers and mothers. At 6 weeks, infants hunched their shoulders and lifted their eyebrows when their fathers appeared in their visual field. The same infants, when they saw or heard their mothers, seemed to expect more routine functional handling such as feeding or diapering and became settled rather than animated.

The involvement in the first year of life of two caring and competent adults appears to have a positive effect on overall cognitive development. [Pedersen et al. \(1979\)](#) found that the more actively involved a 6-month-old had been with his or her father, the higher the infant's scores on the Bailey test of mental and motor development. Also, [Parke \(1979\)](#), in examining children over the first 8 weeks of life, found that the more fathers were involved in everyday, repetitive, boring aspects of care, such as bathing, feeding, dressing, and diapering, the more socially responsive the infants were. It is in the mutual pleasures of this early experience that the adults, who have now moved up a generation and become caretakers to the younger generation, feel their own personal development frequently propelled forward to new levels of empathic—even altruistic—connections, not only with their children but also with other important objects in their lives.

## TODDLERHOOD AND INDIVIDUATION WITHIN THE FAMILY

The child's astounding increase in mental and physical resources propels him or her out of the omnipotence of the first year of life into a much more social context, in which new skills permit more active shaping of the need-satisfying environment. The development of language, increasing sophistication in cognitive structures, mastery over motility and sphincters, and the incorporation of gender identity and gender role expectations all prepare the child for the complex sequences of the vital separation–individuation process ( [Zeanah et al., 2000](#)). Parents are alternately challenged through intense clinging and contentious interchange, giving this era ambivalence as its marquis. Aggression, care taking, love, anger, and sensuous intimacy are now part of the toddler's repertoire ( [Pruett, 1999](#)). This makes limit setting a vital companion to the toddler's adventurous experimentation with challenging, aggressive, and seductive behaviors. At the same time, the parent is wise to be led by the toddler, rather than attempt to lead the toddler ( [Lieberman, 1993](#)). By now, fathering styles are quite differentiated from mothering styles of interaction, with fathers initiating more rough-and-tumble, unusual, unpredictable, physical, and stimulating forms of play. [Billar and Meredith \(1974\)](#) noted that mothers tended to engage in more conventional, toy-mediated play, picking up their children to engage in care-taking and nurturing activities more than fathers. Because the child has increased his or her level of mastery over the internal environment, he or she has a good deal more energy available to explore the boundaries of the external environment, giving separation tasks a much higher valence during waking life. Adjustments to the toddler's new, if clumsy, drive for autonomy are necessary to avoid prolonging the child's functional dependency. Because separation from the mother sometimes is the fuel for the sleep disturbances that are common during the second year of life, the father can help decrease the virulence of nighttime disruption by being the one who goes in to soothe and settle the child, spelling the child from another separation from the mother, while helping the child feel safe and secure.

Clearly, the child's unique temperament and style interact with parental values and experience with regard to personal autonomy, separateness from family of origin, and impulse and bodily control. The unique contributions of the father during these years have been increasingly recognized as vital to the success of this developmental era ( [Greenspan, 1982](#)).

## THE PRESCHOOLER AND THE OEDIPAL CONSTELLATION

The preschool child's appropriate use of personal pronouns, ability to say "no," and increasingly adaptive capacities all draw the family as a whole further into a new domain that will be characterized by the time the stage is complete by three-party rather than two-party relationships. Curiosity, assertiveness, and the capacity to begin to delay gratification help the child regulate and moderate intense instinctual feelings and affects. Cognitive growth, meanwhile, assists the child in learning and remembering what the important objects in his or her life will or will not tolerate. By the beginning of this phase, parents should have largely discarded baby talk because both the child and the parents can have a more satisfactory communicative interaction. Reasonably adept parents are able to respond with affection, empathy, and a minimum of rejection to the preschool child's bid for intimate, controlling attention from the parent of the opposite sex. Appropriate, predictable limit setting and humor play important roles in helping the heterosexual relationships between child and parent withstand the heavy weather of strong rivalrous feelings.

Latent oedipal issues, especially incomplete or conflictual ones, often are resolved with surprising force in the parent metapsychologically. By now, parents are able to yield most control over bodily functions to the child, relinquishing him or her as a physical possession, and admiring and encouraging his or her attributes as a separate, gender-specific human being who is beginning to understand the joys of the delay of gratification.

During these preschool years, fathers interact with their children mainly through play and productivity. Through role modeling, the father provides opportunities for children of both sexes to build increasingly positive self-esteem ( [Sarnoff, 1982](#)).

The press of developmental needs during this period, highlighted in research, shows us a critical relationship between marital satisfaction and parental involvement. There is much research that indicates marital satisfaction to be at its lowest ebb during the childbearing years ( [Glenn and McLanahan, 1982](#)). [Waldron and Routh \(1981\)](#) note that marital satisfaction follows a U-shaped graph, with high levels of marital comfort before children are born and again after they leave home. Frequently, children place such significant demands on the couple that there is little energy left to fuel the marital relationship, although it is not suggested that children destroy marriages (even though in some instances they may be permitted or even encouraged to do so). [Rapoport et al. \(1977\)](#) report that marriage often is experienced by fathers as better than by mothers during this nadir of marital satisfaction because it is mothers who usually have more negative experiences with their children, feel more isolated, and are more vulnerable to psychosomatic stress ailments, including fatigue.

Although the discrepancies between maternal and paternal experience during this phase of the life cycle can be problematic, including occasional envy and jealousy on the part of the parent who is having the more difficult and challenging time with the preschool child, the long-term effects of having both parents involved intimately during this phase are strongly positive. One of the most dramatic findings in the father–infant care research is the relationship between early involvement and subsequent sexual abuse. If a man is involved in the physical care of his child before the age of 3 years, there is a dramatic drop in the probability that man will be involved later on in life with sexual abuse of his own or anyone else's children ( [Parker and Parker, 1984](#)).

## SCHOOL, LATENCY, AND FAMILY UNITY

The timely desexualization of the child–parent relationship allows the child to make powerful psychological investments in adults other than his or her own parents, initiating the first disillusionment in one's parents. Just as the child's body is relinquished from parental control, so is the child's mind. The family now helps the child separate for most of his or her waking hours to attend school and confront its social and cognitive challenges. Large amounts of energy are made available for relating to other children and adults, as well as for learning and problem solving. License must be granted for further exploration; at the same time, limits are placed in a reasonable, comfortable manner. The integration of family and tradition, as guided by societal mythology, serves as the hallmark of this period of development. Themes of internal control begin to compete with the pursuit of pleasure as the integrated 7- or 8-year-old strives for balance. During this period, it often is easier for a family to spend prolonged, uninterrupted segments of time with one another for travel, leisure activities, and neighborhood projects. Interestingly, girls may have only one or two friends they would call “best,” whereas boys may name six or seven friends, who usually turn out to be somewhat more casual acquaintances. It is acknowledged that, as children age, the disciplinary role of the father must increase. But during the school-age and latency period, a father may serve as a confidant, a pal, even a friend or teacher ( [Benson, 1968](#)). The opportunity for shared activities and mastery experiences with adults of the same sex is extremely important in terms of the solidification of gender role behavior and gender identity itself. Father absence, however ( [Pearson et al., 1994](#)), leads teachers to rate both boys and girls as more aggressive relative to mother–father families. Especially poignant was the finding that the protective factors for mother–father families were not as apparent among low-income families.

## ADOLESCENCE AND GENERATIONAL REDEFINITION

Families of adolescents must use boundaries that are qualitatively different from those in families with younger children. The boundaries must now assist the children in managing their own impulses because parents no longer have or can practice complete authority. The boundaries between the family and the outside world must become more permeable without being destroyed. There is a normal careening between independence and dependence. A sense of self is beginning to be consolidated and shaped by values, the search for pleasure, and particular goals and tasks. The primary object relationship between the parents and the teenager now must be retooled for the transition from childhood to adulthood. The teenager is involved in a regular, normative struggle between independence and dependence. The adolescent also is becoming intensely interested in his or her constantly changing body. Values and “do unto others as they would have you do unto them” beliefs are subject to greater scrutiny. Developmental stress does not necessarily doom the family to turmoil during this phase, but profound physical and psychological changes threaten the previous level of homeostasis in the family. An adolescent notices weaknesses and unusual vulnerabilities in the family, as well as in his or her own psychological functioning, but these also may be seen as possible points of departure for new adaptive functioning. But adolescent observations are notoriously selective. Bulimic teenagers and young adults rated their fathers as showing less affection and more control toward them than their nonbulimic siblings, suggesting that the paternal relationship may be a source of nonshared environmental experience associated with bulimia nervosa ( [Wonderlich et al., 1994](#)).

Adolescence often is a period of significant stress in the family because both the adolescents and their parents often are experiencing significant physiologic and mental changes at the same time. Both generations may be scrutinizing their primary attachments anew and questioning their value and trustworthiness. Just as the adolescent is beginning to make choices regarding values and career goals at the beginning of his or her work life, the parents are needing to accept that certain cherished goals may never be achieved, and they may become quite preoccupied by the limited time left in their lives.

As the adolescent begins to pull further away from the nurturing domain through going to college or taking a full-time job, the parental response can be one of either pride in their child's capacity to cope with life's new challenges or sadness over what appears to be the permanent loss of one's own progeny.

The family as a whole can have its homeostatic behavior deeply challenged by an adolescent's need to extract his or her autonomy from the parental nurturing domain.

Young adulthood and emancipation, the marriage of offspring (the middle family), and, finally, aging and senescence are the last three phases of normal family development. The kind of influence parents now have on their children is largely encompassed by their availability for discussion and advice and by bearing witness to their children's integrity, while being careful to plan and sustain their own autonomy. Grandchildren come next, providing a rejuvenation of spirit and body, not to mention occasions of joy.

The developmental tasks faced by the child and the child's caregivers are the same whether the structure of the family is nuclear or reconstituted. The energy and resources, both emotional and physical, to attend to those tasks is strongly influenced, however, by a particular family's structural limits and flexibility. Adoptive, single-parent, foster, and recombined family groupings are all subject to the same leadership, boundary, affectivity, communicative, and task and goal requirements. The issues of attachment, separation, emancipation, loss, and response to change are largely the same. Each exerts its own particular spin on normal development, but none is by dint of family structure alone doomed to trouble.

Adoptive families do not have the same biological preparation time, although given some luck, they can follow a similar psychological preparation sequence. The separate biological parents' narrative must be integrated into the family's mental history of itself in some fashion. Single-parent families, whether male- or female-headed, face depletion and isolation early and often and work best when social support systems are early and flexible enough to supplement parental and child care. Even oedipal phase resolution need not be impossible, given the child's regular access to familiar and caring adults of the opposite sex.

Recombined and reconstituted families, when a divorced, widowed, or never-married single parent forms a household with a new partner who may or may not be a parent, also are increasing exponentially. These families do face risks. Depending on the mechanism of parental singleness (death, divorce, abandonment), the new parent may be seen as a threat or solution to intimacy between parent and child. Rivalry and jealousy frequently stimulate guilt and anxiety, especially when the same-sex parent has been displaced or replaced. Interestingly, [Black and Pedro-Carroll \(1993\)](#) have shown that the effects of interparental conflict on the psychological well-being of children were mediated more by the overall quality of parent–child relationships than by interadult conflict itself. Stepparents frequently are in risk situations, being tested by their “new children” while simultaneously feeling special loyalty to their “old children” and trying to sustain a new spousal relationship. Time (measured in years), patience, and liberal, frequent communication (sometimes new to everyone as a process) plus permission to parent are all essential. Society's myths do not help either. *Stepmother* in English conjures up Cinderella's stepmother, and *stepfather* in Spanish is *padraastro*, which also means *hangnail*.

On a smaller scale, all families face some of the same issues because families are always reconstituting, biologically and psychologically. Because of the relentless push of developmental and maturational forces in the individuals of our species, like the river, one can never step into one family in the same place twice.



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## 22 INFANCY

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By definition, infancy is the period of life between birth and the emergence of language, 1.5 to 2 years later. Despite its brevity, this phase of development has long attracted a disproportionate amount of attention and interest, but infancy as we know it was not considered a distinctive and important stage of life until late in the 18th century ([Aries, 1962](#); [Plumb, 1972](#)), and it was not until the 1960s that a variety of scientific, social, medical, and political trends converged to generate professional interest in human infancy ([Kessen et al., 1970](#); [Lamb et al., in press](#)). In the three decades of intensive theorizing and research since then, a number of core issues have emerged, and these issues constitute the centerpiece of this chapter. Among the most important of these issues are the following: To what extent do “innate” as opposed to experiential factors influence infant behavior and development? How great an impact do early experiences have on later development?

### THE NATURE–NURTURE DEBATE

Although the nature–nurture debate is now centuries old, the issues at its core remain central to the study of infancy. Historically, the study of development—especially cognitive and perceptual development—was driven by the nature–nurture debate. Extreme views were put forward by the empiricists on the one hand and the nativists on the other. Empiricists like John Locke and William James asserted that there is no endowed knowledge at birth, that all knowledge comes through the senses, and that perceptual development reflects learned associations. They argued that external stimuli naturally provoke bodily “sensation” and that, through association, separate raw sensations can fuse into meaningful perceptions.

The belief that humans begin life “empty headed” was considered both philosophically intolerable and logically indefensible by nativists like René Descartes and Immanuel Kant, who proposed that humans were endowed at birth with ideas or “categories of knowledge” that assist early perception and cognition. They postulated that human beings possess innate perceptual abilities to tell size, form, position, and motion, as well as more abstract conceptions, such as knowledge of space and time.

Historically, a gradual shift in orientation can be discerned in the debates between nativists and empiricists. Initially, a so-called main effects model applied: Either environment or constitution was considered to be important. In the middle of the 20th century, however, an interactional model achieved popularity ([Anastasi, 1958](#)). Unlike proponents of the main effect model, interactionists offered an additive view, in which nature and nurture were believed to *interact* to shape development. Twenty years later, the transactional model was articulated by [Sameroff and Chandler \(1975\)](#); they argued that inherent characteristics are shaped by experience and vice versa and that a constant process of mutual influence continues throughout the life span.

Although developmental psychologists now generally agree that neither experience nor biology alone determines behavior, the 1990s have seen a renewed, transformed nature–nurture debate. Advances in genetics have led to renewed interest in behavioral genetics, and researchers with diverse interests are now collaborating on large-scale longitudinal studies, typically studying adopted children and their parents (e.g., [Deater-Deckard and Plomin, 1999](#)) or twins (e.g., [Saudino et al., 1999](#); [Segal, 1997](#)) to assess the magnitude of genetic and environmental contributions. [Scarr \(1993\)](#) and [Harris \(1998\)](#) have taken the more extreme position that, within the range of normal environments, behavioral variability among individuals is largely accounted for by genetic influences, and indeed, a surprising range of individual characteristics are heritable ([Collins et al., 2000](#)). Others argue that “the range of normal environments” is too imprecise a term and that, in fact, it is just these details of socialization patterns that are crucial to an understanding of development (e.g., [Baumrind, 1993](#)).

Like Collins, we take the view that biological adaptation and experience codetermine the course of development; infants are born with simple yet important behavioral proclivities that both constrain and direct development by delimiting the potential for experientially driven change. An infant who is congenitally distractible, for example, is likely to learn slowly about the objects seen or heard because he or she is unable to attend to or concentrate on them for long periods. Biological factors also determine which events may reinforce infant behavior because certain events inherently complement certain experiences. A bad taste, for example, will make an

infant stop eating a certain type of food much more rapidly than would a loud noise that repeatedly accompanied ingestion of that food (cf. [Garcia and Koelling, 1966](#)).

Two kinds of biologically based tendencies exist. Species-typical tendencies are those that all humans share. These include predispositions to cry when distressed; to respond to others' cries so as to alleviate them; to attend to novel sounds, smells, or sights; and to ignore those that have become boringly familiar. By contrast, heritable influences are those that distinguish one person's tendencies from those of another and are the basis of genetically rooted individual differences. Both species-typical and heritable tendencies have important influences on development. [Rothbart and Bates \(1998\)](#), for example, have reviewed recent research documenting the extent to which early differences in temperament predict later psychological maladjustment, and [Kagan \(1998\)](#) has conducted research suggesting that individual differences in behavioral inhibition are stable over time as well.

As far as experiential or environmental influences are concerned, [Gilbert Gottlieb \(1983\)](#) suggests a useful distinction among inductive, facilitative, and maintenance functions. Induction is the most dramatic form of influence. It occurs when a particular experience or set of experiences completely determines whether a capacity, behavior, or tendency will emerge. Facilitation occurs when certain experiences speed up or slow down the emergence of capacities, behaviors, or tendencies. Finally, maintenance describes a situation in which experiences preserve already developed capacities, behaviors, or tendencies.

We view both experiential and environmental influences as functioning together, so that it is impossible to assess the extent of their involvement. This chapter repeatedly notes the ways in which biology (in the form of predispositions and innate propensities) constrains or facilitates experience and learning.

## THE CONTINUITY DEBATE

The continuity debate focuses on the importance of early experiences. Some view the experiences and behavior patterns developed in infancy of crucial importance to later life. Social orientations, motivations, and intellectual predilections established in infancy set lifelong patterns. Others argue that infancy is not of overriding importance because experiences in infancy have little (if any) long-term predictive significance.

### Proponents of Continuity

Early-experience proponents arrived at this position from a diverse array of theoretical starting points. [Sigmund Freud \(1940/1968\)](#) was the first major theorist to focus attention on infancy, justifying this focus by suggesting that the ways infants are treated establish lifelong orientations and personality traits. Freud proposed that there are critical phases in development when certain sorts of experience—affecting specific types of traits—are of special significance. Infancy falls within Freud's oral phase of development, during which feeding experiences and other activities centered on the mouth are particularly relevant. Toward the end of infancy, Freud continued, the oral phase yields to the anal phase. During this period, caretaker–infant interactions center on toilet training and are likely to have long-term consequences as well. However smoothly development unfolds in this phase, though, it is never possible to override any oral problems established earlier; difficulties can be overcome only by “reliving” earlier experiences through lengthy psychotherapy.

[Erik Erikson \(1963\)](#) portrayed early experiences and their effects rather differently. From the early feeding experiences, he suggested, children develop a degree of trust or mistrust in the caretaker rather than concrete oral traits. He also believed that the quality of early interactions (i.e., how much “basic trust” the infant develops) has implications for the way the infant will negotiate the next stage of development, in which the key issue is establishing autonomy or shame. With respect to toilet training, therefore, Erikson emphasized not the anal organs but the status of toilet training as a battleground of wills as the child tries to exert initial control (by determining when to give the caretaker the prize the caretaker seeks). Erikson described eight developmental stages, each marked by a crucial issue: Basic trust/mistrust is at issue in the first stage; autonomy/shame is at issue in the second.

Erikson's view of how early experiences affect the child's later personality substantially improved on Freud's in two major ways. First, Erikson portrayed the lessons learned in each phase in more abstract, general terms (e.g., trust, autonomy) than did Freud (e.g., orality, anality), and the psychological issues described indeed seem pertinent to the stages concerned. Second, Erikson explicitly proposed that the ways in which different stages are resolved are somewhat interrelated. From Erikson's perspective, how much the infant trusts the caretaker may affect the infant's willingness to cooperate in toilet training and other matters. This initial mistrust may yield not only a mistrustful adult but one plagued by unsuccessful resolution of later developmental issues as well.

Unfortunately, the ideas of Freud and his followers, including Erikson, were phrased in mentalistic terms that rarely evoked respect from research scientists. Behaviorists and learning theorists like John B. Watson, Clark Hull, and Neal Miller dominated developmental psychology from the second to the sixth decade of the 20th century. Like the psychoanalysts, these theorists emphasized the importance of infant experiences, and Miller even attempted to rephrase many of Freud's ideas in terms compatible with behaviorism ([Dollard and Miller, 1950](#)). Learning theorists treated early experience in a fashion very different from that of Freud and Erikson, however. Behaviorists eschewed the notions of stages and phases of development. For the learning theorists, early experiences are important because they are first, have no competing propensities to replace, and, thus, yield easy and rapid learning. Moreover, early behavior patterns are believed to serve as foundations for more complex behavior patterns, such as personality traits.

A third group of theorists, most of them ethologists and students of animal behavior, adopted a notion of “critical periods” that also emphasized early experiences ([Bornstein, 1987](#)). The ethologists argue that there are predetermined periods in the maturation of organisms during which development is maximally susceptible to influence by specific types of experiences. Just as Freud spoke of an oral phase during which feeding experiences make the greatest impact on the developing personality, the ethologists spoke of a critical period for imprinting and for various other behavioral tendencies. During critical periods, lessons are learned more easily and demonstrate greater endurance than hitherto or later possible. This notion accorded biological and scientific support to Freud's own and was later integrated into [Bowlby's \(1969\)](#) theory of attachment (see later) and [Lenneberg's \(1967\)](#) theory of language development (see later). The critical periods concept, and its modern “sensitive periods” emendation, assigned great importance to early experiences because it held that once a particular critical period had passed, it was no longer possible for specific experiences to exert formative influences on the developing organism.

Although this diverse group of theorists underscored the formative importance of early experiences, popular commitment to the proposition developed as a result of some dramatic empirical observations published between 1930 and 1950. At that time, reports that children who were reared in impersonal institutions were psychologically stunted led to widespread belief that children needed close relationships in infancy and that, if they were denied such relationships, they would not develop into psychologically healthy individuals ([Bowlby, 1951](#)).

An additional perspective on the continuing importance of infancy was offered by students of cognitive and intellectual development. [Jean Piaget \(1936/1953, 1937/1954\)](#) theorized that all intellectual capacities are built on the simple developments that take place very early in life, and thus, by the early 1960s, hardly anyone doubted that early experiences were especially important. Two trends resulted: a massive increase in the amount of research on infancy and the first attempts to engineer enriching experiences for deprived children in the form of such programs as Head Start. By capitalizing on the special sensitivity of the very young, politicians hoped to “immunize” children against the debilitating effects of later deprivation. More than anything else, the apparent failure of these interventions triggered a decline of confidence in the notion that early experiences were especially influential.

### Proponents of Discontinuity

One of the most vocal opponents of the overemphasis on early experiences was [Jerome Kagan \(1971, 1980; Kagan et al., 1978\)](#), who more recently has come to emphasize the importance of continuity in biogenetic tendencies ([Kagan, 1998](#)). Kagan argued that maturation—the unfolding of genetically determined capacities and individual differences—has been undervalued, and he pointed to research indicating that major differences in rearing environments have little apparent effect on the way children behave. For example, he argued that day care and home care have remarkably similar effects on developing infants ([Kagan et al., 1978](#)) and that even the extreme impoverishment of the rural Guatemalan environment does not retard intellectual development ([Kagan and Klein, 1973](#)).

Kagan's arguments were roundly criticized by researchers who argue that his measures may not be sensitive and that the problem therefore lies in the assessment, not in the concept, of experiential influence. Nevertheless, claims that there is little continuity in development have attracted a great deal of attention. Such conclusions were inevitable because of earlier, oversimplified notions about how infant experiences influenced later development. The difficulty lies in overreliance on the main effects models of development (like Freud's), holding that early experiences have obvious and direct short- and long-term effects. Unfortunately, simple linear relations almost never are empirically substantiated ([Caldwell, 1964; Lamb et al., 1985; Sameroff, 1975; Sameroff and Chandler, 1975](#)).

### The “Transactional” View



Sameroff's transactional model has now replaced the linear or main effects model. Both child and parent are believed to bring distinctive characteristics to every interaction. The child's characteristics affect how the child behaves, how parents treat the child, and how the parents' behaviors affect the child. The parents' characteristics have the same consequences. During each interaction, both parent and child are psychologically changed, and so they enter the next round of interaction as different individuals. From this perspective, it is naive to expect any single experience to have direct long-term effects, for its impact will be diffuse, triggering multiple indirect effects (e.g., also changing the adult's behavior). Nevertheless, events in infancy are important because they initiate such multiple processes of development.

Using this model, [Sameroff and Chandler \(1975\)](#) were able to explain why prematurity, like other types of perinatal risk ([Siegel, 1981](#)), does not always have ill effects. Parents cope with atypical infants in different ways, some providing deprived environments that do not afford the types of experiences that prematurely born children need to offset their potential for developmental delay. Premature infants would be at risk over the long term only if reared in such environments. Thus, to make long-term predictions, it is necessary to consider at least the characteristics of the child, of the parents, and of the family's physical and social environment. Because linear models typically focus on only one of these factors, it is not surprising that their predictive power is poor. One implication of the transactional perspective is that continuity from infancy may be carried *indirectly*, through continuity in the supporting environment, or continuity from infancy may be carried *directly*, through continuity in the infant, or it may be that both are important ([Lamb et al., 1984, 1985](#); [Nash and Hay, 1993](#); [Thompson, 2000](#)). Grossmann and Grossmann (1986), for example, found that there was a significant level of stability in neonatal performance from day to day on the Brazelton Scales ([Brazelton, 1973](#)) and that it was possible to divide their sample into "good" and "bad" orienters. Subsequent observations of the mothers and infants at home allowed the Grossmanns to assess the mothers' behavioral sensitivity. All six of the infants who were good orienters and who had sensitive mothers developed "secure" attachment relationships at 12 months; however, poor orienters with tender and sensitive mothers did so only approximately one-third of the time. Good orienters with sober-talking and less sensitive mothers had only a 38% chance of being "securely attached," whereas only one of the eight infants (13%) who were poor orienters and whose mothers were insensitive developed "secure" attachments. Viewed together, these data suggest that the characteristics of the child and the characteristics of the mother jointly determined what sort of attachment they form—just as Sameroff and Chandler predicted. Of course, the Grossmanns' small sample made it imperative to replicate these findings independently, but they do constitute an elegant confirmation of the transactional model. In this chapter, the ways in which genetically determined tendencies interact with both early and later experiences to shape development are repeatedly described; consistent with the transactional theorists, the authors see development as a continuing process of consolidation and change.

The focus now shifts, however, from overarching issues to specific aspects of development, beginning with perception.

## PERCEPTUAL DEVELOPMENT

Perception constitutes a necessary first step in experiencing and interpreting the world, and for this reason philosophers, psychologists, physiologists, and physicists alike have been attracted to the study of perception. In fact, the study of perception was initiated by philosophers who were interested in the nature–nurture debate, but it also provides information about the quality, limits, and capacities of the sensory systems at the start of life. In this section, we selectively review research on visual and auditory perception, the modalities that have been studied most extensively.

### Visual Perception

## PATTERN PERCEPTION

In the 1960s, [William Kessen \(1967\)](#) reasoned that it ought to be possible to assess infant vision, even at birth, simply by "looking at looking." He and his colleagues photographed the reflection of a stimulus on infants' corneas and found that, even in the first hours after birth, infants looked selectively at parts of stimuli where there was information—usually high-contrast features—instead of scanning the background or central parts of figures randomly.

Subsequent research using this corneal reflection technique has asked not only what the infant looks at but how scan patterns develop and how infants scan different visual shapes, such as faces and geometric forms. Younger infants scan in a limited fashion, whereas older infants scan patterns more widely and efficiently ([Slater and Johnson, 1998](#)). From research on infant scanning, [Haith \(1980\)](#) determined that newborns actively seek visual stimulation and input, scanning the environment to find things to inspect. Newborns also focus most of their attention on the boundaries of figures, where the greatest amount of information is contained. Studies of organized scanning do not tell us *how well* infants see or *what* they see, however. To examine visual acuity in infants, [Fantz et al. \(1962\)](#) capitalized on the observation that infants prefer heterogeneous to homogeneous patterns, posting pairs of patterns for infants to look at; one member of the pair was always gray and the other a set of black and white stripes that varied systematically in width. (The two stimuli were always matched in overall brightness.) The stripe width that failed to evoke a preference was viewed as the one delimiting the infant's ability to tell stripes from the solid gray. By this measure, 2-week-olds showed 20/800 vision (in Snellen notation), whereas 5½-month-olds showed 20/70 vision. In the 40 years since Fantz and colleagues' original study, techniques for measuring infant visual acuity have grown in sophistication, but the results agree well with their findings ([Kellman and Banks, 1998](#); [Slater and Johnson, 1998](#)). Acuity improves steadily from birth and is nearly as good as that of adults by 6 months of age ([Slater and Butterworth, 1997](#)).

Several studies suggest that young infants can perceive some patterns as whole forms. [Slater et al. \(1991\)](#) showed that even newborns perceived and habituated to stimulus compounds, and [Ghim \(1990\)](#) showed that infants as young as 3 months of age recognized (and thus habituated to) the "subjective contours" of figures that did not actually exist on the page but appeared to do so. Further, [Philip Kellman \(1984; Kellman and Spelke, 1983\)](#) studied infants' knowledge of two- and three-dimensional objects when habituated to partly occluded versions of the objects or to multiple perspectives; 4-month-olds who experienced objects moving under these conditions treated them as wholes. Infants also are highly attracted to faces; either because of some innate predisposition ([Mondloch et al., 1999; Slater and Butterworth, 1997](#)) or because faces contain features such as three-dimensionality, movement, sound production, and areas of high contrast that infants find intrinsically interesting ([Slater, 1993](#)). Through everyday experiences, infants then learn to discriminate among different faces ([Bushnell, 1998; Johnson and Gilmore, 1996](#)).

Infants also seem to discriminate orientation well: They discriminate vertical from horizontal ([Bornstein, 1982; Hayes and Watson, 1981](#)) as well as other orientations ([Bremner, 1994](#)), and even adjust a reaching hand properly to match the orientation of a target ([Von Hofsten and Fazel-Zandy, 1984](#)). Patterns that are aligned vertically or horizontally are preferred and detected more readily than those aligned obliquely ([Bornstein, 1982; Held, 1989](#)), with vertical stimuli appearing to be the most preferred, perhaps because infants visually scan predominantly along the horizontal ([Haith, 1991](#)).

## DEPTH PERCEPTION

Depth perception is crucial to determining the spatial layout of the environment, to recognizing objects, and to guiding motor action. The study of depth perception also addresses an interesting psychophysical question, namely, how we perceive the three-dimensional information. Depth perception has served as a kind of battleground between nativists and empiricists. Debate on this question exemplifies the typical historical course: It began with hotly contested disputes between nativists and empiricists that spanned the 17th to 19th centuries and ended only with experimentation in the 20th century.

In the early 1960s, [Gibson and Walk \(1960\)](#) began to investigate depth perception in infants experimentally, using a "visual cliff." One side of the cliff shows the infant an illusory drop, but the other side does not. Gibson and Walk found that almost all infants they tested between 6 and 14 months of age crawled over the glass continuation of the center board on the shallow side when their mothers called them, whereas only a very small minority of infants crawled across the "deep" side. From these results, Gibson and Walk concluded that depth perception must be present in infants as young as 6 months of age. By 6 months, however, children have had plenty of experience perceiving depth. [Campos and colleagues \(1970\)](#) therefore studied precrawling infants by monitoring heart rate when the infants were exposed to shallow and deep sides of the visual cliff. They found that infants as young as 2 months of age showed defensive changes in heart rate when exposed to the deep side. These differences suggest that infants may perceive depth long before they crawl.

The visual cliff experiments represent one way investigators have sought to explore the capacity to perceive depth. Another mode of study involves observing avoidant or "looming" responses to objects approaching on a hit path. [Yonas \(1981\)](#) reported that infants as young as 1 month blinked their eyes in response to approaching objects but that the "looming response" could not be demonstrated reliably until children reached approximately 4 months of age.

A third strategy for investigating depth involves studying sensitivities to isolated visual cues that typically signal depth. Convergence, for example, depends on an organism's capacities to fixate an object binocularly and to fuse the two disparate retinal images into one. If infants rely on convergence of the eyes to cue depth, as [Descartes \(1638/1824\)](#) and [Berkeley \(1709/1901\)](#) proposed, theorists need to show that the eyes indeed converge. We have all noted, however, that infants often

seem unable to fix both eyes on an object (called *strabismus*). Researchers have observed only minimal convergence of the two eyes at 1 month, but regular convergence by 2 to 3 months ([Hainline et al., 1992](#); [Kellman and Banks, 1998](#)). It is at this time that infants also first display sensitivity to the deep side of the visual cliff ([Campos et al., 1970](#)).

In summary, studies of the visual cliff, of looming, and of monocular and binocular sensitivity in infants converge to suggest that depth perception may be relatively poor until approximately 2 months after birth. The development of depth perception is not complete by 4 to 6 months of age, however, although infants have the capacity for *relative* distance perception by this age and are known to use monocular depth cues such as relative size, interpretation, and shading by 7 months of age ([Granrud, 1993](#)), and kinetic cues, such as changes in texture, by the same age ([Kellman and Banks, 1998](#)). Because binocularity can provide information about absolute distance only if interocular distance is known, *absolute* distance calibration may depend on nonvisual sources of information. Theorists such as [Jean Piaget \(1937/1954\)](#) have argued that infants can tell the absolute distance of objects in near space only after they have had experience reaching, and the distance of objects in far space (i.e., beyond reach) only after having experienced crawling.

We know that young infants can *locate* stimuli in space, in part from studies of reaching for objects. Recording following motions, goal-directed behaviors, and types of reaches to objects located at different distances and moving at different velocities, [Von Hofsten \(1984\)](#) reported that infants as young as 4½ months reach and contact an object, even if it is moving, and that their reaching is accomplished in a way that indicates good predictive targeting of location. Until recently, it was believed that such reaching and grasping of objects resulted from visual guidance of the hand toward the object. However, [Ashmead et al. \(1993\)](#) found that it is not until approximately 9 months of age that the opportunity to see the hand helps infants make adjustments to a change in target locations. Similarly, [Clifton et al. \(1993\)](#), tested very young infants' (up to 6 months of age) abilities to reach for objects in the dark (only the targets were illuminated), and found that sight of their own hands was not necessary for reaching and grasping. Nevertheless, infants' initial knowledge of location is highly egocentric; only by the end of the second year does a more environment-centered representation of space become well developed ([Bremner et al., 1994](#); [Hermer and Spelke, 1996](#)).

Locomotion also affects depth perception: Infants who crawl or have experience in a "walker" extract form from a fluctuating display and avoid the deep side of the visual cliff earlier than those without comparable experience ([Campos et al., 1992](#)). [Adolph \(1997\)](#) subsequently showed that locomoting infants use perceptual information to adapt their locomotion and exploration "on foot" of novel surfaces.

## COLOR

By 3 months of age, infants are nearly as acute as adults when the task is to compare brightness differences between stimuli presented simultaneously ([Kellman and Banks, 1998](#)). Thus, in studies where the task is wavelength discrimination *per se*, it is possible to unconfound hue and brightness either (a) by matching colors in brightness using an adult standard in regions of the spectrum where infants and adults are known to match or in conditions testing successive discrimination (as [Chase, 1937](#), and [Bornstein, 1975](#), did); or (b) by varying brightness against hue, so that brightness differences are irrelevant (as [Peeples and Teller, 1975](#), [Mercer et al., 1991](#), and [Adams et al., 1994](#), did). In each case, infants of 2 to 4 months of age were shown to perceive color, and [Adams et al. \(1994\)](#) showed that neonates could distinguish red from gray but not blue, green, or yellow from gray.

So-called color-blind people make identifiable color discrimination errors, and they also confuse certain wavelengths with white. [Bornstein \(1976\)](#), [Teller et al. \(1978\)](#), and [Adams et al. \(1986\)](#) found that newborns, 1-month-olds, and 3-month-olds could discriminate blue-green from white and could discriminate between yellow and green, suggesting that infants are not green-blind, and even 1-month-olds are not blue-blind, a rare third kind of color blindness ([Varner et al., 1984](#)). In addition, the red/green and yellow/blue sensitivity of 1- to 3-month-olds is similar to that of adults ([Banks and Shannon, 1993](#); [Teller and Lindsey, 1993](#)). The development of color vision right after birth has not been studied very well, but it is clear that some sensitivity to color is apparent in most newborns and that these capacities improve quite rapidly.

Adults do not just see colors; they perceive the color spectrum as organized qualitatively into categories of hue, and the infant's color perceptions seems to be similarly organized. Infants as young as 4 months of age perceive the hues blue, green, yellow, and red categorically and in ways similar to those of adults ([Bornstein, 1981a](#); [Bornstein et al., 1976](#)). We do not know whether newborns also perceive color categorically because the phenomenon has not yet been studied at that age. However, after reviewing the physiologic, anthropologic, and psychological literatures, [Bornstein \(1981a, 1984\)](#) concluded that the ability to perceive color categorically probably is both universal and early appearing, reflecting the normal physiologic function of the visual system. Furthermore, there is evidence for a preference for chromatic over achromatic stimuli from birth, although preferences for particular colors do not emerge until the third month; unlike adults, 3-month-olds prefer red and yellow to blue and green ([Adams, 1987](#)).

## Auditory Perception

Even though audition is of major importance to the infant, much less is known about audition than about vision in early life ([Aslin et al., 1998](#)). Newborns can hear sounds: Make a sudden loud noise, and a neonate will startle. Further, acuity of hearing improves rapidly over the first few days after birth as amniotic fluid drains from the ear.

## BASIC AUDITORY PROCESSES

Sound is specified principally by two variables: frequency, the rate at which sound waves vibrate, and amplitude, the intensity of sound waves. Humans hear sounds in the frequency range of approximately 20 to 20,000 cycles per second, or hertz (Hz). For adults, auditory threshold varies with the frequency of the sound: Both low and high frequencies (above or below 1,000 Hz) require more energy than middle frequencies (around 1,000 Hz) to be heard. Infants clearly discriminate among sounds that differ in frequency, complicity, or pattern; they appear to hear low-pitch sounds less well and high-pitch sounds better than adults ([Olsho, 1984](#); [Schneider et al., 1980](#)), and even newborns prefer music over noise ([Trainor and Trehub, 1993](#)). Nearly continuous developmental improvements in hearing at low and high frequencies occur during the first 2 years ([Aslin et al., 1998](#)). Changes in ear structure or nervous system maturation could account for this increasing developmental sensitivity.

In addition, even though infants have smaller heads than do adults, and the locations of sound depends on the distance between the ears, infants are remarkably good at finding the sources of sounds in their environments. By 4 months of age, infants reach in the dark toward a sound source ([Clifton et al., 1994](#)), and by 6 months of age, localization responses are comparable with those of adults ([Clifton et al., 1991](#); [Hillier et al., 1992](#)).

## SPEECH PERCEPTION

Infants' excellent hearing abilities ([Aslin et al., 1998](#)) are applied almost immediately to the highly complex task of perceiving speech. In general, human speech is centered at relatively low frequencies, and numerous investigators have suggested that human speech is special for infants, who seem to be especially "tuned" to the speech register ([Eisenberg, 1976](#); [Fernald, 1984](#)). [Berg and Smith \(1983\)](#) found that even 6-month-olds are more sensitive to 500- and 2,000-Hz tones than to 8,000-Hz tones. In addition, infant-directed speech unusually includes higher pitch, more exaggerated intonation, more rhythmicity, and more repetition than adult-directed speech, and infants clearly maintain attention to infant-directed speech ([Kaplan et al., 1995](#); [Werker et al., 1994](#)).

The topic that has intrigued researchers most, however, has to do with the infant's ability to perceive special aspects of speech. Adults perceive differences in voicing more or less *categorically*; that is, although we can distinguish fine differences in the onset times of low- and high-frequency sounds, we tend to classify different examples of the same voiced (/b/) or voiceless (/p/) sounds as similar, but easily discriminate between voiced and voiceless sounds. Of course, different people say /b/ and /p/ in different ways, yet adult listeners seldom misidentify speech sounds because they use implicit category definitions to determine whether a sound falls within the /b/ or /p/ categories. Many people have assumed that phenomena so ubiquitous and significant as the categorical perception of speech sounds might have a biological foundation. To test this assumption, [Eimas et al. \(1971\)](#) asked whether preverbal infants perceive voicing categorically and found that they did: 1- and 4-month-olds distinguished between variants of /b/ and variants of /p/.

Not all languages use all voicing categories. [Eimas et al. \(1971\)](#) tested an English category distinction in American infants, but if categorical perception is innate, it ought to be universal, and infants from communities where different patterns of voicing prevail should all make the same categorical distinctions. In general, they appear to do so ([Bertoncini, 1998](#); [Plunkett and Schafer, 1999](#)). For example, Spanish has a voiced-voiceless distinction that is not in the same frequency location as the English one, yet Spanish 4- and 6-month-olds discriminate the English voiced-voiceless sound contrast rather than the Spanish one ([Lasky et al., 1975](#)). Likewise, Kikuyu (Kenyan) 2-month-olds categorize both an English voicing contrast not present in Kikuyu, as well as an idiosyncratic Kikuyu prevoiced-voiced contrast not present in English ([Streeter, 1976](#)). Interestingly, Spanish and Kikuyu adults perceive (although they do not use) the English voicing contrast, but they



perceive it only weakly; between infancy and maturity; therefore, a perceptual discrimination that is present at birth atrophies (but is not lost) if not used ( [Lively et al., 1994](#)). According to [Best \(1995\)](#) and [Jusczyk \(1995\)](#), 6-month-olds can discriminate among many phonetic contrasts but are already screening out categorical variations that are meaningless in their native language ( [Kuhl et al., 1992](#)). Thus, certain perceptions seem to be universal and developed at birth; they are maintained by linguistic experiences but may decay if absent from the language heard by the child, whereas other perceptions can be induced or altered in infants and children by exposure to certain speech sounds.

The existence of innate capacities to perceive speech sounds and color categorically underscores the perceptual competence of newborn infants. Together with tendencies to seek out stimulation and rapid developmental increases in perceptual acuity, they show that infants are designed for the efficient extraction of information about the environment. Acuity in the other perceptual modalities—the tactile and chemical (olfaction and gustation) senses—also is impressively good ([Brand, 1997](#); [Mennella and Beauchamp, 1996](#)), but unfortunately, these modalities have received little attention from researchers. The next section examines the ways in which infants build on their perceptual experiences by learning.

## CONDITIONING, HABITUATION, AND NOVELTY PREFERENCE

Learning “refers to a more or less permanent change in behavior which occurs as a result of practice” ( [Kimble, 1961](#), p. 2), and there has been considerable debate concerning the process and content of infant learning. Mechanistic theorists like Watson and Skinner believed that cognitive and emotional development depended on the infant’s ability to learn associations between contiguous actions or events. Moreover, they argued that any environmental stimulus could be associated with any response, given a sufficient amount of practice. By contrast, organismic theorists like Jean Piaget argued that learning could be understood only in light of the organism’s phylogenetic and experiential history. As is shown in this section, infants can and do form stimulus–response associations, but not between any stimulus and response. First, classical and operant conditioning and the conditions under which they have been demonstrated in human infants are discussed, followed by habituation and novelty preference and their use in investigating detection/discrimination, categorization, and concept formation.

### Classical Conditioning

The classical conditioning paradigm was developed by [Ivan Pavlov \(1927\)](#), who reported that the salivation response could be elicited in dogs by a ringing bell alone after several trials in which the bell was followed by placement of food in the dogs’ mouths. In this paradigm, salivation was the *unconditioned response*, which was reliably elicited by food powder, the *unconditioned stimulus*. The ringing bell initially had no effects on salivation and was said to be a *neutral stimulus*. However, the bell acquired the power to elicit salivation after being repeatedly paired with the presentation of food. The bell was then termed the *conditioned stimulus*, and salivation became the *conditioned response*.

Classical conditioning deals exclusively with reflexive and autonomic response systems over which the organism apparently has little control. Pavlov and his followers theorized about the importance of classical conditioning for the acquisition of language, culturally prescribed behaviors, normal and neurotic habits, and cognitive processes like thought and problem solving, but few today believe that classical conditioning plays a central role in the development of thought and language. However, many believe that it can help explain how specific emotions become associated with specific stimuli (such as in the development of phobias). Moreover, classical conditioning has provided important information about the types of stimulus–response associations newborns and older infants are “prepared” to make, allowing insights into how the infant mind is organized.

Classical conditioning is not easily demonstrated in newborns, but researchers have successfully conditioned sucking ( [Blass et al., 1984](#); [Lipsitt and Kaye, 1964](#)), heart rate ([Clifton, 1974](#); [Stamps and Porges, 1975](#)), the Babkin reflex ([Cantor et al., 1983](#); [Kaye, 1965](#)), pupillary reflexes ([Brackbill et al., 1967](#)), and head turning ([Clifton et al., 1972](#); [Siqueland and Lipsitt, 1966](#)). The difficulty in establishing conditioned responses in newborns probably is due in part to the rapidity with which newborns pass through different arousal states; state may influence the likelihood of response elicitation, latency, and magnitude ( [Fitzgerald and Brackbill, 1976](#); [Sameroff and Cavanagh, 1979](#)). [Clifton and Nelson \(1976\)](#) indicated that most periods of alertness in neonates last less than 10 minutes.

Questioning the traditional assumption that any stimulus could be associated with any response, [Seligman \(1970\)](#) suggested that organisms are prepared, unprepared, or contraprepared to associate specific stimuli and responses. Prepared associations are those that apparently require little learning and that are directly related to an organism’s survival (linking certain stimuli with feeding or food seeking). Unprepared stimulus–response combinations become associated only through learning, whereas contraprepared stimulus–response combinations are hard to associate no matter how extensive the attempts to condition them. [Sameroff and Cavanagh \(1979\)](#) argued that the most direct means of empirically distinguishing learned from prepared stimulus–response combinations is by examining the speed of association: Prepared associations, like those demonstrated in newborns, should occur rapidly, whereas learned associations should take more time. They concluded that newborns were not capable of learning by conditioning.

[Rovee-Collier and Lipsitt \(1982\)](#) argued in response that the ability to form stimulus–response associations rapidly is biologically adaptive for newborns, and thus that rapidity of association is not a sufficient criterion to judge whether learning has taken place. To bolster their argument, they noted that duration of sucking increases when followed by bursts of music of similar duration ( [Cairns and Butterfield, 1975](#)), demonstrating that changes in a biological response system can be effected by nonbiological events. According to [Rovee-Collier \(1996](#); [Weisberg and Rovee-Collier, 1998](#)), all infants are capable of conditioning, but the economics of the response demanded of the infants may sometimes require too great an expenditure of effort to be worthwhile.

After 3 months of age, classical conditioning is more easily demonstrated than in the newborn period, although the concepts of preparedness and biological constraints continue to be relevant. Instead of being able to associate any neutral stimulus with any response, infants appear predisposed to associate temporal conditioned stimuli (i.e., a specified interval of time) with autonomic responses (e.g., pupillary reflexes) and to associate tactile, auditory, and visual conditioned stimuli with somatic reflexes (e.g., eye blinks, sucking) ( [Fitzgerald and Brackbill, 1976](#); [Ivkovich et al., 1999](#)). Thus, specific classes of stimuli are most readily associated with specific classes of responses.

### Operant Conditioning

Unlike classical conditioning, in which a neutral signal comes to elicit an autonomic or reflexive response through repeated pairings with an unconditioned stimulus, operant conditioning increases or decreases the probability of a voluntary, nonreflexive “operant” response by controlling its consequences ( [Skinner, 1932](#)). The underlying principle is the “law of effect,” which states that actions followed by positive consequences (reinforcement) are more likely to be repeated, whereas actions followed by negative consequences (punishment) are less likely to be repeated.

Operant conditioning is more easily demonstrated in newborns than is classical conditioning, although here again the associations that have survival value are learned most readily. Among newborns, operant conditioning experiments typically involve sucking ( [Floccia et al., 1997](#); [Lipsitt and Kaye, 1965](#); [Sameroff, 1968](#)) or head turning ( [Siqueland, 1968](#)), two systems that are critical for newborn survival. After obtaining baseline measures of sucking rate for each 3-day-old infant, for example, [DeCasper and Fifer \(1980\)](#) divided their subjects into two groups. Infants in one group were presented with the sound of their mothers’ voice whenever the interval between successive sucking bursts was greater than the infant’s own average interval, and with the voice of another infant’s mother when the interburst interval was less than average. Infants in the second group heard their mothers’ voice when the intersucking interval was less than average, and the voice of another mother when the intersucking interval was greater than average. Eight of the 10 infants modified their sucking pattern to produce their own mother’s voice more often.

A basic principle of operant conditioning in infancy appears to be that neonatally conditionable responses are those that produce maximum gain for the infant at relative low energy cost ( [Rovee-Collier, 1996](#); [Weisberg and Rovee-Collier, 1998](#)). Higher-energy responses, such as foot kicking, do not appear to be conditionable until after the infant becomes capable of homeostatic thermoregulation, which occurs during the second month.

The propensity to learn associations that have functional significance and require minimal energy expenditure is illustrated by conjugate reinforcement ( [Hayne, 1996](#)), which involves making the frequency and intensity of reinforcement (e.g., the movement of a mobile) contingent on the frequency and intensity of a particular response (e.g., kicking). Learning is easy under conjugate reinforcement, suggest [Rovee-Collier and Gekoski \(1979\)](#), because the contingencies are similar to those that infants encounter most frequently in the real world. For example, intense infant cries are more likely to elicit responses than are weak cries, whereas intense sucking produces more milk than weak sucking. In addition, the behaviors lending themselves most readily to conjugate reinforcement impose low energy costs on the infant while providing information about the parameters that guide the infant’s manipulation of and control over the environment. In addition, merely perceiving the contingency between behavior and an environmental event becomes reinforcing for many infants ( [Weisberg and Rovee-Collier, 1998](#)).



## Habituation and Novelty Preference

Habituation refers to the progressive decline in responding to repeated presentations of a stimulus. The habituation task begins with the presentation of a stimulus, usually visual or auditory, to which the infant's attention is measured. Infants typically respond with longer attention spans during the initial presentations, presumably because the stimulus is novel, but attention declines over subsequent presentations. [Sokolov's \(1958/1963\)](#) theory of orienting and habituation, which stimulated a great deal of work on habituation, postulates that the degree of orientation to a stimulus is a direct function of whatever traces of that stimulus are in memory. The weaker the trace, the stronger the orienting reflex.

Few studies of habituation were conducted in humans before the 1960s, perhaps because it was viewed as a very primitive kind of learning, essentially unrelated to the association process considered central to perceptual, cognitive, and emotional development. In the 1950s and 1960s, however, research suggested that habituation depended on cortical processes that actually were more complicated than those involved in either classical or operant conditioning. For example, [Briullova \(cited in Lynn, 1966\)](#) found that habituation was severely disrupted in humans who had sustained cortical damage, and [Jouvet \(cited in Lynn, 1966\)](#) reported the complete absence of habituation in cats without a neocortex. The role of cortical processes in habituation is not yet clear, however. Research on anencephalic infant, for example, revealed substantial habituation of heart rate responses, suggesting that subcortical brain structures can mediate habituation in at least some instances ([Graham et al., 1978](#)). Other work on the habituation of visual attention in infants ([Cohen, 1973](#); [Jeffrey, 1976](#); [Lewis and Baldini, 1979](#); [McCall, 1971](#); [Olson, 1976](#)) suggests that habituation is a kind of "exposure" learning that reflects underlying brain plasticity. The habituation of visual attention, for example, seems to require some sort of mental representation, memory, internal comparison, and a variety of related perceptual and cognitive activities ([Rose and Tamis-LeMonda, 1999](#)).

Novelty preference involves a straightforward adaptation of the habituation paradigm. In this procedure, the infant is familiarized with two simultaneously presented exemplars of one stimulus for a fixed time and is then tested with a novel stimulus that is paired with the familiar stimulus. The proportion of time that the infant looks at the novel stimulus during the test trial is taken as a measure of novelty preference. Greater attention to the novel stimulus is believed to indicate a memory for the familiar stimulus.

Generally speaking, habituation and novelty preference are easily demonstrable in newborns ([Slater, 1995](#); [Slater et al., 1996](#)) and have been used more extensively than operant and classical paradigms to document individual differences and developmental changes in infant learning abilities. [Slater et al. \(1984\)](#) found habituation and recovery in infants as young as 3 days of age using both simple black-and-white and complexly colored stimuli. By 3 months of age, nervous system maturation and improved state organization ([Parmelee and Stern, 1972](#)) permit longer periods of alertness and improvements in learning ability and memory. These improvements continue with increasing age, along with concomitant developments in infants' information-processing abilities and motor coordination.

Habituation, novelty preference, and variants of these procedures have been used with infants to investigate the processes of detection and discrimination, categorization, and concept formation. Detection and discrimination are denoted by dishabituation when a novel stimulus is presented after habituation to the familiar stimulus. As described earlier, infants in the first few months of life successfully discriminate among various geometric forms, colors faces, sounds, odors, and phonemes ([Aslin et al., 1998](#); [Bushnell, 1998](#); [Kellman and Banks, 1998](#)). Categorization and concept formation (i.e., clustering a group of perceptually unrelated stimuli according to some underlying function, theme, or construct) usually are explored by habituating infants to several exemplars of a concept or category ([Hayne, 1996](#); [Oakes et al., 1997](#)). The formation of a concept or category is assumed when infants display habituation to familiar exemplars of the "old" concept or category, generalize habituation to novel exemplars of the old concept or category, and dishabituate to exemplars of an unrelated concept. Using this procedure, infants have been shown to form many different concepts or categories, including chromaticity, numerosity, gender, sounds, facial expressions ([Caron and Caron, 1981](#)), letters, furniture, and types of animals ([Bahrick et al., 1997](#); [Bornstein, 1981b, 1998](#); [Colombo et al., 1997](#); [Eimas, 1975](#); [Oakes et al., 1997](#); [Quinn, 1999](#); [Quinn and Bhatt, 1998](#); [Strauss and Curtis, 1981](#); [Younger and Fearing, 1999](#)). In general, it seems that infants first categorize using global categories within which they gradually recognize subcategories ([Mandler, 2000](#); [Quinn and Johnson, 2000](#); [Younger and Fearing, 2000](#)). The abilities to categorize items and to represent them internally are, of course, fundamental cognitive skills.

Overall, then, we see that infants are born with the ability not only to perceive objects and events but also to remember and associate them as well (see [Chapter 16](#)). Initially, memory span is quite limited, but improvements take place throughout the first year; within the first trimester, infants can remember an association for as long as 2 weeks, especially when prompted ([Rovee-Collier et al., 1980](#)). The easiest associations for infants to learn are those that are biologically prepared, and while these may comprise the limits of learning capacity in the neonatal period, older infants are capable of learning unprepared associations, especially when the energetic costs of responding are low. Perception and learning are the basic building blocks of cognitive development, the topic to which we now turn.

## INFANT INTELLIGENCE

Interest in the definition and assessment of infant intelligence stems from three conceptually distinct perspectives on the nature of intelligence. The Piagetians believe that intelligence exists on the "plane of action" for much of the infant's life, with true thought emerging only by the end of the second year. In the sensorimotor period of intelligence (0 to 24 months), six qualitatively different substages are distinguished (see [Chapter 12](#)). The psychometric perspective is most closely associated with the assessment of infant intelligence, guiding the construction of such measures as the Gesell and Bayley Scales (see [Chapter 40](#)). The following section selectively reviews research identifying environmental predictors of individual differences in infant intelligence and discusses the relation between effectance motivation and infant intelligence. The information-processing perspective on infant intelligence has evolved more recently and is concerned with how efficiently infants encode and store environmental stimuli in memory (see [Chapter 16](#)). It has been useful in sharpening our understanding of infant intelligence and its relation to intelligence measured at later developmental periods.

### Piaget's View of Infant Intelligence

Born in Switzerland in 1896, Jean Piaget was formally trained in biology and philosophy and was interested in the processes by which children acquire knowledge about the world. Piaget's interest in cognition stemmed from his early work in the Binet laboratory in Paris, France, where the first intelligence test was developed. Piaget found the general orientation and methodology of psychometric testing deficient in several respects. First, he found incorrect responses to be just as interesting as correct responses. Children of the same age frequently produced the same type of error; moreover, there seemed to be reliable age-related variations in the types of errors children made, variations that were qualitative, not quantitative, in nature. Second, the inflexible methodology used in administering these tests did not allow examiners to probe the "inner workings" of a child's mind. Piaget was interested in the thinking that guided children's answers and in how these processes changed with age.

Frustrated by psychometric procedures, Piaget developed a less structured means of questioning children and learned to tap thought processes by observing how children experiment with and solve simple problems. The latter approach was used extensively to probe for and document changes in intelligence during the first 2 years, using his own children as subjects. Piaget's observations and theoretical notions regarding the sensorimotor period appeared in three extremely influential books: *The Origins of Intelligence in the Child* (1936/1953), *The Construction of Reality in the Child* (1937/1954), and *Play, Dreams, and Imitation in Childhood* (1946/1962).

Piaget viewed development as a continuous process of adaptation to an environment in which the infant plays an active role. Piaget conceived of maturation as a primary driving force behind development. In Piaget's view, behavior is guided and organized around sets of psychological structures or "schemes" that become intercoordinated gradually. For example, the initially uncoordinated schemes of "looking" and "grasping" eventually become coordinated into a higher-order scheme of "visually guided reaching." According to Piaget, all infant schemes are action oriented; he considered infants incapable of true thought (imagery and the mental manipulation of symbols) until late in the second year. By contrast, older children and adults possess schemes that are thought oriented as well as action oriented.

In the view of Piagetians, adaptation to environmental demands depends on the complementary processes of assimilation and accommodation. Assimilation involves the application of an existing scheme to a novel external stimulus. By contrast, accommodation involves the modification of an existing structure to permit its application to a new, unfamiliar stimulus. For example, accommodation of the grasping scheme occurs when the infant attempts to grasp a novel object and has to modify the width of the grasp and the tightness of the grip. Of course, grasping the new object also involves some assimilation because a generalized grasping scheme already has been established. All actions are believed to involve both assimilation and accommodation.

Piaget believed that all infants passed through the six substages of the sensorimotor phase (0 to 24 months) in an invariant sequence, although there could be substantial individual differences in the rate at which infants negotiate each stage. The progression through the six substages is associated with a decline in

sensorimotor egocentrism, as the infant becomes increasingly aware that he or she is a unique entity, separate and distinct from other people and objects in the environment. Despite the fact that Piaget may have underestimated the age at which some abilities emerged (e.g., the intercoordination of perceptual schemes like looking and hearing appears to exist at birth), subsequent research has generally confirmed the developmental sequence he proposed. In addition, there is now widespread acceptance of the view that children are active participants in their development, not passive reactors to environmental stimuli. Key features of the six substages are provided in [Table 22.1](#).

Substage	Approximate Age	Major Developments	Key Concepts	Typical Activities
1	0-1 month	Reflexes, sucking, grasping, looking, hearing, crying, smiling	Reflexes, basic sensory-motor coordination	Feeding, holding, looking at faces, hearing voices
2	1-4 months	Primary circular reactions, reaching, grasping, sucking, looking, hearing, crying, smiling	Primary circular reactions, object permanence (beginning)	Feeding, playing with objects, looking at faces, hearing voices
3	4-8 months	Secondary circular reactions, reaching, grasping, sucking, looking, hearing, crying, smiling	Secondary circular reactions, object permanence (beginning)	Feeding, playing with objects, looking at faces, hearing voices
4	8-12 months	Tertiary circular reactions, reaching, grasping, sucking, looking, hearing, crying, smiling	Tertiary circular reactions, object permanence (beginning)	Feeding, playing with objects, looking at faces, hearing voices
5	12-18 months	Symbolic play, language, problem solving, social interaction	Symbolic play, language, problem solving, social interaction	Feeding, playing with objects, looking at faces, hearing voices
6	18-24 months	Symbolic play, language, problem solving, social interaction	Symbolic play, language, problem solving, social interaction	Feeding, playing with objects, looking at faces, hearing voices

**Table 22.1. Sequence of Some Important Sensorimotor Developments in Piagetian Theory**

The Piagetian perspective is not commonly associated with formal assessments of infant intelligence because Piaget was not concerned with assessing individual differences in intelligence or in identifying the conditions that speed up cognitive development. Of the few Piagetian tests that have developed, the [Uzgiris and Hunt \(1975\)](#) Ordinal Scales of Psychological Development have been most widely used (see [Chapter 40](#)).

### Environmental Correlates of Infant Intelligence

Interest in the links between infant intelligence and environmental quality stemmed from early studies demonstrating that older children from middle-class environments score significantly higher on IQ tests than do children from lower-class environments (see [Duncan and Brooks-Gunn, 1998](#); [Gottfried et al., 1995](#); [Huston, 1999](#), for reviews). Of course, socioeconomic status (SES) is a marker variable based on parental income, education, and occupation and reveals little about the types of experiences enjoyed by infants of different social classes. Compared with lower-class parents, middle-class parents experience qualitatively different family life stresses ([Hunt, 1969](#); [Ramey et al., 1976](#)), put greater emphasis on their children's motives, feelings, self-direction, and academic success ([Hoff-Ginsberg and Tardif, 1995](#); [Kohn, 1987](#)), are more aware of their influence on their children's intellectual development ([Tulkin and Kagan, 1975](#)), provide more verbal and object-mediated stimulation contingent on infant behavior ([Hoff-Ginsberg, 1991](#); [Hunt, 1969](#); [Kagan and Tulkin, 1971](#); [Klaus and Gray, 1968](#); [Tulkin, 1977](#); [Tulkin and Kagan, 1975](#); [Wachs et al., 1971](#)), provide less punitive modes of discipline ([McLoyd, 1998](#)), and provide a greater variety of daily experiences ([Kagan et al., 1978](#)). Interestingly, reliable SES differences in standardized measures of infant intelligence are more easily demonstrated after rather than before 12 months of age ([Ramey et al., 1982](#)), suggesting that these tests are not as sensitive to variations in environmental stimulation during the first year as during the second. Infant intelligence tests during the first year tap sensory and motor functions whose development may be more influenced by maturational rather than by environmental factors, whereas environmental quality may have more readily observable effects on functions like language, symbolic play, and problem solving, which emerge during the second year. Environmental quality during the first year is important, but enduring environmental influences have the most pervasive and lasting impact on intellectual development.

By the second year, SES differences in parental behavior and in infant intelligence suggest that cognitive performance is in part associated with the quality of infant–parent interaction. However, there also is considerable variability in the cognitive outcomes of infants of lower SES, highlighting the importance of identifying specific social experiences that reliably predict intellectual performance within and across social class. Global measures of maternal involvement, verbal responsiveness, organization of the physical environment, variety in daily stimulation, and provision of play materials at 6 months predict 3-year intellectual performance ([Bradley and Caldwell, 1976](#); [Elardo et al., 1975](#)), and intellectual performance after the first year is predicted by earlier measures of mothers' verbal stimulation, positive toy play, and contingent responsiveness to infant social cues ([Clarke-Stewart et al., 1979](#); [Cohen and Beckwith, 1979](#); [Feldman and Greenbaum, 1997](#); [Tamis-LeMonda et al., 1996](#)). Mothers who respond to their toddlers in an “options-promoting manner” encourage their children to play in more sophisticated ways ([Stilson and Harding, 1997](#)). [Belsky et al. \(1980\)](#) speculated that dyadic play with toys may foster the ability to engage in competent, self-directed explorations, and [Lewis and Goldberg \(1969\)](#) suggested that positive social experiences that are contingent on infant behavior facilitate cognitive growth by fostering the sense of control over and motivation to master the environment. Eight-week-olds who perceived a contingency between their head movements and the movement of an overhead mobile not only increased the frequency of their head movements but showed a much higher frequency of smiling and cooing, an apparent indication of the pleasure derived from perception of the contingency ([Watson, 1972](#)). Conversely, infants may show displeasure and distress when their efforts to elicit contingent feedback are violated, as when mothers respond noncontingently to distress and positive social bids ([Lamb and Malkin, 1986](#); [Trevarthen, 1977](#)). Competent infants, therefore, are those who have learned from past experiences of contingency to explore, adapt to, and control their environments ([Goldberg, 1977](#)).

Studies by Bornstein and his colleagues revealed that the types of activities and experiences provided by parents influence specific areas of mental growth, such as visual, tactual, and perceptual–cognitive competence in infants younger than 6 months, and language and play in 13-month-olds ([Bornstein, 1989](#); [Bornstein and Tamis-LeMonda, 1997](#); [Tamis-LeMonda et al., 1996](#)).

### Continuity of Infant Intelligence

Despite the environmental correlates of intelligence in the second year, the relation between measures of intelligence in infancy and in later life is not at all straightforward. With few exceptions, correlations between measures of intelligence during the first year and IQ scores in later childhood are very low ([Harris and Langkamp, 1994](#); [Palti and Adler, 1994](#); [Wyly, 1997](#)). Prediction from scores obtained in the second year of life is somewhat better ([Reich et al., 1984](#); [Wilson, 1983](#)), perhaps because by that time scales like the Bayley Mental Development Index tap verbal, symbolic, and problem-solving skills that are similar to the abilities assessed by childhood intelligence tests. In addition, predictive relations tend to be higher for high-risk infants ([Siegel, 1989](#)).

The lack of relation between infant and later intelligence has spurred a heated debate about the nature of intelligence across the life span. Some theorists argued that there was no general intelligence factor, *g*, whereas others argued that if *g* exists, it is not fixed or stable across the life span. Still others argued that *g* may exist but that infant intelligence, with its basis in sensory and motor activity, differs from later intelligence. In any case, being highly intelligent in infancy would not ensure high intelligence later (see [Eysenck and Kamin, 1981](#); [Kopp and McCall, 1980](#); [Lewis, 1983](#); [Vernon, 1980](#), for discussions of these issues). A third perspective on intelligence, the information-processing view, demonstrates that the apparent discontinuity in intelligence from infancy to later periods is due in part to the conceptual dissimilarity between the abilities tapped by standardized tests administered to infants and older children. When conceptually similar abilities are assessed on both occasions, the degree of prediction improves.

### Information Processing and Infant Intelligence

The information-processing view of intelligence holds that attention is an important aspect of infant cognitive functioning, conceptually related to measures of intelligence across the life span ([Rose and Tamis-LaMonda, 1999](#)). Recall from our earlier discussion of habituation and novelty preference that infant attention to a stimulus event traditionally has been indexed in two ways: decrement of attention to repeated presentations of the same stimulus (habituation) and recovery of attention to a novel stimulus after habituation (novelty preference). Efficient information processing is assumed when habituation is rapid or when novelty preference is especially marked ([Rose and Tamis-LaMonda, 1999](#)).

Reliable individual differences in habituation rates in infants younger than 6 months have been reported using such stimulus configurations as geometric shapes, multicolored drawings, and social patterns ([Bornstein and Benasich, 1986](#); [DeLoache, 1976](#); [McCall, 1979](#)). Further, [McCall \(1979\)](#), and later, [Bornstein and Benasich \(1986\)](#) identified three basic habituation patterns in 5-month-olds: infants who habituated rapidly, infants who first increased looking and subsequently habituated, and infants who showed idiosyncratic and highly individualized looking patterns. These individual differences covary with various other measures of infant cognition. For



example, rapid habituation in infancy and childhood is positively associated with preference for complex patterns ( [Greenberg et al., 1973](#)), advanced sensorimotor development ( [Johnson and Brody, 1977](#)), competent exploration of the environment ( [Tamis-LeMonda and Bornstein, 1989, 1993](#)), sophisticated play ( [Kagan, 1971](#); [Riksen-Walraven, 1978](#)), and rapid problem solving ( [Lewis et al., 1969](#)). In addition, perinatal risk and developmental disability adversely affect decrement of attention and its recovery ( [Carlson and Werkman, 1996](#); [Jacobson et al., 1993](#); [Rose, 1994](#); [Rose et al., 1998](#); [Zelazo and Stack, 1997](#)).

Of course, infants also show individual differences in standardized test performance before 12 months of age, but the predictive validity of the attentional measures is better than that for the more traditional infant measures. Several recent studies document that infants who show efficient decrement or recovery of attention in the first 6 months of life perform significantly better on traditional assessments of cognitive and linguistic competence in early to middle childhood ( [Bornstein et al., 1997](#); [Colombo, 1993](#); [Dougherty and Haith, 1997](#); [Fagan and Haiken-Vasen, 1997](#); [McCall and Mash, 1995](#); [Rose et al., 1998](#); [Sigman et al., 1997](#)). There also is evidence to suggest that infant novelty responsiveness across modalities, such as being familiarized with a stimulus tactually and then distinguishing a novel stimulus from the familiar one visually, also is predictive of later cognitive competence ( [Rose et al., 1998](#)).

In a comprehensive review, [Bornstein and Sigman \(1986\)](#) noted that the median percentage variance shared between infant attentional measures and childhood intelligence is 21% (a correlation of approximately 0.44). Although the predictive validity of attentional measures is far from perfect, it certainly is better than that of traditional tests. Moreover, it indicates that, when researchers choose measures of early intelligence that tap the same processes as the measures used to assess intelligence later, some continuity is found.

## COGNITIVE CONTINUITY AND THE NATURE–NURTURE ISSUE

It is virtually impossible to demonstrate either an environmental or a genetic basis for any characteristic in children reared by their biological parents because genetic endowment and environmental influences are so closely intertwined ( [Scarr and Weinberg, 1983](#)). The significant correlations between parental IQ and infant intelligence ( [Rose et al., 1979, 1980](#)) could be due to a shared gene pool or to parental behavior. There is some evidence, however, that there are inherent individual differences in infant attention. Newborns show individual differences in habituation ( [Field et al., 1982](#); [Friedman, 1975](#); [Siqueland, 1981](#); [Slater et al., 1984](#)), and identical twins are more alike in the developmental pattern of scores on standardized tests from 3 months to 6 years than are fraternal twins ( [Wilson, 1983](#)). In addition, [Hardy-Brown et al. \(1981\)](#) found that the linguistic and communicative competence of adopted infants at 12 months of age related more strongly to the cognitive abilities of their biological mothers than to the cognitive abilities of the adoptive parents or to measures of the adoptive parents' communicative styles.

The fact that individual differences exist in relatively homogeneous samples, however, suggests that at least some variation is not accounted for by heredity, and several investigators have found concurrent associations between parental behavior and infant attention. For example, [Bornstein \(1985\)](#) found that mothers' tendency to encourage infants to attend to properties, objects, and events in the environment was related to habituation efficiency in 4-month-olds. [Sigman and Beckwith \(1980\)](#) reported relations between fixation time in 4-month-old preterm female infants and concurrent measures of verbal and social interaction. Of course, correlations between contemporaneous variables preclude conclusions about causal influences, but studies designed to help identify causal patterns suggest that the direction of influence is from parent to child, not the reverse. [Belsky et al. \(1980\)](#), for example, showed experimentally that parents who focus their infants' attention on objects promote infant competence. In addition, [Riksen-Walraven \(1978\)](#) demonstrated that infants of mothers who participated in a stimulation program habituated more efficiently than infants of control mothers.

It appears, then, that both genetic and environmental factors influence information-processing capacities. Of course, no direct measures of “genetic quality” exist, rendering moot questions about how much of an individual contribution genetics and environment actually make to intelligence. As [Yeates et al. \(1983\)](#) point out, however, questions about the relative contributions of genetics and environment to a developmental process can be asked using measures such as parental IQ, which presumably reflects some genetic variation. [Yeates et al. \(1983\)](#) examined the relative contributions of maternal IQ and measures of environmental quality of the home (e.g., maternal involvement, provision of play materials) to child IQ from 2 to 4 years of age, statistically controlling for the effects of one while assessing the effects of the other at various age points. They found that environmental quality became a more important predictor and maternal IQ a less important predictor over the 2-year period. Although infant cognitive capacities were not assessed, the results suggest that early variation in cognitive abilities may be more attributable to genetic than to environmental factors, with environmental factors playing an increasing role with age.

Although the predictive validity of infant attention to childhood intelligence is better than that of more traditional tests, [Bornstein and Sigman \(1986\)](#) reported predictive correlations ranging in absolute magnitude from 0.28 to 0.77. Some of this variation may be due to differences among the techniques used to measure attention, the measures used to assess childhood intelligence, the intervals between the ages at which infant attention and later intelligence were assessed, and measurement error. However, a potentially important source of variance that has not yet been addressed systematically is the degree of stability in children's environments from infancy to the later assessment. [Lewis and Goldberg \(1969\)](#) have argued that effectance motivation, and hence cognitive development, is facilitated by social environments that are emotionally warm, sensitive, and contingently responsive to children's needs. Further, [Bond \(1982\)](#) has noted that pervasive effects on cognitive development seem to occur when environmental influences also are enduring and long lasting. Thus, the predictive validity of infant attention measures should be relatively high when environmental stability over time also is high, whereas weaker predictive validity should be found when there are fluctuations in environmental quality (e.g., variations in the quality of the parent–child relationship).

Overall, the qualitative (Piagetian) and quantitative (information-processing and psychometric) perspectives on intelligence are complementary, addressing different questions about infant cognition. Although the Piagetian approach has proven useful for normative descriptions of infant cognitions, the information-processing approach is clearly in the ascendancy today, whereas the psychometric approach remains useful for assessing the current developmental status of individual infants. Except at the extremes, however, these scores have no predictive validity, whereas measures of information processing are predictive of performance on later measures of symbolic and analytic functioning. Perhaps the most dramatic aspect of symbolic function is represented by language, the topic of the next section (for a general overview of the development of communication, see [Chapter 17](#)).

## LANGUAGE DEVELOPMENT

As mentioned earlier, very young infants seem to be especially tuned to frequencies within the range of human speech sounds. Infants like to listen to voices and will perform some act in order to do so: When speech (but not other sound) is contingent on nonnutritive sucking, the amount of sucking increases ( [Trehub and Chang, 1977](#)). In addition, 4- to 16-week-old infants are able to perceive categorically many of the acoustic cues that underlie speech ( [Aslin et al., 1983](#); [Eimas et al., 1971](#)). Thus, it appears that young infants are primed for hearing the components of language. These perceptual capacities presumably play a role in shaping the infant's vocal output, facilitating the development of language.

### Vocal Production

During the first month of life, infants make reflexive cries, fusses, and vegetative sounds ( [Lenneberg, 1967](#); [Oller, 1980](#); [Tal and Bornstein, 1992](#)). As early as 5 weeks to 3 months of age, cooing and laughter emerge, typically in response to the voices and faces of others ( [Lenneberg, 1967](#)). These cooing sounds involve repeating the same vowel or velar consonant (“g,” “k”), sounds in which tongue movements are relatively undifferentiated ( [Kent, 1981](#)). Between 4 and 6 months of age, infants explore a variety of vocalizations, such as squeals, yells, growls, and vowel-like sounds. Thereafter (7 to 10 months of age), canonical or reduplicated babbling occurs, with infants producing syllables and reduplicating the same consonant and vowel (e.g., “da, da, da”). Between 11 and 12 months, nonreduplicated babbling (also termed *jargon* or *protowords*) emerges; at this stage, infants produce a variety of sounds in which syllables, consonants, and vowels may vary, such as “dakee,” “babe.” Finally, at approximately 12 months, infants produce referential words.

Changes in these vocal abilities are paralleled by changes in the anatomy and physiology of the vocal apparatus, which suggests that the developmental changes in vocal behavior are related to structural changes in oropharyngeal anatomy ( [Kent, 1981](#); [Lester and Bourkydis, 1991](#)), as does evidence that initial sound production follows a universal pattern ( [Jakobson, 1941/1968, 1971](#); [Vihman, 1991](#)). Infants brought up in Spanish-, Japanese-, Arabic-, or English-speaking homes show no great differences in their basic sound production repertoire, suggesting that very different linguistic experiences do not affect the elements of babbling very much ( [Nakazima, 1975](#); [Oller and Eilers, 1982](#); [Vihman, 1993](#)). In addition, infants with little language experience (e.g., infants with deaf parents) vocalize much like infants in sound-rich environments until they are approximately 6 months old ( [Lenneberg, 1969](#)). Thus, infants appear to use a universal core of sounds.

This does not mean that sound production is unaffected by environmental input: Infants eventually do produce the sounds they commonly hear, and they correctly mimic the rhythm and intonation of their native language long before they know what the words mean ( [Crystal, 1979](#); [de Boysson-Bardies et al., 1984](#); [Weir, 1962](#)). Routh (in [Oller, 1981](#)) stimulated 2- to 7-month-olds with either consonant- or vowel-like sounds and thereby selectively increased the number of consonants or vowels the infants produced. By contrast, [Webster \(1969\)](#) found that when 6-month-olds were stimulated with vowel sounds, they increased the proportion of



consonants produced, and vice versa. Taken together, these studies suggest that infants can modify their own vocalizations in response to the vocalizations of others, by either matching or contrasting the sounds that they hear ([Oller, 1981](#)).

The amount and patterning of babbling is affected by environmental factors as well. Infants vocalize substantially more when they are socially stimulated by others than when they are not, regardless of whether the stimulation is contingent on the infants' own vocalizations ([Bloom, 1979](#); [Rheingold et al., 1959](#)). In addition, when adult vocalizations or adult silences are contingent on infant vocalizations, infants suppress their own vocalizations just after the contingent stimulation or silence, as though listening for something. Responding contingently to infant vocalizations thus allows infants to learn the basic conversational pattern: talk, then listen ([Bloom, 1979](#); [Bloom and Esposito, 1975](#)).

The influence of social input on sound production is highlighted in studies contrasting the vocalizations of deaf infants with those of normal-hearing infants; the former appear to fall behind the latter ([Gilbert, 1982](#); [Oller, 1981](#); [Stoel-Gammon and Cooper, 1984](#)). Hearing-impaired infants produce fewer consonant types, show a decrease in types over time, and use a lower proportion of multisyllabic utterances than normal-hearing infants ([Oller and Eilers, 1988](#)). It, thus, appears that in the first half-year of life, infants do not require acoustic information to produce common precanonical sounds, but that the acoustic environment does influence vocalizations, such as babbling, later. Babbling, however, is not restricted to vocalizations. When deaf infants are exposed to sign language from birth, they babble manually in much the same way that hearing infants do through speech ([Petitto and Marentette, 1991](#)).

### First Words

Gesell ([Gesell and Armatruda, 1962](#)) observed that children regularly say their first words approximately the time they celebrate their first birthday. At this age, children are interested in movements and actions they can perform, both with and without objects, and their first words typically reflect their sensorimotor schema; often they refer to objects that move or can be held rather than to equally common words on which infants seldom act (e.g., diaper, crib, shoe) ([Nelson, 1973](#)). In addition, early words (e.g., bye-bye, bang) often express and are accompanied by actions (e.g., waving, hitting) ([Corrigan, 1979](#)).

Although many early words refer to objects ([Benedict, 1979](#); [Bowerman, 1976](#); [Leopold, 1949](#)), infants also use “expressive” words to engage in social interactions. [Nelson \(1981\)](#) found that infants' early vocabularies consisted of both kinds of words and that infants could be categorized according to the proportion of each in their repertoires. For most children, early vocabularies contain a large proportion of object names, with some verbs, proper names, and adjectives; Nelson termed this style of early word use *referential*. A large minority of children, however, have a more diverse vocabulary, with a large number of social routines, such as “stop it” and “what’s that” included among the nouns, verbs, and adjectives; Nelson called this style of early word use *expressive*. Although children may use one style more than the other, only rarely do they exclusively use either one ([Bretherton et al., 1983](#)).

In addition to individual differences in the style of first words used, individual differences in the timing of early word acquisition are common. [Bayley \(1969\)](#), in her assessment of 1,200 infants, found that the most advanced imitated a word by 9 months of age, and the least advanced by 18 months of age. Similar variation was found in the number of words in the vocabularies of 13-month-olds; production ranged from 0 to 45 words, and comprehension varied from 11 to 97 words ([Snyder et al., 1981](#)). In general, comprehension of words precedes their production.

### Sharing a Referent: Development of Gestural Communication

Producing and comprehending words necessitates an understanding of shared referents. Before language, such shared reference to objects and events first comes about through gestural communication. During the second half of the first year, infants begin to use and understand various communicative signals to potentiate mutual attention to external topics ([Bakeman and Adamson, 1984](#)). Joint attention and external reference are the bases of symbolic communication, be it gestural or vocal ([Bates, 1979](#); [Schaffer, 1984](#)). There is some evidence indicating that infants who experience more of these joint attention experiences tend to talk earlier and show faster vocabulary development ([Dunham and Dunham, 1992](#); [Tomasello, 1990](#)).

Toward the end of the first year, infants begin to share objects and events with others by using conventional gestures such as pointing, offering, and showing objects ([Hay and Rheingold, 1983](#)). [Leung and Rheingold \(1981\)](#) note that pointing and reaching function to direct another's attention to objects that arouse the infant's interest. Reaching is characteristic of 9- to 10-month-olds, entering the repertoire before pointing, which is first displayed by most children at 12.5 months and is a common gesture at 16.5 months of age ([Leung and Rheingold, 1981](#)). Because pointing is modeled and reinforced by adults, it replaces reaching as a referential gesture ([Leung and Rheingold, 1981](#); [Murphy and Messer, 1977](#)), gradually being refined until it assumes the conventional form. Language acquisition involves a similar process of learning the conventional means by which to refer to phenomena (Acredolo and Goodwyn, 1990). As with language, the ability to comprehend or to follow another's points precedes the infant's own pointing. By the start of the second year of life, infants frequently use conventional gestures (such as showing and offering objects) with mothers and fathers, as well as with unfamiliar men and women ([Hay, 1979](#); [Rheingold et al., 1976](#)) and with peers ([Bakeman and Adamson, 1986](#); [Eckerman et al., 1975](#); [Nash, 1985](#)).

Conventional gestures and language follow a parallel developmental course. Infants begin to use shared referents, both gestural and verbal, at approximately 1 year of age, the age at which Piaget suggests that the capacity for symbolic representation first emerges. Other forms of symbolic representation, such as play, also emerge at approximately this time ([Nicholich, 1981](#)). In addition, social gestures (such as pointing) and speech both pass through an initial noncommunicative phase ([E. Bates et al., 1982](#); [Leung and Rheingold, 1981](#); [Schaffer, 1984](#)). Both are first tied to specific routines or “action formats” (routines between adult and infant such as peek-a-boo, “So Big,” and reading) and occur only in these contexts ([Acredolo and Goodwyn, 1985, 1990](#); [Bakeman and Adamson, 1984](#); [Bates, 1979](#); [Ratner and Bruner, 1978](#)). Later, they are used in other contexts to stand for objects and events that are not present, thus serving a symbolic communicative function.

### Prelinguistic Conversations and Behavioral Dialogues

Besides helping infants understand semantics, interactive routines also help infants learn the pragmatics of language and the rules of conversation, such as mutual attention, sharing a topic, and taking turns ([Bruner, 1977](#); [Schaffer, 1984](#)). Dialogues between infants and caretakers begin in the newborn period: The communicative exchanges between parents and 3-day-old infants are smooth, with a high degree of turn taking ([Bakeman and Brown, 1977](#)). At this stage, however, adults more frequently initiate, follow through, and complete behavioral sequences.

At 2 months of age, infants regularly respond to attentive, talking adults by orienting to their faces, focusing on their eyes, smiling, becoming more active, and vocalizing, then shifting their gaze to the adults' mouths or away from their faces ([Rheingold, 1961](#); [Trevarthen, 1977](#)). Infants, thus, seem to alternate between attending and expressing themselves. During the expressive phase, they make mouth movements, often accompanied by sounds and gestures. Mothers frequently respond to such prespeech with talk of their own. By 3 months of age, infants and parents tend to alternate their vocalizations, taking turns ([Bloom, 1988](#)). Mothers are primarily responsible for initiating exchanges and attempting to elicit responses from infants, but infants at this age sometimes vocalize after silences, as if attempting to keep the “conversations” going. Turn taking becomes more and more refined and predominates in the vocal exchanges of 6- to 16-month-olds and their mothers ([Davis, 1978](#)). Similarly, [Schaffer et al. \(1977\)](#) found that turn taking occurs equally often in the vocal exchanges of 12- and 24-month-old children. At both ages, overlaps were rare and brief.

During the first 6 months, conversations and dialogues are sustained mainly by adults, but after this, infants begin to take more responsibility. [Rogoff et al. \(1984\)](#) observed 7-month-olds reaching toward an adult's arm while looking at the handle of a jack-in-the-box, as though attempting to make the adult wind the handle and thus continue the game. The role of infants in the management of joint activities was shown to increase rapidly from 6 to 13 months ([Mosier and Rogoff, 1994](#)). [H.S. Ross \(1980\)](#) reported that, when an adult stopped a game by not taking her turn, 12-month-olds reacted as though they had some understanding of the rules of turn taking; for example, they guided the adult's hand to the appropriate object, thus helping the adult to take her turn. The infant's role in taking turns is illuminated by behavior with peers from the start of the second year, when “games” may include turn taking ([Goldman and Ross, 1978](#); [Nash, 1986](#)).

[Snow \(1977\)](#) suggests that 1-year-olds understand the rules of conversational turn taking as well, and [Schaffer \(1984\)](#) proposed a mechanism by which even very young infants help sustain a vocal dialogue: They suppress their own vocalizations when stimulated by the sounds of others. [Webster et al. \(1972\)](#) found that infants vocalize less when presented with other sounds and increase them when the sounds stop. Furthermore, infants reduce their own vocalizations, particularly when they hear preferred voices ([Barrett-Goldfarb and Whitehurst, 1973](#)).

Although 12-month-olds understand some rules of social exchange and take an active role in keeping exchanges going, adults still take the major responsibility. [Schaffer et al. \(1977\)](#) examined speaker-switch pauses in vocal interchanges between mothers and 1-year-olds and found that child–mother switches were characterized by shorter pauses than mother–child switches, thus highlighting the mothers' greater competence in responding. In addition, by keeping their own



vocalizations very brief and pausing often, parents give infants a chance to join in ( [Schaffer, 1984](#)).

That vocal and behavioral interactive routines follow the same developmental course is illustrated by [Bruner's \(1977\)](#) descriptions of "Give and Take" games and [Snow's \(1977\)](#) examinations of vocal conversations. At 3 months, Bruner found the game of "Give and Take" to be one sided, with mothers offering objects to their infants most frequently. At 6 months, the infants played a more active role, accepting the objects. By 12 months, children both gave and took objects. The number of exchanges and offers of objects initiated by the children increased from 0% to 50%, so that "Give and Take" became a game involving reciprocal roles. Similarly, Snow found that mothers took the major responsibility for maintaining conversations by responding to their 3-month-olds' smiles and vocalizations as if they were intending to communicate. When infants did not respond, mothers often filled in. By the end of the first year, children more often initiated turns, actively assuming the roles of speaker and listener.

Early interactive routines may thus teach the basic pragmatics of speech. In addition, [Bruner \(1977, 1983\)](#) suggested that they also provide a strong foundation on which infants build syntax, while learning the sequence of agent, action, object, and recipient. Thus, Bruner argues that, before actually speaking, infants, with adult guidance, have learned language formats. This language support system may thus provide a stepping stone to [Chomsky's \(1965, 1986\)](#) notion of a language acquisition device, an innate mechanism that provides toddlers and preschoolers with an understanding of grammar.

Gestures, nonverbal sounds, and words are important means whereby infants communicate with others. Emotional expressions, discussed in the next section, also serve important communicative functions. Whether the signals are gestural, verbal, or emotional, furthermore, their prevalence and universality underscore the intrinsically social nature of human infants.

## SOCIAL AND EMOTIONAL DEVELOPMENT

### Emotional Development

Many theorists consider the ability to produce and respond appropriately to emotional expressions to be innate and cite four types of evidence to support this view ([Campos et al., 1983](#); [Darwin, 1872](#); [Eibl-Eibesfeldt, 1970](#); [Ekman and Oster, 1979](#); [Izard, 1971, 1979](#)). First, facial expressions are indeed quite similar in different cultures ([Camras et al., 1992](#)), and expressions posed by Westerners are correctly identified by people in other cultures, including preliterate societies that have had little or no exposure to Westerners ([Ekman, 1994](#)). Second, the development of emotional expressiveness seems to be under maturational control; blind infants begin full smiling at the same age as sighted infants ([Fraiberg, 1979](#)), and premature infants begin to smile at environmental stimuli at the same conceptional age as full-term infants ([Dittrichova, cited in Bower, 1977](#)). Third, monozygotic twins are more alike than dizygotic twins with respect to fear of strangers and smiling over the first 4 months ([Freedman, 1974](#); [Plomin and Rowe, 1978](#)), suggesting a genetic component to these emotions. Finally, the ability to recognize certain expressive displays has an innate basis in nonhuman primates. [Sackett \(1966\)](#) found that rhesus monkeys raised in total isolation responded with fear to pictures of monkeys making threats and showed positive reactions to pictures of infant monkeys. [Mason \(1985\)](#) found that experience has little influence on several facial and vocal emotional displays in rhesus monkeys.

To study the development of emotions, the investigator must decide whether to focus on internal state or on outward expression because there may not be a direct correspondence between the two ([Saarni et al., 1998](#)). Some theorists assume a correspondence ([Darwin, 1872](#); [Ekman 1972](#); [Izard, 1979](#); [Malatesta, 1985](#)), and there is some physiologic evidence to support this assumption. Correlations have been found between various facial expressions and distinct patterns of autonomic nervous system activity in adults ([Ekman et al., 1983](#)), and between the amount of separation distress ([Tennes, 1982](#)) and distress to inoculations ([Lewis et al., 1993](#)) and levels of infants' cortisol production. Other researchers have emphasized the lack of synchrony between infant expressions and internal states, however. Citing examples of early asynchrony in relation to infant smiling ([Emde and Koenig, 1969](#); [Wolff, 1963](#)), [Lewis and Michalson \(1985\)](#) argue that the relation between internal state and outward expression during infancy is in fact U-shaped, with no synchrony at first, a high degree of synchrony later in infancy, and then less correspondence as infants get old enough to use expressions to communicate intentionally or withhold them to mask certain states. This concern about the correspondence between emotional state and expression is important not only to theorists but to parents and caretakers, who must understand infant signals and act appropriately, deciding whether such signals necessarily correspond to particular emotional states.

To clarify the relation between expressions and underlying emotions, it is helpful to ask about the likely function of a correspondence between feelings and expressions. [Campos and colleagues \(1983](#); [Saarni et al., 1998](#)) argue that emotions play a primary role in organizing the individual's behavior and signaling others about the individual's tendencies. They assume that the communicative functions of emotions are evident from birth and that there is, as a result, a direct although imperfect correspondence between emotional expressions and internal "feeling" states.

## DEVELOPMENT OF FACIAL AND VOCAL EXPRESSIONS

By responding "appropriately" to infants' facial and vocal expressions, parents indicate that infants are able to communicate effectively with others. The same facial movements used to express distinct emotions in adults are used by infants ([Ekman and Friesen, 1976, 1978](#); [Izard and Dougherty, 1982](#)). As a result, even untrained observers can accurately identify the emotion displayed by photographed infants ([Emde et al., 1978, 1985](#); [Izard et al. 1980](#)) and show differentiated psychophysiological responses to infant smiles and cries, as well as to cries with different characteristics ([Frodi et al., 1978a, 1978b](#)). As infancy proceeds, expressions change from basically reflexive signals to more flexible, graded, instrumental signals, which may be used intentionally as well as unintentionally in social communication ([Thompson et al., 1998](#)). Indeed, one of the tasks of infancy is to modify expressions in light of societal expectations and rules. The frequency of facial expressions also changes, with decreases over the first 6 months ([Malatesta and Haviland, 1982](#)) and increases in the next 2 months ([Malatesta et al., 1986](#)).

The first emotional expressions involve crying. [Brazelton \(1962\)](#) and [Rebelsky and Black \(1972\)](#) have charted the developmental course of crying, noting an increase over the first 6 weeks and then a decrease, with maximum crying initially in the evening, but later near feeding times. Both researchers also noted much variability in and between infants. The communicative function of crying is indicated by the ability of others to identify different kinds of cries. Adults are able to distinguish among and interpret the cries, not only of 6-month-olds, but of 1-month-olds and newborns ([Leger et al., 1998](#); [Thompson et al., 1998](#); [Thompson and Leger, 1998](#); [Zeskind et al., 1985](#)). It also is possible to distinguish spectrographically among the cries of newborn infants with malnutrition, asphyxiation, neurologic abnormalities, and chromosomal deviations ([Hollien, 1980](#); [Lester and Boukydis, 1985](#); [Zeskind, 1981](#)).

Newborns can communicate emotions through facial expressions as well ([Oster and Rosenstein, 1998](#)). [Rosenstein and Oster \(1988\)](#) recorded the facial expressions of 2-hour-old infants who were given sour, sweet, salty, and bitter substances to taste. They responded to pleasant tastes with apparently positive facial expressions and to unpleasant tastes with negative expressions. [Field \(1982\)](#) reported that facial expressions of surprise were elicited from newborns by some items in the Brazelton Neonatal Behavioral Assessment Scale examination. And, of course, newborns are able to smile, although unlike later smiling, which involves both ocular and lower face muscles, neonatal reflex smiling involves only the lower face muscles ([Korner, 1969](#); [Wolff, 1963](#)). Newborns smile quite readily, but only during rapid eye movement states in sleep ([Emde and Harmon, 1972](#)). According to [Sroufe and Waters \(1976\)](#), who charted the ontogeny of smiling and laughter, smiling progresses from internal to external stimulation. At first it occurs during sleep, later in drowsiness, and finally in attentive states. Sounds are initially the most effective external stimuli, but by the fifth week, visual stimuli become more effective. Six- to 12-week-old infants smile at faces, as well as at a wide range of nonsocial stimuli, such as bull's-eye patterns and bells ([Emde and Harmon, 1972](#)). Gradually, smiling becomes more selective, progressing from any human face, to particular faces, to particular expressions. Newborns do not laugh; the development of laughter trails the developmental course of elicited smiling by approximately 1 month ([Rothbart, 1973](#)). [Sroufe and Wunsch \(1972\)](#) reported that tactile and kinesthetic cues were the strongest elicitors of laughter, that 7- to 9-month-olds laughed more and to a greater variety of stimuli than did 4- to 6-month-olds, and that the frequency of laughing at visual and social stimuli (such as during a peek-a-boo game) increased with age.

One- to 9-month-old infants display all the facial expressions displayed by adults—interest, joy, surprise, sadness, anger, disgust, contempt, and fear ([Izard et al., 1980](#); [Malatesta and Haviland, 1982](#))—although the initial expressions are not always complete or used appropriately. Stenberg and coworkers ([Stenberg et al., 1983](#); [Stenberg and Campos, 1990](#)) took a closer look at the development of anger expressions by examining infants' responses to the removal of teething biscuits and to restraints of their arm movements. They found the 1-month-olds showed few clear expressions of anger, but 4- to 7-month-olds showed clear expressions of anger. The oldest infants directed their anger expressions toward the person who was restraining their arms or removing the biscuit. In addition, anger in response to inoculations increases with age ([Izard et al., 1983](#)).

The last emotional expression to occur appropriately is fear. Between 6 and 9 months, infants begin to display fear to a variety of social (e.g., separation from a parent, approach of a stranger) and nonsocial (e.g., heights, looming stimuli) stimuli ([Campos and Stenberg, 1981](#); [Cicchetti and Hesse, 1983](#)). Not all infants display fear in response to each of these stimuli, however. In particular, fearful reactions to strangers, once thought of as a developmental milestone ([Bronson, 1972](#);



Freedman, 1974; Hess, 1970; Spitz, 1950a), vary depending on the particular stranger involved, his or her behavior, and the context in which infant and stranger meet (Clarke-Stewart, 1978; Rheingold and Eckerman, 1973; Thompson and Limber, 1991). Nevertheless, although fear of strangers is not universal, it emerges during the second half of the first year if it does occur (Bronson, 1972; Haith and Campos, 1977).

Nonhuman primates go through the same developmental sequence, with positive expressions toward social stimuli being used appropriately before negative ones. In this way, sociability is fostered; nonhuman and human infants form ties with fellow conspecifics before they begin to fear particular individuals. In addition, because young infants do not have the motor skills to protect themselves from potentially dangerous situations, fear is not adaptive for protection until later (Izard and Malatesta, 1987). Indeed, although infants can distinguish the two sides of a visual cliff (a shallow and deep side) by 2 months of age (Campos et al., 1970), they show no fear of the deep side until the third quarter-year of life; that is, they begin to show fear of heights at approximately the same time they are beginning to locomote and thus are able to flee (Campos and Bertenthal, 1989). This is one example of a tendency for changes in the child's motor capacities to bring about changes in social relationship and the manifestation of emotion. Biringen and colleagues (1995) reported that upright locomotion also had profound effects.

In summary, infants are able to express all the primary emotions at or soon after birth; during the next 6 to 12 months, infants learn to express these emotions fully and appropriately, so as to communicate successfully with others. Next, the ability to respond to the emotional expressions of others is considered.

## INFANTS' RESPONSES TO THE EMOTIONAL EXPRESSIONS OF OTHERS

As members of a social species, humans must constantly modulate their emotions in response to the emotions of others (Nelson, 1987). Newborns are able to discriminate among happy, sad, and surprised faces (Field et al., 1982), and at 3 to 5 months of age they are able to discriminate among expressions of joy, surprise, anger, and sadness (Barrera and Maurer, 1981; LaBarbera et al., 1976; Young-Browne et al., 1977), as well as degrees of such expressions (Kuchuk et al., 1986). Between 5½ and 7 months of age, infants can discriminate happy and surprised expressions (Caron et al., 1982), and at 7 months, infants also show generalized discrimination (across several models) of happy and frightened faces and prefer looking at frightened rather than happy faces (Nelson and Dolgin, 1985). Interestingly, this preference for looking at frightened faces emerges at approximately the age when infants themselves begin to express fear appropriately.

Emotional contagion occurs in a variety of social species (Hinde, 1970), and human newborns may be capable of some such modulation; they appear to match ("imitate") both facial expressions (Field et al., 1982; Meltzoff and Moore, 1977, 1983) and vocal expressions (crying; Martin and Clarke, 1982; Sagi and Hoffman, 1976; Simner, 1971). In addition, 3-month-olds become inactive when their mothers act depressed (Cohn and Tronick, 1983; Lamb and Malkin, 1986). As infancy proceeds, this immediate response to the emotion of others becomes less automatic. Although newborns readily cry when they hear other newborns crying (Martin and Clarke, 1982; Sagi and Hoffman, 1976; Simner, 1971), 6-month-olds do not cry so rapidly in response to the cries of their peers. Hay et al. (1981) found that the probability of a 6-month-old's crying in response to the distress of a peer increased the longer the peer cried; if the peer cried for 2½ minutes, the listening infant was sure to cry. Further demonstrating that automatic responding decreases as infants age, Nelson and Horowitz (1983) found that 2-month-olds respond to expression changes in holograms of faces, whereas 5-month-olds do not.

Older infants use more deliberate means to match the emotions of others than do younger infants. By 12 months of age, infants in ambiguous situations use other peoples' emotional expressions as cues for their own, a phenomenon termed *social referencing* (see Feinman, 1992; Mumme et al., 1996). Infants are more likely to cross a visual cliff of ambiguous height when their mothers look happy than when they look afraid (Sorice et al., 1985) and are more likely to approach and explore novel toys and animals when an adult signals joy and to seek reassurance from the adult when she or he signals fear (Hornik et al., 1987; Hornik and Gunnar, 1988; Klinnert et al., 1983; Mumme et al., 1996; Zabatany and Lamb, 1985). By 18 months of age, infants are able to respond with recognizable empathy to the distress of their companions, offering teddies or restoring desired toys to distressed peers (Hoffman, 1978).

Responses to particular auditory displays are more difficult to document, although most theorists agree that emotions are reflected in vocalizations (Darwin, 1872; Scherer et al., 1972; Zahavi, 1982). Newborns cry in response to the distress of others, whereas exposure to recordings of their own cries calms newborns, indicating that they can discriminate their own cries from those of others and do not merely become distressed because they find crying irritating (Martin and Clarke, 1982). This differential responding may be an early precursor of empathy. Twelve-month-olds avoid crossing a visual cliff of ambiguous depth when their mothers' vocal tone is negative (angry or fearful) but cross immediately when the mothers speak positively (Svejda and Campos, 1982). Such social referencing in response to vocal expressions of emotion has been found in other situations as well (Walden and Baxter, 1989). Studies have indicated that infants begin to coordinate vocal and facial features of emotional expression early in infancy—between 4 and 9 months of age (Caron et al., 1988).

## SOCIALIZATION OF EMOTIONS

The fact that the production and recognition of emotional expression is innate does not mean that experience plays no role in emotional development. As Fraiberg (1979) reported, social smiling in response to familiar voices first emerges at approximately the same age for blind and sighted infants, but smiling by blind infants subsequently becomes less frequent and more muted. Unlike sighted infants, furthermore, blind infants do not initiate interaction by smiling. Similarly, Ambrose (1969) reported that smiling to external stimuli occurred later in institution-reared infants, who receive little social feedback, than in home-reared infants.

Experimental studies indicate that emotional expressions can be modified by conditioning and social learning (reviewed by Campos et al., 1983), processes that characterize interactions between mothers and infants throughout the first year of life (Goldberg et al., 1994; Malatesta and Haviland, 1982, 1985; Malatesta et al., 1986). According to Malatesta, maternal expressions typically changed less than a half second after the infants' expressions changed, the optimal delay for instrumental conditioning. Mothers contingently responded to their infants' positive expressions more than to their negative expressions, thus producing an increase in positive emotional expressions and a decrease in negative expressions. Contingent maternal responses also led to increases in the overall rate of infants' facial expression changes.

Malatesta's studies also implicate observational learning in the modification of infants' emotional expressiveness. Malatesta and Haviland (1985) reported that mothers on average changed their expressions eight times per minute. From this they estimated that mothers change their expression 362 times per day in face-to-face play, and 32,580 times over the 3-month period of peak face-to-face play. As these investigators remarked, "This is not a trivial learning opportunity for the infant" (p. 104). The amount of joy and interest modeled by mothers at 5 months predicted increases in the amount of joy and interest expressed by infants at 7½ months (Malatesta et al., 1986).

The infant's role in the socialization of emotions is underscored by comparing the face-to-face interactions of preterm and full-term infants. Mothers of preterm infants matched their infants' facial expressions less and ignored their anger expressions more than did mothers of full-term infants. The former also responded randomly rather than contingently to their infants' sad expressions. These differences in maternal behavior were attributed to the fact that preterm infants expressed more negative emotions and gazed at their mothers less than full-term infants did (Malatesta et al., 1986).

In addition to being an arena in which instrumental and social learning can take place, early face-to-face play reflects cultural values concerning emotional expression (Super and Harkness, 1982). Among the Gusii of Kenya, for example, social interaction is characterized by bland countenances. Face-to-face play is rare, and when it does occur, eye contact is avoided (as it is between adults). When infants become excited, mothers respond with a variety of strategies to dampen the infants' emotional displays, looking away or blinking rapidly to break visual contact. The cultural norm of avoiding emotional displays is thus taught in the earliest social interactions (Dixon et al., 1981).

During early face-to-face interactions, North Americans instill cultural norms concerning the greater acceptability of emotional displays by girls (Malatesta, 1985). Malatesta and Haviland (1982) found that, although female and male infants display similar facial expressions, American mothers respond differently to them. As infants grow older, mothers smile increasingly in response to their sons' smiles and decreasingly in response to their daughters' smiles. Similarly, Lewis and Michalson (1985) found that after 6 months mothers responded less to boys' crying than to girls', were more likely to imitate their sons' expressions, and were more likely to show a wider range of expressions, both imitative and dissimilar, to their daughters. Girls were reinforced for a wider range of emotional expressions than were boys, which may explain why girls are better at decoding emotional expressions than are boys at all ages (Hall, 1978).

In summary, infants' abilities to communicate emotionally are well documented. These abilities allow for the formation of close, special, relationships. Such attachments to particular individuals typically emerge during the second half of the first year of life. We next discuss the development of such attachments.

## Development of Attachment

The most popular explanation of attachment formation was provided by [Bowlby \(1969, 1988\)](#), a psychoanalyst who was much impressed by the capacity of ethologic theorists to explain early emotional communication between nonhuman infants and their parents. Bowlby's influential theory consequently emphasizes the infant's innate capacity to emit signals to which adults are biologically predisposed to respond. Adults' responses to these signals may account for the development of attachments, as we show shortly.

Bowlby began with the assumption that the behavioral propensities of infants and parents are most profitably considered in the context of the environment in which our species evolved. In that "environment of evolutionary adaptedness," the survival of infants would have depended on their ability to maintain proximity to protective adults. Unlike the young of many other species, however, human infants are unable to move closer to or to follow adults for several months, and they are even incapable of clinging to adults. Instead, human infants rely on signals to entice adults to approach them. For these signals to be effective, adults must be predisposed to respond to them. The best example of such a prepotent signal is the infant cry, which very effectively entices adults to approach, pick up, and soothe the infant ( [Bell and Ainsworth, 1972](#); [Frodi et al., 1978b](#); [Korner and Thoman, 1970, 1972](#); [Murray, 1979](#)). As they grow older, infants develop a variety of means of achieving proximity or contact, including independent locomotion. Throughout infancy, children seek proximity to protective adults to obtain nourishment, comfort, and security. Gradually, infants come to focus their bids on those people with whom they are familiar and on whom they can count, and this is one crucial aspect of the process called *attachment formation*.

## BASIC PHASES OF SOCIAL DEVELOPMENT

[Bowlby \(1969\)](#) described four phases in the development of infant–parent attachments. The first phase in the attachment process (indiscriminate social responsiveness, 1 to 2 months) is marked by the development of a repertoire of signals, labeled *attachment behaviors*. The defining or common characteristic of these behaviors is that they all help achieve comfort and security by bringing the infant close to protective, caretaking adults.

From the time of birth, there is one very effective signal at the infant's disposal—the cry. A second potent attachment behavior enters the infant's repertoire in the second month of life—smiling. Like crying, smiling is a signal that powerfully affects adult behavior. However, smiles are effective because they encourage adults to stay near and continue interacting with the infant because adults find the interaction pleasant, whereas cries encourage adults to approach the infant to terminate a signal that they find aversive ( [Ambrose, 1969](#); [Frodi et al., 1978b](#)).

From birth, therefore, infants are capable of affecting the people around them, but in this phase, infants use proximity-promoting signals indiscriminately. They are satisfied when anyone responds to their attachment behaviors.

Frequent encounters with caretakers at times when infants are alert also may facilitate infants' capacities to recognize caretakers, and Bowlby suggested that acquisition of the ability to recognize specific people marks the transition to the second phase of attachment development (discriminating sociability, 2 to 7 months). In fact, infants seem able to recognize their parents much earlier than Bowlby believed. [Macfarlane \(1975\)](#) provided anecdotal evidence that infants could distinguish between their mothers' smell and that of other nursing mothers within the first 2 weeks of life. After teaching infants to suck in one way to turn on a tape recording of their mothers' voices and in another way to produce the voice of another woman, [DeCasper and Fifer \(1980\)](#) found that 2-day-old infants not only could discriminate between two voices but worked harder (i.e., sucked in a special way) to hear their own mothers' voices.

Presumably because significant others (such as parents) have been associated with pleasurable experiences (e.g., cuddling and play) and with the relief of distress from early in life, familiar people become persons with whom infants prefer to interact. Initially, these preferences evidence themselves in fairly subtle ways: Certain people are able to soothe the infant more easily and to elicit smiles and coos more readily, broadly, and frequently. According to Bowlby, these preferences are initially based on recognition only: Infants do not even have a primitive conception of the permanent existence of their parents. Following various psychoanalytic theorists ( [Freud, 1965](#); [Spitz 1950b, 1965](#)), [Bowlby \(1969\)](#) and [Ainsworth \(1973\)](#) believe that infants cannot be said to love parents until they understand that their parents exist even when they are neither interacting with nor visible or audible to the infants. Consequently, Bowlby argued, we cannot claim that 3- or 4-month-olds have formed attachments, although they have taken another step toward this achievement.

The third of Bowlby's phases (attachment, 6 to 7, to 24 to 30 months) is marked by the emergence of intentional and focused attachment behavior. Seven-month-olds clearly understand and respect the rule of reciprocity in their interactions. Their confidence in others reinforced, 7-month-olds enjoy the newly acquired ability to creep around and to take responsibility for getting close to their parents at will, instead of waiting for others to come in response to their cries or coos. Intentional social behavior is now possible ( [Lamb, 1981a](#)), and 6- to 12-month-olds are increasingly likely to initiate interaction using directed social behaviors, whereas mothers more frequently initiate games, terminate or redirect their infants' activities, and issue verbal requests ( [Green et al., 1980](#)). Caretaking becomes less prominent. One sign of the increased intentionality of infant behavior is that 7-month-olds begin to protest more reliably when parents leave. Wariness of strange adults also may become more prominent around this time.

Especially from the parents' point of view, the transition between phase 2 and phase 3 is not abrupt because infants have been showing preferences for parents over others for several months. Nevertheless, the beginning of phase 3 is marked by two major behavioral changes. First, when infants protest when left by an attachment figure, they are no longer satisfied by the appearance of substitute interactive partners ( [Stayton et al., 1973](#)). According to Bowlby, separation protest should be viewed as a signal aimed at making attachment figures come back to the infant, and its emergence can be linked to the attainment of some primitive conception of person permanence—the notion that people have a permanent existence independent of the infant ( [Ainsworth, 1973](#)). Second, the emergent ability to crawl permits exploration of the environment on a scale hitherto inconceivable, affording infants the capacity to move toward attachment figures when they want to be near them. From this point on, infants assume an increasingly active role in their relationships with attachment figures.

Of course, major changes in social relationships occur between 6 and 7 months (the beginning of phase 3) and approximately 24 to 30 months (the end of this phase): Infants become increasingly sophisticated in their abilities to behave intentionally, to communicate linguistically, and to respond appropriately in a variety of different contexts. As infants grow older, they initiate an increasing proportion of their interactions. They can tolerate a growing distance from attachment figures as they grow older ( [Anderson, 1972](#); [Rheingold and Eckerman, 1970](#)).

According to [Bowlby \(1969\)](#), the next major transition occurs at the beginning of the third year of life, when children become able to take their parents' needs into account when interacting with them (phase 4, goal-corrected partnerships). For example, they are now able to respect others' differing perspectives and appear to recognize for the first time that parents must sometimes give priority to other activities, while the child's needs or wants wait ( [Marvin, 1977](#); [Mossler et al., 1976](#)). The beginning of phase 3 is, thus, the time at which the first infant–adult attachments are formed. Most theorists define attachment as a specific, enduring, emotional bond whose existence is of major importance in the process of sociopersonality development.

To whom do attachments form? In many places in the world, infants are cared for primarily by their mothers. The universality of traditional parental roles has led psychologists to assume that the first persons to whom infants become attached are their mothers. Until very recently, in fact, most theorists assumed that the infant–mother relationship was not simply most important but uniquely important ( [Ainsworth, 1973](#); [Bowlby, 1969](#); [Maccoby and Masters, 1970](#)), thus accepting [Freud's \(1940/1968, p. 48\)](#) dictum that the mother–infant relationship is "unique, without parallel, established unalterably as the prototype of all later love relations . . . for both sexes." Fathers were not considered important until the oedipal period began, around 3 years of age.

In fact, most infants become attached to their fathers at approximately 7 months of age, the same age at which they form attachments to their mothers ( [Lamb, 1976b, 1977, 1981b](#), in pressb), although many U.S. mothers are the primary or preferred attachment figures in the sense that infants prefer mothers at times of stress ( [Lamb, 1976a, 1976c](#)). In many parts of the world, infants may have several caregivers who are emotionally involved in their lives, and, thus, may form several attachments ( [Dunn, 1993](#); [Nash and Hay, 1993](#)).

In the United States, infant–mother and infant–father relationships offer different sorts of experiences for young infants. A detailed analysis of face-to-face play between parents and very young infants (2- to 6-month-olds) shows that fathers provide more unpredictable, less rhythmic, and more exciting (rather than calming) physical and vocal stimulation than do mothers ( [Yogman, 1982](#)). Naturalistic home observations of older infants and their parents yield similar findings ( [Lamb, 1976b](#)). In addition, mothers hold their infants most often for caretaking purposes; fathers hold their infants most often to play with them ( [Lamb, 1976b, 1977](#)).

These differences are significant because they show parental roles being translated into distinctive styles of interaction. Children learn to expect playful stimulating interactions with their fathers and, thus, come to prefer playing with them ( [Clarke-Stewart, 1980](#); [Lamb, 1977](#); [Lynn and Cross, 1974](#)). In addition, the playfulness of



fathers may increase the salience and importance of the limited amounts of time that many infants spend with their fathers.

## SECURITY OF INFANT–PARENT ATTACHMENTS

Attachment theory was developed by clinically oriented scholars primarily interested in how early attachment relationships might affect subsequent development. Much research on this issue involves use of the “Strange Situation” for assessing “the security of attachment” ( [Ainsworth and Wittig, 1969](#); [Ainsworth et al., 1978](#)). [Ainsworth et al. \(1978; Ainsworth and Bowlby, 1991\)](#) also provided an elegant and persuasive account of the relation among prior infant–mother interaction, security of infant attachment, and subsequent child development. We emphasize these developmental associations in this chapter, even though a great deal of research has recently focused on the ways in which different aspects of parental behavior affect child development (Bornstein, in press).

The Strange Situation can be used only when infants are old enough to have formed attachments and be mobile (e.g., older than 10 months), yet are young enough to be stressed by strangers and brief separations from their parents (e.g., younger than 24 months). The procedure has seven episodes, which are outlined in [Table 22.2](#), and is designed to expose infants to increasing amounts of stress to observe how they organize their attachment behaviors around their parents when distressed. Stress is stimulated by the unfamiliar environment, by the entrance of an unfamiliar adult, and by two brief separations from the parent. This procedure has been used widely, although increasing numbers of researchers instead use an alternative procedure, the Attachment Q-set ( [Vaughn and Waters, 1990](#); [Waters and Deane, 1985](#)). Interestingly, scores on the Attachment Q-set are much more highly correlated with temperament measures than are Strange Situation measures, perhaps because both measures of temperament are verbal report measures ( [Seifer et al., 1996](#); [Teti et al., 1996](#); [Vaughn et al., 1992](#); [Wachs and Desai, 1993](#)).

Episode*	Persons Present	Change
1	Parent, infant	Enter room
2	Parent, infant, stranger	Unfamiliar adult joins the dyad
3	Infant, stranger	Parent leaves
4	Parent, infant	Parent returns
		Stranger leaves
		Parent leaves
5	Infant	Stranger returns
6	Infant, stranger	Parent returns
7	Parent, infant	Stranger leaves

\*All episodes are usually 3 minutes long, but episodes 3, 5, and 6 can be curtailed if the infant becomes too distressed, and episodes 4 and 7 are sometimes extended. Adapted from Ainsworth MD, Wittig BA: Attachment and the exploratory behavior of one-year-olds in a strange situation. In: Fox RM (ed): *Determinants of Infant Behavior*, vol 4. London, Methuen, 1969, pp 113–136.

**Table 22.2. The Strange Situation**

### A Typology of Infants

According to Bowlby and Ainsworth, infants in the Strange Situation should be able to use their attachment figures as secure bases from which to explore the novel environment. The stranger's entrance should lead infants to inhibit exploration and draw a little closer to their parents, at least temporarily. The parents' departure should lead to attempts to bring them back by crying or searching, and to less exploration and affiliation. After the parents' return, infants should seek to reengage in interaction and, if distressed, may wish to be cuddled and comforted. The same responses should occur, with somewhat greater intensity, after the second separation and reunion. In fact, this is how approximately 65% of the infants studied in the United States in the Strange Situation behave. Following the practices of Ainsworth and her colleagues, these “securely attached” infants are designated type B.

By contrast, some infants seem unable to use their parents as secure bases from which to explore. Furthermore, although they are distressed by their parents' absence, they behave ambivalently on reunion, both seeking contact and interaction and angrily rejecting it when it is offered. These infants are conventionally labeled type C and typically account for approximately 15% of the infants in American research samples. Another group of infants seem little concerned by their parents' absence. Instead of greeting their parents on reunion, they actively avoid interaction and ignore the parents' bids. These infants are said to exhibit type A attachments; they typically constitute approximately 20% of the infants in American samples.

Although the behavior of most infants allows them to be classified into one of these categories, some infants are difficult to classify ( [Main and Solomon, 1986, 1990](#)). [Main and Solomon \(1986\)](#) observed such infants and found that, rather than using different patterns of behavioral organization, these infants appear disorganized. They have designated a new category, type D attachment, to reflect infants who show behavioral indices of disorganization and disorientation in the Strange Situation. Main and Solomon have compiled a list of behaviors that reflect such disorganization and disorientation, thought to reflect a conflict between behavioral systems (e.g., “cries while moving away from parent” or “falls prone on reunion”).

### Determinants of Strange Situation Behavior

According to ethologic attachment theorists, infants count on attachment figures to protect them and to be accessible when needed and so use them as secure bases from which to explore and interact with other people. However, not all infants trust their attachment figures equally, and these differences in security of attachment might affect how willingly infants use their attachment figures as bases of security. In fact, Ainsworth has argued that the A, B, and C patterns of attachment behavior in the Strange Situation indeed reflect individual differences in attachment security. Moreover, Ainsworth has developed hypotheses concerning the ontogeny of these individual differences.

Almost from birth, infants learn about people from their interactions with them ( [Lamb, 1981a; Watson, 1985](#)). The extent to which their behaviors—particularly their cries—are answered depends, of course, on the responsiveness of the infants' caretakers. When caretakers hear the infants' signals, interpret them correctly, and make consistently appropriate responses, infants develop confidence in their own “effectance” (ability to act on the environment successfully) and trust in the reliability or predictability of the persons concerned. Evidence gathered in an exploratory longitudinal study ( [Ainsworth and Bell, 1974; Blehar et al., 1977](#)) led Ainsworth to propose that caretakers' sensitivity is important from the first months of life—the months during which infants come to understand what it means to be social. Because adults differ in their sensitivity, there would be differences among infants in the extent to which infants had confidence in their own effectance and in the reliability of others. Ainsworth equates these with differences in the security of attachment ( [Ainsworth et al., 1974; Ainsworth and Bowlby, 1991](#)). Since Ainsworth's hypotheses were proposed, many other researchers (see reviews by [Belsky and Cassidy, 1994; Thompson, 1998](#)) have attempted to test them in independent longitudinal studies (e.g., [Bates et al., 1985; Belsky et al., 1984; Egeland and Faber, 1984; Erickson et al., 1985; Grossmann et al., 1985; Miyake et al., 1985; Sagi et al., 1985](#)). After reviewing the complicated results of these many studies, [Lamb and his coauthors \(1985\)](#) concluded that there was general support for the notion that more socially desirable parental behavior, that is, nurturant, attentive, nonrestrictive parental care, was associated with type B infant behavior in the Strange Situation, at least when studies were conducted in the United States. [DeWolff and van IJzendoorn \(1997\)](#) reached the same conclusion after a metaanalysis of the available data. Both reviews agreed that the mothers of infants who behave in type A or type C fashion (often called *insecurely attached*) manifest less socially desirable patterns of behavior: They may overstimulate or understimulate, fail to make their behaviors contingent on infant behavior, appear cold or rejecting, and sometimes appear inept to observers. Unfortunately, there is too much variability in the results to identify precisely what aspects of parental behavior are formatively important. Some studies identify warmth but not sensitivity; some identify level of stimulation; some, patterning of stimulation but not warmth or amount of stimulation, and so forth. Infants who behave in a disorganized fashion often have experienced abuse at the hands of their parents ( [Carlson, 1998](#)).

### Stability and Prediction

**Stability.** The notion that Strange Situation behavior reflects something meaningful is supported by findings showing stability over time in patterns of infant behavior. Both [Waters \(1978\)](#) and [Connell \(1976\)](#) reported remarkable stability between 12 and 18 months of age in the way infants behave in the Strange Situation when observed twice with the same parent. According to Waters, 48 of 50 infants (96%) obtained the same classification on both occasions! However, test–retest reliability is not always so high. [Vaughn et al. \(1979\)](#) showed that, in an economically disadvantaged sample, many infants changed from one mode of behavior to another between 12 and 18 months of age. Changes were systematic: When the families had experienced considerable social stress during the 6-month period, type B attachments often changed to types A or C, although when families experienced a low degree of stress, types A or C attachments did not necessarily become type B. Later, [Thompson et al. \(1982\)](#) found equivalent instability in a middle-class sample. In this study, major changes in family circumstances or caretaking arrangements

(e.g., the onset of maternal employment) led to changes in Strange Situation behavior, but they did not necessarily engender change from the more desirable type B to the less desirable types A and C any more often than the reverse. Because parents' sensitivity to the infants' signals appears to influence Strange Situation behavior ([Ainsworth et al., 1974, 1978](#)), these data might mean that changes in the amount of stress to which the mothers were subjected affected the manner in which they interacted with their infants. [Teti et al. \(1996\)](#) showed that this might be true in a study concerned with the effects of a second child's birth on the attachments between older siblings and their mothers. Attachment security declined most dramatically when mothers were most psychologically distressed and had the poorest marital relationships.

**Predictive Validity.** Another reason why attachment classifications intrigue developmentalists is that they sometimes predict aspects of the child's future behavior ([Thompson, 1998](#)). Attachment theorists believe that when infants encounter people for the first time, they tend to assume that those persons will treat them in the same way that others have treated them in the past. Thus, infants who have developed trust in their attachment figures tend to regard new people they encounter as trustworthy too. As infants get to know each individual, of course, they develop a set of expectations about that specific individual.

The relation between Strange Situation behavior and styles of interaction with unfamiliar adults has been well documented (see [Thompson, 1998, 1999](#) for reviews). [Main \(1983\)](#), for example, showed that infants who had type B attachments to their mothers were later more cooperatively playful when interacting with a friendly stranger than were type A or C infants. Type A infants (who were avoidant of their mothers) later avoided the stranger, whereas type C infants were later resistant with the stranger. [Main and Weston \(1981\)](#), as well as [Thompson and Lamb \(1983\)](#), confirmed that type B infants were more sociable with unfamiliar adults than type A or C infants were. Similarly, [Lieberman \(1977\)](#), [Easterbrooks and Lamb \(1979\)](#), and [Pastor \(1981\)](#) showed that Strange Situation behavior was associated with social behavior in initial encounters with peers, as well as over longer periods in preschool play groups ([Booth et al., 1991](#); [Sroufe, 1983](#); [Waters et al., 1979](#)) and summer camp at 11 years of age ([Elicker et al., 1992](#)). Type B infants engaged in more frequent and more mature forms of interaction with their peers, sharing more and showing a greater capacity to initiate and maintain interactions, for example. For reasons that are not clear, however, [Jacobson and colleagues \(Jacobson and Wille, 1984; Jacobson et al., 1983, 1986\)](#) have reported contrasting results, suggesting that the relation between attachment classification and peer competence is neither direct nor powerful (see [Lamb and Nash, 1989](#), for a review of the studies and issues concerning the influence of attachment relationships in infancy on later peer relationships).

Other research ([Arend et al., 1979](#); [Frankel and Bates, 1990](#); [Matas et al., 1978](#); [Sroufe, 1983](#); [Suess et al., 1992](#)) addresses the relation between attachment classifications and aspects of achievement motivation. These researchers found that Strange Situation behavior at 12 or 18 months predicts cognitive performance in problem-solving situations and in a variety of stressful and challenging contexts, at least until kindergarten age. Placed in cognitively challenging situations, children who, as infants, had type B attachments to their mothers persisted longer and more enthusiastically than did children who had type A or C attachments. Type B infants also seemed to be more resilient and robust when stressed or challenged ([Block and Block, 1980](#)). In addition, type B infants appeared more socially competent and independent when they later entered preschool ([Sroufe, 1983](#); [Sroufe et al., 1984](#)) and also showed fewer behavior problems ([Bates et al., 1985](#); [Erickson et al., 1985](#); [Lewis et al., 1984](#)). In general, although some researchers have found that insecure attachments may be associated with severe behavior and adjustment problems, others have not ([Belsky and Cassidy, 1994](#); [Belsky and Nezworski, 1988](#); [Fagot and Kavanaugh, 1990](#); [Lyons-Ruth et al., 1993](#); [NICHD Early Child Care Network, 1998](#)).

Evidence concerning the temporal stability of Strange Situation behavior and its relation to measures of earlier infant–mother interaction and later child achievement and personality suggests that the Strange Situation measures some meaningful aspect of mother–infant attachment and has important implications for understanding and predicting development. Presumably, Strange Situation behavior with fathers affects development in analogous ways, particularly because paternal sensitivity significantly affects the security of infant–father attachments ([van IJzendoorn and DeWolff, 1997](#)). However, the recent attention given to multiple attachment figures for many infants—mothers, fathers ([Lamb, 1981b, Lamb, in pressb](#)), siblings ([Teti and Ablard, 1989](#)), grandparents ([Myers et al., 1987](#)), day care providers ([Howes et al., 1988](#))—highlights the need for a comprehensive model of such combined influences ([Nash and Hay, 1993](#); [Tavecchio and van IJzendoorn, 1987](#); [Thompson, 1990](#)). Are these influences additive, such that a secure attachment to one parent and an insecure attachment to the other leads to intermediary levels of functioning later on ([Easterbrooks and Goldberg, 1990](#); [Pipp et al., 1992](#))? Or are these influences compensatory, such that a secure relationship with a day care provider, for example, can compensate for an insecure attachment to a parent ([Goossens and van IJzendoorn, 1990](#))? (See [Nash and Hay, 1993](#) for a fuller discussion of the issue of combining influences from several early close relationships.)

Even for the relatively simpler infant–mother predictions, [Lamb et al. \(1985\)](#) and [Thompson \(1998\)](#) have pointed out that the predictive validity of longitudinal associations is far from perfect. Rather, the relation between Strange Situation behavior and subsequent child behavior is found only when there is stability in the caretaking arrangements and family circumstances, which (as we noted previously) seems to maintain stability in patterns of parent–child interaction. Thus, the question first encountered when considering the stability of cognition in infancy arises again: Is the prediction over time attributable to individual differences in the quality of early parent–child interaction, or is it attributable to the continuing quality of child–parent interactions? If the latter were true, it would imply that the quality of early relationships was predictively valuable, not because it caused later differences directly, but because it presaged later differences in the quality of relationships, which, in turn, would cause differences in the child's behavior. Such a pattern of findings would place the locus of stability in the continuing quality of parent–child interactions rather than in some aspect of the child's personality. Research ([Thompson, 2000](#)) has shown that both early and later relationship patterns appear to shape the child's development

Although attachment security, measures of prior infant–parent interaction, and measures of the child's later behavior are all interrelated, the relations are not very strong. This suggests that factors other than quality of attachment, such as temperament or familiarity with strangers and brief separations, influence children's Strange Situation behavior. In turn, this means that researchers and clinicians need to rely on multiple convergent methods to assess constructs as complex and as important as the quality of infant attachments, rather than relying on a single measure like the Strange Situation in which behavior is influenced by factors other than quality of attachment.

### Cross-Cultural Research

Some of the most provocatively informative recent work on Strange Situation behavior has been conducted outside the United States. The distribution of infants across the A, B, and C categories in many other countries differs from that typically found in American samples ([Lamb et al., 1985](#)). One could interpret these results by concluding that parents in the cultures are either much more or much less sensitive than American parents, but that ethnocentric interpretation seems improbable. Rather, these results may demonstrate the importance of factors other than the quality of parental behavior in explaining Strange Situation behavior ([Lamb, in pressb; Lamb et al., 1985](#)). For example, the high degrees of stress manifested by Japanese and Israeli infants leads to increases in the proportion of infants classified as type C. It may be that Japanese infants appear inordinately distressed because they have much less experience with separations from their mothers than American infants typically have; thus, the situation is not psychologically similar for Japanese and American infants. For infants growing up on Israeli kibbutzim, encounters with strangers are unusual and may thus elicit great distress. Again, therefore, even though the procedure was structurally the same for Japanese, Israeli, and American infants, the psychological experiences or meanings for infants from each culture might be very different. In addition, [Miyake et al. \(1985\)](#) reported that the Japanese infants who were later classified as type C were temperamentally more irritable than type B infants from birth. Thus, it appears that culture-specific rearing practices or temperamental differences account for at least some of the variance in Strange Situation classifications across cultures. Scholars have developed an increasingly sophisticated understanding of the ways culture may shape human development, and this is likely to refashion much of developmental psychology in the next decade ([Parke and Buriel, 1998](#)).

The picture emerging from the many studies in which Strange Situation behavior was assessed is a complicated one. Strange Situation behavior appears to reflect individual differences in patterns of infant–parent interaction, with type B attachments potentiated by warm, sensitive, and supportive parental behavior. However, other factors seem to be important as well, notably culture-specific rearing practices and (perhaps) infant temperament. To understand the formative importance of infant–parent attachment, we need to obtain multiple measures of attachment, rather than rely exclusively on the observation of Strange Situation behavior. In addition, we need to understand better how several early attachment relationships together may affect later functioning.

The next two sections take into account such a diverse social milieu, which for many infants involves not only parents but grandparents and other adults, siblings, and peers. We now discuss infants' relationships with nonparental figures and illustrate similarities and differences among the infant–adult, infant–sibling, and infant–peer systems.

### Sibling Relationships

Siblings in many nonindustrialized countries assume a major responsibility for infant caretaking ([Zukow-Goldring, 1995](#)). These siblings may spend relatively little time playing with the infants; most of their interactions, like those of Western parents, involve protection or caretaking. In our society, by contrast, siblings seldom assume any responsibility for infant caretaking, and sibling relationships appear to incorporate features of both the infant–adult and infant–peer systems (Teti, in press). On



the one hand, sibling dyads share common interests and have more similar behavioral repertoires than do infant–adult dyads. On the other hand, sibling pairs resemble infant–adult pairs to the extent that they differ with respect to amount of experience and levels of both cognitive and social ability. These discrepancies often lead to differences in the ways younger and older siblings relate to each other, differences that distinguish them from the infant–peer system (see later). Observational studies of sibling interaction in a laboratory playroom reveal consistent asymmetries between the roles assumed by older and younger children ( [Abramovitch et al., 1979](#); [Lamb, 1978a, 1978b](#); [Teti et al., 1986](#)). Older siblings “led” the interaction: They engaged in more dominant, assertive, and directive behaviors than their younger siblings. The infants, meanwhile, appeared inordinately interested in what their siblings were doing; they followed them around, attempting to imitate or at least explore the toys just abandoned by the older children. This is, of course, a strategy that maximizes the amount the infant can learn about the environment from the older child. As [Zajonc \(1983\)](#) suggests, older siblings offer young children cognitive models, albeit less sophisticated ones than do adults. In a home observational study, [Abramovitch and colleagues \(1979\)](#) reported patterns of interaction similar to those reported by [Lamb \(1978b\)](#), although siblings at home engaged in much more interaction than in the laboratory. In a longitudinal study, [Lamb \(1978a\)](#) found remarkable stability across time in the amount of interaction engaged in by siblings. The pattern of correlations, furthermore, suggested that the sociability of the younger infants determined the amount of attention they later received from their siblings, rather than the reverse.

Individual differences in the quality of sibling relationships also were the focus of a longitudinal study conducted in England by [Dunn and Kendrick \(1980, 1981a, 1981b, 1982a, 1982b\)](#). [Dunn et al. \(1998\)](#) found that same-sex siblings got along better than different-sex siblings. In addition, siblings interacted more poorly when mothers and first-born girls had very positive relationships before and immediately after the birth of the second child and when there was frequent interaction and play between the siblings and their mothers. These findings suggest that parents exert an important influence on the mutual affective involvement of siblings who may compete for parental attention.

In a variety of studies, researchers have found that older siblings spend at least some of their time teaching object-related and social skills to their younger siblings (including infants) and that the amount of teaching increases with the age of the older child ( [Cicirelli, 1973, 1974](#); [Minnett et al., 1983](#); [Pepler, 1981](#); [Stewart, 1983](#)). These studies, along with the finding that infants monitor and imitate their older siblings ( [Lamb, 1978a, 1978b](#)), confirm that older siblings may influence the cognitive and social skills of infants through some combination of teaching and modeling ( [Zajonc, 1983](#)). [Dunn \(1988\)](#) very richly documents the enhanced arena for the development of social understanding provided by siblings, and infants' considerable practical understanding of the feelings and intentions of other family members, siblings and parents alike. Infants also form attachments to their older siblings ( [Dunn, 1993](#)), which affect the infant's levels of aggressiveness, distress, and conflict ( [Teti and Ablard, 1989](#); [Volling and Belsky, 1993](#)).

Not only do older siblings influence infants, but the reverse occurs. For example, [Howe and Ross \(1990\)](#) found that friendly interactions between preschoolers and their infant siblings increased the older children's perspective-taking abilities.

The effects of age spacing on the patterns of interactions between infants and their preschool-aged older siblings are unclear; [Abramovitch and her colleagues \(1979\)](#), like [Dunn and Kendrick \(1981b, 1982a\)](#), found no age-spacing differences in amounts of positive, negative, and imitative sibling behaviors. Suggesting that broad summary measures may not have been sensitive enough to detect actual differences, [Teti et al. \(1986\)](#) used a more fine-grained coding scheme and found that older preschool-aged first-borns indeed created more “intellectual” (e.g., language mastery) and “social” (e.g., game) experiences for their infant siblings than did younger preschool-aged first-borns. In addition, [Gibbs et al. \(1985\)](#) found that widely spaced siblings were more interactive and responsive to one another than were closely spaced siblings. In neither of these investigations, however, did the researchers uncover relations between measures of infant cognitive or linguistic level and either sibling age or measures of sibling interaction. Thus, the net impact of siblings on infant development remains unclear, partly because such influences depend on sex of the siblings, birth order, and family size ( [Wagner et al., 1993](#)), and partly because both parents and the children themselves help to create very different rearing experiences for children in the same family ( [Dunn and Plomin, 1991](#); [Kowal and Kramer, 1997](#)), and behave differently before and after the birth of later children ( [Bayder et al., 1997](#); [Sakin et al., 1996](#)).

### Infants' Interactions with New People

Before the 1970s, it was commonly believed that infants were uninterested in their peers and afraid of strangers. Studies conducted since then, considered alongside earlier studies, indicate otherwise ( [Bridges, 1933](#); [Buhler, 1933](#); [Maudry and Nekula, 1939](#); [Ross and Goldman, 1977](#)). In fact, infants are both interested and competent in their interactions with strangers, whether peers or adults.

## INFANT–PEER INTERACTIONS

Although infants younger than 1 year of age do not have sustained interactions with peers ( [Rubin et al., 1999](#)), even newborns are responsive to their peers. As described previously, they respond to the distress of their peers by becoming distressed themselves ( [Martin and Clarke, 1982](#)). Two- and 3-month-olds attend to the activities of their peers ( [Bridges, 1933](#)) and increase their own activity level in a peer's presence ( [Field, 1979](#); [Fogel, 1979](#)). By 6 months of age, peers do not simply elicit responses in infants; infants respond to one another in socially complex ways. They initiate interactions by touching, vocalizing, or smiling at peers ( [Hay et al., 1983](#); [Vandell, 1980](#); [Vandell et al., 1980](#)), and their increased sensitivity to social cues is indicated by tendencies to continue interactions when peers are responsive and to stop when they are not ( [Hay et al., 1983](#)). In addition, 6-month-olds do not automatically cry in response to their peers' cries but instead attend to a peer's distress, becoming distressed themselves only if the other's distress is prolonged ( [Hay et al., 1981](#)).

Between 6 and 12 months of age, infants direct increasing numbers of social behaviors toward peers ( [Jacobson, 1981](#); [Maudry and Nekula, 1939](#); [Vandell, 1980](#)), although the amount of physical contact decreases ( [Vandell, 1980](#); [Vandell et al., 1980](#)). Perhaps this reflects an increasing reliance on distal rather than proximal modes of interaction, for during the second half of the first year infants begin to use conventional acts (gestures and words) to communicate with their peers ( [Bakeman and Adamson, 1986](#)). The structural complexity of peer-directed acts also increases with age so that by 9 months infants can synchronize several social behaviors ( [Becker, 1977](#); [Vandell et al., 1980](#)) and can attend simultaneously to objects and peers and thus engage in joint play ( [Adamson and Bakeman, 1985](#)). Infants at this age also express more affect during periods of joint engagement with peers than when alone ( [Adamson and Bakeman, 1985](#)) and begin to distinguish between familiar and unfamiliar peers ( [Dontas et al., 1985](#); [Jacobson, 1981](#)).

At the start of the second year of life, both cooperation and conflict begin to occur in interactions between peers ( [Eckerman and Whatley, 1977](#); [Goldman and Ross, 1978](#); [Hay et al., 1991](#); [Howes, 1988](#); [Nash, 1985, 1986](#)). The ability to engage in conflicts over toys and personal space, not present among 6-month-olds ( [Hay et al., 1983](#)), is found at the start of the second year of life ( [Caplan et al., 1991](#); [Nash, 1986](#)). The social nature of such early conflicts is suggested by [Hay and Ross \(1982\)](#), who found that 21-month-olds did not simply grab toys from one another but used gestures and vocalizations to resolve disputes. In addition, when children yielded possession of objects to peers, the winners often turned their attention elsewhere, as though the interaction itself, and not obtaining the object, was important. These infants spent only 5% of their time with one another in conflicts.

Studies comparing interactions with peers and mothers indicate that infants demonstrate similar social competencies in their interactions with peers and mothers ( [Adamson and Bakeman, 1985](#); [Bakeman and Adamson, 1984, 1986](#); [Nash, 1985, 1986](#); [Vandell and Wilson, 1982](#)). In addition, several studies of 10- to 19-month-olds revealed that interactions with peers were more frequent than those with mothers when both were present ( [Eckerman and Whatley, 1977](#); [Eckerman et al., 1975](#); [Nash, 1986](#); [Rubenstein and Howes, 1976](#); [Rubenstein et al., 1982](#)), even though mothers of 13- to 15-month-olds initiate more interactions with their infants than vice versa and initiate as many interactions as do peers ( [Nash, 1986](#)). Novelty is not the issue either: When given the opportunity to interact with peers, mothers, and unfamiliar adults, infants interact more frequently with peers than with unfamiliar adults ( [Eckerman and Whatley, 1977](#); [Lewis et al., 1975](#)). On the other hand, parents are more sensitive to infants' developmental needs and thus are more likely to act in ways that foster their cognitive development ( [Tamis-LeMonda et al., 1994](#)). Some researchers believe that individual differences in peer instruction reflect differences in the security of prior parent–child attachments ( [Belsky and Cassidy, 1994](#); [Rose-Krasnor et al., 1996](#)), others suggest that skills honed in interaction with nonparental care providers are crucial ( [Howes, 1989](#); [Oppenheim et al., 1988](#)), whereas others point to generalized and perhaps inherent differences in sociability ( [Kagan, 1997](#); [Lamb and Nash, 1989](#)).

## INFANT–ADULT INTERACTIONS

The fact that infants prefer to socialize with peers rather than with adults does not mean that they are uninterested in, or wary of, unfamiliar adults. Indeed, they are quite adept in their social relations with new adults as well. When infants are approximately 8 months of age, they begin to distinguish between unfamiliar and familiar adults by showing signs of apprehension about the former ( [Bronson, 1972](#); [Emde et al., 1976](#)). This wariness was at one time termed *stranger anxiety* and was treated as a developmental milestone. More recent studies have shown that in many situations, infants do not always behave anxiously and that infants' responses to strangers depend to a large extent on the strangers' behavior. When an unfamiliar adult approaches an infant in a warm, friendly manner, the infant usually responds in kind ( [Bretherton, 1978](#); [Bretherton et al., 1981](#); [Clarke-Stewart, 1978](#); [Rheingold and Eckerman, 1973](#)). The parent's behavior, too, can affect the infant's response:



Infants whose mothers talked to them in a happy tone about a stranger were friendlier toward the stranger than infants whose mothers talked about the stranger in a neutral tone ([Feinman et al., 1986](#)). Thus, when placed in a situation conducive to socializing (i.e., when those present act in a friendly manner), infants respond accordingly.

A variety of studies have indicated that infants are quite interested in and able to interact with new people. [Rheingold and Eckerman \(1973\)](#) found that nearly all the 8- to 12-month-olds in their study looked and smiled at strangers and appeared comfortable in their presence. Many let the stranger pick them up while their mothers were out of the room. With both their mothers and a friendly, interactive stranger present, 12-month-olds approached the stranger and spent more time with her than with their mothers ([Ross and Goldman, 1977](#)). Furthermore, 9- to 12-month-old infants were as likely to follow an unfamiliar woman as their mothers into another room ([Hay, 1977](#)) and also actively interacted with strangers, willingly sharing objects with them ([Hay and Murray, 1982](#); [Rheingold et al., 1976](#); [Ross and Goldman, 1977](#)). One-year-olds engaged in cooperative play ([Bretherton, 1978](#)) and games ([Ross and Goldman, 1977](#)) with unfamiliar adults, whereas 18-month-olds imitated unfamiliar adults as much as they imitate their mothers ([Hay et al., 1985](#)), and will help unfamiliar adults to shelve groceries ([Rheingold, 1982](#)). Infants also can request help from unfamiliar adults effectively ([Mosier and Rogoff, 1994](#); [Rogoff et al., 1984](#)), and in one study used social referencing with adults they had known for only 10 minutes when confronted with a mildly frightening toy ([Klinnert et al., 1986](#)).

Infants' interest in and abilities to interact with people other than their parents can set the stage for the formation of relationships with others. Thus, 10- to 14-month-olds who were cared for by an unfamiliar adult for 2½ hours during each of 3 consecutive days formed bonds to the caretaker: They consistently approached her and cried when she left them with a person they had seen for only a few minutes ([Fleener, 1973](#)). One-year-olds who had interacted with a stranger for 8 minutes either did not cry when left alone with the stranger or were more effectively soothed by her than were infants who had interacted with a stranger for only 1 minute ([Bretherton, 1978](#)). Furthermore, infants cried when a stranger whom they had only recently met departed, leaving them alone in an unfamiliar room ([Ipsa, 1981](#)). Not surprisingly, infants can feel secure with people in addition to their parents. Thus, infants formed attachments to care providers both in day care centers ([Ahnert and Lamb, 2001](#); [Ainslie and Anderson, 1984](#); [Goossens and van Ijzendoorn, 1990](#); [Ricciuti, 1974](#)) and in Israeli kibbutzim ([Fox, 1977](#); [Sagi et al., 1985](#)). As with parents, the type of attachments infants formed with major care providers affected aspects of their personality several years later ([Howes, 1990](#); [Howes et al., 1988](#); [Oppenheim et al., 1988](#), reviewed by [Nash and Hay, 1993](#)).

Findings like these highlight the roles played by social interaction in shaping development in infancy. Born with the ability to express emotions, infants effectively engage adults from the start. Over time, the infant's expressive and social skills improve as a result of social experiences; the interactions between infants and their care providers establish dispositions that, when maintained by a stable social environment, may predict aspects of the children's functioning years later.

We began this chapter with a discussion of the transactional model; its portrayal of development as a joint function of infant and exogenous influences improves on the more static main effects and interactional models. It is fitting, then, to conclude the chapter with a discussion of infant temperament. From a transactional perspective, temperament appears to be an early emerging and direct way in which infants affect other individuals and, thereby, their own development.

## INFANT TEMPERAMENT

Developmentalists traditionally believed that individual differences in infant behavior were determined by differences in infant–mother interactions, with genetic/constitutional factors playing a minor role, such as in explaining susceptibility to separation distress. Today, however, theorists and researchers agree that individual differences may play a far more important role than this, and consequently issues concerning the definition, measurement, and origins of temperament have moved to the forefront of infant research ([Rothbart and Bates, 1998](#)). Temperament is generally viewed as having a genetic/constitutional base and as being somewhat stable over time, although theorists disagree over the stability and modifiability of temperament and the relation between infant temperament and later personality ([Buss and Plomin, 1975](#); [Caspi, 1998](#); [Rothbart and Bates, 1998](#); [Thomas and Chess, 1977](#)) (see [Chapter 15](#) for a general historical account of the concept of temperament).

### Conceptualizations of Infant Temperament

Temperament can be thought of as a constitutionally based source of individual variation in personality functioning, emerging early in life. Most measures of temperament sample aspects of infant affect, attention, and activity, attending to temporal (e.g., latency of response) and intensive (e.g., strength and sensitivity of response) characteristics of certain behaviors.

Although [Gesell and Ames \(1937\)](#) first suggested research on infant temperament, it was not until Thomas and Chess began the New York Longitudinal Study (NYLS) in the 1950s that research on infant temperament began in earnest ([Thomas and Chess, 1977](#); [Thomas et al., 1963, 1968](#)). The subjects of the NYLS were followed at regular intervals from infancy through adolescence and adulthood. Mothers were first interviewed when the infants were 2 to 3 months of age, and from these interviews Thomas and Chess derived nine dimensions of infant temperament: activity level, rhythmicity, approach/withdrawal, adaptability, intensity of reaction, attention span and persistence, distractibility, quality of mood, and threshold of responsiveness. Four behavior styles were ultimately identified. “Easy infants” were positive in mood, regular in body functions, and adaptable; these infants (comprising approximately 40% of the entire sample) approached new situations positively and reacted with low to moderate intensity. By contrast, “difficult infants” (10%) were negative in mood, irregular, and slow to adapt; they withdrew from new situations and reacted with high intensity. “Slow-to-warm-up infants” (15%) were negative in mood and slow to adapt; they withdrew from new situations, reacted with low to moderate intensity, and were low in activity. “Average infants” (35%) were not rated highly on any dimension.

Thomas and Chess reported that individual differences in infant temperament were fairly stable over time. However, one of their most important and provocative findings was that a child's behavioral style was not always predictable from knowledge of behavioral style in infancy. [Thomas et al. \(1968\)](#) reported that in approximately 70% of infants characterized as “difficult,” behavior problems eventually developed. Not surprisingly, these “difficult” children created special challenges for parents, who were described as behaviorally and emotionally inconsistent with their children; they frequently blamed themselves for their children's problems. Parents of those “difficult” infants who had no significant problems in later childhood adapted much more successfully to their children's difficult behavior, however; they were tolerant, patient, and consistent. Rather than blaming themselves, these parents accepted their children's temperamental difficulties with good humor. The major differences between these two groups of “difficult” children suggested that temperamental characteristics early in life can be modified by environmental experience. [Thomas et al. \(1968\)](#) argued that the “goodness of fit” between the child's temperament and the demands of the environment determined whether outcomes would be favorable or unfavorable.

Like many pioneering studies, the NYLS has been the target of much criticism. One major question concerns the close contact the investigators maintained with their subjects, raising the possibility that knowledge of the child's temperament at one point may have inadvertently influenced investigators' judgments of the same child's later behavior. Second, most of the information about temperament and behavior was obtained from parental interviews, raising questions concerning the extent to which data were influenced by parental perceptions. A third criticism arose from the conception of temperament as a behavioral “style,” which implies a kind of pervasiveness that seldom occurs in human behavior. Fourth, other researchers have failed to find evidence of the nine NYLS divisions. Instead, researchers have suggested that five or six independent dimensions of temperament are distinguishable ([Caspi, 1998](#); [Rothbart and Bates, 1998](#)).

Despite these criticisms, however, the growing recognition of constitutional differences at birth led [Brazelton \(1973\)](#) to develop an objectively administered Neonatal Behavioral Assessment Scale (NBAS). This scale was originally designed to assess neonatal behaviors having clear social significance to parents. Thus, it has many of the features of a measure of temperament, although the scale was not specifically designed as such. NBAS items assess states of alertness, capacities to self-soothe, responses to adult ministrations, and the abilities to respond and cease responding to animate and inanimate stimuli of various types. The NBAS also assesses motor abilities, muscle tone, and some neurologic reflexes. Early reports suggested that NBAS scores tended to correlate with later parental reports of temperamental traits, such as distractibility ([Sostek and Anders, 1977](#)).

During the 1970s, the NBAS became popular for documenting individual differences in the social capacities of neonates. Unfortunately, however, researchers failed to find significant reliability in neonatal performance on the NBAS, even from day to day ([Sameroff, 1978](#)). This could have a variety of explanations. First, infant social capacities may be genuinely unstable. Second, these social capacities may be stable, but fluctuations in infant state may mask stability. Third, the type and dosage of medication administered to mothers during childbirth, as well as the time elapsed between birth and testing, also may affect the temporal stability of the NBAS. Despite these limitations, the NBAS has become popular as an intervention tool for demonstrating infant capacities to parents ([Nugent et al., 1986](#); [Nurcombe et al., 1984](#); [Rauh et al., 1986](#)).

From the original interview responses obtained by Thomas and Chess, [Carey \(1970\)](#) developed the Infant Temperament Questionnaire (ITQ), which was later refined by [Carey and McDevitt \(RITQ; 1978a, 1978b\)](#). The ITQ and RITQ measure the nine dimensions derived by Thomas and Chess and also allow summary



characterizations of infants as easy, slow to warm up, difficult, intermediate low, and intermediate high. [Bates et al. \(1979\)](#) also have developed a parent-report measure, the Infant Characteristics Questionnaire (ICQ), measuring the dimensions of “fussiness-difficult” (on which they place greatest emphasis), “unadaptable,” “dull,” and “unpredictable.” [Rothbart \(1981\)](#) developed a third parent questionnaire, the Infant Behavior Questionnaire (IBQ), which measures the dimensions of activity level, soothability, fear, distress to limitations, smiling and laughter, and duration of orienting. Finally, [Buss and Plomin \(1975, 1984\)](#) have developed a parent questionnaire measuring the dimensions of emotionality, activity, sociability, and impulsiveness. Three additional temperament measures involve more objective home or laboratory assessments. [Bornstein et al. \(1986\)](#) have developed a measure that couples home observations and parent reports, which is built around infants' spontaneous activity and infant responsivity in structured vignettes. This measure assesses the dimensions of positive affect, negative affect, persistence, and motor activity. [Matheny and Wilson \(1984; Matheny, 1980, 1983\)](#) have created a laboratory-based assessment of temperament, measuring the dimensions of test extraversion, activity level, and task orientation. Finally, [Goldsmith and Campos \(1982, 1986\)](#) have developed a laboratory observation technique, tapping 10 task-specific dimensions. All of these measures remain in use today, despite concern about their validity and reliability ( [Rothbart and Bates, 1998](#)).

Overall, little consensus exists regarding the prominent characteristics of infant temperament, and the psychometric properties and convergent validity of temperament measures are poor ([Hubert et al., 1982](#)). Most instruments also show only moderate temporal stability, low interparent and parent–observer agreement, and inconsistent relations to observed interaction patterns. Nevertheless, it may be desirable to obtain information from both parents and observers, even if scores on the two measures differ, because temperament is so complex a construct. Both may measure valid aspects of infant temperament, and although parental ratings may be colored by idiosyncratic perceptions and parental personality ( [Goldsmith and Campos, 1982; Kagan, 1998; Sameroff et al., 1982](#)), these perceptions may be more important predictors of infant–parent relationships than “objective” measures of the infant's temperament would be ( [Bates, 1980, 1983](#)).

During the 1990s, investigators began focusing on physiologic measures of temperament, after its successful use in studies of toddlers and preschoolers during the 1980s (see [Kagan, 1998](#), for a review). Individual differences in positive and negative emotionality are thought to reflect difference in temperament. Thus, measures of neural and endocrine processes (related to genetic differences) should correlate with other measures of emotionality. In support of this, [Gunnar and Nelson \(1994\)](#) have found that differences in event-related potentials were associated with differences in emotionality and cortisol levels. Higher cortisol levels have been reported for behaviorally inhibited infants compared with uninhibited infants in several studies by Gunnar and associates ( [Gunnar et al., 1992, 1995; Larson et al., 1991; Nachmias et al., 1996](#)). [Stifter and Fox \(1990; Fox and Stifter, 1989\)](#) included measurements of vagal tone in their longitudinal study of emotional reactivity in infants and found that at 5 months infants with high vagal tone were emotionally more reactive to negative events, and at 14 months they were quicker positively to approach a stranger and a new toy than were infants with lower vagal tone. [Porques et al. \(1994\)](#) reported that vagal tone was higher in fussy infants, and that 9-month vagal tone predicted temperamental difficulty at 3 years of age. Apparently, individual differences in parasympathetic control (as indexed by vagal tone) affect socioemotional organization, such that infants with higher parasympathetic control (higher vagal tone) are better organized socioemotionally and more behaviorally responsive to others ([Porques, 1998](#)).

### Stability of Infant Temperament

Given the expected relation between infant temperament and later personality, temporal stability was initially seen as a necessary component of temperament ( [Buss and Plomin, 1984](#)). However, the age-to-age correlations for different dimensions of temperament are variable and may be especially low in the first years of life ([Rothbart and Bates, 1998](#)). This apparent instability was first attributed to measurement error or to environmental flux, but most recently, researchers have begun to ascribe the fluctuations to the discontinuous actions of genes over time ( [Matheny, 1980, 1983; Plomin, 1983; Scarr and McCartney, 1983; Wilson, 1983, 1984](#)). In fact, temperament seems to follow a complex fluctuating course in the earliest years, as befits characteristics regulated in part by “on again, off again” gene programs ([Plomin, 1983; Plomin et al., 1993](#)). The patterns of developmental acceleration and lag also appear to be more synchronous in more genetically similar organisms, suggesting that the fluctuations are meaningful, not random ( [Matheny, 1983; Wilson, 1978, 1983, 1984](#)). Most likely, certain dimensions of temperament [e.g., behavior inhibition ( [Kagan, 1998](#))] may show greater longitudinal stability than others.

### Temperament and Infant–Parent Interaction

It was widely assumed for many years that individual differences in parent–infant interaction were determined by prior parental behaviors, but parental behavior may itself be influenced by the infant's temperament and behavior ( [Bell, 1968, 1971](#)). Parents are extremely sensitive to their infants' temperaments, and different temperaments can be expected to elicit different behaviors from caretakers. Difficult infants demand and receive more attention than easy infants in the United States ([Bates et al., 1982; Petit and Bates, 1984](#)), Israel ([Klein, 1984](#)), and Kenya ([deVries, 1984](#)). Moreover, the perception of difficultness in early infancy increases the likelihood of later maltreatment ([Sherrod et al., 1984; Vietze et al., 1980](#)). Furthermore, [Vaughn and Bost \(1999\)](#) have argued that parental perceptions affect parent response, which in turn shapes the infant's future behavior.

Temperament also is constructive. An infant's temperament affects those around the infant, who in turn are likely to treat the infant in consistent ways that help construct the infant's own personality. A temperamentally active infant might be considered “inquisitive”; a temperamentally inactive infant, by contrast, might be seen as “thoughtful” or “passive.” Insofar as parents believe that their infants' temperaments fit a particular mold, they may mold their infant's environment to suit these expectations. In this sense, parental perceptions are important regardless of their relationship to objectively defined characteristics.

More than any other investigators, [J. Bates \(1980, 1983, 1986\)](#) and colleagues ([Bates et al., 1985a, 1985b; Petit and Bates, 1984; Rothbart and Bates, 1998](#)) have explored the assessment and clinical implications of infant “difficultness.” Difficultness has at its core frequent and intense expressions of negative emotion that are part of the child's disposition. If difficultness is dispositional, it ought to appear early, have a biological basis, and endure. Bates has confirmed that difficultness manifests itself in infant crying, and [Lounsbury and Bates \(1982\)](#) found that hunger cries of difficult infants are higher pitched and are perceived as aversive and demanding even by unrelated adults. [Bates et al. \(1984\)](#) believe that the excessive demands for social interaction made by difficult infants also lie at the root of their mothers' attributions of difficulty, because mothers interpret this “coercive control” negatively. Further, difficultness is relatively stable between 6 and 24 months ( [Fish and Crockenberg, 1981; Lee and Bates, 1985; Snow et al., 1980](#)). Finally, difficultness assessed by maternal report at 6 months predicted maternal perceptions of aggressiveness and anxiety at 3 to 5 years ([Bates et al., 1985a, 1985b](#)). Therefore, it is possible that difficult infants have troublesome dispositions that endure, or it could be that, as infants, they behave in ways that encourage their parents to treat them so that they act out, years later, as a result.

In addition, difficultness can have distinctly positive connotations in some circumstances. [DeVries \(1984\)](#) studied 4-month-old Masai infants in Kenya, where drought and malnutrition take the lives of many infants. In an admittedly small sample, difficult infants—those who were intense, irregular, unadaptable, and fussy—tended to survive, whereas easy infants tended not to survive. DeVries hypothesized that the demandingness of difficult infants is an advantage in the Masai society. The moral here is that temperamental concepts like difficultness must be understood in context; in our culture, difficulty may be associated with behavior problems, but in the Masai environment, it may promote survival and health.

Of course, difficultness is not the only infant characteristic that may predict later behavior problems. Activity level could be associated with a propensity to childhood accidents, whereas many psychiatric syndromes, including manic–depression and anxiety neurosis, could involve premorbid characteristics even in infancy. Perhaps the study of infant temperament, in conjunction with a certain parental personality or disciplinary styles, will lead to the identification of significant early indicators of risk, and, more generally, of the diverse ways infants contribute to their development.

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## 23 DEVELOPMENT OF SCHOOL-AGE CHILDREN

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The middle years of childhood, spanning the age from when a child enters primary school through age 10 years, are also called the *school-age period* because of the critical importance of school in development in our society. This time has been characterized by many classical theorists as the period when a child enters society and begins to establish the basis for becoming a contributing member of his or her community. Sigmund Freud proposed that this was a sexually quiet “latent” period following the mastery of oedipal strivings and establishment of the superego. He likened the period to the glacial epoch in the earth's history, both quiescent periods necessary for the birth of civilization ( [Buxbaum, 1980](#)). It has since been established, however, that latency is a myth. [Erik Erikson \(1950\)](#) lent a more enduring characterization of this period when he described the critical psychological issue: “Industry versus Inferiority.” In formal schooling, a child is attempting to master the basics of the industry of our society, to build on academic abilities. Failure to progress in school and in the peer context can establish a sense of inferiority rather than support the momentum of a drive for competence. Neofreudians (i.e., Sullivan, Horney, Thompson) added an emphasis on social context as the critical shaping force on how this developmental period is negotiated. Harry Stack Sullivan, for example, observed that this “juvenile era” provides the first opportunity for society to correct the influence of the family.

Freud, Erikson, and Sullivan have defined the important work of this period. Research into the details of cognitive, emotional, and social development confirm that civilization and being-in-society are the developmental challenges of the school-age period. Thus, thinking about the child's psychological well-being must extend beyond the child to include not only the family but the social, economic, and political contexts that define its functioning. Vygotsky's concept of “mind in society” and Bateson's “ecology of mind” point to the dynamic exchange of experience, accomplishment, and social response and effectiveness.

The best way to emphasize the remarkable growth that occurs during middle childhood is to contrast the skills of children when they enter and exit grade school ([Table 23.1](#)).

**Table 23.1. Neuropsychiatric Examination of the School-Age Child**

[Table 23.1](#) describes what can be seen in normal, average children in the United States, whether they come from poor, inner-city neighborhoods, rural settings, or more privileged educated family backgrounds. Children may be more advanced if they have been provided with educationally stimulating experiences. Children who do not perform at the levels indicated in the table bear closer examination to determine whether “delays” are due to disabilities, emotional factors, or contextual factors.

In this chapter, we review current information about how children advance from preschool to preadolescence, examining the areas of physical maturation, emotion, gender differences, moral development, social development, and cognition. These areas must be understood in the contexts of the child's internal life and in relation to family, peers, and school. We focus greater attention on issues of schooling—because school-aged children spend most of their waking lives in school, the school environment is vital to every aspect of the child's development, including how the child views the family and how the family views itself in relation to the child.

### MATURATION OF THE CENTRAL NERVOUS SYSTEM

The brain undergoes a period of rapid growth to 2 years of age and grows at a much slower rate until puberty. At birth, the brain is estimated to be approximately 10% of adult volume: it grows to 90% of adult volume by 5 years of age and completes its growth slowly over the next 9 years. What is more significant than actual volume, however, is the modification of anatomic structures and myelination, which is almost completed by the age of 7 years ( [Shapiro and Perry, 1976](#)). Synaptic pruning in the prefrontal cortex (the area affecting social judgment) continues as an ongoing process through adolescence.

Magnetic resonance imaging studies of school-aged children's brains have confirmed these observations. By 7 years of age, the child's brain is approximately the size of the adult brain. Boys' brains are approximately 10% larger than girls', and this total volume difference persists into adulthood ( [Giedd et al. 1997](#); [Reiss et al., 1996](#)). Differences, too, are found in the basal ganglia, where the globus pallidus is larger in boys, whereas the caudate is larger in girls. Boys show a relatively greater increase in size of the amygdala, whereas girls have more growth of the hippocampus. These relative differences are consistent with findings of androgen receptors in the amygdala and of estrogen receptors in the hippocampus. There also are findings of greater lateral ventricular expansion in boys ( [Giedd et al., 1997](#)). [Caveness et al. \(1996\)](#) found that subcortical gray structures are at adult volume in girls and are greater than their adult volumes in men, whereas the volume of central white matter is smaller in the female brain. The significance of these findings in terms of social development, regulation of focus and attention, management of activity and aggression, and the like is not yet described. However, the stage of concrete operations characterizing the cognitive functioning of school-aged children may be correlated with findings of reduced slow frequencies in the delta and theta bands and an increase in higher frequencies in the alpha band of the electroencephalogram between 5 and 7 years of age (for more detail, see [Chapter 5](#)).

Some data on neurotransmitter development may further sharpen our view of the intrinsic maturational schedule of the child and elucidate the emergence of the abilities of school-age children. In contrast to noradrenergic systems, which develop early and exert early influence on the formation of the cortex, dopaminergic systems, associated with attention regulation, and serotonergic systems, associated with mood and aggression, have a more gradual establishment of crucial



connections between brain stem nuclei and cortical structures. Cholinergic systems, associated with memory and higher cortical functions, develop relatively late ([Coyle and Harris, 1987](#)).

These refinements in brain structure and function result in the maturation of higher cortical functions, correlating with improved abilities in motor coordination, increased attention and focus, increased self-regulation, and expanded consideration for others. For example, tasks of writing, organizing work on a page, coordinating sounds with visual cues (as in deciphering words and spelling phonetically) require control in ear and eye–hand coordination. Children who enter the school-aged period with the ability to write letters or words backward or who may write their names with the correct letters but in a different order, are not necessarily showing signs of learning disability. Rather, they have not yet developed mastery of the conventions involving direction and order that are necessary to read and write and perform arithmetic functions. It is normal and expectable not to master these conventions until second grade.

## GENDER DIFFERENCES IN DEVELOPMENT

### Psychosexual Development

According to psychoanalytic theory, sexual development is biphasic, with a “latent” period during the school-age years. Freud believed that latency was a distinguishing feature of humans over animals and hoped to discover anatomic evidence of this through then-promising studies of changes in the interstitial portions of the “sex glands” ([Buxbaum, 1980](#)). Contemporary studies of sex hormones do not support the biphasic theory, however. Infants have relatively high but varying proportions of sex hormones in cord blood. The levels of sex hormones fall after birth and begin to rise, owing to endogenous production, during the school-age period (ages 7 to 8 years in girls, and approximately 2 years later in boys). Sex hormones begin a gradual upsurge at approximately 8 years of age, continuing through the pubertal peak, children engage in some sexual play with self and others, and retrospective interviews with homosexual adults usually pinpoint middle childhood as the time when sexual preference was established. Some have postulated that school-age children's greater sexual awareness may be reflected in their expressed feelings of disgust and shame and the strong sense of modesty that develops during the school years. Others have reported that sex play among school children is a natural extension of that of preschoolers ([Rutter, 1971](#)). Infants and toddlers masturbate; preschoolers also engage in mutual exploration. Psychoanalytic theory posits that sexual strivings in the oedipal child are overcome with the development of defense mechanisms. Children often enter middle childhood with a few good friends of the opposite sex. Around 8 years of age, however, the same-sex groupings become polarized, with the opposite gender having “cooties” and generally avoided or teased. Moving toward preadolescence, however, the “yuckiness” of the opposite sex gradually gives way to admiring certain individuals from a distance. Older school-age girls' budding attraction to movie stars and rock idols serves a group interactional function, as well as helping to define identity. As with preschoolers, if school-age children are preoccupied with sexual themes, it is wise to look for the possibility of sexually stimulating experiences, such as sexual abuse or witnessing sex acts.

There is a wide range in both the timing and tempo of pubertal onset, and it begins for many girls near the end of what traditionally is termed middle childhood. The onset of puberty at 9 to 11 years of age, as measured through breast growth, correlates with a positive body image, positive peer relationships, and superior adjustment in girls ([Brooks-Gunn, 1988](#)).

It is well established that children adopt a firm gender identity by age 3 years. This expression of their maleness or femaleness is manifested early by choices of role models and friends. With gender differences, as with so many of the other issues discussed in this chapter, the question of what is innate and what is the outcome of socialization is very intriguing.

Money's studies of infants with ambiguous genitalia established that gender identity was primarily determined through socialization. But explorations of differences in sexual preference and transsexual behaviors have raised questions about the roles of sex hormones in determining gendered behaviors. [Carol Jacklin \(1989\)](#) reviewed a number of purported relationships of hormones with behavior. There were no consistent findings and many weaknesses with the studies. Jacklin proposed that the internal representation of a person's gender, which she refers to as *schemata* (changing and evolving networks of associations to filter and organize information about oneself, one's gender, and its meaning) evolve out of diverse information, including modes of behavior, properties of objects, attitudes, and feeling states. Information is both presented and processed differently for boys and girls. For example, Jacklin notes, it is rare to see someone compliment a girl on how strong she is getting or a boy on how nurturing he is. Gender schemata and their expressions are evolved and maintained in a gendered society where it is most likely that girls will keep the company of women and other girls, and boys will do likewise with men. Cross-cultural studies do show male and female characteristic distinctions, but it appears that association with children and adults of the same sex is a powerful influence. We become the company we keep.

A number of characteristics are associated with being male or female in the school-age period. [Carol Gilligan \(1982, p. 9\)](#) cites Janet Lever's studies of 181 fifth-grade children at play. She observed that boys play outdoors in large and heterogeneous groups, and they play competitive games that last longer than those of girls. The games played by boys are full of disputes that seem to add interest to the interaction and do not derail the game. Similar observations of children playing led Jean Piaget to conclude that boys were more advanced in moral development because of their fascination with legal procedures and experience at generating fair arbitration of disputes. Many others concluded similarly that in the area of moral development and the development of the capacity to exert effective leadership in complex groups, boys preceded girls; few girls ever caught up.

In proposing that women listen to the demands of socialization and morality “in a different voice,” [Gilligan \(1982\)](#) added new value to this “instrumental- versus expressive” gender dichotomy. Gilligan's descriptions of different lines of moral development for boys and girls is discussed later in the review of moral development during school age.

Recognizing that in general boys develop instrumental functions and girls develop expressive ones has provided explanations for other observed differences between boys and girls. In academic achievement, for example, girls traditionally tend to do better in verbal areas, whereas boys have done better in math and science. Even as the causes of these differences are being explored, they also are being denied and recharacterized. Feminism apparently has had its effects on gender identity and gender role behaviors in both girls and their mothers. For example, the widely held view that boys are more mathematically capable than girls has been demonstrated to be more an effect of socialization than innate capability. Math anxiety was related to gender-stereotyped beliefs of parents, the mothers being most influential ([Eccles and Jacobs, 1986](#)). Furthermore, gender differences in academic skills that had been previously noted now are not found on many tests of academic competence ([Jacklin, 1989](#)). It is likely that other so-called innate gender differences will be similarly reevaluated in the future. Still, [Gilligan et al. \(1990\)](#), in a Harvard research study, describe “hitting the cultural wall”—when preadolescent girls realize that society values appearances more than accomplishment, they become more self-critical and worry about their weight. A negative body image was found to be associated with high IQ. In the American Association of University Women's “Shortchanging Girls, Shortchanging America,” a study of 3,000 girls and boys in fourth through tenth grade, concluded that girls lose their positive self-esteem and switch to appearance as the primary way to measure themselves ([Greenberg-Lake Analysis Group, 1991](#)). Other studies ([Debold et al., 1993](#); [Sadker, 1994](#)) support that preadolescent girls are more likely to get depressed, have their IQ scores drop, and decline in math and science.

### Cognitive Development

The standard by which school-age children's cognitive competence has been evaluated has been the achievement of what Jean Piaget termed *concrete operations*. The preschooler's preoperational thought is a creative effort to grasp causality and make meaning of experience using idiosyncratic and egocentric logic. In contrast, school-age children master important operations that increase their objectivity and their ability to be conventional. For example, 4-year-old Jocelyn demonstrates “jumping” by doing calisthenics, and when asked to stand still and hold her hands out, quickly evolves the motion to a pirouette. Seven-year-old James makes a concentrated effort to do exactly what was requested and refuses to lift his shirt, so the examiner can look for periumbilical movements, because he does not show naked parts of his body to strangers.

Classification and conservation are the two crucial achievements of concrete operational thinking. Classification is the ability to group objects or concepts; conservation is the ability to recognize constant qualities/quantities of material even when the material undergoes changes in morphology. The concrete logical operations are

1. Composition—combining elements leads to another class (e.g., red triangles and blue triangles leads to another group, i.e., triangles)
2. Associativity—combinations may be made in different orders with the same result
3. Reversibility—being able to return mentally to an earlier point in the process

The concrete logical operations enable the child to deal systematically with hierarchies and categories, series and sequences, alternative and equivalent ways of

getting to the same place, and reciprocal relationships ( [Minuchin, 1977](#), p. 14).

Logical operations are crucial to mastering basic reading and mathematics skills, and they also are necessary for conducting social interaction, with its increasing complexity of groups, games, and rules. Acquisition of the ability to do conventional and objective mental operations is associated with an interest in the scientific workings of the birth process, and a grasp of the finality, universality, and inevitability of death.

Successful school-age children have not just the ability to perform the specific concrete operations themselves but the ability to communicate about them in conventional ways. They understand that there are conventions of conversation, responses to questions on tests, and social comportment. With cognition, as with almost every other aspect of the school-age youngster's development, joining society and sharing conventions is the key to success. This interest in conventions and rules frequently is accompanied by a fascination with ordering and ritual. For example, school-age children often develop favorite numbers, magical rituals ("Step on a crack, break your grandmother's back") or the need to do things in even pairs. They also may become collectors of coins, stamps, insects, baseball cards, comic books, and the like, and may spend a great deal of time reviewing and ordering their collections.

## Morality

Along with the development of concrete operations, a child's sense of morality, that is, the appreciation of consequences and justice, evolves from an egocentric and idiosyncratic, and often harsh system of evaluation of behaviors by punishment, to the adoption of internalized rules for evaluating behavior.

[Piaget \(1948\)](#) posited that school-age children's morality is in the "interpretation of rules" stage. This accomplishment permits the child to understand the spirit of a rule and to make subjective moral judgments.

[Kohlberg \(1969\)](#) described the moral development that most school-age children reach as the level of "conventional morality." Conventional morality contains two stages: "interpersonal concordance" and "orientation toward authority." In the stage of "interpersonal concordance," a child measures behavior and judges it on the basis of whether it pleases those he looks up to. These mutual interpersonal expectations are those of a "good girl" or "good boy" who wants to please her or his parents and teachers and obeys the Golden Rule ("Do unto others as you would have them do unto you"). The next stage in conventional morality, "orientation toward authority," reflects the societal values of duty, respect, and law and order. This differs from the stage of interpersonal concordance in that the child's moral compass is now set by the social system instead of the immediate social context of family, school, or neighborhood. The child supports the rules of society, believes that it is essential not to break these rules for society to function, and makes moral judgments based on how well an individual situation conforms to the rules of the social system.

Gilligan's studies of girls' and women's moral development led her to emphasize the importance of relationship. In contrast to the masculine quasimathematical system for evaluating moral choices, Gilligan finds that girls use a form of narrative that evolves solutions within conversations and interpersonal action. Thus, Gilligan interprets the fifth-grade girls' play observed by Lever (described earlier) not as poorly developed or socially immature but as valuing different aspects of the social experience. Gilligan describes the different responses of an 11-year old boy and girl, both of whom were at the top of their sixth grade class in a private elementary school in an academic community. The moral test question was that of the man whose wife is gravely ill and whose survival depends on receiving a specific medicine. The medicine is too expensive and the pharmacist will not reduce the price. The man breaks into the pharmacy and steals the medicine for his wife. In responding to the question of what should happen to the man, the boy thoughtfully weighed the problem of laws against stealing and a higher law valuing life. The girl, on the other hand, felt that the various parties needed talk to each other, and could not render an opinion about what should happen to him. She was aware of the rules but found the conflict was such that she felt that mediation was needed to reach a resolution.

There has been significant critical reanalysis of Gilligan's data and conceptualization of the gender differences in moral reasoning. For both girls and boys, the development of morality reflects conventional thinking and measuring their evaluations against the rules of their society, as they understand them. Although moral reasoning continues to evolve through and beyond adolescence, many of the standards that are developed for our own behavior during middle childhood are likely to remain internalized and used as self-evaluation measures into adulthood.

[Stilwell et al. \(1997\)](#), in a study of 132 students aged 5 to 17 years, describe moral development as a natural outgrowth of attachment, evolving through five stages. First, the child's sense of security and experience of empathic responsiveness become paired with a sense of moral obligation. Next, the caretaker's rules are incorporated. Then, an understanding develops of how empathy can modify strict rule-following. Next, ideals and role models are selected that reflect earlier learning in attachment relationships. Finally, the self is visualized as a keeper of moral standards. These stages roughly correlate with Kohlberg's stages of morality, but emphasize the grounding of morality and conscience in the early and fundamental experience of attachment and a secure base, out of which empathy develops.

## EMOTIONAL ISSUES

The most significant emotional issues in the lives of school-aged children concern personal worth, which is determined by a sense of competence and place (in family, peer group, and communities). Competence is reflected in all of the places a child may live, at home by accomplishing tasks of caring for self (completing dressing, including tying shoes) and at school by addressing the academic material presented. [Robert White \(1959, 1960\)](#) postulated a "drive" to competence that he thought was as important as libidinal drives. In the school-age period, a sense of competence is determined not just by the child succeeding at a task, but by others' evaluation of his or her performance.

As Erikson warned, the emotional risk for the school-age child is the possibility of feeling inferior if the child evaluates himself or herself as not being able to accomplish tasks. This evaluation comes first from outside, from a teacher expressing disappointment or frustration, from other children laughing, from parents' disappointment with grades or a teacher's report. Increasingly through the school-age period, children can evaluate their own performance and measure it against that of others. Failures in one area may be compensated by accomplishments in another, eventually, but the early school-age child who has not yet learned about compensation simply may feel bad. By the end of middle childhood, each child has constructed a composite evaluation of his or her own relative areas of competence and weakness and has come up with his or her own answer to the questions, "What am I good at? Can I get the job done?" Again, these characterizations tend to persist into adulthood.

The fears of a school-age child are quite different from those of a preschooler. Because school-aged children are out and about in society, they are much more likely to witness or hear about catastrophic events that could happen to them. Their vulnerability to catastrophic fears is increased by the development, during the school-age period, of understanding of the irreversibility and inevitability of death. Many school-age children's dreams reflect efforts to master these fears by setting themselves up as heroes who save whole families or communities from robbers, murderers, fires, storms, or other disasters. Children who do not feel competent may be overwhelmed by these fears and have repeated dreams in which they are attacked and victimized and helpless.

[Harry Stack Sullivan \(1953\)](#) was one of the first to emphasize the social influence on development. He described a series of internal processes by which the child gradually substitutes his or her own standards of evaluation for those of family members. Stimulated by models outside the family, these processes unfold throughout the early school period.

- *Social subordination* reflects a change in the child's acceptance of authority from the specifics of personal caretakers to general categories such as principal, police, crossing guards, and teachers. The child first evaluates peers in terms of how they are regarded by these authority figures.
- *Social accommodation* is a process of acknowledging that there are differences between people. Early school-age children are intolerant of differences and can be cruel, but with socialization and education, differences gradually may come to be respected.
- *Differentiation of authority figures* is the child's emerging ability to compare adults, comparing parents with school-based authorities.
- *Control of focal awareness* refers to the child's response to social pressure to abandon some of his or her egocentric ideas and to adopt a more conventional stance.
- *Sublimatory reformation* refers to the reorientation of focal awareness to the group-approved satisfactory behavior.
- *Supervisory patterns* reflect an awareness of one's behavior in groups. The supervisory patterns are almost like imaginary characters who develop, so that the child can monitor himself or herself and eventually become internalized.

## SELF IN SOCIETY

The most salient achievement of the school-age period is a sense of oneself as a member of society. To accomplish this, maturation is required, as has been



described. But the most significant arenas for advancing and refining the sense of self, both in the present and in anticipating the future, are the interpersonal arenas of family, peers, and school.

## Home and Family

According to Heinz Kohut, the development of self occurs through a process of mirroring and idealization. To develop healthy narcissism, the child needs grown-ups to admire him or her and demonstrate attunement to his feelings (“mirroring”). Children also need to be able to look up to their parents and to other role models and aspire to be like them without being unduly distracted by their faults and shortcomings (“idealization”).

Similarly, parenting styles have been classified by [Diana Baumrind \(1996\)](#) according to “responsivity” (accurately assessing and responding to children’s needs) and “demandingness” (setting high expectations). Parents with high responsivity and high demandingness (“authoritative” style) tend to have the best outcome, with children who do well academically and socially. Low responsivity/low demandingness describes the neglectful or uninvolved parent; and high responsivity/low demandingness describes the permissive parent. Low responsivity/high demandingness is characteristic of an authoritarian style, which may be predictive of a positive outcome in some minority families.

The parent’s optimal role in middle childhood may be that of a consultant or facilitator, coaching the child’s development of his or her own skills and opinions, assisting as needed when help is requested, but allowing mistakes to be made and independent striving to occur in a supportive environment whenever feasible. This can be congruent with an authoritative style, in that high responsivity and high expectations can coexist with allowing self-exploration on the part of the child.

The familiar social context of the family and neighborhood (which also includes involved extended family and religious communities with which the family may be involved) is altered in the school-age period through several processes. The first is a practical one: When children spend more time in school, parents may spend more time doing things other than caring for their children. For parents who already were working or who remain at home caring for younger children or who choose to home-school their children, this may not be a significant change. In general, however, this creates a significant shift from the family organized for care of itself and its young children, drawn inward by primarily centripetal forces, to the more outwardly oriented family of adolescent children, where forces seem to be centripetal, drawing family members out into interactions with the society at large. [Combrinck-Graham \(1985\)](#) described the school-age child’s family: “The family’s opening up is like a house in the summertime; it is sturdy but has doors and windows open for circulation. Everyone comes in to share the family meal, to take shelter from the rain, and to sleep” (p.147).

A second process that changes the family environment is the evaluation that comes from children bringing home their experiences with other children, other children’s parents, and other adults whom they meet independently of their own families. Children make statements: John’s mother lets him do this; Martha’s mother doesn’t do that; Sandy’s father doesn’t live with them. Or they ask questions: Why doesn’t Daddy stay home with us they way George’s father does? How come you don’t pick me up at school, Randy’s mother does? Are we rich or poor? Are we Republican or Democrat? Why don’t we celebrate Christmas? And so on. Or children reflect frank criticism: You don’t know as much as my teacher. Smoking is bad for you; you shouldn’t smoke.

A third process of family change is through children’s relatively greater involvement in activities outside the home. Visits to friends, after-school activities, membership in clubs, or participation on teams takes time out from family routines after school and on weekends. Adolescents are far more involved in activities outside the home, but most school-age children’s families have the opportunity to assemble for dinner and an evening routine that allows for completing homework and some form of age-appropriate bedtime routine. Exceptions are largely due to complicated work schedules of caregivers who may work second shift or overtime.

A fourth process of family change is the possible social enrichment of all family life through involvement with the school as a community. This can be through socializing with families of other children, involvement in school and after-school activities, and through advocacy about school issues (e.g., PTA or participation on the school board). All family members usually become more involved in social experiences outside the family during the school-age period (not just the children).

Family/community relationships that had been stable before the school-age period are subject to change in similar ways. In the early school-age period, children are developing skills that facilitate interaction with peers in the neighborhood (e.g., mastering a two-wheel bicycle; accomplishments in the pick-up sport of the neighborhood, such as soccer or basketball). Children may have different experiences with other children in the home area and in school. At home, in the neighborhood, a child may be included in a group of different-aged children and accepted because of familiarity or the relationships between the families. For example, children may be a part of a Sunday school group that also is part of the social context of the congregation. This peer group reflects the child’s place in a family’s place in a community. As the school-age child grows older in such a community, he or she becomes more identified by distinct contributions to the community (e.g., participating in the music program, mastery of religious lessons, contributions to recreational activities) rather than just by membership in a family.

Children may assume more distinct roles in their neighborhood society or Sunday school group as they develop in school, or they may be less involved, as they become more interested in other things. Hobbies and collections are characteristic passions of school-age children and often become the basis for formation of new social groupings. Social contacts may be conducted online as children come together around a particular interest.

## Peers

The peer group can be one of the most facilitating influences in school-age children’s development, or it can be disastrously inhibiting. As with other developmental tasks, each child brings a particular pattern of prior experience to the task of developing a social self. There is considerable evidence that peers themselves have their own attractions and that a substantial if not primary influence over a child’s social self-development comes from the outside, predominantly through peer culture and its particular draw on the child’s drives for mastery and competence. Robert White’s ( [1959, 1960](#)) description of the growth of competence drive in the school-age period emphasizes how to get along with others in the sense of competing, compromising, learning the rules of the game, and protecting oneself from injury. He points out that other children afford an opportunity to do something interesting with the environment and that gradually the world of contemporaries competes with the family circle. [Bemporad \(1984\)](#) describes the juvenile era as a period between separation and procreation in which peers are the intermediaries. Erikson’s concentration only on how children master industry places too much emphasis on individual development and gives insufficient attention to cooperation. In a cross-cultural comparison, [Kagan and Klein \(1973\)](#) credit the society of peers for advancing the development of children in San Marcos, Guatemala. They describe rural Guatemalan Indian infants and preschoolers who by all culturally relevant measures are “retarded” but who at 11 years of age perform at the same level as American children in tests of cognitive functioning. The Guatemalan infant is unstimulated and left alone; only basic physical needs are attended to. Thus, Guatemalan infants do not have the assertive interaction with environment that is so prized in American infants and preschoolers. Kagan and Klein suggest that in the school-age period, where the relative neglect of these children by adults leaves them to form their own social groupings, Guatemalan youngsters begin to practice and learn assertiveness through jockeying for social position. [Grunebaum and Solomon \(1980\)](#) refer to Harry Harlow’s studies, concluding that young rhesus monkeys leave their mothers because peer relationships are interesting, not because their mothers reject them. There has been some recent debate about the relative influence of peer group versus parents on development, with some taking the view that the peer effect is significant and the parental effect is negligible ( [Harris, 1998](#)). Regardless of the relative weight placed on each of these factors, it is clear that the drive for inclusion and acceptance, and the judgments of the other children whom the child selects as his or her peer group, have a heavy impact on the school-age child’s development of self-image and values. At the same time, the child’s ongoing internal self-definition in turn influences his or her selection of peers to identify with and measure himself or herself against. By approximately 8 years of age, there has been a significant shift in a child’s ability to compare and assess his or her own skills, both through comparing him or herself with others, and through using feedback from parents, teachers, and peers. The child begins to rank himself or herself in various arenas and to combine these multiple assessments into his or her own ongoing “report card.” This constant evaluation of self in social context becomes internalized into the child’s own kinetic sense of identity. For better or worse, the opinions and descriptions we form of ourselves in middle childhood tend to continue throughout life. Personal “style,” preferences, values, and self-assessment in comparison with others all have their foundation during the school-age period.

Stages of peer development have been identified and described by [Grunebaum and Solomon \(1982\)](#):

- Unilateral partners and one-way assistance—the preschool child
- Bilateral partners and fair-weather cooperation—middle childhood
- Chumship and consensual exchange—preadolescence (pp. 288–291).

The first school-age phase is characterized by membership in peer groups and is based on playmates’ willingness and ability to play the way the child wishes. The second school-age stage (from approximately age 9 years) advances friendship to a closeness that Sullivan referred to as *chumship* with a peer of the same sex with whom an intimacy is formed, which paves the way for heterosexual intimacy and caring beginning in adolescence. A study that examined second, fifth, and eighth



graders' attitudes about and choices for companionship and intimacy found that family members were the most important sources for companionship for both second and fifth graders. Same-sex peers were important throughout the school-age period but were increasingly important as the subjects grew older. Only for eighth graders did opposite-sex peers become at all interesting. Girls tended to report intimate disclosure to peers earlier than boys, probably reflecting that girls may value intimacy more than boys ([Buhrmester and Furman, 1987](#)).

Just as peer interaction and the view of self in relation to others is vital to cognitive and intellectual development, so cognitive operations are vital to a child's emerging social self in the school-age period. [Minuchin \(1977\)](#) points out that children move from games such as "Simon Says," "Mother May I," and "Follow the Leader," in which the children in groups follow the directions of a leader, to games in which the rules are set and governed by the players themselves, to games that involve contributions to the efforts of a team. This evolution involves shifts in the ways in which others are evaluated. Children begin the period by deeming others good if they give them things and bad if they take things away, and move to recognizing skills and personal attributes, to finally acknowledging and valuing social attributes, such as fairness. This shift, in turn, requires the expansion of perspective, which permits a child to see a situation from another's point of view. This decentering may be seen as a cognitive component to empathy and the development of more sophisticated morality.

With social development, as with intellectual development, preparation and prior experience are substantial influences. Patterns of behavior involving aggression are established early, and generalized aggressive disposition and the tendency to exhibit aggression in the context of specific relationships are quite stable ([Cummings et al., 1989](#)). Aggressiveness also is associated with social rejection in the school-age period ([Boivin and Begin, 1989](#); [Rogosch and Newcomb, 1989](#)). One view of the stability of aggression is that it is constitutionally determined. Another view is that aggression develops and is maintained in interpersonal sequences. For example, one researcher reported that "early-timing" mothers, those whose first children are born when they are in their 20s, have more difficulty setting limits on their children than "late-timing" mothers do ([Hartup, 1989](#)). Children of early-timing mothers are more likely to be aggressive, and in the absence of good limit setting in the family, the aggression becomes less amenable to social intervention. It is most likely that aggression levels are determined by an ongoing interaction of biological tendency with psychosocial context.

Sociometric studies of school-age children yield up to five groups: popular, average, rejected, neglected, and controversial. There are two subgroups of rejected children, those who undervalue themselves and have low self-esteem, even compared with their teachers' evaluations of them; and those who have a positive view of themselves but are seen as defensive and aggressive ([Boivin and Begin, 1989](#)). When children in different sociometric groups were asked to evaluate themselves and one another, it was expected that aggressive children would show attributional biases not shown by nonaggressive children. In fact, however, these children's evaluations of others were not out of line, even though other children clearly identified the reputation of rejected children. In addition, negative reputation increasingly separates the rejected group at older ages ([Rogosch and Newcomb, 1989](#)). Aggression and consequent rejection and social isolation during middle childhood is a primary predictor of maladjustment in later years.

### Schooling

Schooling refers to the ecological setting in which children learn. It refers to the environment, the size, the philosophy, the characteristic transactions between teachers and students, and the culture of the school. How the child *can* function, what he or she can do, how the child perceives himself or herself, especially in terms of competence and the accompanying confidence in the ability to accomplish tasks, and, finally, how the child is a part of communities/societies, are substantially forged and reshaped in the school environment.

We discuss four aspects of schooling:

1. Preparation: the effects of prior experience
2. Attunement to children's learning styles and needs
3. Concordance or discordance with the patient's family/community ethos
4. How the school serves as a model for a community in which a child finds a role

## THE EFFECT OF EXPERIENCE AND PREPARATION ON CHILDREN'S SCHOOL FUNCTIONING

Children arrive at school with diverse experiences. Most dramatically different are the experiences of children from middle-class, educated families and those from poor, minority, inner-city families. The former are more likely to have attended educationally oriented preschools, have traveled at least in their own communities, have visited libraries and museums, and have been read to by their parents and teachers, whereas the latter have more likely been involved with complex family and "adoptive" family relationships ("play" mamas and many "aunts" and "uncles"), have experienced comings and goings of people in their daily worlds, and have been exposed to situations of danger and hardship with little sense of control over these situations. Children from immigrant families may not speak English, or, alternatively, may be the only members of their family who do speak English. Some minority youngsters may never have seen a book or a piece of paper or a crayon by the time they enter school. But many of them may have been assuming responsibilities in the household, such as caring for younger children, getting meals, caring for themselves while adults are away, and translating for their parents. The former group has been prepared to enter school since toddlerhood, whereas the latter group is prepared to manage an entirely different set of experiences, which may not be compatible with what is expected in school ([Heath, 1989](#); [Miller-Jones, 1989](#); [Wilson, 1989](#)) (see [Chapter 106](#)).

Studies of the effects of model preschool programs and general application of Head Start programs for poor and minority children demonstrate that there is some advantage to having had a preschool experience, and this advantage is more dramatic and more lasting if the experience was in a model preschool program ([Haskins, 1989](#)). In follow-up throughout the remainder of their school lives, children from model preschools were significantly less likely to be placed in special education programs than control subjects, but most often the effects disappeared in 3 to 6 years, after the children entered formal public school ([Bradley et al., 1988](#)). This suggests that preparation, alone, does not suffice to ensure a positive school experience. The most effective early education programs involved the parents in the school effort, so that the fit between home culture and school culture was enhanced.

Reading comprehension largely is thought to be text based (i.e., content oriented) and interactional (i.e., develops within a relationship). It is increasingly evident that reading success is heavily influenced by the preparation of the reader, who brings to the task his or her expectations, prior knowledge of the content and structure of the material, and cultural background ([Hall, 1989](#)). Studies support this observation. One study, for example, reported that a group of 4-year-olds given simple instructions in segmenting and blending words of two and three syllables 10 minutes a day for 13 weeks resulted in dramatically higher reading scores than those of children involved in nonspecific reading-related activities ([Coles, 1987](#)). Coles reported that a study of the families of children with reading disabilities found that there is a significant lack of preparation of these children for reading. Other studies found that in some families, messages about expectations of failure were transmitted, in others there were failures to provide exposure to preparatory material, and in still others there was obvious evidence of "communication deviance" whereby the explanatory frameworks of language were so odd or idiosyncratic to the family that the child had unusual difficulty mastering conventional rules needed to learn to read ([Ditton et al., 1987](#)). In addition to insufficient preparation, other etiologies of reading difficulty must be considered. These include learning disability (due to underlying processing deficits, auditory discrimination problems, decoding difficulties), mental retardation, and knowledge deficits in oral language or vocabulary. Basic prereading skills include the ability to bring background knowledge to bear on a new situation, self-questioning behavior, and predictive skills.

## ATTUNEMENT TO CHILDREN'S LEARNING STYLES AND NEEDS

The second aspect of schooling involves the interaction around learning. Many use Vygotsky's ideas about the evolving mind in society to understand better how children learn successfully. In this framework, learning represents the transfer of responsibility for reaching a particular goal ([Belmont, 1989](#)). This transfer takes place in the "zone of proximal development" (ZPD), which is defined as "the distance between the actual developmental level as determined by independent problem solving and the level of potential development as determined through problem solving under adult guidance or in collaboration with more capable peers" (Vygotsky, cited in [Slavin, 1987](#), p. 1162). The ZPD is a useful measure of learning potential because it includes the instructional context (or, in the case of psychometrics, the relationship between child and examiner) as indispensable to the measured achievement. When children are presented with strategies in the process of "strategy instruction" (whether children can adopt a strategy and apply it to other, similar situations without further instruction), those presented in the lower area of the ZPD are readily generalized, those in the midrange are adopted and gradually generalized, and those toward the higher end of the zone may not be adopted at all ([Belmont, 1989](#)). The child's readiness and the instructor's attunement to presenting material at a level that stretches already established abilities but is not so novel as to be overwhelming, are crucial to the "transfer of responsibility" that defines successful learning.

Fellow students, as well as teachers, provide assistance with developing more sophisticated problem solving, as is stated in Vygotsky's definition of the ZPD. There are specific methods of peer involvement in learning that have come to be known as *cooperative learning*. Cooperative learning refers to any number of types of

student groupings for learning but differs from peer tutoring in that the material is presented by the teacher rather than by the peers. Students are given problems to solve or projects to complete, and the incentive to work together is encouraged by either rewarding the group's efforts, rewarding each individual child on the basis of the group's efforts, or rewarding the group on the basis of each child's achievement. Children can and will positively influence one another's progress. This is particularly true when some children in the group are more advanced than others, but it also is true when all children are struggling to solve a new challenge. For example, using the Piagetian description of the accomplishment of conservation, nonconserving children learn from peers who have mastered conservation, but they also progress in conservation skills when struggling with conservation problems with other nonconserving children ( [Slavin, 1987](#)). The process of working together must be specifically supervised and rewarded because otherwise the more competent children take most of the responsibility for accomplishing the objectives or completing the project, while the less competent children do not contribute. Properly conceived, however, the value of cooperative teaching extends beyond the opportunities it creates for learning. It also provides a framework for learning about others, valuing differences, observing and using the strengths of others, helping one another, and making a contribution to a community goal.

Regardless of the rate at which children learn and the specific strengths and weaknesses they may have in mastering certain materials, attunement to each child's ZPD and pitching the new material to the appropriate level will inevitably enhance not only the child's success but his or her enthusiasm for learning.

[Howard Gardner's \(1999\)](#) theory of multiple intelligences is echoed in Mel Levine's neurodevelopmental profiling ( [Levine et al., 1993](#)). In these and other approaches being used in innovative educational strategies, the principle is that different children have specific learning styles. According to these theories, it is incumbent on the teacher to ask, "How does this particular child learn best?" and to design an educational strategy that uses the appropriate mode.

## CONCORDANCE OR DISCORDANCE WITH THE PATIENT'S FAMILY/COMMUNITY ETHOS

Two facets of congruence between the style of school and family as interfacing systems have been studied. The first involves expectations in the areas of educational goals, what is expected of the child, rules, and areas of permissiveness. Because the education system has been established by the majority culture, goals in general are congruent between school and majority families. Parents expect their children to attend school regularly, to be respectful, to be motivated, and to achieve. Problems come up when children cannot or do not fulfill these expectations, which are ordinarily shared by family and school systems. They also come up, however, when these expectations are not shared by family and school systems. Then, the all-too-common complaint that the child misbehaves at school but is fine at home, or, less commonly, the reverse, is brought to the attention of a counselor or mental health professional. This type of problem is most likely to be found in children from ethnic and cultural minority families ( [Phinney and Rotheram, 1986](#)) and can be ameliorated when parents are intimately involved in school life, as James Comer has so elegantly demonstrated (see [Chapter 106](#)). The second facet concerns the congruence of the way school and family systems are organized. In general systems terms, interpersonal systems can be open and relatively closed, referring to characteristics of freedom of exchange with other systems, definition of system boundaries, and amount of variety that is encouraged or tolerated in the system. Some schools that have been founded around specific religions are examples of closed systems, and these often serve a specific population with shared rules and values, constituting a good fit. Rules about conduct, limits, and privacy are consistent across school and family and may differentiate each from the rest of society. Some schools have a more closed system than many of the families of children attending. That is, the schools have clear rules and expectations about everything from dress to punctuality, whereas the families' rules are more loosely organized. The school personnel tend to see these families as irresponsible and incompetent, and themselves as more capable caretakers. Open families sending their children to closed schools feel criticized and defensive, and a child is caught between the two systems because he or she may want to conform to the school's expectations but depends on the family to provide appropriate support. Open school systems allow for variability and have the potential for being flexible. In many instances, however, open school systems interfacing with open family systems may have such a lack of definition that the children have no clear framework within which to define themselves. This kind of "congruence" between open family and open school systems often results in the involvement of the child with more systems, such as welfare, juvenile justice, or mental health ( [Rotheram, 1989](#)).

## HOW THE SCHOOL SERVES AS A MODEL FOR A COMMUNITY IN WHICH A CHILD FINDS A ROLE

A list of characteristics of effective schools includes strong leadership, an atmosphere that is orderly and not oppressive, teachers who participate in decision making, school staff that has high expectations of students, and frequent monitoring of student progress ( [Linney and Seidman, 1989](#)). A specific aspect of school environment that has been studied is school size. It has been shown that large schools often are "overmanned," meaning that there are more students than role opportunities. This means that there are not enough opportunities in student government, arts, or sports programs, or for individual distinction to recognize more than a very few children. In "undermanned" schools, there are opportunities for students to be involved in activities and to take more initiative. The environmental role demands on the students in undermanned schools increase the levels of student participation so that they can contribute to the school community, develop identified roles in this community, and become known to themselves and each other as distinctive individuals. In large schools, there is the danger of anonymity and ultimately a high rate of dropping out and involvement in antisocial behavior and substance abuse. The movement to consolidate schools thus increases the chances that students will not have a positive experience in school, unless the student population is broken down into smaller units within which students experience a manageably sized community ( [Linney and Seidman, 1989](#)).

## SUMMARY OF SALIENT FEATURES OF SCHOOLING

For school to be most effective at supporting the crucial development in school-age children, there has to be attention to the preparation children have had before entering school, and assistance to those whose experience has not primed them to take advantage of the school experience. Second, each child's learning style and readiness needs to be understood sufficiently that educational material is presented that stimulates a child's drive to competence, without being so overwhelming that the child gives up. Most curricula are established to meet the levels of most children in the grade, but there are always children who are either more advanced or slower to whom learning tasks need to be thoughtfully and individually offered. Third, attention needs to be given to the congruence between expectations of school personnel and those of parents. Involving parents in school activities is the most effective way to collaborate and close any gaps that could cause confusion and loyalty conflicts for the children. Finally, schools need to form manageably sized communities in which children can distinguish themselves. Even if the total school size is very large, there are ways to subdivide into smaller communities.

There are cultures where children learn the "industry" of their society other than in school, primarily through various forms of apprenticeship. Schools in our society offer both the opportunity for learning the industry and for learning one's place in society. We would be remiss if we did not point out that children in our Western society are privileged to have time set aside in their development for schooling. Although child labor is almost nonexistent in the United States and Europe, there are currently 250 million child laborers in the world between the ages of 5 and 14 years (90% in Asia and Africa), living in extreme poverty, engaged in repetitive and physically demanding tasks who do not have the opportunity to go to school at all, and have limited opportunity to play and learn ( [Jones, 2000](#)).

## FAILURES OF DEVELOPMENT IN THE SCHOOL-AGE PERIOD

From this discussion of normal developmental processes and influences on developmental outcome, an understanding of the many failures of development is straightforward. maturational deviations or delays (e.g., developmental disabilities, mental retardation, or pervasive developmental disorders) limit a child's developmental progress in all the spheres we have discussed. The child is able to master neither the learning nor social tasks. In addition, the family does not develop the independence and social integration characterized by the school-age family, often because these families are tied into attending to the needs of their special children. Similar limitations of development also often occur in families with children with chronic medical illnesses. The challenge for mental health professionals who use a developmental approach in their work is to find arenas where handicapped children can be competent and interact socially. Lowering expectations of performance, providing ability-appropriate responsibilities for children to meet, and encouraging reciprocal social interaction are interventions that can maximize developmental accomplishments of these special youngsters.

Interference with learning may be based on immaturity, or, as is more commonly diagnosed, may be due to disorders of focus, attention, or impulse management or to specific learning dysfunction. Understanding the crucial developmental issues of establishing a view of oneself as a functioning person in a community can focus the mental health consultant's attention on helping the child, family, and teachers find strategies for managing areas of difficulty. Medication often is used to support such efforts but is most effective when the child and the people closest to him or her are concentrating on competence and strategies for becoming competent.

Inferiority and defeat are the principal emotional pitfalls of school-age children because of the importance of mastery and recognition in the communities in which children participate. Depression is both a cause and an outcome of failure to progress in the manner that the child believes others expect. Assessment and treatment of depression in school-age children always must include assessment of the developmental issues of competence and how a child is viewed by his or her peers so that in addition to psychotherapy and medication, assistance with social functioning and academic mastery will be included. Externalizing behavior disorders drastically interfere with children's developmental progress in this era by disrupting learning and social accomplishment. Many such behaviors may be viewed as a



defense against a sense of failure and inferiority (better to be seen as bad than dumb). Developmental approaches to treatment must include attention to academic progress along with several approaches to helping such children be accountable for their behavior. Behavioral management with consequences may be helpful but will be more so in this age group if there are exercises in putting oneself in others' places and developing empathy, so that these children can function in society.

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## 24 ADOLESCENCE

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We are born, so to speak, twice over. Born in existence and born into life; born a human being and born a man.  
—Rousseau, *Emile*

Adolescence in contemporary Western industrial society is shaped and defined by the interplay of complex biological, cultural, economic, and historical forces. This lengthy transitional state, which may last a decade or more, is a distinctive period in which a youngster is no longer a child nor yet fully adult, but partakes of some of the challenges, privileges, and expectations of both epochs. Adolescence is a period of paradoxes, as youngsters reach physical and sexual maturity well before they are fully cognitively and emotionally mature. On one hand, a secular trend towards earlier and earlier puberty over the past century and half means that by 13 years of age many youngsters are potentially fertile and sexually attuned, if not yet fully active. On the other hand, the educational demands of a complex modern economy have prolonged formal education and raised the age of mandatory school attendance to approximately 16 years, whereas social welfare concerns have abolished child labor and legally restricted adolescent employment, thus postponing entry into the world of work. [Some measure of this shift may be seen in the contrast between 1900, when many Americans still lived on family farms, and only 0% of 14- to 17-year-olds attended high school and the present 95% high school attendance rate ([Arnett, 2000](#)).]

As a result, full economic emancipation usually is not possible until the later teens, at the earliest, and in the case of young people pursuing college or postgraduate education, often not until the middle to late twenties.

In the United States, the legal status of adolescents is a confusing mixture of privileges and strictures that attempts to balance the need for control and protection with the incremental granting of autonomy. For example, a 14-year-old may fly a plane, but not legally drive a car, whereas a 17-year-old may serve in the army, but not vote until 18 years of age, when he or she still is not legally allowed to drink. In many jurisdictions, a 14-year-old may legally obtain an abortion without her parents' knowledge or consent but needs her parents' permission to be absent from school to do so.

Despite the restrictions on their full-time employment, young adolescent consumers are a potent economic force, controlling billions of dollars in disposable income annually. Teenagers, hence, comprise an eagerly sought-after demographic target for marketers, advertisers, and the broadcast, print, and electronic media. In turn, to attract and hold these young viewers and readers, media programming directed to them increasingly emphasizes sex and violence as prominent themes; sexual themes are estimated to make up approximately one-third of the content of prime time shows popular with teens ([Chapin, 2000](#)).

Winnicott once remarked aphoristically, "There is no such thing as a baby," meaning that the baby could not be considered apart from its relationship with its mother. Although adolescence is the epoch *par excellence* of individuation and autonomy striving, it is similarly impossible to have a full understanding of adolescent development apart from its specific biological, family, community, cultural, and historical contexts ([Lerner and Galambos, 1998](#)).

The interactions among these factors are complex and multidirectional. Not only are adolescents influenced by their families, but they reshape their families' dynamics as they grow. Although important aspects of adolescents' development are genetically and biologically determined, the effects of these determinants may be mediated or influenced by psychosocial factors. For example, family factors influence not only the impact of the timing of puberty but may actually affect the timing of puberty itself, with earlier and more rapid maturation in adolescents raised in more conflictual, less supportive homes ([Graber et al., 1995](#), [Graber et al., 1996](#); [Steinberg and Morris, 2001](#)). Behavioral genetics studies that have revealed the importance of nonshared environmental factors suggest that adolescent siblings evoke different interactional and social environments even within the same family ([Reiss, 2000](#)).

It is important to bear in mind the great diversity of social and family contexts in which today's adolescents grow up. In the United States, despite some commonalities, the experiences of adolescent who are immigrants, gay or lesbian, or growing up in poverty, foster care, single-parent, or other nontraditional family structures differ in important ways from the general patterns presented later in this chapter. Even greater differences exist between the majority culture of the West and more traditional societies, with less emphasis on individual autonomy and fewer expectations that adolescence should be a period of vocational choice or attaining full independence from families ([Arnett, 1999](#)). Some anthropologic studies have concluded that in such preindustrial societies, there may be less adolescent turmoil and conflict with parents ([Cote, 2000](#); [Schlegel, 2000](#); [Schlegel and Barry, 1991](#)). One important research question concerns the impact on adolescence in such societies as the process of globalization exposes more and more teenagers to the same media and cultural influences as in the West ([Arnett, 1999](#); [Schlegel, 2000](#)).

### PHYSICAL CHANGES AT ADOLESCENCE

The term *puberty* (from the Latin *pubertas*, meaning "age of manhood") is used to refer to the physiologic and morphologic changes that mark the transition from childhood to adulthood.

#### Hormonally Mediated Changes

The most visibly dramatic aspects of adolescence relate to the hormonally mediated changes of puberty: the development of primary and secondary sexual characteristics; marked growth in stature, muscle mass, and strength; and increased sebaceous gland activity.

Adrenarche, the steady increase in adrenally produced androgens, begins as early as 6 to 8 years of age, leading to increased skeletal growth and the beginning appearance of body hair even before the surge of gonadal hormones associated with puberty proper.

Puberty proper is marked by the pulsatile release of gonadotropin-releasing hormone, producing increased pituitary release of follicle-stimulating hormone and luteinizing hormone that in turn drive the production of gonadal hormones (primarily testosterone in boys, estrogen in girls) ([Petersen, 1987](#)). Together with these gonadal hormones, increased release of growth hormone stimulates the pubertal growth spurt.

The triggers for this activation of the pituitary–gonadal axis are unclear, but have been speculated to include leptin (serving as a metabolic signal of adequate body weight/composition), neurotransmitter-mediated attenuation of inhibitory tone or increased excitatory tone at the level of the hypothalamic gonadostat, and altered

hypothalamus–amygdala interactions (for review, see [Spear, 2000](#)).

The process of puberty takes approximately 4 to 5 years from start to finish, with girls (in the industrialized world) beginning the process on average at 9 to 11 years of age, approximately 2 years earlier than the average onset for boys. The various stages of this process, as indicated by pubic hair, breast development, height spurt, and menarche in girls and pubic hair, penile and testicular growth, and height spurt in boys, have been classified by [Tanner \(1974\)](#) into stages I through V. Although Tanner staging of a child's pubertal development is done most accurately by direct physical examination, alternative methods include the self-report Pubertal Development Questionnaire or asking youngsters to identify their stage of development using a set of standard, gender-specific photographs ([Graber et al., 1996](#)).

The first harbinger of impending puberty usually is an acceleration in linear growth, as much as 10 cm per year, which usually precedes increases in muscle mass and strength, thereby producing the gangling appearance of many early adolescents.

For girls, the initial stages of puberty are the beginnings of breast development [mean age 8.87 years (Standard deviation [SD], 1.93) for African-American girls and 9.96 years (SD, 1.82) for white girls] and the appearance of pubic hair [mean age 8.78 years (SD, 2.00) and 10.51 years (SD 1.67), respectively] ([Herman-Giddens et al., 1997](#)).

The clearest marker of puberty in girls is the onset of menses, or *menarche*. Girls' periods initially remain irregular for some time, and despite the high rates of early teen pregnancy, ovulation and full fertility may require 2 years to develop. Most modern girls have been well prepared for menarche by health classes, peers, and mothers, and news of who has (or has not) yet begun her periods is the topic of excited exchanges of confidences among middle school girls.

A critical body weight and fat/muscle ratio appears to be a necessary condition for menarche; hence, girls who train intensively for athletics or dance or who are anorectic may have delayed menarche. Probably related to the permissive role of adequate nutrition and body weight, there has been a steady secular decrease in the age of menarche since the Industrial Revolution, at the rate of approximately 2.3 months per decade. Currently, the average age of menarche is 12.88 years (SD, 1.20) in white girls and 12.16 years (SD, 1.21) in African-American girls ([Herman-Giddens et al., 1997](#)). (As in preindustrial Europe, the age of menarche remains approximately 17 years in many developing countries). In recent years, there has been controversy about the appropriate norms for deciding at what age female pubertal development should be considered premature because one large study found that by 7 to 8 years of age, 5 % of white girls and 15.4 % of African-American girls were at Tanner stage II or greater for breast development and 2.8% and 17.7%, respectively, were at Tanner II stage or greater for pubic hair. Further study is needed as to whether this represents an increased prevalence of very early puberty in girls, and if so, what its implications are regarding potential causes and indications for suppressive treatment ([Kaplowitz and Oberfield, 1999](#); [Endocrine Society and Lawson Wilkins Pediatric Endocrine Society, 2001](#)).

In boys, growth of the penis and testes and beginning spermatogenesis occur in early and middle adolescence. In contrast to menarche, however, “semenarche” or the beginning of ejaculation, whether by masturbation or spontaneous nocturnal emissions, usually remains a very private matter among Western boys ([Stein and Reiser, 1994](#)).

Detailed longitudinal studies reveal considerable variation in the onset and progress of the various stages of puberty, both within and between genders. Thus, peak growth velocity in girls occurs approximately 2 years earlier than in boys, whereas pubic hair appearance often is only approximately 9 months earlier.

Much research has examined the question of the developmental impact on adjustment of early versus late maturation in boys and girls ([Brooks-Gunn and Paikoff, 1997](#); [Graber et al., 1997](#)). In general, these studies show that, for boys, early maturation is advantageous in terms of popularity, self-esteem, and intellectual abilities, but does confer some increased risk for delinquent or problem behaviors, perhaps because of friendships with older peers ([Steinberg and Morris, 2001](#)). For girls, the picture is more complex, with early-maturing girls tending to have more adjustment difficulties (including lower self-image and greater vulnerability to depression, anxiety, and eating disorders), to engage in more risky behaviors, and to experience early sexual intercourse. The impact of early versus late maturation in girls, however, also depends on social context variables such as social class, pubertal status of peers, cultural norms, and timing of concomitant changes (e.g., school transition), as well as prepubertal adjustment ([Ge et al., 1996](#); [Steinberg and Morris, 2001](#)).

For many decades, an emphasis on the psychological effects of pubertal hormonal changes dominated discussions of the psychobiology of adolescence—what might be termed the “raging hormones” theory of adolescent psychology. However, only limited and equivocal associations have been found between various forms of adolescent psychopathology and gonadal hormonal levels ([Brooks-Gunn et al., 1994](#); [Buchanan et al., 1992](#)), with hormonal levels accounting for only a very small proportion of the variance in negative affects, compared with the influence of social factors.

### Neurobiological Changes in Adolescence

Recent research has underlined the magnitude of neurobiological changes in the adolescent brain, especially in the forebrain and mesocortical and limbic regions. To what extent these are influenced by or dependent on prenatal or pubertal hormonal factors is not clear. Studies of schizophrenia and mood disorders have shed light on the pathogenic potential of these adolescent brain developments (see extensive review by [Spear, 2000](#)). For example, [Weinberger's \(1995\)](#) developmental theory of schizophrenia draws on the observation that although the infectious, neuromigratory, and nutritional insults predisposing to the disorder occur prenatally (usually in the second trimester), overt schizophrenic symptoms typically do not appear until late adolescence. Drawing on various animal models, Weinberger and colleagues suggest that the behavioral effects of early lesions remain largely silent until unmasked by abnormalities in the usual late-adolescent maturational changes in the prefrontal cortex, hippocampus, or other limbic regions; these maturational changes are hypothesized to lead to the overt symptoms of schizophrenia, perhaps because of increased sensitivity to normative adolescent stressors.

One of the most dramatic changes in adolescent brain reorganization is a massive elimination or “pruning” of cortical synapses, with an estimated loss of up to 30,000 synapses per second during adolescence ([Bourgeois and Rakic, 1993](#); [Rakic et al., 1994](#)). The resulting loss of approximately half of the cortical synaptic connections present before puberty is believed to affect preferentially excitatory synapses and is accompanied by declines in brain glucose metabolism, oxygen utilization, and blood flow; decreased overall electroencephalographic amplitude; and more complex and focal patterns of brain activation. The neuropsychological and neurochemical consequences of this synaptic remodeling are especially prominent in the prefrontal cortex, with loss of excitatory glutamatergic inputs, but also marked changes in dopaminergic input.

Longitudinal scanning magnetic resonance imaging studies find a rostrocaudal wave of growth in the corpus callosum during childhood, with peak growth rates in the fibers connecting the temporoparietal cortical association and language areas peaking in early adolescence and then declining (perhaps paralleling the ending of the critical period for second-language acquisition). Cortical volume changes varied by region, with enlargement in the temporoparietal regions, but up to 50% loss in the subcortical gray matter of the head of the caudate ([Thompson et al., 2000](#)).

The full extent and significance of these changes in brain architecture and functioning are not yet clear ([Giedd et al., 1999](#); [Sowell et al., 1999](#); [Spear, 2000](#)). It seems likely, however, that they are reflected in the adolescent's burgeoning intellectual capacities, as well as a shift in various motivational, attentional, and emotional realms. For example, various neuropsychological tasks of executive functioning and inhibition that are believed to involve prefrontal cortical functioning continue to improve through adolescence ([Welsh et al., 1991](#)).

### Other Biological Changes

Along with puberty come changes in appetite and sleep patterns.

Across species, the adolescence-associated growth spurt results in more time spent feeding and foraging for food. Most families with teenagers can attest to their youngsters' elevated metabolic rate and what has been termed *developmental hyperphagia* ([Post and Kemper, 1993](#); [Spear, 2000](#)).

Adolescence also sees a shift in sleep patterns, with a sleep phase delay or tendency to fall asleep later and wake up later ([National Research Council and Institute of Medicine, 2000](#)). On average, 10- to 12-year-old children sleep approximately 9.3 hours a night and awaken spontaneously. In contrast, the mean length of sleep for high school students is 7.5 hours per night, with one-fourth of students sleeping 6.5 hours or less per night. Adolescents also have greater difficulty getting up and suffer from frequent daytime drowsiness, accompanied by impaired alertness and cognitive functioning.

Part of this phase shift appears to be biological; later night-onset and later morning-termination of melatonin secretion make it difficult for the adolescent to go to sleep earlier or to wake up alert in time for school, which, deleteriously for many teenagers, may begin as early as 7:20 AM. This shift in sleep patterns also has a



psychosocial component. Adolescents are given greater autonomy by their parents in controlling their own bedtimes, whereas the expansion of social contacts outside the home and increased social stimulation (in the form of phone and e-mail) keep the teenager up later.

## COGNITIVE CHANGES IN ADOLESCENCE

Adolescence is marked by dramatic quantitative and qualitative growth in cognitive abilities ( [Keating, 1990](#)). Although not a universal achievement, adolescence marks the attainment for many youngsters of what Piaget termed the stage of formal operations, with the ability to construct “contrary-to-fact” propositions and a growth in hypothetico-deductive problem-solving ability and understanding of propositional logic and probability ( [Coleman and Hendry, 1990](#)). Along with a greater capacity for abstraction, adolescence often sees the flowering of passionate intellectual and aesthetic interests, with impressive achievements in areas such as music, mathematics, computer science, or physics. Interestingly, adolescent works of genius are more commonly in these abstract areas than in those involving the empirical sciences or the humanities.

Although the validity of Piaget's views have been debated, there is a general consensus that adolescents' cognitive abilities are characterized by growing complexity, the ability to think about possibilities, and increased speed and efficiency of information processing.

These cognitive changes also have their counterpart in the adolescent's social cognition and moral development. The development of formal operational thinking permits a growth in social perspective-taking and a decline in childhood egocentrism; it enables the adolescent to contemplate better what a social situation might look like from another person's point of view ( [Selman, 1980](#)). As described by [Kohlberg \(1969\)](#), moral reasoning becomes more complex and expands to include orientation to interpersonal relationships, maintenance of social order, notions of social contract and general rights, and, finally, reference to universal ethical principles. [ [Gilligan \(1982\)](#) has criticized Kohlberg's purportedly universal hierarchy of stages of moral reasoning as male-oriented in its emphasis on rules and universal principals and “moral logic of justice,” in contrast to what she sees as the more interpersonal and nurturant bases of women's values in the “moral logic of care.”]

Despite greater cognitive abilities, however, adolescents do not always use these capacities for sound decision making, in part, perhaps, because their cognitive performance in real-life situations (as opposed to optimal test conditions) is more vulnerable to disruption by everyday stresses ( [Beyth-Marom and Fischhoff, 1997](#); [Keating, 1990](#)).

On a practical level, the adolescent develops a more mature time sense, a greater awareness of the finality of death, and, along with wider knowledge of the outside world, a keener sense of the diversity and relativity of moral codes. This moral awakening may be accompanied by an intensified interest in and sophistication about politics, ideology, or religion. This wider vision, as most eloquently described in the work of [Erik Erikson \(1963\)](#), provides both opportunities and hazards. Along with a penchant for philosophical musings, the adolescent may experience a sense of moral confusion and at least transient feelings of anomie. The anxieties of what [Seltzer \(1989\)](#) has termed *frameworklessness* may lead some adolescents to a fanatical embrace of some ideology or religion on one hand or a posture of nihilism on the other.

## PSYCHOLOGICAL TASKS OF ADOLESCENCE

The physical, neurobiological, and cognitive changes described previously herald dramatic shifts in the adolescent's relationship to his or her own body, appetites, parents, peers, and self-image. In this next section, we turn to the psychological tasks of adaptation these shifts impose on the developing teenager ( [Table 24.1.](#))

Task	One Third One	Two Thirds	One Third One
1. Independence	Defiance breaks for parents and peers leads to role	Independence about aspects	Integration of independence issues
2. Body image	Adjustments to adolescent changes	“Try on” different images to find self	Integration of existing body image with personal self-perception
3. Sexual drive	Sexual curiosity, attraction, masturbation, sexual arousal in sex	Sexual experimentation, possible	Integration of sexual drive and identity
4. Relationships	Increased peer group social status	High interpersonal peer group relationships	Individual relationships now important for peer group
5. Career plans	Vague and often unrealistic ideas	=	Specific goals and specific steps to attain them
6. Conceptualization	Concrete thinking	=	Ability to abstract
7. Moral system	Deep in values; taking of moral system of parents	Internalized =	Adolescent's own concept of right and wrong (Theoretical decision)

From [H. H. Adelman, \*Seven Crucial Years: How to Deal with Your Adolescent's Developmental Tasks\*, 1983, p. 13-14](#)

**Table 24.1. Growth Tasks by Developmental Phase**

The normative psychological tasks of adolescence are:

1. Developing a satisfactory and realistic body image
2. Developing increased independence from parents and adequate capacities for self-care and regulation
3. Developing satisfying relationships outside the family
4. Developing appropriate control and expression of increased sexual and aggressive drives
5. Identity consolidation, including a personal moral code and at least provisional plans for a vocation and economic self-sufficiency

### Coping with a Changing Body Image

Save for pregnancy or devastating illness, no other epoch sees such dramatic changes in the body and its self-representation as does adolescence. Although often welcome, these changes also are unsettling. Body and facial hair begins to grow. Menstrual discharges, erections, or ejaculation can occur at unexpected and embarrassing times. Acne and body odors make their appearance and are a source of anxiety. Boys' voices may break unexpectedly as they deepen. Changes in the distribution of fat and muscle alter body outlines. Not only must girls deal with breast development, but, to their embarrassment, many boys develop gynecomastia.

Adolescents compare their development carefully with that of their peers and are acutely aware of their self-perceived imperfections. Much time is spent brooding in front of the mirror, examining every potential blemish and trying to catch a glimpse of the self. A single pimple may seem to loom as large as the Matterhorn, its stigma increased by the sense that it is as glaringly obvious to everyone else as it is to the adolescent.

In Western society, girls in particular are very preoccupied with the body image ideal of thinness held up to them by the media. Girls' levels of satisfaction with their bodies and physical appearance decline as they pass through adolescence. This is especially problematic for girls who are earlier maturing. The National Health Examination Survey ( [Gross and Duke, 1983](#)) found that most adolescent boys with body weights less than the top 10th percentile were satisfied with their weight; in contrast, even among girls whose weight was in the 50th percentile, 25% of lower socioeconomic group girls and over 40% of high socioeconomic group girls wanted to be thinner. The Youth Risk Behavior Survey ( [Centers for Disease Control and Prevention, 2000](#)) found that although 29% of high school boys and 22% of girls were overweight or at risk of being overweight (defined as having a body mass index equal to or greater than the 85th percentile for age and sex), only 24% of boys thought they were overweight, compared with 36% of girls; furthermore, only 26% of boys were trying to lose weight during the preceding 30 days, compared with 59% of girls. Compared with black girls, a higher proportion of white and Hispanic girls considered themselves overweight or were trying to lose weight. Pathologic eating behaviors are common in adolescent girls. For example, a survey of two private girls' secondary schools found that 18% of the girls reported at least one major symptom of an eating disorder: 8% to 15% thought about food all the time, 6% to 12% induced vomiting to control their weight, over 2% used laxatives for weight control, and 7% often fasted or starved to lose weight ( [Hendren et al., 1986](#)). These endemically high levels of body dissatisfaction and pathologic eating attitudes provide a large reservoir of vulnerable adolescent girls from whose ranks those with frank bulimia and anorexia are recruited.

More research is needed, however, to understand better the cultural factors that influence the wide variations in the prevalence of disordered eating attitudes and

behaviors across different communities and ethnic groups and over time ([Ponton, 1996](#)).

The adolescent's body also is a representation of the adolescent's self. Hence, not surprisingly, teenagers spend great amounts of time, energy, and money trying to make their appearance conform to some perceived ideal. Boys in middle adolescence may try to bulk up their gangling habitus or firm up a pudgy physique by weight lifting, body-building, nutritional supplements, and even anabolic steroids in an effort to transform their self-image from weak, dependent, or vulnerable to that of a "hard body"—tough, masculine, and strong. Girls endlessly experiment with makeup and consult friends and teen magazines regarding "makeovers." Both sexes may go through a dizzying panoply of clothing and hairstyles, trying on in rapid succession a kaleidoscope of fashions and styles that also represent possible social selves: slut, punk, home-boy, preppie, grunge, jock, goth, and so forth. Multiple body piercings and tattoos convey more permanent, but potentially disfiguring personal statements. [By tattooing on a lover's name, the bearer seeks self-reassuringly to reinforce the permanence of a relationship ([Martin, 1997](#))].

Beyond its role as a source of anxiety or pleasure, the body also may be the vehicle for painful or self-destructive means of reducing psychic tension (e.g., through delicate cutting) or dealing with conflicts over dependency or instinctual longings (e.g., bulimia, anorexia nervosa) ([Ritvo, 1984](#)).

### Changing Relations with Parents

Surveys such as those of [Offer and Schonert-Reichl \(1992\)](#) have emphasized that most adolescents regard their relations with their parents as stable, trusting, and sustaining and continue to turn toward parents as important, primary sources of advice, comfort, and assistance. Although this appears to be objectively true for most adolescents, on a subjective level, for both the parents and youngster, there are important shifts in the emotional terms of the relationship ([Steinberg and Morris, 2001](#)).

#### LOOSENING TIES TO PARENTS

Time spent with family decreases during adolescence, from 25% of waking hours for high school freshmen to only 15% for seniors ([Csikszentmihalyi and Larson, 1984](#)). Furthermore, there is a shift in the affective tone of time spent with parents and in the adolescent's view of the parents.

The teenager's own parents often are de-idealized. This is a painful process for parents as their previously admiring child develops a keen (sometimes distorted, sometimes all-too-accurate) sense of their shortcomings. Even those many teens who retain warm and supportive relations with their parents experience an increased sense of loneliness because the youngster feels no longer able or willing to share many intimate concerns or longings as of old. The adolescent may alternate between wishes for autonomy and wishes to be taken care of. Feelings of dependency may have to be warded off with disparagement, indifference, or oppositionality ([Freud, 1958/1969](#)). As a result, well-meaning parents often are bewildered as to which side of the child's ambivalence they are dealing with at any given moment [hence, the titles to various guides for the parents of teens, such as *Get Out of My Life, but First Could You Drive Me and Cheryl to the Mall?* ([Wolf, 1991](#))].

#### CONFLICT WITH PARENTS

[Csikszentmihalyi and Larson \(1984\)](#) remark that "friction appears to be an endemic feature of family life" in families with adolescents (p. 138), noting that "adults and adolescents live in separate, if overlapping, realities" (p. 140), often viewing the same events quite differently. This is true not only of specific events, but of family life in general, with adolescents tending to underestimate parental influence on them, and parents tending to overestimate their influence. Similarly, parents usually perceive the family's cohesion and adaptability as more satisfactory than do their teenagers ([Coleman and Hendry, 1990](#)) who, in turn, report more conflicts with their parents than do the parents themselves ([Laursen et al., 1998](#)).

Conflicts between parents and children increase with the beginning of adolescence. In early adolescence, these clashes concern household rules, chores, room cleaning, bedtime, diet, friends, dress, and hygiene; later in adolescence, issues such as dating and curfews become more prominent. [Montemayor and Hanson's \(1985\)](#) naturalistic study of early adolescents found that conflicts with parents and siblings occurred at the rate of approximately 20 per month, or 1 every 3 days. Metaanalytic studies find that although the frequency of parent-child conflicts declines from early adolescence through late adolescence, the negative affective intensity of conflicts peaks in mid-adolescence ([Laursen et al., 1998](#)).

Mothers appear to bear the brunt of most of these clashes, especially with early adolescent daughters ([Graber & Brooks-Gunn, 1999](#)); father-son clashes in particular take on greater affective intensity in mid-adolescence ([Laursen et al., 1998](#)). Early maturation in girls and problems such as adolescent depression or substance abuse increase the likelihood of conflict ([Arnett, 1999](#)).

This squabbling and bickering take a toll on the psychological well-being of the parents, as well as the adolescent, with many parents reporting difficulty adjusting to their teenager's strivings for autonomy ([Steinberg and Morris, 2001](#)).

The quality of daily family life, thus, becomes more turbulent in families with adolescents, with minor, but frequent "daily hassles"; for example, as boys approach puberty, there is a deterioration in family communication as both parents and child interrupt each other more frequently and explain themselves less (Steinberg, 1981, cited in [Csikszentmihalyi and Larson, 1984](#)). Time sampling studies find that when with their families, adolescents' negative thoughts outnumber positive ones by 10 to 1 ([Csikszentmihalyi and Larson, 1984](#)).

The apparent decrease in parent-child conflict in middle adolescence coincides with decreased time spent with parents and a turn toward greater involvement and reliance on peers, leading [Laursen et al. \(1998\)](#) to speculate that "it is likely that parents and children disagree less simply because they are together less. Increases in conflict affective intensity coincide with increases in autonomy and emotional dysphoria that occur as adolescents spend more time alone and with peers" (p. 828).

Despite the mutual stresses of increased conflict, most parent-child relationships remain solid. As [Arnett \(1999\)](#) notes, "Even amidst relatively high conflict, parents and adolescents tend to report that overall their relationships are good, that they share a wide range of core values, and that they retain a considerable amount of mutual affection and attachment" (p. 320).

Seen from one perspective, the immediate *causa belli* of many typical parent-adolescent clashes appear seemingly trivial: hairstyle, clothes, chores, curfew, or volume and taste in music. The intensity of conflict, however, usually reflects the parents' or child's perception (accurate or not) that vital issues are at stake: for the parents, issues of loyalty, respect, responsibility, and the dangers of sex, substance abuse, or other risky behavior; for the adolescents, issues of autonomy, control of their own body, and connections to friends.

Adolescents whose parents are able to hold firm and maintain balance in the face of these upheavals, without being overly permissive, harshly authoritarian, or indifferent, appear to do best. Numerous studies conclude that what has been termed *authoritative parenting*—combining warmth and responsiveness on one hand with firmness and demandingness—is associated with a wide range of adolescent competencies, academic achievement, and positive outcomes.

By later adolescence, in most cases, volatility and strife decrease and some degree of equilibrium is restored to the parent-adolescent relationship, albeit on a newer and more egalitarian basis, with the youngster having more autonomy and involvement in family decision making.

#### TRANSITION TO SELF-CARE

As adolescence progresses, youngsters gradually claim or are ceded greater control over their diet, hygiene, sleep schedule, and dress, as well as responsibility for their school work. Nonetheless, these remain topics of frequent minor skirmishes, at least in early adolescence, with much parental nagging about junk food, skipped meals, slovenly or inappropriate dress, time spent on the phone (or Internet), and the like.

Although adolescence is a time of general good physical health, it also is the period during which many attitudes and habits are established with respect to diet, exercise, substance use, smoking, driving, and sexual behaviors that will constitute long-term risk (or protective) factors for health in later life ([DiClemente et al., 1996](#); [Millstein et al., 1993](#)).

Ironically, for many youngsters with serious chronic illnesses, such as type 1 diabetes mellitus or cystic fibrosis, despite their greater cognitive understanding of the exigencies of their condition, the quality of their care frequently deteriorates as they take over the responsibility for adherence to their treatment regimen



([Brooks-Gunn, 1993](#); [King and Lewis, 1994](#)) Thus, many adolescent diabetic patients fail to adhere to their diet, blood glucose testing, and insulin regimen. Adolescent diabetic girls may even deliberately permit themselves to spill sugar in their urine as an ill-advised form of weight control. Some chronically ill youngsters may stop their medication, including chemotherapy or immunosuppressant therapy, with potentially fatal consequences.

Unlike health-conscious adults, many adolescents perceive the need to pay extra attention to their physical condition as anxiety provoking, stigmatizing, or frighteningly threatening to their wish for autonomy and invulnerability; hence, rather than responding to their medical condition with heightened attentiveness, they may try to avoid thinking about or dealing with their illness altogether.

### Developing Satisfying Relationships Outside the Family

As dependence on parents becomes less acceptable to adolescents, they turn increasingly to peers for companionship, advice, support, and intimacy. [Csikszentmihalyi et al. \(1977\)](#) found that during the school year, adolescents spend one-third of their waking time talking with peers, but less than 8% of waking time talking with adults. Talking with friends was the activity that teens reported made them the happiest.

Parents may be bemused or annoyed by their adolescent's intense need to hang out with and be with peers, regardless of any family plans. As [Seltzer \(1989\)](#) points out, this is not merely for the immediate pleasures of the event. Rather, the need for continuing access to peers is driven by an intense need to relate, to compare, and to try out aspects of the developing self. It is this developmental need that explains "why adolescents never seem to tire of being with one another. ... [I]t is not the overt social activity or the content of the event (e.g., a rock concert, a football game, a dance) that feeds the drive. It is *being with one another*—looking, listening, and resultant comparing. Adolescents report details of who was there, what they did or said, and what they wore in far greater detail than they describe the content of the event" (p. 41).

With adolescence, the communicative, supportive, and intimate aspects of friendship take on increased importance. Although nonromantic opposite-sex friendships occur in later adolescence, close friendships in early adolescence tend to be with the same sex. Among girls, intimate conversations are most often the cement of friendships, whereas for boys, it tends to be shared activities. With age, the need for control and conformity decreases, and there is greater tolerance for differences between friends ([Steinberg and Morris, 2001](#)).

The choice of friends is a complex matter. Although the range of possibilities is defined by the given community and school population, the adolescent's specific choices of friends reflect an important and often fateful aspect of self-definition. Adolescents most often choose friends who share their behaviors, attitudes, interests, and identities ([Steinberg and Morris, 2001](#)). Friends, however, also may be chosen on many other grounds, including perceived virtues or aspects of the self that the adolescent consciously repudiates or feels he or she lacks. Friends may serve as sources of support or admiration, as collusive companions for regression or delinquency, objects of sexual or aggressive exploitation, targets for projection—the list is endless. The choice of friend may be used to try on or borrow self-attributes; for example, a girl who perceives herself as unattractive or unpopular may hang out with a girl whom she sees as beautiful or popular. A boy who feels himself to be overly compliant, timid, or passive may choose to hang out for a while with a more venturesome or delinquent peer. Friendships pursued out of such interests may be transient or unstable as the youngster comes to feel in greater possession of the desired attribute himself or herself, or repudiates the wish for it.

Empirical studies have examined what personal attributes of peers are most salient to adolescents. For example, Midwestern high school students were asked to rate a list of attributes as to the frequency with which they were noticed in age mates. The top 10 attributes most frequently rated as "always of interest" were (in decreasing order): cleanliness, loyalty to friends, clothes, dependability, trustworthiness, general physical appearance, maturity, popularity with opposite sex, figure/build/physique, and honesty. Despite some gender and age differences, appearance and dependability were widely viewed as very important, whereas specific skills and abilities were of little interest ([Seltzer, 1989](#)).

Over the course of high school, adolescents shift in the gender and group size with whom they associate ([Brown, 1990](#)). Thus, freshmen hang out predominantly in same-sex groups, sophomores in same-sex dyads, juniors in mixed-sex groups, and seniors predominantly in small groups of heterosexual couples ([Csikszentmihalyi and Larson, 1984](#)). In early adolescence, the adolescent's crowd consists of a large group of peers with similar reputations and role stereotypes (e.g., preppies, goths, brains, jocks, nerds). Younger adolescents value crowd affiliation as fostering friendships, providing support, facilitating interactions, and providing a source of identity and status (according to where the adolescent's particular crowd fits in the school or community hierarchy). Attitudes toward being "part of a crowd" do, however, change over the course of adolescence ([Brown, 1990](#)). Older adolescents are more likely to be dissatisfied with the perceived conformity demands of crowds and prefer smaller, more intimate groups. By the later high school years, the salient peer group may be the clique, a smaller group of peers similar in terms of activities, attitudes, status, age, and race.

Ironically, many adolescents, while resisting parental advice in the name of autonomy, are slavishly compliant with the perceived tastes and values of peers, especially regarding fashions and preferences in dress, slang, music, television, and movies.

As [Steinberg and Morris \(2001\)](#) point out, however, peers influence each other in positive ways, including prosocial behaviors and academic achievement, as well as in negative ways, such as delinquency or substance use. These influences are not necessarily coercive or conformist, but also stem from emulatory admiration and a community of attitudes and interests that form the basis for the friendship in the first place.

Adolescents' relationships with their parents are an important influence on peer relationships. Authoritative parenting styles appear to lessen the negative effects of peer influences; conversely, teenagers from less cohesive families are more likely to be influenced by peers than by parents ([Steinberg and Morris, 2001](#)). A tendency to look to peers rather than to parents for guidance and values, especially when combined with a choice of peers with delinquent behavior, low academic aspirations, or with values markedly divergent from the adolescent's parents, is an important risk factor for a wide range of problem behaviors ([Jessor, 1991](#)).

### Sexual and Aggressive Drives

Adolescence sees the epochal development of experiencing sexual attraction toward others and perceiving oneself as the object of others' sexual desire. How these subjectivities unfold and are given individual and social meanings is a complex process with physiologic, cultural, and individual dimensions. The interplay of these factors has been the focus of much anthropologic, psychoanalytic, and developmental study (see [Chapter 20](#)) ([Cote, 2000](#); [Brooks-Gunn and Paikoff, 1997](#); [Herdt and McClintock, 2000](#); [Katchadourian, 1990](#)).

After a period of fairly open genital interests and play during the preschool years, overt sexual behavior and interests diminish markedly during the school years ([Friedrich et al., 1991](#)). Although even in these years before adolescence, sexual interests are never completely latent, masturbation, if it continues, is more furtive, and the child becomes more modest and inhibited about discussing sexual and romantic matters.

Beginning at approximately 10 years of age, feelings of sexual awareness and attraction make their conscious appearance. This development, which appears to be linked to rising adrenal androgens, occurs even before the onset of gonadal puberty proper. The reticence of children at this age makes the phenomena difficult to study in Western culture, but in many preliterate cultures, such as Melanesia, sexual rites of passage, at least for boys, occur as early as 10 years of age ([Herdt and McClintock, 2000](#)). It is also around the age of 10 to 11 years that some children become aware of same-sex attractions and homoerotic fantasy.

In early adolescence, genital excitement and sexual interests often occur independently of liking, intimacy, or wish for emotional closeness. For young adolescent boys, the objects of sexual fantasy and masturbatory excitement frequently are magazine models or television and movie figures, rather than actual acquaintances. In keeping with their burgeoning attunement to social relationships, early adolescent girls are usually intensely interested in the romantic relationships, real and fantasied, of their peers, with endless discussions of who is "going out" or has broken up with whom. However, the early teen boys who are their age-peers often are unpromising candidates as romantic partners, hence many girls' crushes and eroticized longings focus on media figures, such as the various "boy bands" marketed to this audience. Although movies and television have long provided the raw materials for adolescent sexual fantasy, it is not yet clear what the recent increases in the ubiquity, sexual explicitness, and violence of mass media (including now the Internet) will be on the sexual socialization of teens ([Chapin, 2000](#)).

With adolescence, there also is a resurgence of overt sexual activity (see [Chapter 20](#)) ([Friedrich et al., 1991](#); [Rutter, 1971](#)). The rate of explicit masturbation increases from approximately 10% at age 7 to approximately 80% at age 13 years, whereas that of heterosexual play rises to approximately 65% at age 13 years; homosexual play also is not uncommon in early adolescence, with 25% to 30% of 13-year-old boys reporting at least one episode of same-sex play.

The transition from childhood masturbation to that of adolescence involves more than an increased physiologic capacity for arousal and orgasm. Most teens report



consciously fantasizing when masturbating to orgasm, and even those who do not seem aware of some sort of sexual imagery ([Katchadourian, 1990](#)). Sexual fantasies (which also occur without overt masturbation) become an intense and important part of the adolescent's psychological inner life. Beyond serving as a source of pleasure and compensatory wish fulfillment in lieu of other sexual outlets, these fantasies provide the occasion for the adolescent to elaborate or explicate his or her idiosyncratic and personal erotic scripts: who, doing what, with whom, with what body parts, with what implicit and explicit emotional tone and interpersonal interactions, and with what admixture of dominance or submission, control or abandonment, sadism or masochism, and admiration or degradation. [[Laufer \(1976\)](#) has coined the term *central masturbatory fantasy* to describe the organizing aspects of these not always fully conscious fantasies in relationship to arousal and orgasm.] These fantasies are more than just a form of rehearsal or anticipatory coping; they help the adolescent explore and become aware of what is pleasurable, anxiety provoking, transgressive, or deeply compelling in his or her longings and to become familiar with his or her individual preconditions for erotic excitement and fulfillment.

A key task of adolescence is to bring these erotic longings adaptively into the interpersonal arena as a vehicle for intimacy, emotional closeness, and ultimately the formation of a stable partnership for the conception and rearing of the next generation.

Although early adolescence sees the transition from largely autoerotic sexual activity to sexual interactions with peers, this takes place at different rates in different social and ethnic groups and with different interpersonal meanings (see [Chapter 20](#)) ([Brooks-Gunn & Paikoff, 1997](#); [Katchadourian, 1990](#)). For girls, the relational aspect of sexual involvement usually is paramount, and girls may engage in petting, oral sex, or intercourse as an attempted means of winning or retaining a boy's perceived interest, affection, or commitment. For young adolescent boys, sexual activity often has a more exploitative nature, with less interest in the relational aspect of the activity [for a detailed account of the epidemiology and other aspects of adolescent sexuality, as well the implications for prevention of pregnancy and acquired immunodeficiency syndrome (AIDS), see [Chapter 20](#)].

Falling in love is an important part of adolescence, even when not accompanied by sexual intimacy. Adolescence's most intense longings, keenest pleasures, painful frustrations, and bitterest disappointments center on the quest for a reciprocated love that helps to define the still inchoate self and assuage the loneliness of individuation.

Adolescent sexuality stirs in adults a variable reactive mixture of envy, apprehension, or repressiveness. Parents who may be facing their own midlife crises must contend with their sons' or daughters' burgeoning sexuality. Many of the parent-child conflicts in mid-adolescence, such as those around clothes, friends, dating, curfews, and driving, although seemingly trivial, have the subtext of the parents' attempts to control the pace, scope, and direction of their adolescent's sexual activity. (A few parents who are overidentified with their adolescent's sexuality or take too much vicarious excitement in it collusively encourage their teen's transition to sexual activity.)

Despite much forgetfulness about the travails of their adolescence, most adults retain evocative, bittersweet memories of their own adolescent romantic longings. It is not surprising then, that from *Romeo and Juliet* on, the pangs and passions of adolescent love, whether thwarted or fulfilled, have remained an enduring theme of plays, novels, poetry, movies, and songs.

## Identity

In his seminal work *Childhood and Society*, [Erik Erikson \(1963\)](#) describes the challenge of identity formation as follows:

[I]n puberty and adolescence all samenesses and continuities relied on earlier are more or less questioned again. ... The growing and developing youths, faced with [the] physiological revolution within them, and with tangible adult tasks ahead of them are now primarily concerned with what they appear to be in the eyes of others as compared with what they feel they are, and with the question of how to connect the roles and skills cultivated earlier with the occupational prototypes of the day. ... To integrate all identifications with the vicissitudes of the libido, with the aptitudes developed out of endowment, and with the opportunities offered in social roles. [The desired outcome] is the accrued confidence that the inner sameness and continuity prepared in the past are matched by the sameness and continuity of one's meaning for others (p. 261).

One of the most influential empirical extensions of Erikson's work was Marcia's taxonomy for classifying adolescents into four identity statuses: the *identity-confused* subject who has not yet experienced an identity crisis or made a role commitment; the *foreclosed* subject, who has made unexamined commitments, usually as received from parents and others; the *moratorium* subject, who is actively struggling to define values and commitments; and the *identity-achieved* subject, who has resolved these crises ([Muus, 1988](#)). In contemporary society, many of these identity issues are not fully engaged until the college years or beyond ([Arnett, 2000](#)).

Empirical research has shifted away from global notions of identity to focus more on the development of specific self-concepts ([Offer et al., 1988](#); [Steinberg and Morris, 2001](#)). With their growing cognitive sophistication, adolescents' views of themselves become more differentiated and better organized. [Harter \(1999\)](#) and others have examined the distinct dimensions of adolescents' self-concept across several realms, such as social relations, appearance, academics, athletics, and morality, and their relationship to global self-worth. Adolescents weigh these dimensions differently depending on whether they are interacting with peers, parents, or teachers, and it is only over time that these discrepancies decline to produce a more consonant, better-integrated self-image. [Behavioral genetic studies of siblings suggest that heritable factors may exert more of an influence than do shared environmental factors on perceived scholastic and athletic competence, physical appearance, and general self-worth; perceived social competence appears to be primarily determined by nonshared environmental factors reflecting each sibling's unique family and social experiences ([McGuire et al., 1999](#); [Reiss, 2000](#))].

In addition to the ubiquitous categories of adolescent self-concept described by Harter, ethnic and sexual minority adolescents also must consolidate a sense of identity vis-à-vis both their minority group and the mainstream culture ([Steinberg and Morris, 2001](#)).

## CLINICAL ASPECTS OF ADOLESCENCE

On the basis of many epidemiologic studies, it now appears that only approximately 20% of adolescents have diagnosable clinical disorders. However, it is equally clear that during adolescence, a substantial proportion of youngsters experience increased conflicts with parents and mood difficulties and engage in risk behaviors.

### The Storm and Stress Debate Revisited

For many years, debate has continued over the frequency and severity with which adolescent turmoil occurs and the extent to which it should be considered normative in American culture or universal across epochs and cultures (for a review of this issue, see [Arnett, 1999](#)).

Classical writers, such as Aristotle and Plato, saw the period we now call adolescence as a time of changeability and vulnerability that required careful character education and social constraints. Aristotle noted in his *Rhetorica*:

Young men have strong passions, and tend to gratify them indiscriminately. Of the bodily desires, it is the sexual by which they are most swayed and in which they show absence of self-control... They are changeable and fickle in their desires, which are violent while they last, but quickly over: their impulses are keen but not deep rooted... They cannot bear being slighted, and are indignant if they imagine themselves being unfairly treated" (quoted in [Muus, 1988](#), p. 8).

Although, over the centuries, adolescents have at times been idealized for their beauty and grace or as bearing the hope of the future, they also have been regarded by their elders with deep misgivings and dismay as potentially disruptive and subversive, not only difficult to handle, but threatening to undermine society's strictures regarding sex, aggression, and respect for elders. Hence, the old shepherd laments in *The Winter's Tale*:

I would that there were no age between ten and three-and-twenty, or that youth would sleep out the rest, for there is nothing in between but getting wenches with child, wronging the ancients, stealing, fighting... (Act III, scene iii).

In his seminal 1904 work, G. Stanley Hall noted "a period of semicriminality is normal for all healthy [adolescent] boys" (vol. 1, p. 404, quoted in [Arnett, 1999](#)). Speaking of the internal upheavals of adolescence, rather than overt deviant behavior, Anna Freud noted, "the upholding of a steady equilibrium during the

adolescent process is in itself abnormal" (1958/1969, p. 164). She observed that

it is normal for an adolescent to behave for a considerable length of time in an inconsistent and unpredictable manner; to fight his impulses and to accept them; to ward them off successfully and to be overrun by them; to love his parents and to hate them; to revolt against them and to be dependent on them;...to thrive on imitation of and identification with others while searching unceasingly for his own identity; to be more idealistic, artistic, generous and unselfish than he will ever be again, but also the opposite—self-centered, egoistic, and calculating (pp. 164-165).

In more recent years, this view of adolescence as inherently tumultuous has come under attack as an overgeneralization from mental health professionals' clinical samples or from popular authors' vivid portrayals of their own idiosyncratic adolescent experiences. For example, in an article polemically entitled "Debunking the Myths of Adolescence: Findings from Recent Research," Offer and [Schonert-Reichl \(1992\)](#) characterize the "myth" that normative adolescence is tumultuous as assuming that "[t]he 'typical' adolescent is ... out of control, in constant conflict with his or her family, and incapable of rational thought (p. 1003)." In contrast, they observe, on the basis of nonclinical samples, that "adolescence is not a time of severe disturbance for all adolescents. Moreover, ... a significant percentage of adolescents (80%) do not experience adolescent turmoil, relate well to their families and peers, and are comfortable with their social and cultural values.... [T]eenagers who exhibit little disequilibrium are normal ... [and] ... adolescence is a period of development that can be traversed without turmoil and that the transition to adulthood is accomplished gradually and without undue upheaval" (p. 1004).

As with many debates, the truth lies somewhere in the middle of these dichotomies and depends on the terms of the argument and matters of degree (e.g., what type and severity of turmoil). In a review of the topic, [Arnett \(1999, p. 324\)](#) concluded

[T]here is support for [Hall's \(1904\)](#) view that a tendency toward some aspects of storm and stress exists in adolescence. In their conflicts with parents, in their mood disruptions, and in their heightened rates of a variety of types of risk behavior, many adolescents exhibit a heightened degree of storm and stress compared with other periods of life. Their parents, too, often experience difficulty—from increased conflict when their children are in early adolescence, from mood disruptions during mid-adolescence, and from anxiety over the increased possibilities of risk behavior when children are in late adolescence. However ... there are cultural differences in storm and stress, and within cultures there are individual differences in the extent to which adolescents exhibit the different aspects of it.

[However], [e]ven amidst the storm and stress of adolescence, most adolescents take pleasure in many aspects of their lives, are satisfied with most of their relationships most of the time, and are hopeful about the future.

Having examined conflicts with parents, we turn to a more detailed consideration of mood problems and risk behaviors.

### **Mood Difficulties and Perceived Stress**

Adolescence is a time of rising incidence for major depression, with the risk of depression and the preponderance of affected girls versus boys increasing not only with age, but more specifically with advancing pubertal status.

Only a minority of adolescents develop a full-blown affective disorder. Nonetheless, adolescence sees a marked increase in emotional lability, depressed mood, and negative emotions (e.g., anxiety and self-consciousness), with over one-third of adolescents in nonclinical samples reporting high levels of depressed mood ( [Arnett, 1999](#); [Buchanan et al., 1992](#); [Larson and Richards, 1994](#); [Petersen et al., 1993](#); [Rutter et al., 1976](#)). For example, the national Youth Risk Behavior Survey ( [Centers for Disease Control and Prevention, 2000](#)) of high school students found that, during the preceding year, 36% of girls and 21% of boys felt sad or hopeless almost every day for at least 2 weeks in a row and 25% and 14%, respectively, seriously considered attempting suicide. Mood disruptions are associated with higher levels of negative life events ( [Brooks-Gunn and Warren, 1989](#)). Comparing fifth graders and ninth graders, [Larson and Richards \(1994\)](#) describe the dramatic decline in the proportion of time youngsters feel "very happy," "proud," or "in control" as an emotional fall from grace.

Girls appear especially prone to negative moods. For example, [Offer and colleagues \(1988\)](#) found marked gender differences in emotional vulnerability across adolescence. Compared with boys, girls described themselves as moodier, sadder, lonelier, more prone to uncontrollable crying, more easily hurt, less autonomous and more other-directed, and more ashamed of their bodies.

Adolescents also report a substantial increase in the number of negative life events. It is difficult to determine to what extent this reflects a more stressful environment, greater sensitivity to events, or shifts in the types of situations that precipitate negative emotions ( [Larson and Asmussen, 1991](#); [Wagner and Compas, 1990](#)). The nature and sources of perceived stress change over the course of adolescence, with early adolescents experiencing stress in relationship to peers and older adolescents with respect to academic issues. Especially in early adolescence, girls appear to experience more stress than at other ages and to perceive more stressful events than do boys ( [Ge et al., 1994](#); [Vik and Brown, 1998](#); [Wagner and Compas, 1990](#)). Although significant life events, such as parental separations or unemployment, moves, or deaths, have a serious impact on adolescents, many of the fluctuations in adolescent mood reflect the less dramatic daily hassles—homework, tests, disagreements with friends—the minor disappointments, stresses, and embarrassments that form the fabric of adolescent life. It may be deceptive, however, to try to conceptualize and quantify these hassles as completely independent external variables because unlike many major life events, the intensity, valence, and even the occurrence of such episodes often lie largely in the eye of the adolescent beholder ( [Csikszentmihalyi and Larson, 1984](#)).

Time sampling studies of adolescent mood in community samples find that, compared with adults, adolescents have greater mood variability as measured by both the width of their mood swings (between extreme highs and lows) and the evanescence of these extremes ( [Csikszentmihalyi and Larson, 1984](#)). In the nonclinical high school population studied, greater subjectively experienced mood lability was not associated with poorer adjustment or other pathologic processes.

Neurobiological factors also may influence the changes in mood regulation and stress reactivity observed in adolescence.

A longitudinal study by [Angold and colleagues \(1999\)](#) found that reaching Tanner stage III was associated with increased levels of depression in girls; however, hormonal levels of testosterone and estradiol were more closely associated with levels of depression than age or Tanner stage *per se*. Although this suggests that it is not morphologic body changes themselves that render pubertal girls more prone to depression, it is unclear whether these findings reflect direct depressogenic endocrine influences on the central nervous system or hormonally mediated changes in responsivity to life events and stress.

[Spear \(2000\)](#) proposed that age-related changes in the balance of dopamine regulation in the prefrontal cortex relative to mesolimbic brain regions lead to shifts in the incentive value and motivational power of different reinforcers. Drawing on both human and animal data, Spear proposes a transient relative "reward deficiency syndrome" or "adolescent anhedonia" that results in previously pleasurable activities being experienced as less rewarding and leads to a compensatory search for new and more intense forms of stimulation (e.g., greater novelty seeking, risk taking, and increases in consummatory behaviors, such as food and drugs).

### **Risk-Taking Behaviors**

Risk-taking behavior is a significant source of morbidity and mortality for young people. In contrast to persons 25 years of age or older, for whom two-thirds of all deaths are due to cardiovascular disease and cancer, three-fourths of all deaths for youths 10 to 24 years of age are due to motor vehicle crashes, other unintentional injuries, homicide, and suicide, with AIDS also becoming a rapidly rising cause of death for young adults. Behaviors that increase the risk of these adverse outcomes are common among teenagers. The [Centers for Disease Control and Prevention \(2000\)](#) national Youth Risk Behavior Survey found that for the 30 days preceding the survey, 16.4% of high school students reported rarely or never wearing a seat belt, one-third had ridden with a driver who had been drinking, 17.3% had carried a weapon, 50% had drunk alcohol, 34.8% had smoked tobacco, 26.7% had used marijuana, and 7.8% had attempted suicide in the prior 12 months. Other health-impairing behaviors also were common. Half of the students had had sexual intercourse; of these, 42% had not used a condom at last intercourse.

These risk behaviors are not uniformly distributed across the adolescent population. For example, among ninth graders, 22% are estimated to be at low risk, with no involvement with any risk behaviors such as substance use, sex, depression/suicide, antisocial behavior, school problems, unsafe vehicle use, or bulimia ( [Dryfoos, 1998](#)). Another 29% report only one such risk indicator, 18% report two, and 31% report three or more. High-risk youth (those reporting multiple risk behaviors) are characterized by early onset of high-risk behaviors, absence of nurturing parenting, child abuse, lack of involvement with school, susceptibility to peer influence, depression, disadvantaged neighborhoods, and lack of gainfully employed role models. Certain patterns of autonomic reactivity may also predispose to increased risk-taking under certain circumstances ( [Liang et al., 1995](#)).



It is important to distinguish between occasional experimentation and persistent patterns of dangerous behavior ( [Steinberg and Morris, 2001](#) ). Although most adolescents will experiment with alcohol or minor delinquent behaviors, in most cases these behaviors do not persist into adulthood. The prevalence of problem behaviors does increase in adolescence and early adulthood, but persistence in problem behaviors, such as substance use or antisocial behavior, usually is associated with difficulties in earlier childhood ( [Arnett, 1992](#) ).

The work of [Jessor \(1991\)](#) and others ( [Igra and Irwin, 1996](#) ) suggests that risk-taking behaviors such as sexual activity, substance use, reckless driving, and delinquency not only increase over adolescence but frequently are associated with each other and share common psychosocial antecedents. These behaviors are not simply arbitrary, perverse, or motivated only by sensation-seeking or exploratory motives, but are in part purposeful, meaningful, goal-oriented, and functional. For example, [Jessor \(1991\)](#) observes that such behaviors also can serve the instrumental ends of gaining peer acceptance, establishing autonomy from parents, defying conventional authority, relieving anxiety or frustration, or affirming the transition to a more adult status.

## ETHOLOGIC PERSPECTIVES

Examination of the transition from youth to adulthood in other mammalian (especially nonhuman primate) species suggests animal models for the counterpart of human adolescence. Like human adolescents, the young of other species exhibit increases in peer-directed social interactions and greater novelty-seeking and risk-taking behaviors ( [Wilson and Daly, 1985](#) ). In a thought-provoking review, [Spear \(2000\)](#) has suggested that these shared behavioral features represent ontogenetic adaptations that help individuals in this transitional period in “acquiring the necessary skills to permit survival away from parental caretakers. Increased affiliation with peers and the taking of risks via exploring novel areas, behaviors, and re-enforcers may also help facilitate the dispersal of adolescents away from the natal family unit” ( [Spear, 2000](#), p. 418 ), with the adaptive goal of avoiding inbreeding. Similarly, human adolescents and many pubertal nonhuman primates spend increased time in social interactions with peers, including aggressive fighting, but also in reconciliatory and affiliative behaviors; paralleling this shift in social orientation from adults to peers is an increase in conflicts between the adolescent and parents, which also may help to encourage separation from the natal family unit ( [Steinberg, 1989](#); [Steinberg and Belsky, 1996](#) ). Increased risk taking also is seen across species, with increased exploratory behavior and novelty seeking, but also increased mortality. From an evolutionary perspective, such risk taking “may be—or at least once were—means of securing physical resources, attracting mates, and denying mating opportunities to competitors” ( [Steinberg and Belsky, 1996](#), p.117 ).

## THE CLOSE OF ADOLESCENCE

The pubertal changes that are the hallmark of adolescence provide a relatively clear marker for the beginning of adolescence. In contrast, the close of adolescence in contemporary society is less clearly defined. At one time in the United States and even today in traditional societies, the end of adolescence and assumption of adult status was usually marked by a discrete event, such as marriage, beginning of full-time employment, or military service. Currently, however, the same forces that have helped to create adolescence as a distinctive period of life in industrial and postindustrial society also have blurred the end of adolescence ( [Arnett and Taber, 1994](#) ). College and postgraduate education has become increasingly important, and the proportion of youth pursuing post-high school education has risen from 14% in 1940 to over 60% in the 1990s. Correspondingly, the median age of marriage in the United States rose from 21 years for women and 23 years for men in 1970 to 25 years for women and 27 years for men in 1996. Thus, for many young people, entry into adult roles regarding work, marriage, and parenthood is delayed until the late twenties or even early thirties.

[Arnett \(2000\)](#) has proposed that the period from the late teens through the twenties be considered a distinctive period he terms *emerging adulthood*. In contrast to adolescents, 95% of whom live in a parental home, most young people age 18 years or older in the United States leave home, approximately one-third to live in a college setting and approximately 40% to live independently and work full time. Approximately two-thirds cohabit for a time with a romantic partner. However, despite high rates of residential mobility in the twenties, many young people retain some degree of dependence on their parents. Hence, adolescence gives way to a prolonged period of quasiautonomy and continued identity and vocational exploration that only gradually draws to a close in the third decade of life.

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## 25 DEVELOPMENT OF THE SYMPTOM OF ANXIETY

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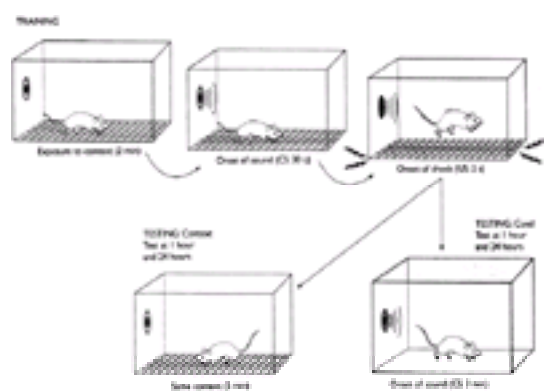
Advances in neuroscience have generated increasingly sophisticated understandings of brain function. This understanding initially applied to basic aspects of perception and motor control and was subsequently extended to cognitive constructs, such as language and attention. More recently, advances in neuroscience have generated novel insights on emotion, and “anxiety” has emerged as an emotion for which a particularly sophisticated understanding has been reached. This has been possible because of the considerable cross-species reservation in the underlying neural circuitry that moderates anxiety, as reflected in cross-species parallels in physiologic and behavioral aspects of anxiety. Using a modern neuroscience approach, this chapter outlines understandings of how symptoms of anxiety arise in human beings. The chapter begins with a summary of current knowledge on the underlying neural circuitry involved in anxiety. This summary is followed by a review of data documenting the wide range of parallels between humans and animals in phenomenologic and neurophysiologic aspects of anxiety. Finally, the basic science and clinical literature is integrated to delineate briefly current views on the origin of anxiety in humans.

It is vital to begin such an integrative perspective on anxiety with a review of current definitions applied across basic science and clinical domains. Because animal-based research cannot rely on subjective reports to define one or another emotion, basic science definitions draw heavily on behavioral factors. For the current chapter, the term *emotion* refers to a brain state associated with perception of either a reward or punishment. The term *reward* refers to any stimulus for which an organism will extend effort to obtain, whereas the term *punishment* refers to any stimulus for which an organism will extend effort to avoid. Some controversy remains over defining individual emotions, such as anxiety or fear, because families of emotions may exist on continua, varying in terms of both valence and arousal. For the current chapter, the term *fear* refers to a family of brain states that result from the perception of punishments or overtly dangerous stimuli; the term *anxiety*, in contrast, refers to a family of brain states associated with fear or apprehension that is inappropriate to the context. As an example, “fear” is the emotion that a person would experience when confronting a large snake in the woods, whereas “anxiety” is the emotion a person would experience when confronting a large snake in the zoo. The current chapter considers the relevance of basic neuroscience research in this area for understandings of normal human fears, as well as for understandings of selective anxiety disorders, comprising phobias, spontaneous panic attacks or panic disorder, separation anxiety disorder, and generalized anxiety disorder. Clinical aspects of these disorders, as well as both clinical and biological aspects of other anxiety disorders, are considered elsewhere in this volume.

### BASIC SCIENCE PERSPECTIVES ON ANXIETY

#### Fear Conditioning

Recent interest among neuroscientists in emotion and particularly in anxiety can be traced to work conducted since the late 1970s on the construct of fear conditioning (Davis, 1998; Davis et al., 1997; LeDoux, 1996, LeDoux, 1998; Maren, 1999). As illustrated in Fig. 25.1, in a fear conditioning experiment, an organism, typically a rodent, is placed in an experimental chamber and an aversive or punishing stimulus, such as a shock or a blast of air, is paired with a neutral stimulus, such as a sound or a light. The aversive stimulus is considered the unconditioned stimulus (UCS), whereas the neutral stimulus is considered the conditioned stimulus (CS). In this situation, an organism's reaction to the CS changes with repeated presentation, such that the organism begins to exhibit behaviors such as freezing and physiologic changes such as altered heart rate and respiration, associated with states of fear. Fear conditioning is an amazingly robust phenomenon, repeatedly demonstrated across a diverse array of animal species (Davis, 1998; Davis et al., 1997; LeDoux, 1996, LeDoux, 1998; Maren, 1999).



**Figure 25.1.** The protocol for two types of fear conditioning that can be produced after a single learning trial. The trial consists of a single foot shock and produces context conditioning (to a box) and cued conditioning (to a tone). CS: conditional stimulus. (From Squire RL, Kandel ER: *Memory: From Mind to Molecules*. New York, Scientific American Library, 1999; with permission).

The conceptual importance of fear conditioning emanates both from its ubiquity among organisms and from the degree to which neuroscientists have delineated the underlying neural circuitry. Through the work of (LeDoux 1996,1998), Davis (1998, 1997), and others, a key role in fear conditioning has been ascribed to a collection of nuclei that lie in the amygdala, a medial temporal lobe structure lying in primates immediately anterior to the hippocampus. Much of this work relies on processing of auditory CS stimuli, which are perceived at the ear and further processed in a circuit extending from the thalamus to the basolateral nucleus of the amygdala. Processing of the aversive UCS stimulus also is thought to extend to the basolateral nucleus, where information on the UCS and CS can be integrated. This information is transmitted to the central nucleus of the amygdala, which, in turn, interfaces with multiple brain systems involved in an organism's physiologic and motor responses to danger. For example, this nucleus interfaces with the paraventricular nucleus of the hypothalamus, which regulates hypothalamic–pituitary–adrenal (HPA) axis activity, the parabrachial nucleus in the brain stem, which regulates respiratory activity, as well as components of the vagus nerve that regulate cardiovascular activity.

Precise delineation of underlying neural circuitry for a behavioral phenomenon, such as fear conditioning, enables a comprehensive understanding of mechanisms behind such a phenomenon. Such understanding, in turn, facilitates molecular and genetic insights into the phenomenon. For example, fear conditioning represents an elemental form of learning, during which contingencies between two stimuli, the CS and UCS, are perceived. Beginning with the insights from Hebb, a series of studies over the past 50 years outline the cellular and molecular basis of such learning (Mayford et al., 1996; Squire, 1992). At a cellular level, learning results from changes in an outflow neuron after the delivery of temporally linked action potentials from two afferent neurons. When these action potentials are delivered in phase, the threshold for stimulating the outflow neuron is lowered, resulting in a state termed *long-term potentiation* (LTP). Applied to fear conditioning, learning the nature of

the relationship between the CS and UCS is thought to involve LTP in neurons in the basolateral amygdala ( [Maren, 1999](#)). Because LTP is understood in molecular terms, work on the role of LTP in fear conditioning generates a set of explicit hypotheses about the role of genetic factors in fear conditioning among various species, including humans.

### Fear and Anxiety as Heterogeneous Constructs

Research on the relatively simple construct of fear conditioning has stimulated an array of experiments focused more broadly on the range of reactions organisms typically exhibit to a range of dangerous stimuli or scenarios. These experiments demonstrate considerable heterogeneity in behaviors associated with distinct stimuli and scenarios. This heterogeneity is thought to reflect differences in the underlying neural circuitry linked to unique brain states associated with one or another form of danger. Considerable disagreement persists on the most appropriate means for categorizing brain states associated with such distinct forms of danger. This disagreement relates to incomplete understandings of the underlying neural circuitry. Nevertheless, general agreement has emerged on the view of fear and anxiety as a family of relatively specific constructs. A brief summary is provided of ranging views on distinctions among fear-related behaviors.

Stimuli differ in the degree to which they are naturally perceived as aversive or punishing. For example, in the fear conditioning experiment, the UCS consists of a stimulus that is innately fear-provoking, capable of eliciting withdrawal in the absence of prior training. The CS, in contrast, comes to elicit fear-related behaviors through learning. Organisms exhibit distinct reactions to stimuli that induce fear innately as opposed to through learning. Moreover, within categories of innate or learned fears, organisms exhibit considerable heterogeneity in their reactions, depending on particular features of the fear-inducing stimulus.

For innately fear-inducing stimuli, organisms' reactions depend on various characteristics of the feared stimulus. Work by Blanchard et al. (1997) has documented such a range in the reactions of rodents when confronted with overt danger. For example, when a rat is placed directly in the presence of a cat, the cat elicits an immediate flight response in the rat that typically is followed by a prolonged period of freezing when the rat reaches the safety of the burrow. In contrast, indirect evidence of a cat, perhaps related to the smell of a cat or signals from other rats suggesting that a cat may be near, typically elicits immediate freezing without flight. Similarly, organisms exhibit a range of reactions to innately aversive stimuli based on the proximity of the stimulus ( [Rodgers, 1997](#)). A distal threat typically elicits freezing, whereas an immediate threat typically elicits either flight or attack.

For learned fears, organisms also exhibit a range of reactions to distinct components of the fear. In the classic fear conditioning experiment, an organism learns to fear the CS, such as the tone or the light, owing to a process known as *cue-specific fear conditioning*. The organism also learns to fear the experimental chamber within which the UCS had been delivered through a second process known as *context conditioning* (see [Fig. 25.1](#)) ( [LeDoux, 1998](#)). Although some controversy persists over the precise details of the underlying neural circuitry for context conditioning ( [Davis, 1998](#); [Davis, 1997](#); [LeDoux, 1998](#); et al.), cue-specific and context conditioning are generally thought to involve distinct components of the nervous system. Whereas the amygdala is involved in both phenomena, either the hippocampus or the bed nucleus of the stria terminalis may play a selective role in context conditioning.

Based on pharmacologic and neuroanatomic data, [Davis \(1998\)](#) devised a scheme for categorizing fear-related behaviors. Learned fears, as classically modeled by cue-specific conditioning, represent one such family of behavioral construct. Such learned fears are acquired through experiences with relatively abrupt or phasic temporal cues, such as a light or sound presented for a brief period, that signal a soon-to-be-delivered aversive stimulus. Such learned fears extinguish relatively easily with repeated presentation of unpaired CS and UCS stimuli. For the second category of fear-related behavior, [Davis \(1998\)](#) uses the term *anxiety*, which is reminiscent of the broader use of this term as defined previously. In contrast to learned fears, anxiety for Davis typically applies to stimuli in animals that are innately dangerous or punishing. Anxiety-provoking stimuli involve more protracted temporal cues, such as a well-lit open field in which a rodent is placed where it would be vulnerable to attack. Similarly, unlike learned fears, the response to anxiety-provoking stimuli is relatively resistant to extinction and may even become sensitized on repeated presentation.

### Neurochemistry of Fear-Related Behaviors

From a clinical perspective, the importance of work by Blanchard et al. (1997), [Davis \(1998\)](#), and others ( [Rodgers, 1997](#)) on the heterogeneity of fear states lies in the neurochemical insights these studies may ultimately provide. Neurochemical regulation of activity in brain circuits associated with fear states is understood in increasingly precise detail. Projection neurons involved in fear states rely on glutamate as a neurotransmitter. This includes, for example, projections involved in fear conditioning from the thalamus to the amygdala and from the amygdala to effector organ systems. Activity in these glutamatergic synapses is, in turn, modulated by local g-aminobutyric acid (GABA) neurons in the amygdala and locally secreted neuropeptides, as well as by distal neurochemical inputs from noradrenergic or serotonergic neurons ascending from the brainstem, hormones such as cortisol, and peripherally secreted compounds, such as epinephrine ( [Blair et al., 1998](#); [Gray and McNaughton, 1996](#); [McNaughton, 1997](#); [Mongeau et al., 1997](#); [Quartermain et al., 1993](#); [Roosendaal et al., 1999](#); [Stutzman et al., 1998](#)).

Although this basic neurochemical architecture applies to many fear-related behaviors, distinct forms of fear are associated with unique forms of neurochemical regulation. For example, Davis has elucidated distinctions in the degree to which various fear-related behaviors are influenced by the neuropeptide, corticotropin-releasing factor (CRF) ( [Davis, 1998](#); [Davis et al., 1997](#)). CRF plays parallel regulatory roles at a hormonal level in HPA axis function, where it stimulates adrenocorticotropic hormone secretion from the pituitary, as well as at a local brain circuitry level, where it acts as a neuromodulator of brain states associated with aversive stimuli. For learned fears, as classically modeled by cue-specific fear conditioning, manipulations of CRF in amygdala nuclei produce less marked effects on fear-related behavior than they do for innate fears. For innate fears, manipulations of CRF in the basolateral nucleus of the amygdala, as well as the bed nucleus of the stria terminalis, produce relatively more robust changes in fear-related behaviors. To index fearfulness, [Davis et al. \(1997\)](#) rely on the degree to which a rat will startle when placed in a well-lit open field. High levels of fear, as induced either by an open field or a fear conditioning experiment, are reflected in potentiation of the startle reflex ( [Davis, 1998](#)). CRF infusions into relevant brain regions produce a further potentiation of this reflex in the open field test, although not in cue-specific fear conditioning experiments. There is evidence of an association between CRF and clinical anxiety, as well as measures of fear among nonhuman primates ( [Coplan et al., 1995b](#); [Pine and Grun, 1999](#); [Sapolsky, 1996](#)). As such, these data suggest a subset of fear behaviors in animals exhibit particularly close relationships with clinical anxiety.

Work on the heterogeneity of fear states also carries considerable psychopharmacologic implications. There is a rich tradition documenting parallels between effects of anxiolytic medications on anxiety in humans and animal models ( [Gray et al., 1996](#); [McNaughton, 1997](#)). For one set of phenomena, there are strong consistencies across species. For example, benzodiazepines exert robust effects on specific forms of acute anticipatory anxiety in humans and animals ( [Gray et al., 1996](#); [McNaughton, 1997](#)). Classically, these forms in animals have included approach-avoidance conflicts and fear learning paradigms, as modeled in fear conditioning. For another set of paradigms, there are less consistent cross-species parallels. For example, many antidepressants exert robust clinical effects on both acute panic and anticipatory anxiety in humans but minimal effects in animal fear paradigms, including fear conditioning experiments ( [Cassella and Davis, 1985](#); [Rodgers, 1997](#)). Closer parallels between animals and humans emerge in more recent studies that rely on ecologically informed animal models. For example, Blanchard et al. (1997) note close parallels in pharmacologic effects on panic disorder and rodents' reactions to select innate fear-provoking stimuli, such as the presence of a predator. Antidepressant medications, including selective serotonin reuptake inhibitors, exhibit parallel effects on such phenomena and on clinical anxiety disorders (Blanchard et al, 1997; [Rodgers, 1997](#)).

Because of the difficulties in conducting studies in nonhuman primates, neurochemical regulation of the circuitry underlying fear-related behaviors has been more extensively mapped in rodents than primates. Nevertheless, there is some lesion-based work examining the role of the amygdala in fear-related processes among nonhuman primates ( [Bachevalier et al., 1999](#); [Rolls, 1999](#); [Webster et al., 1991](#)). Studies have demonstrated involvement of the primate amygdala in socially relevant behaviors ( [Leonard et al., 1985](#); [Rolls, 1984, 1999](#)). Cells in the primate amygdala are sensitive to emotionally salient social cues, such as threat faces ( [Leonard et al., 1985](#)).

### Development and Basic Science Perspectives

Despite considerable advances in many basic science aspects of fear, neurodevelopmental aspects of anxiety remain relatively underexplored. The paucity of research in this area stands in contrast to a wealth of literature on developmental aspects of human fears. Most of the advances reviewed previously in neuroscience derive from the experimental psychology tradition, as illustrated by research on fear conditioning. In the area of developmental neurobiology, the advances that have emerged derive from both the experimental and ethologic traditions.

From the tradition of experimental psychology, most developmentally oriented research on fear states examines the organizational influences of early-life stress on the expression of fear-related behaviors and on the functioning of neural circuits associated with such behaviors. Early in the life of a rodent, stress can exert long-term, potentially permanent effects. [Levine et al. \(1967\)](#) initially noted the long-term effects on stress-reactivity of handling manipulations in the initial weeks of



life. [Meaney and colleagues 1988](#); ([Francis et al., 1999](#)) extended this work by demonstrating the full range of effects from handling and the underlying neural mechanism for the effects. For example, neonatal handling produces long-term increases in feedback sensitivity in the HPA axis, as well as decreases in anxiety on various standardized paradigms. Although relatively mild stressors, such as handling, do not exert such long-term effects in adult organisms, the precise developmental window for these effects has not been defined. It remains unclear if the HPA axis and related fear systems are susceptible to such environmental effects only during the neonatal period.

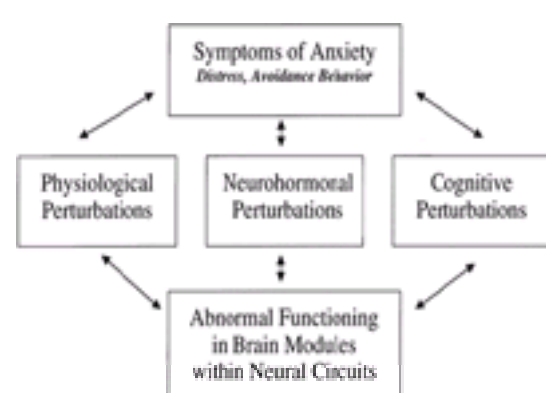
Other related work traces the development in nonhuman primates of neural pathways implicated in fear conditioning. Although the intrinsic connections of the primate amygdala mature very early in life, [Bachevalier and colleagues \(Bachevalier et al., 1999; Rolls, 1999; Webster et al., 1991\)](#) demonstrate considerable plasticity during childhood in the afferents from the inferior temporal region to the amygdala. Such plasticity may play a role in developmental variations in the effects of brain lesions on emotional behavior. Recent lesion studies among nonhuman primates primarily use adult animals, and the effects of amygdala lesions may vary as a function of developmental stage.

From the ethologic perspective, research among nonhuman primates establishes effects similar to those seen in rodents of early-life rearing manipulations on HPA axis function and fear-related behaviors. [Rosenblum, Coplan, and colleagues \(Coplan et al., 1995b, 1996; Smith et al., 1997\)](#) note such effects on infant bonnets in a variable foraging demand paradigm. In this experiment, mother bonnets are required to exert an unpredictable degree of effort to locate food, which affects the mother-child relationship. This produces increases in fear-related behaviors in the child and elevations in cisternal CRF when the former child bonnet is studied at maturity. Other work on nonhuman primates documents the effects of maternal separation on fear-related behaviors. [Suomi and colleagues \(Berman et al., 1994; Suomi, 1997\)](#) suggest that developmental experiences related to separation may interact with underlying genetic predispositions to produce fear states. Finally, work by [Kalin and Shelton \(1998\)](#) considers specificity in the developmental nature of fear responses shown by nonhuman primates. Much like work from Davis delineating differences between “fear” and “anxiety,” Kalin and Shelton find that immature primates show a range of responses to distinct threats, including isolation and social threat. Moreover, much like Davis' concepts of fear and anxiety, these responses in immature infant primates are dissociable in terms of their psychopharmacology.

## PARALLELS BETWEEN HUMAN AND ANIMAL FEAR

Through the mid-1990s, much of the clinical psychobiological research on human anxiety examined relationships between clinical factors and peripheral physiology or monoamine profiles. Underlying pathophysiologic processes were sought in measures of cardiovascular, respiratory, serotonin, or norepinephrine dysregulation. Such work remains an active area of investigation. For example, there is accumulating evidence, reviewed later, to link abnormalities in respiratory physiology to a family of disorders associated with spontaneous panic ([Coplan and Klein, 1996; Gorman et al., 1988; Klein, 1993, 1994; Papp et al., 1989, 1993](#)). Similarly, a wealth of research documents abnormalities in monoamine regulation in various disorders, particularly panic disorder ([Coplan and Klein, 1996](#)). Nevertheless, advances in basic science have provided the novel insights reviewed previously on the role for specific neural circuits in anxiety, and recent clinical studies have attempted explicitly to link such basic findings to clinical constructs.

Basic science conceptualizations of anxiety emphasize the role for neural circuits that involve an array of interconnected brain regions functioning as distinct modules ([Davis, 1998; Davis et al., 1997; Gray et al., 1996; LeDoux, 1996, 1998; Maren, 1999; McNaughton, 1997](#)). These include modules located in limbic structures, such as the amygdala and hippocampus, modules located in the frontal lobe, and modules located in the inferior posterior cortices. This conceptualization resonates with recent views on other brain processes, such as attention allocation, that emphasize the modularity of the brain. In basic science studies, knowledge of the relationship between functional aspects of brain modules and behavioral manifestations of fear states requires a careful delineation of the observable processes associated with fear states. Studies in humans delineate such components at multiple levels. The scheme for such work is illustrated in [Fig. 25.2](#).



**Figure 25.2.** Schematic of delineation of the observable processes associated with fear states.

At the grossest level, considerable research examines overt manifestations of anxiety and fear, considering the degree to which distinct fear states represent truly unique syndromes. Such overt expressions are manifest as avoidance behavior and subjective states of distress. At a slightly deeper level, such overt manifestations are associated with changes in cognitive functioning, autonomic regulation, and neurohormonal tone. Finally, these cognitive or physiologic alterations are, in turn, thought to arise from changes in brain function that may be detectable using modern neuroimaging techniques.

### Overt Manifestations of Fear and Anxiety in Humans

Clinical or behavioral manifestations of anxiety are categorized in the *Diagnostic and Statistical Manual of Mental Disorders* (4th edition, revised; DSM-IV) based on the object of the anxiety and the form of the symptoms. As outlined elsewhere in this volume (see [Chapter 67](#)), distinct clinical disorders are defined based on their differential involvement of feared objects or situations, as well as their distinct constellations of symptoms. Phobias refer to excessive fear related to specific objects or situations; panic disorder involves recurrent episodes of spontaneous sudden increases in fear associated with autonomic symptoms (panic attacks); and generalized anxiety disorder involves a pattern of pervasive worry. Other anxiety disorders not discussed in the current chapter include posttraumatic stress disorder and obsessive-compulsive disorder.

Beyond these clinically defined syndromes, development is characterized by a pattern of normal fears and worries ([Evans et al., 1999; Fredrikson et al., 1996; King et al., 1989; Muris et al., 1998, 1999; Ollendick et al., 1989, 1991; Pine and Grun, 1999](#)). These include fears in toddlers related to separation and strangers, fears during the early school years related to small animals and bodily harm, as well as fears and worries during adolescence related to social concerns. Phenomenologically, behaviors exhibited during such normal fear states resemble those found in the anxiety disorders, both being characterized by avoidance or withdrawal and high levels of physiologic arousal. However, such early childhood fears are considered a normal aspect of development as long as they are not associated with excessive distress or impairment. The relationship between such normal fears and later, clinically significant anxiety disorder remains an area of active study. [Rosen and Schulkin \(1998\)](#) suggest that normal degrees of fear-related behavior during childhood, through effects on the CRF system in the amygdala, may alter the underlying substrate from which later anxiety disorders arise. Similarly, the behaviorally inhibited temperament represents a normal behavioral variant in toddlers that predicts anxiety disorders during childhood and adolescence ([Biederman et al., 1995; Kagan, 1995; Schwartz et al., 1999](#)). Nevertheless, the limited available longitudinal data suggest that high levels of developmentally appropriate fears during childhood show relatively weak associations with later clinical anxiety disorders ([Fyer, 1989; Pine, 1999; Pine et al., 1998a](#)).

Each of the developmentally appropriate and clinical fear states in humans share various similarities in terms of observable behavior with anxiety states in animals. This includes the tendency to avoid objects or situations associated with the fear, the tendency closely to monitor the feared object, and the tendency to experience changes in somatic activity associated with avoidance and monitoring.

Considerable disagreement remains on the degree to which specific fear states or anxiety syndromes described in DSM-IV represent truly distinct categories. Despite clear distinctions in terms of overt symptoms, there is some evidence to suggest that some supposedly distinct anxiety disorders in DSM-IV actually represent subtle variations on a single category of anxiety state. Such evidence includes extremely high comorbidity rates, particularly among children, and similarities in overt symptoms, as well as in demographic correlates of the various disorders ([Cooper and Eke, 1999; Costello and Angold, 1995; Fyer et al., 1995; Klein, 1995; Merikangas et al., 1999](#)). For other disorders, there is more evidence from longitudinal and family-based studies to suggest that the syndrome described in DSM-IV



depicts a relatively specific construct. The most convincing evidence of specificity probably exists for phobias. For both social and specific phobia, there is relatively good evidence to suggest that these syndromes, particularly among children and adolescents, are distinct from each other as well as from other anxiety disorders (Cooper et al., 1999; Fyer, 1989; Klein, 1995; Merikangas et al., 1999; Pine et al., 1998a). For separation anxiety disorder, there also is evidence of distinction from other anxiety disorders, although the evidence appears somewhat less convincing than for phobias. For example, relative to childhood phobias, separation anxiety disorder appears to show a closer longitudinal and family-based tie to adult panic disorder (Costello et al., 1995; Pine, 1999). Generalized anxiety disorder, in contrast, exhibits strong relationships with a range of anxiety disorders (Fyer et al., 1995; Pine, 1999; Pine et al., 1998a). Data on specificity of clinical disorders is consistent with basic science research emphasizing the heterogeneous nature of fear states in animals.

### Cognitive Correlates of Anxiety

Beyond similarities between animals and humans in overt symptomatic manifestations of fear and anxiety, anxiety in humans is associated with particular cognitive attributes that are reminiscent of attributes found in animals. The most consistent cognitive feature associated with fear and anxiety in humans represents an attention bias for stimuli associated with danger (Andrews et al., 1990; Barlow, 1988; Capps et al., 1996; Kendler et al., 1995; McNally, 1996, 1997; Mineka and Sutton, 1992). This bias resembles the high degree of vigilance animals show for feared scenarios or objects. Attention bias in human anxiety states typically is measured with one of two standardized paradigms, either the dot-probe (McNally, 1996; Mineka and Sutton, 1992) or the emotional Stroop task (Andrews et al., 1990). The Stroop task requires subjects to name the color of various potentially threatening words, such as *coronary* or *embarrass*. Individuals with high levels of anxiety show prolonged latency for naming such color-words. This effect presumably arises from interference, such that reactions elicited by a fear-word affects attention to the color of the word. In the dot-probe task, individuals monitor two boxes, one containing either a threatening word or picture and another containing a neutral word or picture, presented on either side of a computer screen. Subjects must press a button when a target appears in one of the boxes, and reaction times to these targets are influenced in individuals with high levels of anxiety by proximity between the threat cue and the target.

Both the results for the Stroop and the dot-probe tasks remain amazingly well replicated across a range of disorders (Andrews et al., 1990; Barlow, 1988; Capps et al., 1996; Kendler et al., 1995; McNally, 1996, 1997; Mineka and Sutton, 1992). Attention bias is found across development and across most anxiety disorders, including phobias, panic disorder, and generalized anxiety disorder. Similar degrees of attention bias are found across these disorders, although each disorder may show particular susceptibility to attention biases associated with words that are most explicitly linked to a particular disorder. For example, subjects with panic disorder may show particularly prolonged latency to panic-related words, such as *smother*, whereas subjects with social phobia may show particularly prolonged latency to phobia-relevant words, such as *embarrass*.

Disruptions in mnemonic functioning represent a second, albeit less well-replicated cognitive correlate of human anxiety (Capps et al., 1996; Mathews and MacLeod, 1994; McNaughton, 1997; Squire, 1992; Taghavi et al., 1999; Williams et al., 1996). Two aspects of memory have been examined. First, overall levels of memory performance are influenced by functional aspects of limbic structures, including the hippocampus, that are implicated in animal models of anxiety (Capps et al., 1996; McNaughton, 1997). As a result, a series of studies in humans consider relationships between clinical anxiety and memory performance. In general, most studies find relatively intact memory function across anxiety disorders, although there are some notable exceptions to these findings. Second, memory is influenced by the emotional salience of perceived stimuli (Pine and Grun, 1999; Roozendaal et al., 1999; Vasey et al., 1996). Both humans and lower animals display a tendency selectively to recall arousing or emotionally evocative stimuli. In anxiety disorders, particularly panic disorder, there is some limited evidence that fear-related words are selectively recalled (McNally, 1997). Nevertheless, the weight of the evidence more convincingly demonstrates mnemonic bias but not attention bias in depressive disorders and attention bias but not mnemonic bias in anxiety disorders. Given the influence of attention on encoding and the influence of anxiety on attention, the lack of a mnemonic bias in anxiety has led to the suggestion that some active cognitive process interferes with encoding or recall in anxiety.

Anxiety-related bias in cognitive functions is thought to index the underlying diathesis for anxiety (Kendler et al., 1995). These cognitive attributes are seen as elemental reflections of dysfunction in limbic brain circuits associated with animal models of anxiety. This conceptualization of cognitive functions may provide a key avenue for integrating basic and clinical science on anxiety. Despite the heuristic importance of these views, effective treatments have been shown to eliminate cognitive bias in anxiety (Andrews et al., 1990; Kendler et al., 1995), which suggests either that cognitive bias is more a reflection than a cause of anxiety or that effective treatments alter both symptoms and the underlying diathesis for anxiety. Longitudinal or high-risk studies documenting an association between prior cognitive bias and later anxiety are needed to clarify the nature of these associations. There is some evidence that self-reported cognitive bias, in the form of anxiety sensitivity, predicts later anxiety reactions to stress (Schmidt et al., 1999). However, such self-reported bias may be confounded with subtle clinical symptoms. No studies have used the Stroop or dot-probe task to predict changes in clinical anxiety over time.

From a neuroscience perspective, clinical work on cognitive biases provides a potentially important avenue for integrative research. Studies of mnemonic bias in animals precisely delineate the role for a neural circuit centering on the basolateral amygdala (Pine and Grun, 1999; Roozendaal et al., 1999; Vasey et al., 1996). Regulation of this circuit is understood in precise detail, involving monoamine and autonomic influences. For attention bias, more neuroscience research examines influences on attention for incentive, rewarding stimuli than for anxiety-related stimuli (Gallagher and Holland, 1994; LeDoux and Muller, 1997; Phelps and Anderson, 1997). Distinct amygdala nuclei influence distinct aspects of such bias for incentives. Nevertheless, given strong influences of the amygdala on attention for rewards, such research has piqued interest in the role of the amygdala in attention for punishments and other anxiety-related stimuli.

### Physiologic and Neurochemical Correlates of Anxiety

As in the area of cognition, research on physiologic and neurochemical correlates of anxiety may provide insights into underlying brain pathways for anxiety in humans, facilitating integration between basic and clinical studies. Changes in autonomic physiology represent key aspects of anxiety in animal models (Davis, 1998; Davis et al., 1997; LeDoux, 1996, 1998). Such changes are linked to specific anxiety-related constructs. For example, in animals, panic-like states associated with flight involve marked increases in both respiratory and cardiovascular arousal that facilitate escape from proximal danger. In contrast, vigilance or orienting responses associated with monitoring of a distal threat involve distinct changes in physiology associated with maximal intake of information from the external environment.

It remains unclear the degree to which research on human autonomic physiology can facilitate an integration of basic and clinical views of anxiety. Clearly, work in this area has established important parallels between anxiety states in humans and animals. For example, both flight reactions in animals and panic attacks in humans share similar autonomic hallmarks. Moreover, there is some evidence that distinct emotional states are each associated with a unique profile in terms of autonomic physiology. For Damasio (1994), distinct emotional states may reflect the evaluation of such unique peripheral physiologic profiles by ventral components of the frontal lobes. Nevertheless, at least for measures of cardiac autonomic function, there exists insufficient evidence of specific relationships between physiology and one or another specific disorder to support the notion that peripheral physiologic profiles provide labels for one or another emotion. Recent physiologic studies on respiration and on startle regulation do suggest that work in these areas may provide more direct insights on the underlying brain pathways involved in human disorders.

In animal models, respiratory changes are intimately connected with specific fear states involving innate, immediate threats. Flight reactions associated with immediate threat involve robust increases in respiration, possibly resulting from activation of regions in the brain stem involved in respiratory control (Pine and Grun, 1999). Separation reactions also are associated with brain stem activation and precise control of respiratory activity associated with distinct separation-related vocalizations. Moreover, both panic and separation anxiety reactions in animals are sensitive to manipulations of the opiate systems (Blanchard et al., 1997; Kalin, 1993; Kalin and Shelton, 1989, 1998; Pine and Grun, 1999). In humans, longitudinal and family-based studies note associations between separation anxiety and panic, suggesting parallels with animal fear states involving precise changes in respiration. Moreover, in humans, both panic disorder and separation anxiety disorder show unique associations with respiratory dysregulation (Klein, 1994; Papp et al., 1989; Pine et al., 1998b). Unlike for work on cognitive bias, respiratory dysregulation in panic disorder relates to the underlying risk for the condition (Gryell, 1997). Abnormalities in respiratory control are found in asymptomatic first-degree relatives of patients with panic disorder. Such abnormalities also characterize panic patients with particularly high family loadings for panic disorder.

In terms of startle regulation, the work by Davis and others reviewed previously relies on the startle reflex to delineate the role for precise neural structures in distinct fear states (Davis, 1998). The circuitry for this primitive reflex is preserved across phyla, and the magnitude of startle provides a conserved, reliable, easily quantifiable index of underlying neural activity. In humans, the effect of distinct manipulations on the startle reflex is thought to index the degree to which underlying brain circuits may place an individual at risk for specific learned or innate forms of fear associated with one or another anxiety disorder. In animals, startle is used during fear conditioning experiments to assess learned components of fear. Among humans, there is some evidence of abnormal fear learning both among adults with clinical anxiety disorders and among children of adults with clinical anxiety disorders (Grillon et al., 1991, 1997a, 1997b, 1998a, 1998b, 1999; Merikangas et al., 1999; Pine and Grun, 1999). In these groups of individuals, enhanced startle potentiation has been found in the context of a fear conditioning experiment. Similarly, in animals, startle also is used during an open-field test to index the degree of innate fear. Among humans (Merikangas et al., 1999), dark-potentiated startle provides an analogous index that also has been linked to both clinical anxiety disorders and to familial risk for anxiety.



Although research on startle, respiration, and autonomic physiology provides insights into underlying brain circuitry, work on neurochemical aspects of anxiety occupies a unique place in clinical research on anxiety. Extensive studies examine monoamine and GABA profiles in patients with various anxiety disorders. Although consistent abnormalities arise, this work provides no more compelling account than other psychobiological studies on the underlying physiology of clinical anxiety. However, unlike physiologic studies, neurochemical studies carry direct therapeutic implications. Various pharmacologic compounds exert parallel effects on anxiety states in humans and animals. These data strongly implicate regulatory dysfunction in brain monoamine and GABA systems in the pathophysiologic process of anxiety (Crestani et al., 1999; Malizia et al., 1998; Roy-Byrne, et al., 1996). As reviewed in chapters on therapeutics, there is consistent evidence that benzodiazepines, selective serotonin reuptake inhibitors (SSRIs), and tricyclic antidepressants alleviate many forms of clinical anxiety. As noted previously, such clinical effects show many parallels with human anxiety states. Research in this area may provide key insights into further integration between basic and clinical studies. Namely, recent basic science work has begun to distinguish different forms of fear among animals based on pharmacologic response profiles. This basic work shows interesting parallels with clinical syndromes. For example, both flight reactions to predators in rodents and spontaneous panic in humans are insensitive to diazepam, potentiated by acute SSRI treatment, and reduced by chronic SSRI treatment (Blanchard et al., 1997; Rodgers, 1997). As normal and pathologic fear states in animals are understood in increasingly precise detail, novel pharmacologic compounds may be uncovered that selectively alleviate specifically deranged components of one or another brain circuit. Documentation of pharmacologic parallels between fear states in animals and humans using existing compounds may facilitate the application of such novel agents to clinical syndromes.

## Imaging Studies

Neuroimaging studies provide one of the most exciting opportunities to document parallels in the underlying neurocircuitry associated with anxiety among humans and animals. Through imaging, structural and functional aspects of particular neural circuits can be evaluated across species, providing neuroanatomically informative research tools. A comprehensive review of neuroimaging studies in anxiety is beyond the scope of the current chapter. A brief summary of available techniques is provided, and findings are summarized that bear on research reviewed in other sections.

Neuroimaging techniques have been used primarily to evaluate three aspects of neural circuits implicated in anxiety: neurochemistry, brain structure, and functional activity. Neurochemistry has been evaluated using techniques that rely on radioactivity, including positron emission tomography (PET) and single-photon emission tomography (SPECT), as well as radiation-free techniques that rely on magnetic resonance spectroscopy (MRS). Although PET and SPECT techniques can assess a wide array of compounds, current MRS techniques are suitable only for evaluating relatively large compounds. GABA and glutamate emerge as two such potentially clinically relevant chemicals. Brain structure has been evaluated primarily with structural MRI (sMRI), and brain function has been evaluated either with PET or functional MRI (fMRI). For studies in children, the magnetic-based techniques of MRS, sMRI, and fMRI represent ideal research tools in that they are safe, relatively inexpensive, and reliable.

Many studies examining the relationship between neurochemistry and anxiety focus on GABA and monoamines. Despite some inconsistencies across studies, at least three groups find evidence in PET or SPECT studies of abnormalities in central nervous system GABA among patients with panic disorder (Kuikka et al., 1995; Malizia et al., 1998; Tiihonen et al., 1997). These findings are consistent with studies that examine behavioral effects of GABA manipulations, where there is evidence of an altered set-point in the GABA complex among patients with panic disorder (Roy-Byrne et al., 1992). Definitive PET/SPECT data on serotonin or norepinephrine are not available, although the availability of appropriate ligands should generate such data in the future. Studies examining brain structure in anxiety focus on limbic regions. There is some evidence of medial temporal lobe abnormalities in panic disorder, which may relate to studies among rodents implicating the amygdala and hippocampus in anxiety (Dantendorfer et al., 1996).

Perhaps the most extensive imaging data relevant to anxiety pertain to the relationship between acute anxiety and changes in brain activity. Initial research in this area demonstrated an inverted-“U”-shaped association between cerebral blood flow and levels of anxiety, reminiscent of the Yerkes-Dodson relationship between arousal and cognitive performance (Mathew et al., 1997). These findings may relate to more recent work from Dager et al. (1999) demonstrating elevated brain lactate in patients with panic disorder during lactate infusion. Elevations in brain lactate may reflect more extreme reductions in blood flow during panic, with effects on the balance between aerobic and anaerobic metabolism. Other studies using brain mapping techniques have detected one or another brain region that might be particularly susceptible to anxiety-associated changes in cerebral blood flow or glucose metabolism. These studies have used a range of techniques in various patient groups to induce anxiety, including fear conditioning experiments in healthy subjects (LaBar et al., 1998), lactate infusions in patients with panic disorder (Dager et al., 1999), and presentation of feared objects in patients with either specific phobias or obsessive-compulsive disorder (Mountz et al., 1989; Reiman, 1990). For these studies, changes in brain activity across diverse regions are correlated with changes in acute anxiety. Although these studies reveal considerable heterogeneity in the degree of activation across various brain regions, limbic structures in the temporal and frontal lobes are relatively consistently implicated. Finally, the most recent imaging studies focus on activation of neural structures implicated in fear among animals during procedures similar to those used in studies of fear among humans. Hence, a series of studies document amygdala activation during fear conditioning experiments in humans (LaBar et al., 1998). Similarly, basic science research implicates the amygdala in the processing of facial cues indicative of threat (Leonard et al., 1985; Rolls, 1984). Neuroimaging studies accordingly document amygdala activation in such processes (Fredrikson et al., 1995; Mountz et al., 1989; Rauch et al., 1997).

## HOW DO SYMPTOMS OF ANXIETY ARISE?

Studies reviewed previously document parallels in the behavioral, physiologic, and anatomic substrate of fear states in animals and humans. During acute fear states, humans and other animals exhibit similar behaviors, similar changes in physiology, and activation in similar brain systems. Moreover, specific and related forms of acute fear or anxiety states in humans and animals exhibit similar susceptibility to distinct pharmacologic manipulations. In clinical terms, anxiety disorders are conceptualized as abnormalities in the regulation of fear states. Such dysregulation might involve a low threshold for arousal of the brain systems involved in one or another form of fear, an inability to turn off these brain systems, or a failure of adaptive learning in these brain systems. This final section uses the parallels between anxiety states in animals and humans to summarize current views on the origins of human anxiety symptoms and disorders.

Anxiety disorders, like most common psychiatric disorders, represent complex conditions. This means that the disorders result from interactions among multiple risk factors and underlying predispositions. Although a comprehensive, accepted pathophysiologic model of anxiety does not exist, three conclusions have received relatively strong support. These conclusions most accurately summarize current views on the underlying origins of anxiety. These conclusions include the realization that (a) anxiety disorders develop against a back-drop of underlying preclinical vulnerability, (b) anxiety disorders are developmental conditions, and (c) environmental factors exert profound direct effects on the symptoms of anxiety. Data supporting these three conclusions are briefly summarized.

Evidence that anxiety disorders develop from an underlying vulnerability includes findings from studies reviewed previously documenting abnormal startle and respiration in healthy relatives of patients with anxiety disorders, as well as from studies on cognitive style or symptomatic precursors of anxiety, including anxiety sensitivity, negative affectivity, or neuroticism. The precise origin of such underlying predispositions remains unclear. Family studies consistently note a strong association between anxiety in parents and their children (Klein, 1995; Pine et al., 1999). Moreover, twin studies among both children and adults document a statistically significant genetic component to various forms of anxiety (Fyer et al., 1995; Klein, 1995; Morris et al., 1998). Finally, association studies for a polymorphism in the serotonin transporter gene provide some evidence of a genetic contribution to potential precursors of anxiety, such as neuroticism (Lesch and Mossner, 1998).

Despite such evidence of a genetic component, anxiety disorders in general involve a smaller genetic component but larger environmental component than typically observed in mood or psychotic disorders. From an environmental perspective, parents with anxiety disorders may use distinctive child-rearing practices that may affect risk for anxiety (Klein, 1995). As such, familial associations between parent and child anxiety could result either through direct effects from modeling of anxiety, effects of parent-child relationships on underlying predispositions for anxiety in children, or through interactions between parent-child relationships and a genetically predetermined diathesis.

A firm understanding of the diathesis for anxiety in humans of all ages requires studies focusing on children at risk for anxiety disorders. This requirement results from the second major tenet on the origins of clinical anxiety, concerning the developmental nature of anxiety in humans. Temperamental factors measurable during the first years of life, such as behavioral inhibition, predict later onset of anxiety disorders (Biederman et al., 1995; Kagan, 1995). Children with the temperamental construct of behavioral inhibition react to novel situations with signs of reticence or withdrawal. Based on peripheral physiologic data, behavioral inhibition is thought to result from an underlying hypersensitivity within amygdala-based neural circuits (Kagan, 1995). Behavioral inhibition is relatively common, found in approximately 15% of American children, and childhood anxiety disorders are even more common, with a general lifetime prevalence greater than 20%. Although most children with behavioral inhibition do not develop clinical anxiety disorders, behavioral inhibition does predict an elevated risk for childhood anxiety disorders, particularly social phobia (Schwartz et al., 1999). Similarly, although most children with anxiety disorders do not exhibit symptoms past adolescence, childhood anxiety disorders do predict adult disorders (Pine et al., 1998a). In fact, most adult anxiety disorders are preceded by an anxiety disorder during childhood. Taken together, such

developmental data suggest that childhood anxiety disorders or behavioral inhibition serve as constraining factors on adult outcomes. Children and adolescents who meet criteria for neither behavioral inhibition nor clinical anxiety disorders are unlikely to have an anxiety disorder as adults. A large proportion of children who do meet such criteria also are free of anxiety disorders as adults. However, among adults with anxiety disorders, most have met such criteria as children. This suggests that a key to understanding the origins of anxiety across the life span lies in gaining insights into factors that determine the degree to which childhood anxiety will remain chronic or transient.

The third major conclusion on the origins of anxiety relates to the role of environmental factors in anxiety disorders. Although compelling data suggest anxiety arises from an underlying diathesis, equally compelling data implicate environmental or contextual factors in anxiety. At an elemental level, fear conditioning experiments in animals demonstrate an effect of context on acute fear states (LeDoux, 1996, 1998). At a more complex level, many forms of stress, such as isolation or unpredictability in the stressor, affect the degree to which an organism reacts to punishment (LeDoux, 1996, 1998). For humans, as highly social organisms, social factors provide particularly compelling influences on fear states. Effects of social factors on fear states are found in both observational and experimental studies. For example, epidemiologic studies find that acute stress in the form of life events predicts an increase in anxiety symptoms over time, whereas twin studies document a large effect of the nonshared environment on risk for anxiety (Kendler et al., 1992). In experimental studies, manipulations of the social environment as well as of expectancies produce changes in acute anxiety reactions to stress (Francis et al., 1999). Moreover, clinical studies that rely on cognitive behavioral techniques document robust acute and long-term effects of experiential factors on anxiety symptoms. A central question to emerge from basic experimental research concerns developmental effects on associations between risk factors and overt manifestations of anxiety. As noted earlier, rearing manipulations in rodents and nonhuman primates can produce long-term alterations in behaviors and neurochemical factors related to anxiety. Although weaker, if any, effects usually result from environmental manipulations in adulthood, the precise shape of these associations across developmental epochs has not been determined. Moreover, clinical studies have only begun to consider the relevance of such basic science work to human anxiety disorders.

## CONCLUSIONS

This chapter reviews prior studies on the neurobiology of fear in animals and humans. Considerable parallels exist in these areas, possibly because of the strong selective pressure on organisms for development of brain circuits involved in perception of and reaction to danger. As modern neuroscience sheds light on the pathophysiologic processes of fear states in animals, the parallels with fear states in humans may ultimately provide important insights for predicting outcomes of anxiety and potential novel treatments.

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## 26 SYMPTOMS OF DEPRESSION

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Depressive syndromes in children and adolescents constitute a group of serious mental disorders with considerable risk for recurrence and subsequent psychosocial impairment with, in some cases, continuity into adult life ( [Birmaher et al., 1996](#); [Harrington and Dubicka, 2001](#) ). Using the American Psychiatric Association *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) clinical criteria ( [American Psychiatric Association, 1994](#) ) successfully identifies the same clinical syndromes in school-age children, adolescents, and adults. Most psychiatric studies have focused on two disorders, major depression and dysthymia, although there is an emerging literature on other affective syndromes, including depressive adjustment reaction, minor depressions, and, more recently, early-onset bipolar disorder ( [Geller et al., 2000](#); [Lewinsohn et al., 1995](#) ).

This chapter discusses the nature of clinical signs and symptoms that in combination result in detectable clinical syndromes. A developmental perspective is taken, examining the frequency, characteristics, and patterning of these symptoms at different ages in girls and boys in the first two decades of life.

### CLINICAL CONSTITUENTS OF DEPRESSION

All affective conditions, including the depressive component of bipolar disorder, share a core set of signs and symptoms that involve undesirable alterations of mood and feelings, thoughts, social behaviors, and physical functions. An essential feature is that of a change in mood from pleasant (euphoric) to unpleasant (dysphoric), even painful. This mood shift is experienced as relatively pervasive, persisting over time and place and sufficiently severe to interrupt everyday functioning. This negative mood state is accompanied by other sets of other features in varying combinations, including negative and distorted cognitions about the self, impaired concentration and attention, and an adverse alteration in a range of physical characteristics, eating, sleeping, energy, and activity. Symptoms usually are elicited at interview through direct questioning of the child or adolescent and his or her parents, with information occasionally obtained from other relatives, teachers, or close friends.

Signs are concurrent observations made by the interviewer about the respondent's general appearance and may include changes in facial expression, increases or decreases in physical activity, general care in dressing and personal hygiene, and diminished speed and fluency of speech. Interviewers also are expected to make a judgment about the subject's general awareness of his or her mental condition, recent and current behavior, and general personal care. This evaluation of the degree of insight a subject has into his or her well-being may have considerable implications for clinical management. For example, the ability of the patient to participate in voluntary treatment may be determined by the interviewer's evaluation of this feature. The extent to which parents, siblings, and the wider environment, such as peers and school personnel, are involved in clinical care also may be determined by the interviewer's judgment of insight and other person-specific characteristics such as motivation.

### DETECTING DEPRESSIVE SIGNS AND SYMPTOMS

The reliability and validity of diagnostic assessments have been the subjects of much investigation in university and research settings using instruments developed for research inquiries in both clinical and community populations ( [Hodges, 1993](#); [Kaufman et al., 1997](#) ). By contrast, little is known of the utility and efficacy of diagnostic procedures in routine clinical practice used throughout the world or the processes through which diagnoses are arrived at in different settings, such as private practice, health care clinics, multidisciplinary teams, or primary care. As a result, the sensitivity and specificity for the detection of mental disorders in child and adolescent health care settings by different professional groups are not known. Furthermore, the same language may be used to convey quite different things about the patient's current well-being. For example, the word *depression* may be used to describe a particular mood state only, a psychological dynamic or causal process, a syndrome of signs and symptoms, a categorical illness state, or even a disease with known pathophysiologic correlates or established etiologic mechanisms.

The focus of most research into affective disorders has been on the detection of syndromes obtained from interview procedures, often delivered by intensively trained research staff ( [Costello et al., 1996b](#); [Lewinsohn et al., 1993](#); [Verhulst and Koot, 1995](#) ). In large-scale epidemiologic studies, quantitative symptom counts of current low mood, negative thoughts, and physical changes obtained from self-report or parent questionnaires have been used as additional proxies for syndromes to improve the power of the investigation ( [Hewitt et al., 1997](#); [Silberg et al., 1999](#) ). In clinical studies where sample sizes of "real" cases are large enough (usually between 80 and 200), multivariate statistics have been applied to obtain distinctive factors or clusters of signs and symptoms as a test of the construct validity of depressive syndromes ( [Kolvin et al., 1991](#); [Ryan et al., 1987](#) ). Community and clinical studies also have paid considerable attention to the signs and symptoms reflecting other disorders accompanying the presence of major unipolar depression. The inclusion of comorbid syndromes has been used to delineate clinical subtypes of depression based on the pattern of diagnoses at presentation. Examples include double depression (dysthymia and subsequent major depression) ( [Kovacs, 1997](#) ), depressive conduct disorder ( [Harrington et al., 1991](#) ), and depressive obsessional disorder ( [Goodyer et al., 1997](#) ). For presenting depressive disorders in young people, comorbid syndromes are the rule rather than the exception, but their validity as clinical markers of etiologic differences within this group of conditions remains unclear ( [Angold et al., 1999](#) ).

### DEPRESSIVE SYNDROMES

The early work in adult populations was successful in delineating the characteristics of somatic (high levels of physical symptoms, weight loss, insomnia, early-morning waking, diurnal variation of mood, persistent feelings of guilt, hopelessness, and psychomotor retardation or agitation) as opposed to psychological depressions (anxious mood, initial insomnia, self-pity rather than self-blame, complaints of anorexia rather than weight loss). A series of criteria for the classification of depression was subsequently proposed by [Feighner and colleagues \(1972\)](#) at Saint Louis. Phenomenologic psychopathology remained the core construct guiding these criteria, with no dependency on putative causation. These suggestions formed the basis for subsequent refinements that led to Research Diagnostic Criteria for the diagnosis of depression ( [Spitzer et al., 1978](#) ). For these purposes, a diagnostic category had to be supported by two sets of mutually exclusive descriptive criteria: those that specify characteristics that lead toward making a diagnosis (inclusion criteria) and those that lead away from making the diagnosis (exclusion criteria). These principles form the basis of the current American Psychiatric Association's classification in the DSM-IV ( [American Psychiatric Association, 1994](#) ) and the International Classification of Diseases (ICD) sponsored by the World Health Organization ( [World Health Organization, 1994](#) ). The adoption of these systems has led to a method of classification that has satisfactory reliability between professional raters for signs, symptoms, and course of depressive disorder. The system has proven highly successful in generating a common language for the diagnosis of depressive disorders that makes findings comparable between investigations despite marked differences in the nature of the populations studied. This categorical approach to classification of depression is not without its detractors, however. For example, it is widely recognized that meeting inclusion criteria still allows for considerable variation in clinical presentation, including high levels of nondepressive symptoms, giving rise to marked individual differences in the descriptive psychopathology of depressions. With the main research emphasis on the quantity of depressive symptoms as predictors of subsequent onset of depression, little attention has been paid as yet to the relative importance of different types of symptoms or their salience to the subject for evolving a depressive disorder. In addition, many children and adolescents present with a range of symptoms that fall below inclusion

threshold for a diagnosis (too few or insufficient duration) but are of sufficient severity to cause personal impairment ( [Costello et al., 1996a](#)).

## Major Depression

According to the DSM-IV ( [American Psychiatric Association, 1994](#)), the diagnosis of major depressive disorder (MDD) requires establishing first the mandatory presence of lowered mood (dysphoria) together with four of eight other possible nonmandatory symptoms from the two broad domains of disordered cognitions and physical changes. These are shown in [Table 26.1](#).

Mood	Cognitive	Physical
Dysphoria or irritability in children	Anhedonia Feelings of worthlessness Inappropriate guilt Diminished ability to think or concentrate Recurrent thoughts of death or suicidal ideation	Weight change (includes a failure to make expected weight gains) Fatigue or loss of energy Psychomotor agitation or retardation

\*The diagnosis can be made only when five or more symptoms are present over a 2-week period. Dysphoric mood must be present.

**Table 26.1. Symptoms of Major Depression in Childhood Adolescence<sup>a</sup>**

It is essential that the five symptoms occur concurrently with each other over a minimum 2-week period. In children and adolescents, but not in adults, the entry criterion of lowered mood can be irritability. Young people often identify their own negative feelings with mood-valent words such as *gloomy*, *grumpy*, or *down*, and these should be used in interviews where there is uncertainty about the salience of the word *depressed*, such as with the prepubertal child. It is important to establish that these symptoms are not accounted for by the direct effects of substance misuse or a general medical condition, particularly one that involves known brain changes, because this reduces the likelihood of reliable and valid mental state assessments. In addition the symptoms should not be accounted for by recent bereavement.<sup>1</sup>

There are no requirements for a particular pattern of cognitions or physical symptoms. Despite the inference from adult studies that physical symptoms reflect a more sustained, severe, or even “biological” syndrome, there is no hierarchy or weighting in favor of these features in the DSM-IV classification system [but see the ICD-10 somatic depression subtype, which has been accorded special status because of the long-standing clinical belief that endogenous depression is a severe and potentially distinctive disorder ( [World Health Organization, 1994](#) )]. Equally, no distinction is made regarding the duration of these disorders, which, providing they have been present for at least 2 weeks, may vary in length for any period of time, even years. They also may differ in their severity or personal psychosocial impairment from mild, indicating only a modest deviation from normal behavioral functioning, to unable to care for oneself and requiring 24-hour intensive psychiatric care.

## Dysthymia

Dysthymia has been described as a chronic mood disturbance of young people characterized by long-standing gloom and dysphoria, brooding about feeling unloved, and affect dysregulation. The dominant negative cognition is self-deprecation or negative self-esteem. There are high rates of irritability and anger in everyday circumstances (i.e., occurring as a hyperemotional response to social hassles in the everyday environment) ( [Kovacs et al., 1994](#) ). According to the DSM-IV, dysthymia is a chronic depressive condition that in childhood or adolescence presents with the same general characteristic of lowered mood (dysphoria or irritability) as major depression but must have been present for at least 1 year or more. In addition to depressed mood, the subject must have two of a further six symptoms from the list shown in [Table 26.1](#) except that feelings of guilt and suicidal behavior are not included because they are for major depression. The implication is that the latter two symptoms are not found in dysthymic disorders and, if present, suggest that the patient is likely to be suffering from major depression.

The best clinical description comes from the work of [Kovacs and colleagues \(1994\)](#) based on children referred to mental health services. Compared with MDD, dysthymia is distinguished by the virtual absence and significantly lower prevalence of anhedonia and social withdrawal, and comparatively lower levels of guilt, morbid preoccupation, and impaired concentration. Practically none of the dysthymic children had reduced appetite, and few had hyposomnia or fatigue ( [Kovacs et al., 1994](#) ).

It is important to establish that the patient does not fulfill criteria for current major depression. If major depression has preceded the onset of dysthymia, then there must have been full remission of all depressive symptoms for at least 2 months before the development of dysthymia. By contrast, episodes of major depression can be superimposed on dysthymia disorder, in which circumstance both diagnoses can be given.

## Subthreshold and Minor Depressions

Aware of the limitations of coverage in the classification of mental disorders, both the DSM and ICD systems include criteria for a “not otherwise specified” category, for those cases not meeting full criteria for a specified syndrome. Despite this important caveat, there have been continual calls for the addition of new “disorders.” During the DSM process, over 150 different new disorders were proposed, with varying levels of evidence supporting their addition to the classification ( [Pincus et al., 1999](#) ). A review and synthesis of studies on minor depression and other “brand names” demonstrated that a myriad of names and definitions for subthreshold depression have been used in the literature, with varying duration, symptom thresholds, and exclusions. Of particular importance was the observation that to date little has been done to bridge the difference between clinical and primary care perceptions of what constitutes a mental disorder. There is little doubt that primary care physicians see individuals of all ages with distressing and dysfunctional mental states that are not well articulated in the psychiatric nosology and often are not a major consideration to mental health specialists ( [Pincus et al., 1999](#) ). Epidemiologic findings in childhood and adolescence also have shown that a significant number of young people between the ages of 6 and 18 years have current symptoms with psychosocial impairment that may warrant treatment, but do not meet criteria for a clinical diagnosis ( [Costello et al., 1996a](#) ). The natural history of these subthreshold conditions remains a matter for further research, ideally in longitudinal designs such that the temporal relationship between impairments and clinical status can be examined as an evolving rather than static process. What little has been done to date suggests that, from the public health perspective, it would be unwise to ignore subthreshold depressions (and indeed other subclinical syndromes) if they present with personal impairment ( [Costello et al., 1999](#) ). The evidence is that such young people are adding to the general burden of affective morbidity in the community at large and may continue to do so over time. Whether there are specific and particular continuities and discontinuities in signs and symptoms between subthreshold conditions and clinical disorders is not known.

## Somatic Depression

A second public health issue that impinges on the diagnostic status of depressions in young people are somatic complaints [not to be confused with the ICD-10 use of the term *somatic (endogenous) depression*, which is synonymous with physical depressogenic symptoms dominating the presenting picture]. In primary care settings, approximately 2% to 10% of children at any one time complain of aches and pains such as headaches, stomach aches, and limb pain, and, somewhat less frequently, tiredness or fatigue ( [Campo and Fritsch, 1994](#); [Garralda, 1996](#) ). In some of these children, this represents a physical presentation of psychiatric disorder. The prevalence of depressive illness in this somatic population is not known. There is no doubt, however, that in the 8- to 11-year-old age group in particular the main concurrent psychological features are internalizing emotional symptoms of anxiety and depression ( [Larson, 1991](#) ). These types of physical symptoms also are relatively common in children and adolescents presenting with depressive disorders, with prevalence estimates in clinical populations as high as 70% ( [McCauley et al., 1991](#) ). The current data are sparse, but it seems that physical symptoms such as abdominal pain, headache, limb pain, and malaise (which are not part of the diagnostic criteria for the depressions) may be an index of affective disorders in young people, even when comorbid anxiety disorders are taken into account. Whether they should be considered as potential components of a more developmentally sensitive set of diagnostic criteria is worth further consideration. In such circumstances, an adequate mental state examination often detects whether psychological or physical signs and symptoms of depression are present.



In the preschool child, for whom there are no really satisfactory criteria for mood disorders, somatic features may be particularly important indices of a current affective illness. The relation between somatic and depressive features deserves further inquiry in this younger population.

## DEPRESSIVE SYMPTOMS

### Patterning of Symptoms

When symptoms of depressive disorders obtained from psychiatric interview data of clinical populations have been subject to multivariate analysis, two components—negative cognitions, consisting of self-blame, hopelessness, low self-worth, and suicidal features, and biological “endogenous” features, consisting of dysphoria, anhedonia, fatigue, and psychomotor retardation—usually have been identified (Kolvin et al., 1991; Ryan et al., 1987). Anxious and conduct factors also have been obtained from these approaches, indicating the heterogeneity of affective disorder presentations. In the study by Ryan and colleagues (1987), suicidal ideation was associated with both negative cognitions and the conduct factor. Inspection of symptom frequency in depressed cases shows that whereas initial insomnia frequently is reported in childhood, terminal insomnia is less so (Carlson and Kashani, 1988; Kolvin et al., 1991; Ryan et al., 1987). Compared with adolescents, somatic symptoms are reported more frequently by younger depressed children, but loss of weight, diurnal variation of mood, and psychomotor retardation are less pronounced in youth than in adulthood (Cooper and Goodyer, 1993; Kolvin et al., 1991). Individual or combinations of symptoms that are rare or uncommon in the community but frequent in patients could be important indices of depressive conditions or those at risk for such conditions. By contrast, some symptoms may be so common in the community at large that they are unlikely to be specific enough to indicate at-risk populations. The importance of specific symptoms and their patterning for the detection of disorders thus depends on a knowledge of the base rates of signs and symptoms in the community in general.

In a community-based study of major depression in adolescent girls, a profile of depression, anxiety, and oppositional/conduct symptoms and disorders was obtained from 1,056 girls aged 11 to 16 years interviewed for the occurrence of one or more episodes of major depression (DSM-III-R criteria) over the previous 12 months (Cooper and Goodyer, 1993). Subjects were selected for interview on the basis of high scores (>80th percentile) on the mood and feelings questionnaire (MFQ), a self-report measure of depressive symptoms, together with a random sample of low and medium scorers. Three hundred sixty-eight girls (45 high scorers) were assessed within 2 weeks of completion of the self-report. A number of core clinical depressive features were estimated to have a prevalence of approximately 20% in this interviewed community sample, notably depressed mood, social withdrawal, and early insomnia. Depressive thoughts were estimated to be less common, with guilt and ideas of worthlessness found in less than 10%, but nihilistic ideas were found in 16%. Although the girls were selected for interview specifically on the sole basis of their self-reported depression scores, there also were anxious and behavioral symptoms. School phobia was particularly common (37%), as was anxiety associated with school attendance (20%). Other anxious symptoms were less common than depressive features with somatic symptoms (aches and pains), which occurred in 10% and were related in most cases to school attendance. A number of oppositional and conduct symptoms also were estimated and shown to be relatively common, with defiance to parents (29%) being markedly prevalent. Stealing, lying, vandalism, and defiance of school teachers all were found in just under 10% of this female sample.

The findings suggest that as many as one in four adolescent girls have differing types of depressive symptoms, some of which will be behavioral as well as anxious. Many of these would be deemed “in scope” for a clinical intervention based on DSM or ICD entry criteria, or reflect a cause for concern to primary care services. Estimates of the syndrome of (DSM-III-R) major depression in this sample showed that the 1-month prevalence was 3.6% and the 12-month prevalence 6.0%, very similar to other published reports (Angold and Costello, 2001). A further 8.9% and 20.7% were classified as subthreshold disorders at 1 and over 12 months, respectively, further demonstrating the potential public health difficulties regarding the targeting of the appropriate at-risk populations.

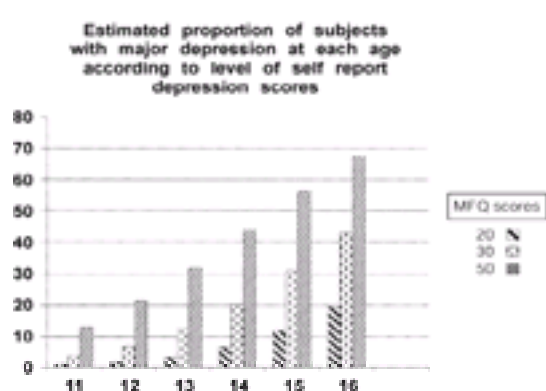
### Age Effects on Symptom Patterns

#### CHILDREN

Because the syndromes of major depression and dysthymia have been described in both prepubertal and in preschool children, high self-report depression scores may provide important clues regarding the detection or prediction of these disorders (Kovacs et al., 1994, 1997). In a study of 1,300 first-graders, the highest endorsed symptoms on a self-report measure were aches and pains (14.7% to 28.0%), followed by sad affect (13% to 17%) and then helplessness/hopelessness (12% to 25%) (Edelsohn et al., 1992). Although it is unclear if children of this age appraise all of these items with the same salience as adults, those with scores in the upper quartile showed considerable stability over a 4-month period. This stability of reporting included negative cognitions of the self and the future. Children as young as 5 years of age have a substantial set of emotions and can report the presence of both guilt (the belief that one has committed a wrongdoing leading to self-blame) and shame (the conscious feeling of humiliation brought about by actions of self or others) (Ferguson et al., 1999), and even preschoolers (especially girls) can manifest guilt-related beliefs and behaviors, including a heightened sense of empathy, a misplaced sense of responsibility, and unrealistic ideas about their own ability to produce or reduce another's plight (Zahn-Waxler and Kochanska, 1988). Stable high scorers also were more likely to show persistent difficulties in areas of personal and academic competence. This suggests that functional impairment and depressive symptoms can be both present and stable in childhood populations and in some cases may be “hidden from view” by somatic complaints (Kashani and Carlson, 1987; Kashani et al., 1983, 1986).

#### ADOLESCENTS

Somewhat similar concerns have been expressed about the nature of depressive symptoms in adolescence where, unlike childhood, persistent sad mood, especially with overt functional impairment, may be an indicator of an evolving depressive disorder (Compas et al., 1993). Increasing self-report depression scores occur across the adolescent age range, suggesting developmental influences on the occurrence of symptoms. For example, Fig. 26.1 shows the proportion of MDD at each MFQ decile point between 11 and 16 years in the community study described previously (Cooper and Goodyer, 1993).



**Figure 26.1.** The legend indicates three levels of mood and feelings (MFQ) self-report scores, 20, 30, and 50, from a possible range of 0 to 66, obtained from 1,056 girls aged 11 to 16 years. The estimated proportion of cases (y axis) is, as expected, greater for higher scores at each age. Importantly, the same level of symptom scores at each age estimates significantly different proportions of cases. This suggests developmentally sensitive differences in adolescent girls for the likelihood for detecting major depression from self-reports. (Data first published in Cooper PJ, Goodyer IM: A community study of depression in adolescent girls.1: Estimates of symptom and syndrome prevalence. *Br J Psychiatry*. 163:369–374, 1993.)

As age increases, high MFQ scores predict major depression with increasing specificity. For example, with an MFQ score of 30, 3.6% of 11-year-olds, 20.4% of 14-year-olds, and 42.9% of 16-year-olds would be cases of major depression (DSM-III-R criteria were used for this data set). With a score of 50 or more, 32.8% of 11-year-olds, 68.1% of 14-year-olds, and 84.1% of 16-year-olds would be such cases. The MFQ items reflect all symptoms that constitute depressive disorders, but there are at least three potentially crucial influences on the relation between scores and development:

1. It may be that some symptoms are consistently associated with major depression at different ages and developmentally insensitive. For example, taking an overview of all studies to date, negative mood (dysphoria and irritability), withdrawal from normal social activities, loss of energy, and early insomnia may be invariant features of current major depression across the school-age years.



2. Equally, because the same scores have such markedly different implications for the liability of depression at different ages, endorsement of some items may be highly dependent on individual differences in maturation in the child. Such items are likely to show increases in reporting with age. Here, the best candidate symptoms appear to be terminal insomnia, suicidal acts, and perhaps negative views about the world (but not necessarily negative thoughts about the self).
3. It also is likely that the salience of the same item is quite different for girls (and probably boys) of different ages. Thus, even if the symptom is endorsed to equal levels by girls between 11 and 16 years of age, the negative intensity and personal impairment of a given symptom may have different effects between ages. This more inferential assumption about salience rather than mere frequency of reporting, might refer particularly to making judgments about self-worth and self-efficacy (such as impaired scholastic and personal performance), impairments in attention and concentration, and also physical state such as motor agitation or retardation.

Although there is general agreement that development does influence endorsement of clinically relevant symptoms, these rather specific suggestions are speculative given the current knowledge base. Very little indeed is known about the interaction between maturation and the liability to endorsing particular symptoms on checklists in the school-age population at large. Such information would be most helpful in determining the base rate of symptoms at different ages for both sexes.

Might there also be developmentally sensitive issues regarding the reporting or endorsement of nondepressive symptoms in such populations? Returning again to the data from the previously mentioned study, one would conclude that this is likely, perhaps in much the same way as described previously for depressive symptoms. Thus, fears and phobias and oppositional behavior with parents look relatively invariant over this age range and may occur with broadly the same frequency and meaning between the ages of 6 and 19 years. With the increasing importance of peer group and school-based environments, both school reluctance and oppositional behavior in social circumstances may reflect a developmentally mediated set of symptoms increasing with age. Finally, endorsement of features such as lying, stealing, and argumentativeness may well have quite different meanings to the subject at different ages.

An interesting group of symptoms in community subjects are those regarding obsessional thinking and compulsive acts. Two percent of the aforementioned sample in the community study of girls reported obsessional thinking, and 8.2% obsessional acts. This makes the thinking relatively uncommon but actions relatively more common in this sample, particularly in relation to other nondepressive symptoms. Given that comorbid obsessive-compulsive disorder with major depression may index a somewhat more severe major depressive condition with decreased likelihood of short-term recovery, the relation between obsessional and depressive symptoms may be worth more detailed investigation ([Goodyer et al., 1997](#)).

Is it possible to use individual or particular combinations of symptoms in the community to identify evolving cases or young people at high risk for depressions? A preliminary multivariate examination of depressed cases among girls in the community suggests that three symptoms, hopelessness, insomnia, and mendacity, in combination are best predictors of current major depression compared with subthreshold cases ([Goodyer and Cooper, 1993](#)). Further examination to predict the presence of current major depression at different ages suggests that, at 11 to 12 years of age, depressed girls are particularly likely to report symptoms of hopelessness and reduced speech compared with age-matched control subjects; at 13 to 14 years, the best symptom predictors were depressed mood, concentration impairment, impaired school performance, guilt, and indecisiveness; and at 15 to 16 years, depressed mood and agitation were the best discriminators. These preliminary results demonstrate that there are developmentally sensitive patterns of symptoms associated with major depression in adolescent girls at different ages. Interestingly, even though hopelessness is a significant predictor of major depression, it appears most specifically associated with younger rather than older adolescent cases. Again, even though concentration impairment and agitation were equally endorsed by depressed girls across this age range, they appear specifically to predict depression only in cases older than 13 years of age. These findings are similar to those reported from the Oregon Adolescent Project, which noted considerable construct validity for the diagnosis of (DSM-III-R) major depression in adolescents, but also that symptom patterns may change across the adolescent age range and within subjects who report recurrent episodes of the disorder ([Roberts et al., 1995](#)).

Much remains to be done to confirm these early results that suggest that the current nosology, although necessary in many respects, is insufficient for detecting depressions in the younger adolescent and therefore even more possibly in the prepubertal child. There is, however, considerable core construct validity for the syndrome of major depression as currently constituted. The most frequent depressive symptoms are depressed mood, hopelessness, social withdrawal, agitation, and nihilistic ideas, all of which are likely to occur in greater than three-fourths of cases of major depressions at all ages across adolescents.

#### *Negative Cognitions, Suicidal Thoughts, And Actions*

A decrease in frequency with age for the self-reporting of the symptom of hopelessness, ranging from approximately 90% in 11- to 14-year-olds with major depression down to nearly 50% in 15- to 16-year-olds, is similar to the observation of [Kashani and colleagues \(1989\)](#) that there is no increase in the reporting of this core negative cognition between prepubertal childhood and adolescence. By contrast, in Cooper and Goodyer's community study, suicidal actions show an increase with age, with the handful ( $n = 5$ ) of depressive cases younger than 13 years of age exhibiting no suicidal acts, compared with 24% ( $n = 21$ ) of the 15- to 16-year-old depressives. The symptom of hopelessness also is a significant predictor of completed suicide among already depressed adults ([Brown et al., 2000](#)), although in younger populations this relationship remains unclear. Given the known correlation between suicidal intent and depression in adults and, to a lesser extent, in adolescents, the apparent inverse relationship between hopelessness and suicidal actions with age seems odd. Suicidal actions appear more likely in juveniles with a history of both dysthymia and major depression compared with depressive adjustment disorders and nondepressed cases ([Kovacs et al., 1994](#)). Whether this is because patients with these double depressions are specifically more likely to report greater levels of hopelessness compared with those with other affective disorders is not clear. Indeed, how negative views of the self evolve as cognitive schemas within a child, how they become translated into "symptoms," and the nature of their prospective relations with subsequent depressions with and without suicidal intentions and actions require much clearer understanding. This is not merely a theoretical issue but has practical implications for detecting young people at risk for suicide among those in the community predisposed to depressions.

A social cognitive model for depression has been proposed that postulates that dysphoric mood and dysfunctional thinking are more apparent among individuals with a previous history of depression, increasing the tendency to further episodes through biases in recall of negative thoughts about the self ([Teasdale and Barnard, 1993](#)). This notion suggests that memory is influenced by dysphoric moods in ways that increase the liability for negative self-referent material to be rehearsed and elaborated during subsequent depressive episodes ([Teasdale, 1999](#)). Using a prospective design, the Oregon Adolescent Depression Program reported that compared with adolescents with a history of first-episode depressions, those with a history of recurrent depression self-reported more depressive symptoms and higher levels of dysfunctional negative cognitions ([Lewinsohn et al., 1999](#)). This study provides the first evidence for the notion that depression is in reality "bad for the mind" through dysphoric effects in the long term on episodic memory and self-schema. The authors relied on self-report data to infer dysfunctional cognitions rather than establish if recurrent disordered subjects were more likely to recall negative views about themselves during induced negative mood. The findings therefore require replication using a similar longitudinal design but with the appropriate experimental procedures for mood induction and measures of self-description. Normal adolescents at risk for depression because of a more emotionally negative temperament also report increased negative cognitions about the self when in dysphoric mood compared with those not at risk in a similar negative mood state ([Kelvin et al., 1999](#)). This suggests that the pattern of negative cognitive symptoms in depressed adolescents may arise from vulnerabilities in emotion processing in a particular subgroup of adolescents. A prospective study of adolescents at risk for depression would determine if negative cognitive symptoms were, in fact, liable to precede the onset of first-episode depression. If so, then such symptoms would be potentially causal and not only the consequences of previous episodes of depression or dysthymia.

#### **Executive Functions, Physical Symptoms, and Depression**

A somewhat different model of recurrent disorder has been proposed by Post and colleagues which suggests that the role of physiologic processes inducing dysphoria and associated physical symptoms are more important in promoting recurrent than first episodes of depression ([Post, 1992](#); [Post et al., 1986](#)). The Oregon study did not contain sensitive measures of such symptoms over time, and therefore, it remains unclear whether there is a difference in the prevalence of physical and neuropsychological symptoms, such as impaired concentration, between first and recurrent episode depressions.

Indeed, although neuropsychological and physical symptoms, including impaired concentration, fatigue, agitation, and early insomnia, are reported as relatively frequent (>75% of cases) in both community and clinical studies, we know little of their origins or whether they carry specific effects for the natural history or treatment of major depression in young people. Although there is consistent evidence for various forms of memory difficulties in depressed patients ([Austin et al., 1999](#); [Kuyken and Dalgleish, 1995](#); [Teasdale, 1999](#)), it is not known whether specific neuropsychological deficits in working or declarative memory, or dysfunctions in attentional processing, are present before the onset of depressions, emerge as the nature of affective syndromes unfolds, or are direct consequences of attempts to deal with other existing prodromal depressive features. Part of the difficulty lies in merging two somewhat different research philosophies and strategies such that developmentally sensitive, experimentally based neuropsychological measures can be realistically applied to large enough samples of children and adolescents both in the community and in the clinic, preferably in longitudinal designs.

It also is unclear if subjects in whom depressive conditions develop have a greater liability for physical disruption of sleeping, eating, and energy before onset. No prospective studies have determined the attributable risk associated with a particular physical symptom or set of symptoms for the onset of juvenile forms of

depression. Thus, the relative contribution of the three domains of negative cognitions, executive dysfunction, and physical signs and symptoms to the onset of first-episode depression or the risk of recurrence for different forms of depression is unclear.

## SEX DIFFERENCES

Most research in affective disorders is on girls and women, and little in reality is known about depression in boys and men. Sex difference in rates of major depression from mid-adolescence through the seventh decade is one of the most robust findings in psychiatric epidemiology ( [Bebbington et al., 1998](#); [Piccinelli and Wilkinson, 2000](#)). Most clinical studies of sequential cases appear to have few or no sex differences in the size of their study populations or in the nature and characteristics of the disorders ( [Goodyer et al., 1997](#); [Mitchell et al., 1988](#)). This may reflect the impact of severity on the probability of referral, suggesting that the sex difference in prevalence in the community is due to there being a greater proportion of mild and moderate clinical depressions in women compared with men.

As yet, however, there is no concise explanation for the sex difference in childhood and adolescence, and little is known about the nature and characteristics of depressions between the sexes in young people.

In a prospective study of a group of 181 male (n = 68) and female (n = 103) adolescents aged 12 to 16 years at high psychosocial risk for psychopathology, there were 30 episodes of major depression (8 male and 23 female) over the 12-month follow-up period, giving a prevalence estimate of 17% overall for this population ( [Goodyer et al., 2000](#)). Determining if self-report MFQ scores at entry were associated with subsequent major depression for each sex revealed that of the 23 cases of major depression in girls, 14 (60%) were high scorers (>80th percentile of the MFQ, = 26 in this study from a range of 0 to 66); by contrast, for the 8 cases of major depression in boys, only 1 (12.5%) had a high MFQ score at entry. These findings suggest that sex, as well as age factors, influence the salience and validity of self-reported depressive symptoms as indicators of major depression. Compared with girls, boys' self-reports of depressive symptoms may be less sensitive in detecting or predicting major depression, at least in community adolescent populations.

## COVARIATION OF ANXIOUS AND BEHAVIORAL SYMPTOMS WITH DEPRESSIVE SYNDROMES

In a comprehensive review of 19 population studies of depressive disorders, [Angold and colleagues \(1999\)](#) reported high rates of comorbid anxiety disorders in nearly half of all such studies. Clinical studies of major depressions have invariably reported very high (up to 80%) estimates of concurrent nondepressive comorbid disorders, particularly anxiety syndromes ( [Birmaher et al., 1996](#); [Herbert et al., 1996](#)). School refusal is more strongly associated with depression than often is recognized ( [Kolvin, 2001](#)), whereas specific fears and phobias may be less so. Separation anxiety disorder appears more likely in younger depressive subjects, suggesting a potential developmental influence on the phenomenologic characteristics of clinical anxiety states accompanying depressive disorders. Anxiety disorders are not the only form of comorbid syndromes found in young people with major depression. Others include oppositional defiant disorder, conduct and dysthymic syndromes, and obsessive-compulsive disorder ( [Goodyer et al., 1997](#); [Kovacs, 1997](#); [Mitchell et al., 1988](#)).

A number of authorities have suggested that anxious states are likely to precede depressive disorders in school-age populations (e.g., [Compas et al., 1993](#); [Kovacs et al., 1989](#)). The cross-sectional findings reported to date cannot unravel the precedence of syndromes to each other. The notion that nondepressive syndromes of different types may precede the onset of depressive syndromes remains an important question in the natural history of the origins of affective illness in young people.

The nature and characteristics of nondepressive features may provide important clues concerning the etiology and outcome of depressive disorders that are clinically heterogeneous. Prospective community studies of the covariation between anxious and depressive symptoms in young people are beginning to appear in the literature, providing new insights into the natural history of emotional symptoms in the school-age years. For example, [Cole and colleagues 1996, \(1997\)](#) have monitored 330 elementary school children and their parents (n = 228) in a repeat-measures design over 3 years, examining the temporal relationship between anxiety and depressive symptoms. Individual differences in the measures of depression and anxiety proved remarkably stable ( [Cole et al., 1998](#)). The findings did not imply that individual levels of anxious and depressive symptoms did not change, but rather that each child's position remained stable in the ranking in relation to others in the sample. Importantly, high scores from self- and parent-reported anxiety scores predicted increases in self- and parent-reported depression scores over time. By contrast, high levels of self- and parent-reported depression scores did not predict increases in anxiety over time. These findings support the hypothesis that anxiety symptoms precede those of depression. Cole and colleagues also showed that high self- and parent-report scores in depression did not predict increases in anxiety and, as a consequence, suggested that increases in depressive symptoms was a precursor to reductions in subsequent anxiety symptoms.

Despite the well-described association between conduct symptoms and depression in child and adolescent populations, there are no reported studies of the nature of the covariation over time between these symptoms in the child or adolescent age range.

Prospective studies of both emotional and behavioral symptom profiles of children and adolescents at high risk for affective psychopathology followed through the adolescent period of risk for onset of major depression (13 to 16 years) would substantially contribute to our understanding of how symptom patterns evolve, organize, and relate to each other. They also would be able to delineate the symptom combinations in the childhood years that are most associated with the onset of subsequent affective psychopathologies in juveniles.

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## 27 DEVELOPMENT OF PSYCHOTIC THINKING IN CHILDREN

Rochelle Caplan, M.D., and Peter Tanguay, M.D.

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### Historical Overview

#### Formal Thought Disorder

##### Definition

##### Clinical Assessment Studies of Formal Thought Disorder in Childhood

##### Etiology and Pathogenesis

##### Differential Diagnosis of Formal Thought Disorder in Children

##### Conclusions

### Hallucinations

#### Definition

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### Delusions

#### Definition

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##### Chapter References

## HISTORICAL OVERVIEW

Impaired thinking or thought disorder has been regarded as the hallmark of schizophrenia since the first descriptions of this illness by [Bleuler \(1951\)](#). Bleuler regarded loosening of associations as a fundamental sign that represents the pathologic process of schizophrenia. In contrast, he regarded hallucinations and delusions as accessory signs that were secondary to the pathologic process of schizophrenia.

From the clinical perspective, earlier theories on thought disorder were all-encompassing and tried to explain thought disorder as a unitary concept ( [Holzman et al., 1986](#)). Since the late 1970s, several studies have demonstrated that thought disorder is not a unitary concept and does not occur specifically in schizophrenia ( [Andreasen and Grove, 1986](#); [Butler and Braff, 1991](#); [Grossman and Harrow, 1996](#); [Holzman et al., 1986](#); [Marengo and Harrow, 1997](#); [Sax et al., 1995](#); [Serper, 1993](#)).

Introduction of the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) ( [American Psychiatric Association, 1980](#)) and DSM-III-R ( [American Psychiatric Association, 1987](#)) provided further clarification of terms by clearly differentiating between disturbances in the form and content of thought. Thus, formal thought disorder describes the clinical manifestations of the manner in which patients present their thoughts to the listener ( [Fish, 1962](#)) and the organization, control, and processing of thoughts ( [Holzman et al., 1986](#)). Delusions, a thought content disturbance, refer to firmly held personal beliefs that are based on incorrect inference about external reality in spite of obvious proof or evidence to the contrary ( [Butler and Braff, 1991](#)).

Unlike earlier studies, the clinical studies that have been conducted since the late 1970s have used reliable and valid instruments for the assessment of well-defined aspects of thought disorder ( [Aloia et al., 1998](#); [Andreasen and Grove, 1986](#); [Docherty and Gordinier, 1999](#); [Goldberg et al., 1998](#); [Harvey and Serper, 1990](#); [Holzman et al., 1986](#); [Mesure et al., 1998](#); [Nestor et al., 1998](#)). These studies have characterized the positive and negative dimensions of formal thought disorder in schizophrenic adults, as well as their relationship with clinical symptoms ( [Andreasen et al., 1990](#)), cognitive/attentional deficits ( [Braff et al., 1991](#); [Cornblatt et al., 1985](#); [Nestor et al., 1998](#); [Nuechterlein et al., 1986](#)), linguistic deficits (see review in [Kuperberg et al., 1998](#)), treatment responses ( [Spohn et al., 1986](#)), and biological measures ( [Fukuzako et al., 1996](#); [Holinger et al., 1999](#); [McGuire et al., 1998](#); [Menon et al., 1995](#); [Shenton et al., 1992](#); [Vita et al., 1995](#)).

In contrast, very few studies were conducted on thought disorder in childhood before the DSM-III. The DSM-III made two important contributions to this field of research. First, it put an end to the historical debate over the relationship between infantile autism and schizophrenia ( [Eisenberg and Kanner, 1956](#); [Fish and Ritvo, 1978](#); [Kanner, 1957](#); [Kolvin et al., 1971](#); [Makita, 1966](#); [Rutter, 1972](#); [Rutter and Lockyer, 1967](#); [Rutter et al., 1967](#)). Second, the DSM-III provided well-defined inclusionary and exclusionary criteria for schizophrenia. Use of these criteria stopped the practice of labeling a variety of clinical manifestations, such as impaired social relationships, language deviance, disturbed reality testing, delusions, and abnormal stream and form of thought as thought disorder. In addition, investigators have become cognizant of the impact of developmental factors on the methods for eliciting and coding thought disorder in childhood ( [Arboleda and Holzman, 1985](#); [Caplan et al., 1989, 1990a, 1992a, 2000](#); [Makowski et al., 1997](#); [Tompson et al., 1990, 1997](#)). As a result, research on schizophrenic children diagnosed based on DSM-III, DSM-III-R, and DSM-IV criteria followed a similar trajectory to that on schizophrenic adults, albeit at a slower pace.

This chapter includes three sections that describe three aspects of psychotic thinking in children: formal thought disorder, hallucinations, and delusions. Each of the chapter's three sections reviews studies on the clinical assessment, underlying mechanisms, and differential diagnosis of formal thought disorder, hallucinations, and delusions, respectively, in childhood-onset schizophrenia.

## FORMAL THOUGHT DISORDER

### Definition

Formal thought disorder represents clinical measures of the form or manner in which the patient presents his or her thoughts to the listener. Numerous clinical signs have been used to describe these communicative characteristics, such as illogical thinking, incoherence, loose associations, digressive speech, circumstantiality, tangentiality, vague speech, overelaborate speech, clanging, neologisms, poverty of speech, poverty of content of speech, echolalia, and others ( [Andreasen, 1979](#)).

The DSM-IV ( [American Psychiatric Association, 1994](#)) uses the term *disorganized speech* (e.g., frequent derailment or incoherence) rather than *formal thought disorder*. The DSM-III-R ( [American Psychiatric Association, 1987](#)) required the presence of loose associations or incoherence to diagnose formal thought disorder. The DSM-III ( [American Psychiatric Association, 1980](#)) included four formal thought disorder signs: illogical thinking, loose associations, incoherence, and poverty of content of speech.

### Clinical Assessment Studies of Formal Thought Disorder in Childhood

A review of the literature demonstrates a dearth of studies on formal thought disorder in childhood schizophrenia and schizophrenia spectrum disorders ( [Arboleda et al., 1985](#); [Cantor et al., 1982](#); [Caplan et al., 1989, 1990a, 1992a, 2000](#); [Caplan, 1994a](#) and [1994b](#); [Green et al., 1984](#); [Kolvin et al., 1971](#); [Russell et al., 1989](#); [Tompson et al., 1990, 1997](#)) and one study on children at risk for schizophrenia ( [Arboleda and Holzman, 1985](#)). Only three groups of researchers have used reliable and valid instruments for the clinical assessment of formal thought disorder in children ( [Arboleda and Holzman, 1985](#); [Caplan et al., 1989, 1990a](#); [Tompson et al., 1990, 1997](#)).

[Kolvin et al. \(1971\)](#) reported that 60% of children with late-onset psychosis had disorder of association, 45% had derailment of thought, and 51% had talking past the point as described by [F.J. Fish \(1962\)](#). The criteria for assessing formal thought disorder, however, were not operationalized for use with children. Cantor identified formal thought disorder signs, such as loose associations, neologisms, illogicality, clanging, poverty of speech, and poverty of content of speech, in both the early-onset and adolescent-onset groups of children with schizophrenia ( [Cantor et al., 1982](#)). Like Kolvin, Cantor did not use a reliable and valid instrument for assessing formal thought disorder in these children. In addition, she did not clarify how she operationalized these formal thought disorder signs for the younger group.



[Green et al. \(1984\)](#) reported that all the schizophrenic children in their sample fulfilled the DSM-III schizophrenic inclusionary criterion for formal thought disorder signs. Because the clinical characteristics of the schizophrenic children in this study were retrospectively obtained from medical charts, it is not clear how the DSM-III formal thought disorder signs were operationalized in this sample of children aged 6 years, 7 months to 11 years, 11 months.

[Russell et al. \(1989\)](#) made reliable global clinical ratings of the DSM-III criteria of formal thought disorder from the Interview for Childhood Disorders and Schizophrenia. These investigators reported that 40% of their schizophrenic sample exhibited incoherence or marked loosening of associations, illogical thinking, or poverty of content of speech accompanied by affect disturbance, delusions, hallucinations, or disorganized behavior ([Russell et al., 1989](#)).

From the developmental perspective, normal children learn to present the listener with their thoughts in a logical and coherent fashion from the toddler period through adolescence ([Shatz, 1982](#)). Formal thought disorder, a clinical measure of the way the child presents his or her thoughts to the listener, involves the maturation of cognitive, linguistic, and pragmatic skills ([Caplan, 1994a](#)). Two studies have addressed the relationship between age and formal thought disorder in childhood.

Using the Thought Disorder Index (TDI), [Arboleda and Holzman \(1985\)](#) conducted the first study that demonstrated the importance of controlling for the level of cognitive development when assessing thought disorder in normal children younger than 10 years of age. This also was the first study to use a formal psychological test with suitable control groups to assess thought disorder in psychotic and high-risk children.

The TDI has demonstrated reliability and validity in schizophrenic, manic, and depressed adults and in their first-degree relatives ([Holzman et al., 1986](#)). It codes 22 categories of verbal responses to the standardized percepts on the Rorschach cards as associative, combinatorial, disorganized, and unconventional verbalizations ([Holzman et al., 1986](#)). The TDI therefore examines a broader range of thought disturbance categories than the DSM-IV (i.e., derailment and incoherence), DSM-III-R (i.e., loosening of associations and incoherence), and DSM-III formal thought disorder signs (i.e., loose associations, illogical thinking, incoherence, and poverty of content of speech). [Arboleda and Holzman \(1985\)](#) found that children with psychosis and psychotic spectrum disorders and those at risk for schizophrenia and affective disorder had more severe thought disorder levels than normal children and children with nonpsychotic psychiatric diagnoses. Using the TDI and the Family Consensus Rorschach Task ([Loveland et al., 1963](#)), [Tompson et al. 1990](#), ([1997](#)) demonstrated that schizophrenic and schizotypal children had significantly more thought disorder and disturbed attention than depressed children and normal children.

In an earlier study, [Asarnow et al. \(1988\)](#) also described a relationship between low Wechsler Intelligence Scale for Children—Revised distractibility factor scores and communication deviance in the parents of children with schizophrenia and schizotypal personality disorder, but not with depression and dysthymia. From the developmental perspective, the findings of [Asarnow and colleagues \(1988\)](#) suggest that the thought disorder of schizophrenic children also might reflect an attentional impairment in both the parents and child, with difficulty establishing a shared focus of attention.

More recently, [Makowski et al. \(1997\)](#) found significantly more idiosyncratic word use, illogical reasoning, perceptual confusion, loss of realistic attunement to the task, and loosely related ideas in the TDI responses of 20 schizophrenic adolescents compared with 12 adolescents with psychotic depression, 34 nonpsychotic adolescents in an inpatient psychiatric unit, and 29 adolescents in a general hospital unit with nonlife-threatening medical illness. These findings highlight that thought disorder is found in both the adolescents with schizophrenia and those with psychosis and depression. The thought disorder of the two groups differed, however, with bizarre content characterizing the speech of the schizophrenic subjects.

[Caplan et al. \(1989\)](#) developed a play procedure, the Story Game, and a coding system, the Kiddie Formal Thought Disorder Scale (K-FTDS), for the assessment of formal thought disorder in middle childhood. Compared with a structured psychiatric interview, the Story Game elicited adequate speech samples that could be reliably rated for formal thought disorder in children ([Caplan et al., 1989](#)). The K-FTDS operationalized the four DSM-III signs of formal thought disorder—illogical thinking, loose associations, incoherence, and poverty of content of speech—so that they could be coded even if children spoke in small paragraphs of one to two utterances.

A study conducted on 88 schizophrenic and 190 normal children demonstrated that illogical thinking and loose associations are reliable, valid, sensitive, and specific measures ([Caplan et al., 2000](#)). Comparison of schizophrenic, schizotypal, and normal children indicates that loose associations occur almost specifically in the schizophrenic and schizotypal children ([Caplan, 1994a](#) and [1994b](#); [Caplan et al., 1989, 1990a, 2000](#)). There are no significant differences in the illogical thinking and loose associations scores of the schizophrenic and schizotypal children ([Caplan, 1994b](#); [Caplan et al., 1990a](#)).

From the developmental perspective, normal children younger than 7 years of age have illogical thinking and loose associations ([Caplan et al., 2000](#)). The young schizophrenic and normal children have significantly higher illogical thinking and loose association scores than the older children with these respective diagnoses ([Caplan et al., 2000](#)).

## Etiology and Pathogenesis

Examination of the relationship of well-defined clinical measures of thought disorder with attention/information processing ([Harvey and Serper, 1990](#); [Nestor et al., 1998](#); [Nuechterlein et al., 1986](#)), linguistic/pragmatic (see review in [Kuperberg et al., 1998](#)), and biological measures ([Fukuzako et al., 1996](#); [Holinger et al., 1999](#); [McGuire et al., 1998](#); [Menon et al., 1995](#); [Shenton et al., 1992](#); [Vita et al., 1995](#)) has been an important step in defining and delineating the components of thought disorder in schizophrenic children. The pathogenesis of formal thought disorder in adult schizophrenia nevertheless remains unclear.

In terms of attention/information processing, negative signs of formal thought disorder have been associated with poor performance on cognitive tasks that demand effortful recruitment of information processing ([Nuechterlein et al., 1986](#)), and working memory deficits ([Nestor et al., 1998](#)). Positive signs of formal thought disorder, however, are related to distractibility ([Cornblatt et al., 1985](#); [Harvey et al., 1986](#)).

Studies in schizophrenic adults with thought disorder highlight deficits in the use of linguistic context to process and produce speech (see review in [Kuperberg et al., 1998](#)), cohesive devices to refer to people, objects, and events ([Docherty and Gordinier, 1999](#); [Harvey et al., 1986](#); [Harvey and Serper, 1990](#)), semantic categorization ([Mesure et al., 1998](#)), and structure of semantic knowledge and network ([Paulsen et al., 1996](#)), as well as aberrant automatic spread of activation or facilitation in semantic networks ([Aloia et al., 1998](#); [Goldberg et al., 1998](#); [McCarley et al., 1999](#); [Weisbrod et al., 1998](#)). The findings of these studies suggest that schizophrenic adults have both receptive and expressive difficulties involving the ability to organize and monitor incoming and outgoing speech (i.e., higher-level linguistic functions). However, there is no evidence for impairment in linguistic functions, such as syntax, in adult schizophrenia (see review in [Kuperberg et al., 1998](#)).

From the biological perspective, several imaging studies have demonstrated involvement of the frontal ([Liddle et al., 1992](#); [Vita et al., 1995](#)), temporal ([Fukuzako et al., 1996](#); [Holinger et al., 1999](#); [Liddle et al., 1992](#); [Menon et al., 1995](#); [Shenton et al., 1992](#)), and anterior cingulate regions ([Liddle et al., 1992](#); [McGuire et al., 1998](#)) in the thought disorder of schizophrenic adults. [Andreasen and Grove \(1986\)](#) proposed that negative formal thought disorder signs of schizophrenic adults, such as poverty of content of speech, are akin to the alogia (i.e., difficulty generating organized speech) found in patients with frontal lobe disease.

Similar to the research on thought disorder in adult schizophrenia, research on the etiology and pathogenesis of formal thought disorder in childhood schizophrenia has included studies on attention/information processing ([Asarnow et al., 1994](#); [Karatekin and Asarnow, 1998](#)), linguistic/pragmatic deficits ([Caplan, 1994a](#) and [1994b](#); [Caplan et al., 1992a, 1996, 2000](#)), and possible biological correlates of thought disorder ([Caplan et al., 1992b, 1993, 1994, 1997](#), in press) in schizophrenic children.

From the *cognitive* perspective, [Caplan et al. \(1990a\)](#) found that illogical thinking and loose associations are related to different aspects of impaired attention/information processing in children with schizophrenia spectrum disorder. Illogical thinking appears to be related to a deficit in momentary processing capacity, measured by the partial report span of apprehension task ([Caplan et al., 1990b](#)), and to the schizophrenic child's difficulty with screening out extraneous stimuli and focusing on the task at hand ([Caplan et al., 1990c](#)). The requirement to present the listener with logical reasoning might tax these cognitive skills in the schizophrenic child ([Caplan, 1994a](#) and [1994b](#)).

In the case of loose associations, the child unpredictably changes the topic of conversation without preparing the listener for the topic change. Loose association is associated with distractibility ([Caplan et al., 1990b, 2000](#)). Thus, the cognitive demands of this situation appear to be different from those eliciting illogical thinking ([Caplan, 1994a](#)).

From the *linguistic/pragmatic* perspective, as demonstrated in schizophrenic adults ([Docherty and Gordinier, 1999](#); [Harvey, 1983](#); [Harvey and Serper, 1990](#)),

schizophrenic children underuse discourse devices that link clauses (sentences) and establish coreference, such as conjunctions and referential cohesion ( [Caplan et al., 1992a, 2000](#)). They make reference to people, objects, or events without previously identifying these referents (i.e., unclear and ambiguous reference). Schizophrenic children also repeatedly disrupt the ongoing conversation to refer to the immediate surroundings (i.e., exophora). This difficulty focusing on the conversational text is associated with illogical thinking and loose associations, unlike other aspects of the discourse deficits of these children ( [Caplan, 1994a](#) and [1994b](#); [Caplan et al., 2000](#)).

Schizophrenic children use fewer self-initiated strategies of repair for on-line correction of errors in conversation involving the organization of ideas or the connections between these ideas across sentences ( [Caplan et al., 1996](#)). In terms of the organization of ideas, they use fewer false starts (i.e., an incomplete clause that begins, but does not complete an idea) and fillers (i.e., words that fill pauses without changing the meaning) compared with normal children. Regarding links between ideas of contiguous sentences, they make fewer clarifications of the referents they use to identify people, objects, or events. Underuse of these repair strategies is associated with loose associations and distractibility ( [Caplan et al., 1996](#)). Thus, in addition to impaired organization and processing of thoughts captured by measures of formal thought disorder, the listener has difficulty following who and what the child is talking about because of abnormal use of linguistic devices that tie together ideas expressed across sentences and impaired correction of online errors in conversation.

Finally, from the *biological* perspective, as demonstrated in adults with complex partial seizure disorder ( [Csernansky et al., 1988](#); [Perez et al., 1985](#); [Slater and Beard, 1963](#); [Taylor and Marsh, 1979](#)), a schizophrenia-like psychosis is found in children with this form of epilepsy ( [Caplan et al., 1991, 1992b, 1997, 2000](#); [Lindsay et al., 1979](#)). In search of a possible biological model of formal thought disorder in childhood-onset schizophrenia ( [Caplan et al., 1992b, 1993, 1994](#)), this association has led to several studies of thought disorder (i.e., formal thought disorder and discourse deficits) in children with complex partial seizure disorder.

The following findings suggest that illogical thinking in middle childhood might reflect frontal lobe dysfunction ( [Caplan et al., 1993, 2001](#), in press b). First, children with complex partial seizure disorder and electroencephalographic evidence for frontotemporal involvement used significantly more illogical thinking and fewer cohesive ties and self-initiated strategies of repair than those without frontal lobe involvement ( [Caplan et al., 1992b, 1994, 1997, 2001](#), in press b). Second, follow-up of seven children who attained seizure control after temporal lobectomy for intractable complex partial seizures revealed postoperative normalization of their high illogical thinking scores ( [Caplan et al., 1993](#)). Seizure control after temporal lobectomy might therefore reduce frontal lobe dysfunction and the illogical thinking found in these children. This hypothesis is based on the propagation patterns of temporal lobe seizures with spread first to the ipsilateral and contralateral frontal lobes and then to the contralateral temporal lobe ( [Lieb et al., 1982](#)), and on intraoperative electrocorticography evidence for functional frontal abnormalities in more than half the patients who undergo temporal lobectomy ( [Mizrahi et al., 1990](#)).

In summary, illogical thinking and loose associations represent different attentional and linguistic impairments in childhood-onset schizophrenia. Preliminary evidence suggests that illogical thinking might reflect frontotemporal dysfunction in the child.

### **Differential Diagnosis of Formal Thought Disorder in Children**

#### *PSYCHOTIC DISORDERS*

Schizophrenic adolescents have both positive (loose associations) and negative formal (i.e., poverty of content of speech, illogical thinking) thought disorder signs. Although infrequent, incoherence sometimes is found in the severely ill adolescent patient. The formal thought disorder manifestations of adolescent manic patients include loose associations, distractibility, tangentiality, and digressiveness, together with pressure of speech. As previously mentioned, despite high levels of thought disorder, the formal thought disorder of adolescents with psychotic depression differs from that of schizophrenic adolescents in the absence of bizarre content ( [Makowski et al., 1997](#)). With the exception of brief episodes of disorganized thinking, children with dissociative disorder have no formal thought disorder ( [Hornstein and Putnam, 1992](#)).

In the child with organic psychosis, the severity of formal thought disorder is related to the severity of global cognitive impairment ( [Caplan et al., 1987, 1992b](#)). Severe incoherence and fragmentation of utterances usually are indicative of "organic" formal thought disorder. Whereas the clinical manifestations of formal thought disorder wax and wane in the child with nonorganic psychosis, they tend to be pervasive in the child with organic psychosis ( [Caplan et al., 1987](#)). Children with complex partial seizure disorder and a schizophrenia-like psychosis have illogical thinking, but no loose associations ( [Caplan et al., 1991](#), in press b).

#### *PERSONALITY DISORDERS*

According to the DSM-IV, patients with a diagnosis of schizotypal personality disorder meet criteria for odd speech, but not for formal thought disorder. However, a sensitive and specific instrument, such as the K-FTDS, can detect formal thought disorder not otherwise identified by structured psychiatric interviews in schizotypal children ( [Caplan et al., 1990a](#)). Although schizotypal children have lower illogical thinking and loose associations scores than schizophrenic children, these differences are not statistically significant ( [Caplan et al., 1990a](#)).

#### *DEVELOPMENTAL DISORDERS*

Children with these disorders might have formal thought disorder secondary to their cognitive or language delay. Because normal children with a mental age under 7 years have illogical thinking and loose associations ( [Caplan et al., 1989, 1990a, 2000](#)), children with mental retardation might exhibit illogical thinking or loose associations by virtue of their low mental age. Evidence for generalized cognitive and developmental delay and the absence of other signs of psychosis would suggest a diagnosis of mental retardation.

Children with specific expressive language disorders and with developmental aphasia have deficits in the social use of language (i.e., pragmatics). Clinically, therefore, their speech could meet criteria for formal thought disorder. The presence of a specific language disorder without other signs of psychosis would support a diagnosis of a specific expressive disorder.

Because of their impaired communication skills, children with pervasive developmental disorder not otherwise specified also could meet criteria for formal thought disorder ( [Van der Gaag, 1993](#)). The presence of a qualitative impairment in reciprocal social interaction and verbal and nonverbal communication, as well as a restricted repertoire of activities and interests, will guide the clinician toward the pervasive developmental disorder diagnosis.

### **Conclusions**

Illogical thinking and loose associations are reliable and valid measures of formal thought disorder in middle-childhood schizophrenia spectrum disorders. Reliable clinical assessment of formal thought disorder involves the use of specific instruments for eliciting speech samples from children (e.g., Rorschach cards, the Story Game) and for measuring formal thought disorder (e.g., the TDI, the K-FTDS). Based on developmental data, 7 years of age is the cutoff point for normal developmental changes in these measures. Different attention/information processing and linguistic/pragmatic deficits underlie illogical thinking and loose associations in schizophrenic children. Underlying pathogenetic mechanisms might involve the frontal/frontotemporal areas.

### **HALLUCINATIONS**

#### **Definition**

The hallucinatory experience has been defined variously as a "false sensory perception not associated with real external stimuli" ( [Kanner, 1957](#); [Kaplan and Sadock, 1985](#)) and as "an apparent perception of an external object when no such object is present." Hallucinations may involve any of the senses and are to be distinguished from vivid eidetic imagery, intensified images or sensory impressions, fantasy productions, and imaginary companions. Hallucinations that occur while falling asleep or waking (hypnagogic and hypnopompic hallucinations) often are categorized as distinct from hallucinations that occur in the fully awake state. Hallucinations may be more or less vivid.

#### *OCCURRENCE*

Hallucinations appear to be rare in children younger than 6 or 7 years of age. [Rothstein \(1981\)](#) reviewed 21 studies published between 1931 and 1975 of children



who experienced auditory or visual hallucinations. The definition of hallucinations and the quality of the behavioral observations varied across these studies. [Rothstein \(1981\)](#) concluded that hallucinations in children were not pathognomonic of psychosis. They were found in normal children suffering from anxiety states, in transient situational responses, in deprived children with personality disorders, and in schizophrenic children. The definition of "schizophrenia" in a number of the studies reviewed by [Rothstein \(1981\)](#) differed from that found in the DSM-III, DSM-III-R, and DSM-IV.

Additional studies of the prevalence of hallucinations in children treated in child and adolescent psychiatry services confirm that hallucinations are not pathognomonic for schizophrenia and that they are infrequent in the young child. [Garralda \(1984\)](#) reviewed the case records for 4,767 children seen at the Children's Department of the Maudsley Hospital between 1954 and 1966. None of the 1.1 % of the children noted to have hallucinations on admission were younger than 8 years of age.

[Kempf \(1987\)](#) examined the hospital records of 331 children admitted to a public psychiatric hospital and diagnosed psychotic. Of the 171 children with hallucinations, three were younger than 8 years of age, and none of the children were younger than 6 years of age. It is unclear if age 6 years was the cutoff age for admission to the hospital in this study.

[Egdell and Kolvin \(1972\)](#) reported hallucinations in only two of 40 inpatients between 5 and 15 years of age who had a variety of psychiatric disorders, excluding psychosomatic conditions. The ages of the two patients were not given. [Kotsopoulos et al. \(1987\)](#) also studied the nature of hallucinations in a population of child psychiatry outpatients. Eleven children (of an approximate sample of 100) were reported to hallucinate, all of them older than age 7 years.

[Kolvin et al. \(1971\)](#), [Green et al. \(1984\)](#), [Russell et al. \(1989\)](#), and [McKenna et al. \(1994\)](#) have all reported finding hallucinations in substantial portions of children having "late-onset psychosis" or DSM-III schizophrenia. The rate of hallucinations in these studies was similar, with 80% or more of the children having hallucinations. Even in this highly selected sample, very few children younger than 8 years of age could be identified as having hallucinations.

[Ulloa et al. \(2000\)](#) found psychotic symptoms in 4.5% of 2,031 children referred to a mood and anxiety disorder clinic. Among these children, 80% had hallucinations, mainly auditory, and there was a diagnosis of bipolar disorder in 24%, major depression in 41%, subsyndromal depression in 21%, and schizophrenia spectrum disorders (schizophrenia and schizoaffective disorders) in 14%.

## TYPES OF HALLUCINATIONS

Like adults, children have auditory, visual, olfactory, and tactile hallucinations. The rate of these different types of hallucinations is similar across studies, with auditory hallucinations in 80% to 100% ([Gordon et al., 1994](#); [Green et al., 1984](#); [Kolvin et al., 1971](#); [McKenna et al., 1994](#); [Russell et al., 1989](#)), visual hallucinations in 30.3% to 78.9% ([Gordon et al., 1994](#); [Green et al., 1984](#); [Kolvin et al., 1971](#); [McKenna et al., 1994](#); [Russell et al., 1989](#)), olfactory hallucinations in 21%, and tactile hallucinations in 37% ([Gordon et al., 1994](#); [McKenna et al., 1994](#)).

Hallucinations in children resemble hallucinations in adults. [Kempf \(1987\)](#) described children as reporting voices "ordering me to kill my sister," "telling me to run away," "calling my name," "telling me to do bad things," "telling me to steal," and "telling me to destroy." He also described children who heard voices calling them "faggot," "punk," and "prostitute." One child heard a voice telling him to do good things. Sometimes the voice was identified as that of a relative; sometimes it was not identifiable. The context of the visual hallucinations comprised people, but most children reported seeing monsters.

[Kotsopoulos et al. \(1987\)](#) described a child who believed he had "a bunch of kids in the head" or who heard voices talking of murder or lurking monsters, "a friendly man talking from the closet," or "the voice of a man urging disobedience."

[Burke et al. \(1985\)](#) reported mumbling voices and voices telling the child to hurt himself or others, for example, "a voice telling him to stab his sister" and "the voice of a man and a woman, one telling him to hurt other children, one telling him not to." Visual hallucinations varied from menacing figures to shapes and scenes.

## FOLLOW-UP

Few studies have provided diagnostic outcome data. [Garralda \(1984\)](#) followed up 20 subjects who had been identified in childhood as having hallucinations and compared them with a control group. After a mean follow-up period of 17 years, Garralda retested 16 of the 20 subjects whose mean age was then 30 years. Sixty-seven percent of the index subjects continued to have hallucinatory experiences. Garralda's findings indicate that continued hallucinations were not associated with an increased risk for later psychosis, depressive illness, organic brain damage, or other psychiatric disorder.

Like [Garralda \(1984\)](#), [Del Beccaro et al. \(1988\)](#) reported hallucinations in 50% of their adolescent sample (mean age, 17 years) after a 4-year follow-up. Eighty percent of these subjects had required further psychiatric care during the 4 years.

## Etiology and Pathogenesis

### NEUROBIOLOGICAL MECHANISMS

The findings of biochemical, imaging, and genetic studies in schizophrenia suggest that the hallucinations of schizophrenic patients might be associated with dysfunction of the primary auditory cortex in the anterior and middle superior temporal gyrus ([Rajarethinam et al., 2000](#)), increased cortical activity in Heschl's gyrus (i.e., transverse temporal gyrus) ([Dierks et al., 1999](#)), the glutamatergic N-methyl-D-aspartate receptor ([Vollenweider et al., 2000](#)), and the cholecystokinin type A receptor gene ([Wei and Hemmings, 1999](#)). Studies are needed on the interaction among hallucinations and neuroimaging, biochemical, and genetics findings to delineate the mechanisms underlying hallucinations.

### PSYCHOLOGICAL MODELS

At an empirical level, nonpsychotic forms of hallucinations would appear to be associated with states of extreme stress and altered consciousness. Illusions and fleeting pseudohallucinations have been reported in posttraumatic stress disorder in adults, although the phenomena have not been especially marked or vivid, even in severe cases ([Horowitz, 1987](#)). [Terr \(1985\)](#) has reported that, after a traumatic event that contributes to the death of someone close, a survivor may see or hear the dead friend or family member. Hallucinations of a more vivid type also have been reported in children who have lost close relatives ([Andrade and Srinath, 1986](#); [Yates and Bannard, 1988](#)). Children with other traumatic experiences, such as sexual and physical abuse, can present with hallucinations ([Hornstein and Putnam, 1991](#); [Read and Argyle, 1999](#)). Such effects might be mediated through increased cortical arousal secondary to emotional activation, although this is speculative.

Earlier psychoanalytic writers have suggested that hallucinations could be viewed as the breakthrough of preconscious or unconscious material into consciousness in response to psychological stress and conflict. As such, the material would be projected onto the world and then perceived as coming from outside. Although somewhat metaphorical in nature, these suggestions allow a bridging between the biological events that underlie hallucinations and the nature of the hallucinatory experience, reflecting as it must a person's prior experiences and current unconscious and preconscious fears, conflicts, defenses, and general mental state.

## Differential Diagnosis of Hallucinations in Children

### PSYCHOTIC DISORDERS

The hallucinations of schizophrenic children wax and wane, change in form and nature, cause subjective distress to the patient, are associated with poor reality testing, and are not mood congruent. In contrast, the hallucinations of children with an affective psychosis are mood congruent, associated with bizarre ideation, and variable. The hallucinations of children with dissociative psychosis, however, often involve a threatening voice and a protecting voice, usually of people known to the patient ([Hornstein and Putnam, 1992](#)). Children with organic psychoses have fixed hallucinations. The child with epilepsy and hallucinations does not remember the content of auditory hallucinations that are ictal.

### PERSONALITY DISORDERS

By definition ([American Psychiatric Association, 1994](#)), patients with schizotypal personality disorder (Russell, 1994) do not have hallucinations but might have

perceptual experiences, like illusions. Unlike schizophrenic children, these children vacillate as to the reality of their unusual perceptual experiences. Children with multidimensionally impaired disorder also have illusionary experiences ([Kumra et al., 1998](#))

#### ANXIETY DISORDERS

Children with anxiety might report that they see and hear monsters in the night, but not in the day. Their reality testing for the nature of these experiences is intact during the day, but not at night. Their hallucinatory experiences cause subjective distress.

Children with obsessive-compulsive disorder often present with mental images. When associated with auditory hallucinations, they are more akin to visual hallucinations. When the patient experiences repeated thoughts rather than voices associated with the mental images, they are most likely to be obsessive-compulsive phenomena.

#### DEVELOPMENTAL DISORDERS

Some children with high-functioning autism or Asperger's syndrome have hallucinatory experiences associated with their circumscribed interests. The children have poor reality testing regarding these experiences, act on the basis of their experiences, and do not experience subjective distress.

#### IATROGENIC CAUSES

Hallucinations in children have been reported as a side effect of promethazine hydrochloride ([Hickson et al., 1990](#); [Zametkin et al., 1986](#)), decongestants, such as pseudoephedrine and triprolidine ([Sills et al., 1984](#)), methylphenidate ([Bloom et al., 1988](#)), as well as anesthetic agents, such as ketamine ([Garcia-Velasco et al., 1998](#)) and isoflurane ([Kelsall et al., 1994](#)).

#### Conclusions

Hallucinations occur in children, although they appear to be rare before 7 years of age. They may be associated with a wide variety of diagnoses, ranging from response to stress and bereavement to more serious disorders, such as schizophrenia and affective disorders.

### DELUSIONS

#### Definition

A delusion is a belief that appears quite false (and sometimes bizarre) to others of the person's same cultural, social, and religious group and that cannot be changed by logical argument or evidence against it. [Mullen \(1979\)](#) has suggested that the characteristics of a delusion include that the belief (a) is held with total conviction, (b) may have great personal significance to the individual, and (c) is not amenable to reason or modifiable by experience. In adults, delusions often are of a persecutory or grandiose nature. Persecutory delusions include the idea that one is threatened with personal harm, one is being systematically robbed, or one's spouse is unfaithful. They may be simple beliefs or complex ideas with multiple false inferences.

#### OCCURRENCE

In contrast to studies of hallucinations, few studies have focused on the phenomenon of delusions in children. It is interesting that none of the studies that dealt with milder hallucinations or with hallucinations in "nonpsychotic" children mention delusions ([Andrade and Srinath, 1986](#); [Burke et al., 1985](#); [Edgell and Kolvin, 1972](#); [Kotsopoulos et al., 1987](#); [Yates and Bannard, 1988](#)). The studies that have focused on psychotic symptoms in childhood schizophrenia ([Gordon et al., 1994](#); [Greene et al., 1985](#); [Kolvin et al., 1971](#); [McKenna et al., 1994](#); [Russell et al., 1989](#); [Spence et al., 1994](#)) and affective disorder ([Chambers et al., 1982](#); [Ulloa et al., 2000](#)) all mention the presence of delusions in their results. [Garralda \(1984\)](#) found that paranoid and persecutory delusions were common in her 20 "late-onset" psychotic children. The children complained that they were being poisoned or that someone was following them. One boy claimed that people were shooting at him from under the floor.

[Kolvin et al. \(1971\)](#), [Green et al. \(1984\)](#), [Russell et al. \(1989\)](#), [Gordon et al. \(1994\)](#) and [McKenna et al. \(1994\)](#) all reported that prepubertal children with DSM-III schizophrenia had delusions similar to those seen in adult schizophrenia. [Bettes and Walker's \(1987\)](#) earlier findings suggested a low rate of positive symptoms, such as delusions and hallucinations, in schizophrenia during childhood and early adolescence. However, the incidence of delusions in most studies varied from 36% to 94% ([Gordon et al., 1994](#); [McKenna et al., 1994](#)), with similar base rates reported by [Kolvin et al. \(1971\)](#), [Green et al. \(1985\)](#), and [Russell et al. \(1989\)](#) of 57.6%, 54.2%, and 53%, respectively.

[Chambers et al. \(1982\)](#) reported delusions of control, persecution, or sin in children with major depressive disorder. Ideas of reference or thought insertion were not noted in these samples. Similarly, [Ulloa et al. \(2000\)](#) found delusions in 22% of the children who were seen at a mood disorder and anxiety disorder outpatient clinic and who met criteria for psychosis. As previously mentioned, 24% had bipolar disorder and 62% major or subsyndromic depression.

#### TYPES OF DELUSIONS IN CHILDHOOD

Like adults, children have bizarre, persecutory, somatic, grandiose delusions and ideas of reference ([Gordon et al., 1994](#); [Greene et al., 1984](#); [McKenna et al., 1994](#); [Russell et al., 1989](#)). Unlike adults, their delusions are unsystematized in the form of irrational fears and cosmic threats ([Russell et al., 1989](#)). The persecutory delusions found in schizophrenic adolescents are less elaborate and systematized than in adults, but more than in children ([Apter et al. 1991](#)).

Unlike adults and adolescents, children also have magical thinking and morbid fantasies ([Russell et al., 1989](#)). In our clinical experience, these are precursors to delusions if they are fixed and pervasive, the child acts on them, or the child does not accept the imaginary quality of these thoughts (i.e., poor reality testing).

#### Etiology and Pathogenesis

##### NEUROBIOLOGICAL FACTORS

Few hypotheses have been advanced to explain the neurobiological origin of delusions or to specify what brain mechanisms might be at their root. Imaging and cognitive studies shed some light on the underlying pathogenesis. In schizophrenic adults, delusions are associated with the relationship between volume of the orbital surface of the frontal lobe on the right and the anterior cingulate and superior frontal gyri ([Szeszko et al., 1999](#)). [Spence et al. \(1997\)](#) suggest that misattribution of significance to sensory information as indicated by hypoactivation of parietal and cingulate cortices in an H<sub>2</sub>[<sup>15</sup>O] positron emission tomography study might underlie delusions. [Andreassen et al. \(1997\)](#) have demonstrated decreased perfusion in lateral, orbital, and medial frontal lobe regions as well as in inferior temporal and parietal cortex, and increased perfusion in subcortical circuits, such as the thalamus, cerebellum, and retrosplenial cingulate gyrus. They suggest that these dysfunctional neural circuits might underlie cognitive impairment that prevents efficient processing of input and production of output, leading to symptoms such as delusions, hallucinations, and thought disorder

##### PSYCHOLOGICAL FACTORS

There have been no studies on psychological factors in the delusions of children.

#### Differential Diagnosis of Delusions in Children

##### PSYCHOTIC DISORDERS

As previously mentioned, the delusions of children with schizophrenia are bizarre, persecutory, somatic, and referential. Children with manic disorders have grandiose



delusions. Children with psychotic depression have mood-congruent delusions of guilt and nihilism that are not bizarre. Children with psychosis associated with epilepsy can have persecutory or mood-congruent delusions (i.e., grandiose, nihilistic). Children with an organic psychosis associated with a degenerative disorder have unsystematized and changing delusions.

#### PERSONALITY DISORDERS

Children with schizotypal personality disorder do not meet criteria for delusions. They have vacillating reality testing for unusual ideas (i.e., suspicious, grandiose ideas) that come and go.

#### ANXIETY DISORDERS

If children with obsessive-compulsive disorder present with bizarre ideation associated with their compulsions and poor reality testing for these thoughts, these most probably are delusions—for example, a child who coughs repeatedly to protect himself because he knows from the look in people's eye that their saliva will infect him.

#### DEVELOPMENTAL DISORDERS

Similarly, the circumscribed interests of children with high-functioning autism and Asperger's syndrome, such as preoccupations with dinosaurs, trains, numbers, road maps, and so forth, are not delusions. However, if associated with bizarre ideation and poor reality testing, they are delusions.

#### IATROGENIC CAUSES

Stimulant toxicity can cause delusions of persecution in children.

### CONCLUSIONS

From a cognitive viewpoint, children continue to develop their understanding of the world and of others well into their early school years. Despite “erroneous” notions about events, causes, and relationships between phenomena in their world, children rarely develop delusions in the absence of a more serious disorder, such as schizophrenia or affective disorder.

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# 28 DEVELOPMENTAL, NEUROBIOLOGICAL, AND PSYCHOSOCIAL ASPECTS OF HYPERACTIVITY, IMPULSIVITY, AND ATTENTION

Lily Hechtman, M.D., F.R.C.P.(C)

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In this chapter, we examine the developmental, neurobiological, and psychosocial aspects of hyperactivity, impulsivity, and attention. These three symptoms are grouped together because, although they are distinct in many ways, there is a great deal of overlap among them. It is, thus, difficult, particularly in young children, to determine if the behavior one observes is purely a problem of attention, hyperactivity, or impulsivity, or a conglomerate of all three. This chapter presents a brief developmental description of how these symptoms can be expressed at various ages. This is followed by a summary of key neurobiological aspects that may underlie these symptoms and, finally, psychosocial factors that may contribute or complicate the symptoms are discussed.

## H3>DEVELOPMENTAL ASPECTS OF HYPERACTIVITY, IMPULSIVITY, AND ATTENTION

### Temperament

The developmental aspects of symptoms such as hyperactivity, impulsivity, and attention cannot be discussed without an appreciation of the role of temperament in these behavioral characteristics. One of the first researchers to investigate temperamental differences in young children was [Gesell \(1937\)](#). Using films and direct observation, he identified some 15 behavioral characteristics that could be identified in the first year and remained relatively stable until the fifth year. The work on temperament by Thomas and Chess and their coworkers ([Thomas et al., 1968](#)) is well known. They interviewed and observed parents and their infants periodically, beginning when the children were approximately 3 months of age and delineated various behavioral or temperamental profiles. These profiles were composed of the following behavioral aspects: activity level, rhythmicity of biological functions, approach or withdrawal to new situations, intensity of emotional reactions, threshold of sensory responsiveness, adaptability, quality of mood, distractibility, attention span, and persistence. We, thus, see that activity and attention are key elements in the temperamental profiles. It was further shown that children, even in the first few months, differ greatly on the aforementioned temperamental characteristics. In discussing the symptoms of hyperactivity, impulsivity, and attention, it is necessary to be aware of normal temperamental variability in these behaviors.

The individual variability of temperamental characteristics may have a number of different origins. Hereditary factors certainly play an important role. Several twin studies ([Freedman and Keller, 1963](#); [Rutter et al., 1963](#); [Torgersen and Kringlen, 1978](#); [Vandenberg, 1969](#)) have shown that monozygotic twins were significantly more alike than dizygotic twins on various behavioral and temperamental characteristics, including those delineated by [Thomas and colleagues \(1968\)](#). As [Dobzhansky \(1967\)](#) suggested, these behavioral aspects probably are polygenetically inherited and constitute patterns of growth and ways of responding to the environment rather than specific traits. There also is a suggestion that some minor congenital physical anomalies may be associated with behavioral differences (e.g., impulsivity and hyperactivity in boys) ([Quinn and Rapoport, 1974](#); [Waldrop et al., 1968, 1978](#)).

Organic brain damage, whether prenatal, perinatal, or postnatal, has a significant effect on behavioral development and may influence temperamental characteristics. The marked irritability, low frustration tolerance, and short attention span of the brain-injured child has been well documented.

Psychosocial factors such as parenting style, family and environmental stressors, and psychological intervention may influence and modify behavioral, developmental, and temperamental characteristics. This dynamic interplay among genetic, biological, and psychosocial influences on behavioral and temperamental profiles results in the individual variability seen clinically. An extreme expression of this variability gives rise to symptoms that prove problematic to the child and his or her family. A description of how the symptoms of hyperactivity, impulsivity, and inattention can be expressed at various ages follows.

### Hyperactivity, Impulsivity, and Inattention in Infancy

In infancy, the aforementioned symptoms often are expressed by sleep disturbance, feeding problems, increased fidgetiness (not cuddly), excessive irritability, and crying.

Sleep disturbance may take the form of sleeping very little or for very short periods with the result that the infant often is awake and demanding. Sleep, when it does occur, often is very restless. The infant also may wake with a startle reaction followed by screaming.

Feeding problems may include poor sucking or crying during feedings, needing to be fed for brief periods fairly often, and not being able to settle into a workable sucking or feeding rhythm. The infant may be irregular in his or her wish for food or easily distracted from feeding. Infants sometimes become picky eaters, and these problems may give rise to problems in the mother–infant relationship. Increased irritability, fidgetiness, crying, or colic make it difficult to soothe or cuddle the child or have him or her settle down for any length of time. Sometimes the child may develop self-soothing or self-stimulating behaviors such as excessive thumb sucking, head rolling, head banging, or rocking. Once the infant begins to crawl, he or she may be in constant motion with relative disregard to the mother's presence or absence, or other dangers. This makes the child extremely accident prone, requiring constant close supervision.

The infant may be hypertonic or may not enjoy being held. All these behaviors make it difficult to establish a harmony with the child and feel effective as a parent.

The constant activity, which is forever changing and often dangerous, is evident, but at this stage, it is difficult to delineate the relative contributions of impulsivity and



inattention to the observed behavior.

### Symptoms in the Toddler and Preschooler

Normal preschoolers are very active, have short attention spans, and are fairly impulsive. It therefore often is difficult to identify children whose behavior in these areas constitutes symptoms as opposed to normal variability. Two researchers who have looked at these problems in this age group in great detail are Weiss ([Schleifer et al., 1975](#)) and Campbell ([Campbell et al., 1982](#)).

#### HYPERACTIVITY

Direct observations ([Schleifer et al., 1975](#)) or actometer measures ([Campbell et al., 1982](#)) did not show “hyperactive” preschoolers to be more active than normal control subjects in free play situations. However, the two groups were significantly different in more structured settings. In general, in this age group, hyperactivity presents with the child always running, never walking, always being on the go, changing the focus of his or her activity frequently so it appears to be without purpose or goal. The child may have difficulty sitting still for any length of time. For example, he or she frequently leaves the table during meals, cannot sit through the reading of a story, or is in constant state of motion, even when watching television.. This inappropriate activity is particularly evident in structured situations.

Despite the marked gross motor activity, which sometimes is developmentally precocious, fine motor coordination and language often are delayed. The marked hyperactivity and inattention may contribute to these delays. The child may thus be very active but poorly coordinated and clumsy, making him or her quite accident-prone. Sleep disturbances may continue, with children sleeping little, restlessly, or for short periods.

#### IMPULSIVITY

In this age group, impulsivity (often referred to as *cognitive impulsivity*) has been measured directly by various laboratory tests and described by careful observations ([Campbell et al., 1982](#); [Schleifer et al., 1975](#)). Tests of impulsivity in this age group have included the Early Childhood Matching Familiar Figures Test, which measures dimensions of reflectivity–impulsivity, Draw a Line Slowly Test, which measures motor impulsivity, and the Cookie Delay Test, which measures more global impulsive responses. All these tests have shown that some preschoolers are much more impulsive than others. Observational ([Schleifer et al., 1975](#)) and descriptive data show that children with this symptom shift their activities unpredictably: They dash out into street for no apparent reason, grab toys, or hit other children without any discernible provocation. The impulsivity makes their behavior unpredictable, very disruptive, and often dangerous. It also presents problems in behavioral management, limit setting, and discipline. Parents frequently complain that children with these symptoms “do not listen,” do not learn from their mistakes because they seem to be unresponsive to praise or punishment, and continue to repeat behaviors parents try to curtail.

#### PROBLEMS OF ATTENTION

Attention also has been studied by laboratory means [e.g., Auditory Continuous Performance Test ([Schleifer et al., 1975](#))] and direct observations ([Campbell et al., 1982](#); [Schleifer et al., 1975](#)). Children in this age group with attention problems shift their activities frequently, are inattentive during structured tasks, do not complete activities they begin, cannot play alone, and are easily distracted.

### Symptoms in the Elementary School–Age Child

#### HYPERACTIVITY

Hyperactivity in this age group is measured by parents' and teachers' rating scales, direct observational measures, or direct mechanical measures using actometers, stabilimeters, electrical mats, wrist microcomputers, or ultrasonic or photoelectric systems. In general, these measures can identify some children who are significantly more active than age-matched control children. Perhaps the longest and most naturalistic study that focused on measuring activity in this age group was carried out by [Porino and coworkers \(1983\)](#). Children in this study wore an actometer with a memory continuously 24 hours a day for 7 days. The authors showed that hyperactive children were, in fact, more active in all situations, structured or free, even during sleep, than were matched control subjects.

However, overall activity levels tell only part of the story. The activity often is “off task,” disruptive, and random, with no apparent goal or purpose. Much of it may be fidgetiness and restlessness, which take the form of leaving one's seat in the classroom, walking around, or engaging in some other somewhat inappropriate activity.

Hyperactivity also may be expressed in being unable to sit through a meal, a story, a game, or a television program. The marked disruptive nature of the activity ([Klein and Young, 1979](#)) is what is often disturbing to parents and teachers and what distinguishes it from high normal activity, frequently resulting in referrals for professional help. In a free play environment, the increased activity may be expressed by disruptive physical contact with other children (running into them, pushing, hitting, disrupting their play). This may be done without any particular anger, hostility, or malicious intent but often results in quarrels and fights with other children.

#### IMPULSIVITY

As the child matures, there is the general expectation that he or she will acquire greater inhibiting control over his or her behavior and become progressively less impulsive. Impulsivity in this age group again can be divided into an impulsive, cognitive, or problem-solving style ([Kagan et al., 1964](#)) and general behavioral impulsivity. The Matching Familiar Figures Test ([Kagan et al., 1964](#)) is a frequently used measure for assessing an impulsive cognitive style. As [Weiss and Hechtman \(1993\)](#) have pointed out, any test (e.g., Continuous Performance Test) that requires some inhibition or delay in response also may measure an element of impulsivity. This impulsive cognitive style may be seen in poor, incomplete, or error-ridden schoolwork because the child rarely follows a careful, systematic, stepwise approach. It also may express itself in a variety of games, particularly those requiring some strategy or ones where moves that are impulsive and not well thought out result in failure (e.g., chess).

However, it is the impulsive behavior that often proves the most difficult and dangerous. The impulsivity often is characterized by uncontrolled behavior with no thought to the consequences. It may be dangerous, such as walking on a high ledge or leaning from a balcony, or not socially accepted, such as taking another child's toy or stealing a candy bar or comic on the spur of the moment. As the child matures, his or her impulsive behavior can become more dangerous and problematic. Some authors have linked this characteristic to adult antisocial personality characteristics ([Clouston, 1982](#); [Lewis, 1974](#); [Weiss and Hechtman, 1993](#)).

#### PROBLEMS OF ATTENTION

Attention is not a uniform dimension or a single cognitive process. As [Taylor \(1985\)](#) has pointed out, the many different functions involved in attention include “the ability to resist distraction; to maintain one's performance on a long task; to focus intensely on specific stimuli; and to explore complex stimuli in a planned and efficient way” (p. 427). The school-age child who has attentional problems may have difficulty in some or all of these aspects of attention.

Distractibility often is described in children with attentional problems, but several investigators ([Davidson and Prior, 1978](#); [Taylor, 1982](#)) have been unable to document this difficulty in a laboratory setting. However, [Radosh and Gittelman \(1981\)](#) found that a very distracting stimulus affected performance on a very boring arithmetic task in children with attentional problems.

It has been argued that the apparent distractibility of these children really is a problem of *sustained attention*. They cannot focus on anything for any extended time and so appear easily distractible. Developmentally, as the child matures, his or her attention span should lengthen, and he or she should be able to concentrate for progressively longer periods. This developmental task is not readily achieved in children with attentional problems. Their short attention span results in not completing tasks, games, or projects, frequently shifting from one activity to another, and in general doing poorly on any task requiring sustained attention (e.g., Continuous Performance Test).

Children with attentional problems often are very disorganized in problem-solving activities and cannot deal with complex stimuli or tasks in a planned and efficient way. Tests such as the Embedded Figures Test clearly show the deficits that exist in this area. Attempts to teach more organized and effective cognitive strategies to children with attentional difficulties have met with limited success ([Abikoff, 1987](#)).

## Symptoms in the Adolescent

In adolescence, symptoms such as hyperactivity, impulsivity, and inattention may change in presentation because of the child's developmental stage and because of psychosocial aspects of the child's life (e.g., school failure, social ostracism), which also affect how symptoms are expressed.

### HYPERACTIVITY

There clearly is a decrease in gross motor hyperactivity. The adolescent usually can sit through a meal and is not grossly running around all over the place like the younger child. However, he or she is still very restless and fidgety. Hyperactivity takes the form of a lot of small muscle versus large muscle movements (e.g., tapping one's fingers, shaking one's leg, shifting positions in one's chair), as opposed to getting up and running around. This change in gross motor hyperactivity has led some authors ([Eisenberg, 1966](#); [Laufer and Denhoff, 1957](#)) to suggest that the symptoms are limited to childhood and that, by adolescence, the child may "outgrow" the problem. This became a common belief perpetuated by clinicians to parents.

Prospective follow-up studies of hyperactive adolescents by [Gittelman-Klein \(1987\)](#) and [Lambert et al. \(1987\)](#) suggest that 20% to 30% of adolescents may no longer have problems; however, most (70%) ([Biederman et al., 1998](#); [Barkley et al., 1990](#)) continue to have difficulties. Their hyperactivity merely takes another form. In addition to the restlessness and fidgetiness already described, they may become involved in many sport activities (if coordination problems are not too severe) or ride their bikes for long periods. They seem always on the go, going to and from places and rarely staying put for any extended time. They may listen to music but are in almost constant motion while doing so. Thus, the hyperactivity persists but may have found more acceptable channels (e.g., sports), and more often involves small muscle movement expressed as restlessness or fidgetiness.

### IMPULSIVITY

As the child enters adolescence, persistent impulsivity may result in increasingly dangerous impulsive acts, leading to ever-greater problems for the adolescent and his or her family. Again, for 20% to 30% of adolescents who had problems with impulsivity in childhood, this difficulty is no longer significant ([Gittelman-Klein, 1987](#); [Lambert et al., 1987](#)). However, for most children ([Barkley et al., 1990](#); [Biederman et al., 1998](#); [Weiss et al., 1971](#)), the continued impulsivity makes them appear very immature and leads to ever greater problems. The impulsivity may be expressed in dangerous and at times socially maladaptive ways. For example, playing the class clown and impulsively calling out, making jokes, or otherwise disrupting the class is not uncommon.

The adolescent may impulsively take a car and go on a joy ride. He may blow up at teachers, peers, or parents. Sometimes these blowups may be physical. Impulsive, excessive drug or alcohol use may occur from time to time. Some of these impulsive behaviors are affected by the child's poor self-esteem arising from chronic school failure and social ostracism by parents, teachers, and peers. The impulsive cognitive style also persists into adolescence, with the result of decreased organized problem-solving capabilities and continuing poor academic achievement.

### ATTENTIONAL PROBLEMS

Attentional problems often persist into adolescence and are expressed in easy distractibility, failure to complete tasks, short attention span for a particular activity, and frequent shifting from one activity to another. Continued difficulties with tasks requiring sustained attention or organized problem-solving and planning capacities often are seen. Thus, the attentional problems continue to be experienced in adolescence, causing the same deficits as they did earlier. However, the continued difficulties and failures may lead to poor self-esteem and poor motivation with a sense of hopelessness and helplessness. An adolescent may then give up and make few attempts to tackle difficult tasks. This giving up, in turn, contributes to the adolescent falling further behind and experiencing a greater sense of failure.

## Symptoms in Adulthood

Symptoms of hyperactivity, impulsivity, and inattention in adulthood may present in a form different from that seen in previous age groups because of the person's developmental state, life circumstances, and psychosocial factors influencing his or her previous and current situation.

Our knowledge regarding the adult picture of these symptoms comes from very different data sources. These sources include (a) controlled, prospective follow-up studies, which follow children with these symptoms prospectively into adult life; (b) retrospective studies, which look at old records, try to reconstruct childhood diagnoses from them, and then attempt to contact these adults; (c) studies that look at adults with these or similar symptoms; and (d) family studies of parents and relatives of children with these symptoms.

Space does not permit a detailed description of the aforementioned studies, but a brief mention of some key ones in each category is made here.

### CONTROLLED, PROSPECTIVE STUDIES

[Weiss and Hechtman \(1993; Weiss et al., 1985\)](#) conducted one of the first controlled, prospective 10-, 12-, and 15-year follow-up studies on 75 young adults who had symptoms of hyperactivity, impulsivity, and inattention during childhood and 45 matched normal control subjects (mean age, 19 years at 10-year follow-up, 25 years at 15-year follow-up). The evaluations were comprehensive, assessing academic, work, psychiatric, social, physiologic, and psychological parameters. The psychological parameters addressed key symptoms of hyperactivity, impulsivity, and attention. In addition to interviews with subjects and parents, reports were obtained from schools, employers, and the court system.

[Gittelman and colleagues \(1985; Mannuzza et al., 1993\)](#) also conducted a controlled, prospective study of 101 subjects who had these symptoms in childhood and 100 matched normal control subjects (mean age at follow-ups, 18.3 and 26 years). Comprehensive evaluations included interviews with subjects and parents and various rating scales. Forty percent of the subjects continued to have two or more symptoms of hyperactivity, impulsivity, or inattention. It also appeared that those subjects who continued to have these symptoms were more likely to have conduct disorder and substance abuse.

[Barkley \(1998\)](#) in his 15-year, prospective follow-up study of 148 hyperactive and 76 matched control subjects (mean age, 21 years), found that although 3% met diagnostic criteria for attention deficit hyperactivity disorder (ADHD) by self-report, 43% met these criteria by parental report, suggesting continuation of the symptoms of inattention, impulsivity, and hyperactivity in many adults with similar childhood symptoms.

### RETROSPECTIVE STUDIES

Retrospective studies [e.g., [Borland and Heckman \(1976\)](#), [Feldman et al., \(1979\)](#)] usually have not focused on the key symptoms of hyperactivity, impulsivity, and inattention, instead focusing on overall educational, employment, social, and emotional outcomes of adults who had these symptoms in childhood.

[Wood and coworkers \(1976\)](#) selected 15 adults from a psychiatric outpatient clinic whose main presenting complaints were impulsivity, restlessness, irritability, inattentiveness, and emotional lability. Some of these patients also had childhood histories of these symptoms, and these subjects tended to respond to stimulant treatment for the symptoms outlined in adulthood.

Similarly, [Mattes and colleagues \(1984\)](#) selected 66 patients from an outpatient psychiatry clinic with symptoms of restlessness, excitability, impulsivity, and irritability. Twenty-nine of these subjects had similar symptoms in childhood, but unlike the study by [Wender et al. \(1981\)](#), this fact did not predict responsiveness to stimulant medication.

### FAMILY STUDIES

In general, studies of parents and other relatives of children with symptoms of hyperactivity, impulsivity, and inattention have looked at various diagnostic categories as opposed to these specific symptoms in the adult relatives under study. These family studies were comprehensively reviewed by [Hechtman \(1996\)](#). Because they did not focus particularly on the symptoms under discussion, they are not detailed here.



## HYPERACTIVITY

The significant gross motor hyperactivity described at younger ages usually is much less frequent and pronounced in adulthood. Prospective studies by [Weiss et al. \(1985\)](#), [Gittelman et al. \(1985\)](#), [Mannuzza et al. \(1993\)](#), and [Barkley \(1998\)](#) that have followed subjects into adulthood have shown that 35% to 40% no longer have these symptoms as adults. The symptoms of hyperactivity that are expressed usually take the form of restlessness and fidgetiness and always being “on the go.” Adults with these symptoms tend to gravitate toward jobs that require a good deal of physical activity ([Weiss et al., 1985](#)) (e.g., sales, construction, mechanics) as opposed to sitting at a desk. Their leisure time also is spent actively doing various sports or going from one place to another. These subjects have difficulty with any endeavor requiring long periods of inactivity, such as reading a long novel. During interviews, adults who continued to be hyperactive were observed to be more restless and fidgety, with many small muscle movements or frequent changes in their sitting positions ([Weiss et al., 1985](#)).

## IMPULSIVITY

In adulthood, impulsivity is expressed socially and cognitively. Socially, it may take the form of frequent, sudden changes of job or place of residence. The sudden job changes often follow some dispute with colleagues or supervisors in which the subject simply suddenly quits without warning. Changes of residence similarly are frequent and sudden, often following some dispute with family or roommates.

It is difficult to pinpoint if the increased number of car accidents reflects problems of impulsivity, inattention, or both. Characteristically, it is coupled with some aspects of poor judgment. Other, perhaps less significant, examples of impulsive actions these subjects are likely to engage in include suddenly leaving an interview, work, a movie, or a party for no apparent reason. Restlessness may be associated with this behavior, but impulsivity is a key factor. Cognitive impulsivity is reflected in a continuing cognitive style that is impulsive and somewhat disorganized. This affects problem-solving capabilities, which may then affect work performance.

The impulsivity in this age group has serious social and work consequences because it may result in changing unstable social relationships and a poorer work record ([Barkley, 1998](#); [Weiss and Hechtman, 1993](#)).

## PROBLEMS OF ATTENTION

In adulthood, attentional problems can be seen in leisure and work activities. In terms of leisure activity, these subjects do not enjoy anything that requires sustained concentration or attention, such as reading a long novel or playing chess. In the work setting, activities requiring sustained attention often are difficult or boring for these individuals. They can be easily distracted, and completing such tasks quickly and accurately may present problems. At university, subjects tend to study for short periods with frequent breaks to cope with the difficulties of sustained attention.

In general, these symptoms in adulthood continue to affect the person's work, social, and emotional adjustment. The problems result in poor self-esteem, hopelessness, depression, and anxiety and are at times accompanied by excessive drug or alcohol use. This causes the situation to deteriorate still further. Unraveling this complex picture of pain and problems to detect the key underlying symptoms often is difficult, and so these basic handicaps frequently remain undetected in adulthood.

## NEUROBIOLOGICAL ASPECTS OF HYPERACTIVITY, IMPULSIVITY, AND INATTENTION

In general, the neurobiological aspects of the symptoms outlined have not been worked out comprehensively. Several excellent recent reviews on the subject ([Biederman and Spencer, 1999](#); [Castellanos, 1997](#); [Faraone and Biederman, 1998, 1999](#); [Swanson et al., 1998a](#); [Zametkin and Liotta, 1998](#)) clearly illustrate the complexity of the area, the divergent findings, and the many questions yet to be resolved. The symptoms are not unidimensional and probably involve various interrelated neuroanatomic and neurochemical systems. This complexity and interrelatedness make it unlikely that any one area or neurochemical system will be found to be solely or primarily involved in any particular symptom. What follows is a brief summary of the current evidence suggesting involvement of various neuroanatomic and neurochemical systems in different aspects of these symptoms.

The neurobiological aspects of the symptoms outlined include the following areas:

1. Genetics
2. Neuroanatomic aspects
3. Neurochemical aspects, including:
  - Neuroanatomic aspects of neurotransmitters
  - Nonpharmacologic studies of neurotransmitters
  - Psychopharmacologic studies of neurotransmitters
  - Neurotransmitter theories and neuroendocrine studies
4. Neurophysiologic aspects, including data from computed tomography (CT), magnetic resonance imaging (MRI), single-photon emission tomography (SPECT), and positron emission tomography (PET) scans, electroencephalography (EEG), and evoked potential studies

## GENETIC STUDIES

Four types of studies suggest that genetic factors play a role in problems of attention, hyperactivity, and impulsivity: twin, siblings and half-siblings, adoption, and family studies.

### Twin Studies

Genetically based disorders should be concordant in twins, and more so in monozygotic (MZ) than in dizygotic (DZ) twins. A number of twin studies have looked at the concordance of symptoms of inattention, hyperactivity, and impulsivity in twins.

In an early study, [Rutter et al. \(1963\)](#) reported that MZ twins were more similar to each other than DZ twins in psychomotor activity. Similarly, [Willerman \(1973\)](#) reported the heritability of activity scores to be 0.77 for 54 MZ and DZ twin pairs. [Torgersen and Kringlen \(1978\)](#) also found evidence for a genetic component in both activity levels and distractibility.

[Stevenson \(1992\)](#), using multiple regression analysis on data obtained from 91 pairs of MZ twins and 105 pairs of same-sex DZ twins, concluded that results were consistent with a significant genetic contribution to individual differences in activity level and attention abilities. More recently, [Edelbrock and colleagues \(1995\)](#) evaluated 99 MZ and 82 same-sex DZ twin pairs aged 4 to 15 years using the Child Behavior Checklist completed by parents. They found correlations of 0.68 (MZ) and 0.29 (DZ) for attentional problems. In general, using multiple regression analysis, they found significant genetic influences on competence in school and on all areas of problem behavior. Significant shared environmental influences were detected for participation in activities, quality of social relationship, performance in school, anxiety/depression, and delinquent behavior.

Most recently, [Hudziak et al. \(2000\)](#), using parent ratings on the Child Behavior Checklist, evaluated 492 twin pairs aged 8 to 12 years on attention problems and aggressive and anxious/depressed behaviors. Estimates of genetic influences were 60% to 68% for attention problems, 70% to 77% for aggressive behaviors, and 61% to 65% for anxious/depressed behaviors.

[Goodman and Stevenson \(1989\)](#) studied 570 twins 13 years of age. These authors focused particular attention on 29 MZ and 45 same-sex DZ twin pairs in which at least one twin met criteria for pervasive hyperactivity. MZ twins were more alike than same-sex DZ pairs on objective measures of attentiveness and on parent and teacher ratings of hyperactivity (59% vs. 33%). In their careful study, these authors also explored the possible effects of stereotyping (i.e., the tendency to rate identical twins similarly); adverse family factors (e.g., marital discord, parental criticism, and malaise); and perinatal adversity (e.g., low birth weight). They concluded that genetic effects accounted for approximately half of the explainable variance in hyperactivity and inattentiveness.

In their extensive study of reading-disabled twins in the Colorado Reading Project, [Gillis et al. \(1992\)](#) and [Gilger et al. \(1992\)](#) attempted to diagnose ADHD in the



twins by parental responses on the Diagnostic Interview of Children and Adolescents [DICA ( [Herjanic et al., 1982](#))]. They examined 81 MZ and 52 same-sex DZ pairs of a reading-disabled sample of twins and found that for reading disability, the concordance rate was 84% for MZ twins and 66% for DZ twins. The concordance rate for both reading disability and ADHD was 44% for MZ twins and 30% for DZ twins. The data suggest that reading disability and ADHD may have strong although independent genetic components.

[Gillis et al. \(1992\)](#) examined the same group of subjects but focused particularly on 37 MZ and 37 same-sex DZ twin pairs in which one twin had been diagnosed with ADHD using the DICA. The authors used a basic regression model for analysis and found that 79% of MZ twins and 32% of DZ twins were concordant for ADHD ( $p < 0.001$ ). Furthermore, adjustment for IQ or reading performance differences did not substantially change the results. The authors, thus, conclude that the results of their analysis suggest that ADHD is highly heritable (0.91).

One of the largest twin studies is being carried out by [Levy et al. \(1997\)](#) in Australia. They evaluated 1,938 pairs of twins aged 4 to 12 years, and their siblings and determined the perinatal and developmental history, as well as the incidence and concordance of ADHD, conduct disorder, and separation anxiety disorder. Only same-sex twins were used in the analysis, and of these, 849 were classified as MZ and 555 as DZ. Concordance for ADHD for MZ twins was 82% compared with 38% for DZ twins, suggesting a heritability of 0.75 to 0.91 ( $p < 0.001$ ).

In another large twin study originated in Minnesota, [Sherman et al. \(1997\)](#) obtained mother (DICA, revised) and teacher ratings (Teacher Rating Form) of inattention and hyperactivity/impulsivity on 194 pairs of MZ twins and 94 pairs of DZ twins (all boys aged 11 to 12 years). Concordance rates for teacher ratings of inattention were 0.78 for MZ and 0.57 for DZ twins ( $p = 0.001$ ). For mother ratings of inattention, the concordance rates were 0.70 for MZ and .30 for DZ twins ( $p < 0.001$ ). For teacher ratings of hyperactivity and impulsivity, the concordance rates were 0.69 for MZ and 0.42 for DZ twins ( $p < 0.001$ ). For mother ratings of hyperactivity and impulsivity, the concordance rates were 0.92 for MZ and 0.32 for DZ twins ( $p < 0.001$ ). These ratings suggest a genetic effect of 0.39 to 0.69 for inattention and 0.69 to 0.91 for hyperactivity/impulsivity, with teachers reflecting a lower genetic rating than mothers.

Generally, the twin studies have shown that regardless of informant and method of assessment (questionnaires or interviews), there is a consistently and significantly higher rate of concordance for attentional, hyperactive/impulsive symptoms in MZ versus DZ twins. As pointed out by [Rutter et al. \(1999\)](#), their review estimates of heritability for these symptoms have ranged between 0.54 to 0.98, suggesting a strong genetic component.

### Sibling and Half-Sibling Studies

[Welner et al. \(1977\)](#) evaluated 53 hyperactive children and their siblings and compared them with 38 nonhyperactive control subjects and their siblings. The authors found that the hyperactive child syndrome was more common among the brothers of the hyperactive children than among brothers of control subjects (26% vs. 9%). Furthermore, the hyperactive children and their brothers presented with more symptoms of anxiety and depression than did the control subjects (16% vs. 6%). The probands, but not their siblings, also presented with more antisocial symptoms than control subjects. This lends support to a family–genetic risk in this condition and suggests that hyperactive children also may show comorbid conditions of depression and anxiety.

In another early study, [Safer \(1973\)](#) compared the incidence of ADHD in 19 full-sibling and 22 half-sibling pairs. Each pair had been raised together by a common mother. One member of each pair was known to have “minimal brain dysfunction,” now known as ADHD. Over half (10) of the full-sibling pairs were concordant for ADHD, compared with only two of the 22 half-sibling pairs. This significant difference between full and half-siblings further supports a genetic component of hyperactivity.

### Adoption Studies

Early studies by [Morrison and Stewart \(1973\)](#), [Cantwell \(1975\)](#), and [Alberts-Corush et al. \(1986\)](#), showed that adoptive relatives of ADHD children are less likely to have ADHD or associated disorders than are biological relatives of such children. In addition, biological relatives of ADHD children perform worse on standardized measures of attention than do adoptive relatives of ADHD children. In a study of international adoptees, age 10-15, [Van den Oord et al. \(1994\)](#) estimated that genes accounted for 47% of the variance of inattention scores on the Child Behavior Checklist.

In another adoption study, [Cadoret and Stewart \(1991\)](#) studied 283 adoptees aged 18 to 40 years. The adoptees were divided into two groups based on whether biological parents showed evidence (from adoption agency records) of psychiatric problems or behavioral disturbances. In addition to these evaluations, direct evaluations of adoptees and adoptive parents were performed. The authors concluded that adult adoptees had to have both a biological parent with a history of criminality/delinquency and a placement in a lower-socioeconomic–status adoptive home to have an increased likelihood for development of antisocial personality disorder. This suggests that although in general, adoptive studies support a genetic component in hyperactivity, there is always an important interplay between genetic and environmental factors. Thus, in general, adoption studies also support a genetic component in these symptoms.

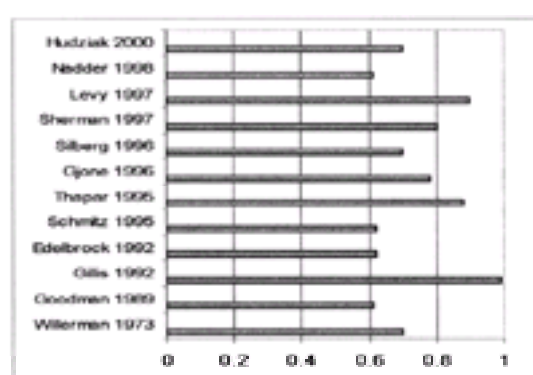
### Family Studies

Family studies of children with symptoms of inattention and hyperactivity/impulsivity have been based on the assumption that a genetic component of these symptoms will be reflected in a higher familial rate of the disorder for probands. Thus, in an early study, [Morrison and Stewart \(1971\)](#) found that 20% of hyperactive children had a parent who could be (retrospectively) diagnosed as hyperactive, compared with 5% of their medical control subjects. Similarly, [Cantwell \(1972\)](#) reported that 20% of hyperactive boys in his sample had a parent who could be classified as being hyperactive/antisocial in childhood, as opposed to 2% of the pediatric clinic control subjects.

The most extensive family studies to date have been carried out by Biederman and colleagues ( [1990](#), [1992](#)). In the first of these studies, [Biederman et al. \(1990\)](#) compared 73 male ADHD probands and 264 of their first-degree relatives with 26 psychiatrically referred but not ADHD children and 101 of their first-degree relatives, as well as 26 normal pediatric clinic control subjects and 92 of their first-degree relatives. The authors used interviewers who were unaware of the children's diagnostic status, and employed structured psychiatric interviews; they also controlled for sex, generation of relative, age of proband, social class, and the intactness of the family. Relatives of ADHD probands had higher morbidity risks for ADHD (25.1% vs. 5.3% vs. 4.6%,  $p < 0.00001$ ), antisocial disorders (24.3% vs. 6.9% vs. 4.2%,  $p < 0.00001$ ), and mood disorders (27.1% vs. 13.9% vs. 3.6%) than did relatives of psychiatric and normal control subjects. These findings indicated the importance of family–genetic factors in ADHD.

In an expanded study of 140 probands, 120 normal control subjects, and 822 first-degree relatives, [Biederman et al. \(1992\)](#) found nearly half (49%) of the subjects with ADHD had no comorbidity with conduct disorder, major depression, or multiple anxiety disorders. However, compared with control subjects, ADHD probands were more likely to have these conditions. Similarly, relatives of ADHD probands had a higher risk for ADHD (25% vs. 8%), antisocial disorders, major depression (26% vs. 9%), substance dependence, and anxiety disorders. Biederman and colleagues suggest that ADHD and major depression may show common familial vulnerabilities, that ADHD and conduct disorder may be distinct subtypes, and that ADHD and anxiety disorders are transmitted independently in families. The authors conclude that these results extend previous findings indicating family–genetic influences.

In summary, twin, sibling, adoption, and family studies all suggest a strong genetic component in the development of hyperactivity, inattention, and impulsivity. [Figure 28.1](#) clearly shows that these studies suggest heritability ranging from 0.5 to 0.98. This still leaves questions regarding the role and importance of environmental factors and the mode of inheritance.



**Figure 28.1.** Estimates of heritability from twin and family genetic studies. (Modified from Faraone SV, Biederman J: Neurobiology of attention deficit hyperactivity disorder. *Biol Psychiatry* 44:951–958, 1998.)

### Mode of Inheritance

Given the strong evidence of genetic influence in ADHD, several hypotheses have been presented as to the possible mode of genetic transmission. [Omenn \(1973\)](#) examined the possibility of sex-linked transmission, given the male preponderance in the condition. However, the author concluded that this was unlikely because of the high frequency of father-to-son transmissions. Morrison and Stewart suggested a polygenic mode of transmission, but could not substantiate it because of limitations of their sample size. A polygenic multiple-threshold model was suggested by [Rhee et al. \(1999\)](#) after analyzing sex differences in the extensive Australian twin and sibling pair study. [Deutsch et al. \(1990\)](#), studying dysmorphic children with ADHD, stated that the dysmorphic changes were consistent with a single-gene autosomal dominant inheritance. Tests of this model would require larger samples and more definitive diagnoses of both ADHD and dysmorphic phenotypes.

[Faraone et al. \(1992\)](#), using the data obtained from subjects and relatives studied by [Biederman et al. \(1992\)](#), applied segregation analysis to these data and suggested the data were consistent with the effects of a single gene. Similar results have been reported in a twin study by [Eaves et al. \(1993\)](#), a pedigree study by [Hess et al. \(1995\)](#), and a family study by Bailey et al. (1997a). Despite these findings suggesting a single gene, as [Faraone and Biederman \(1998\)](#) pointed out, differences in fit between genetic models were modest. This was particularly true for the comparison of multifactorial and single-gene inheritance. This has raised the suggestion that symptoms of ADHD may be caused by several interacting genes of modest effect. Such an idea is consistent with ADHD's high population prevalence and high concordance in monozygotic twins but modest recurrence risk to first-degree relatives.

To date, a specific definitive mode of inheritance has not been established, but work in this area is proceeding. However, [Pauls \(1991\)](#) points out that diagnostic uncertainty impedes progress in developing genetic models that address the type of genetic transmission involved. He argues for the importance of longitudinal studies of prospectively identified subjects and careful observation of their offspring as the best way to resolve some of the thorny methodologic difficulties of family and genetic studies.

### MOLECULAR GENETIC STUDIES

There has been a growing focus on molecular genetic studies to discover or pinpoint the abnormal gene or genes that cause symptoms of inattention, hyperactivity, and impulsivity and, thus, are implicated in the etiology of ADHD.

The molecular genetic studies, in general, can be divided into two large groups, the human studies ( [Table 28.1](#) ) and the animal studies ( [Table 28.2](#) ).

**Table 28.1. Summary of Human Molecular Genetic Findings**

Study	Gene	Findings
Gao et al., 1996	Dopamine transporter gene (DAT)	Hyperactive mice
	Downregulation of mRNA for D1 and D2 receptors	
Jacob et al., 1998	Plasma membrane dopamine transporter gene (DAT)	Hyperactive mice
	50% increase in striatal dopamine	
	300% increase in striatal dopamine	
Schneiders et al., 1998	Plasma membrane transporter gene (DAT)	Hyperactive mice
	Impaired spatial cognitive function	
	Locomotor response to psychostimulants depends on autoregulatory feedback	
Ku et al., 1994	Dopamine 1 receptor gene (D1)	Hyperactive mice
Ishii et al., 1995	Dopamine 2 receptor gene (D2)	Hyperactive mice
Aceti et al., 1996	Dopamine 2 receptor gene (D2)	Hyperactive mice
Rubenstein et al., 1996	Dopamine 4 receptor gene (D4)	Hyperactive mice, better coordinated
	Increased synthesis of dopamine in caudate and putamen	

**Table 28.2. Summary of Animal Molecular Genetic Studies—Dopamine Knockout Models**

In one of the early molecular genetic studies, [Hauser et al. \(1993\)](#) showed that a mutation in the thyroid receptor- $\beta$  gene, which causes generalized resistance to thyroid hormone, was associated with a high rate (61%) of hyperactivity and impulsivity (but not inattention) in affected individuals. However, the thyroid receptor- $\beta$  gene cannot be a major cause of ADHD because the prevalence of generalized resistance to thyroid hormone is very low (1/2,500) in patients with ADHD ( [Weiss et al., 1993](#) ), and the association between ADHD and the thyroid receptor- $\beta$  gene has not been found consistently ( [Weiss et al., 1994](#) ). However, the study sparked a great deal of interest and may have opened the door to the identification of specific genetic abnormalities in this condition.

Many of the molecular genetic studies have focused on genes involving dopamine because this neurotransmitter has been implicated in ADHD and its symptoms, both in neuroanatomic and pharmacologic treatment studies.

### Dopamine D2 Receptor Gene

In an early population-based association study, [Comings et al. \(1991\)](#) found that a genetic variant of the dopamine D2 receptor gene (DRD2; D2-A1 allele) was significantly increased in patients with Tourette's syndrome (44%,  $n = 147$ ), ADHD (46.2%,  $n = 104$ ), autism (54.5%,  $n = 33$ ), alcoholism (42.3%,  $n = 104$ ), and posttraumatic stress disorder (45.7%,  $n = 33$ ) compared with normal control subjects (24.5%,  $n = 77$ ). The prevalence of this allele was not significantly increased in patients with depression, panic attacks, Parkinson's disease, or obesity. However, because the D2-A1 gene is present in fewer than half of the individuals affected, the gene is not thought to be the primary cause of these disorders. The authors suggest that the DRD2 gene acts to modify the expression (making the symptoms better or worse) of the major gene or genes (yet to be discovered) that cause the condition.

More recently, [Rowe et al. \(1999\)](#), exploring the same DRD2 gene and using parental genotypes as controls to control for population heterogeneity, found no significant association or linkage between subjects with ADHD and this gene.

[Winsberg and Comings \(1999\)](#) investigated different dopamine genes and their relationship to methylphenidate response in African-American children with ADHD.



There was no relationship between the DRD2 gene and methylphenidate response.

We thus see that the association of the DRD2 gene with ADHD is neither specific nor consistent and, at present, lacks the promise of other dopamine genes [e.g., dopamine transport gene (DAT1) and the dopamine 4 receptor gene (DRD4)].

### Dopamine Transport Gene

In an early family-based association study, using the haplotype relative risk (HRR) design, [Cook et al. \(1995\)](#) showed an association between ADHD and the 480-bp allele of DAT1. This finding was replicated in a population-based study by [Waldman et al. \(1996\)](#). However, the authors found that the gene was associated not only with ADHD but also with conduct disorder, Tourette's syndrome, and obsessions. In a subsequent family-based study, which examined association and linkage, [Waldman et al. \(1998\)](#) found strong associations and linkage of ADHD with DAT1 with the combined but not inattentive subtype. [Cook and colleagues' \(1995\)](#) findings were further confirmed by [Gill et al. \(1997\)](#) in a family-based study of 40 probands and their parents using the same HRR design. These authors also found an association between ADHD and the DAT1 gene. Confirmation of the association of the DAT1 gene with ADHD also came from [Daly et al. \(1999\)](#) in another family-based study originating in Ireland.

The importance of the DAT1 gene (10-repeat allele) in ADHD was demonstrated by [Winsberg and Comings \(1999\)](#), who showed that homozygosity of this allele characterized nonresponse to methylphenidate in a group of African-American children with ADHD.

However, not all molecular genetic studies involving the DAT1 gene have shown a positive association with ADHD. [Crowe and Vieland \(1998\)](#) failed to replicate an association between ADHD and DAT1 with the 480-bp allele. Similarly, [Palmer et al. \(1999\)](#) indicated that in their large sample, they found no evidence of linkage or linkage disequilibrium between DAT1 and ADHD.

The aforementioned studies clearly suggest that the DAT1 gene may have an important role in the etiology of symptoms of ADHD. However, it may need to interact with a number of other genes to exert its effects. This would account for some of the divergent findings to date.

### Dopamine 4 Receptor Gene

Early interest in the DRD4 gene came from a population study by [Ebstein et al. \(1996\)](#), in which the authors showed that the seven-repeat allele of DRD4 was associated with novelty seeking. People with this trait are described as impulsive, exploratory, excitable, and quick tempered, characteristics frequently seen in people with ADHD. The association between the DRD4 gene and novelty seeking was replicated in a study by [Benjamin et al. \(1996\)](#), where population- and family-based measures were combined. However, another population-based study by [Malhotra et al. \(1996\)](#) failed to confirm this association.

[LaHoste et al. \(1996\)](#), in a population study, found higher rates of the seven-repeat allele of the DRD4 gene among children with ADHD compared with control children matched for sex and ethnicity. These results were maintained when the sample was increased by 50% ([Sunohara et al., 1997](#)). In a family-based study of 52 families, using a HRR design, [Swanson et al. \(1998b\)](#) also found a significant association between ADHD and the DRD4 (seven-repeat allele) gene. Confirmation also came from a study by [Smalley et al. \(1998\)](#) using affected sibling pairs and singleton families and the transmission disequilibrium test (TDT) and mean level of identity descent sharing. The TDT showed the seven-repeat allele is differentially transmitted to children with ADHD. [Rowe et al. \(1998\)](#) found a significant association with the seven-repeat allele of the DRD4 gene for combined and inattentive subtypes, but not the hyperactive/impulsive subtype of ADHD. [Bailey et al. \(1997b\)](#) found a similar association between ADHD and the DRD4 gene. [Faraone et al. \(1999\)](#) studied triads composed of an adult subject with ADHD, his or her spouse, and their child with ADHD. Using a multiallele TDT, they also reported an association between the DRD4 gene and ADHD.

Here too, however, the findings are not all consistent and uniformly positive. [Comings et al. \(1999b\)](#), in a population study involving 737 individuals from four different groups of control subjects and 707 index subjects from four different groups with impulsive, compulsive, addictive, drug abuse, and pathologic gambling behaviors, Tourette's syndrome, and ADHD, found no difference regarding the seven-repeat alleles of DRD4 in any of the groups, although some differences in those carrying any of the five and eight alleles were associated with pathologic gambling and ADHD. [Castellanos et al. \(1998\)](#) also found no association between the DRD4 seven-repeat allele and the 41 children with ADHD and 56 control subjects matched for ethnicity and sex. Furthermore, behavioral and anatomic MRI measures that previously discriminated ADHD and control subjects showed no significant difference between those with and without the DRD4 seven-repeat allele. Thus, this study did not support the association between DRD4 and ADHD. Finally, [Winsberg and Comings \(1999\)](#) found no association between DRD4 receptors and methylphenidate response in a group of African-American children with ADHD.

All these findings suggest an important role for the DRD4 seven-repeat receptor in ADHD. However, as with the DAT gene, the DRD4 gene may exert its influence in ADHD in combination with other genes and in conjunction with other neurotransmitter systems. There have been relatively few molecular genetic studies that have explored other genes and other neurotransmitter systems. Some of these studies are described in the following sections.

### Other Genes

#### DOPAMINE B-HYDROXYLASE GENE

[Daly et al. \(1999\)](#) conducted a family-based study using both the TDT method and HRR analysis. The authors found an association between ADHD and the dopamine b-hydroxylase (DBH) gene.

#### DOPAMINE 5 RECEPTOR GENE

In the same study, [Daly et al. \(1999\)](#) also explored the connection between the dopamine 5 receptor gene (DRD5) and ADHD and found a significant association, particularly in nonfamilial cases.

#### DOPAMINE 1 RECEPTOR GENE

[Thompson et al. \(1998\)](#) explored the association between the dopamine 1 receptor gene (DRD1) and Tourette's syndrome (n = 50), Tourette's syndrome with comorbid ADHD (n = 35), obsessive-compulsive disorder (n = 30), and alcohol dependence (n = 72), with 50 normal control subjects. The authors found no significant association between the DRD1 gene and these disorders and concluded that this gene was unlikely to contribute to the inheritance of these disorders.

#### CATECHOL-O-METHYLTRANSFERASE GENE

[Eisenberg et al. \(1999\)](#) conducted a family-based study of 48 child/parent triads with ADHD using a HRR design and a parent-to-proband allele transmission test. The authors found an association between the catechol- O-methyltransferase (COMT) gene (high activity COMT val allele) and the ADHD.

It is suggested that the high enzyme activity of the COMT val allele increases central nervous system (CNS) clearance of dopamine (and norepinephrine), thus resulting in lower CNS dopamine/norepinephrine levels, which may be important in the pathogenesis of the disorder because methylphenidate, which increases dopamine and norepinephrine turnover, is used in the treatment of the condition.

However, [Barr et al. \(1999\)](#) tested for linkage in 77 ADHD families, using functional polymorphism at codon 158, which determines COMT activity. The authors analyzed the data with the TDT and found no evidence of linkage of this polymorphism in their ADHD sample.

Clearly, more studies are needed to clarify the role of the COMT gene in ADHD.

#### ANDROGEN RECEPTOR GENE

The male predominance of externalizing behaviors suggest that the X-linked androgen receptor (AR) gene might be involved. [Comings et al. \(1999a\)](#) evaluated 302 Tourette's syndrome and control subjects. There was a significant association between AR gene haplotypes and ADHD, conduct disorder, and oppositional defiant

disorder (ODD), suggesting that variation in the human AR gene plays a role in externalizing disorders, including ADHD.

#### IMMUNE FUNCTION AND REGULATION GENES

[Odell et al. \(1997\)](#) explored the association of ADHD with alleles of two genes, the null allele of the C4B gene and the b-1 allele of the DR gene, both coding for products involved in immune function and regulation. The family-based study evaluated 31 subjects with ADHD and their parents and 10 normal control subjects. DR and C4B typing were performed by serologic human leukocyte antigen typing techniques and the DNA method, polymerase chain reaction–restriction fragment length polymorphism. Each of the alleles was found to be significantly associated with ADHD. Approximately 55% of subjects with ADHD carried both these alleles on one of their chromosomes, compared with 8% of normal control subjects. The authors conclude that genes related to the immune system may be associated with development of the symptom of ADHD.

#### Animal Molecular Genetic Studies (Dopamine Knockout Model)

A series of molecular genetic studies that have focused on dopamine genes and ADHD have involved animals (mostly mice) using a gene knockout techniques (see [Table 28.2](#)). These studies have explored the role of dopamine transporter and five dopamine receptors. [Giros et al. \(1996\)](#) showed that knocking out the dopamine transport gene resulted in a hyperactive mouse with some evidence of downregulation of the messenger RNA for D1 and D2 receptors. Thus, a relationship between hyperactivity and the dopamine system is suggested. [Jaber et al. \(1998\)](#) knocked out the plasma membrane dopamine transport gene and produced a hyperactive mouse with a 5% increase in intracellular dopamine and a 300% increase in extracellular dopamine. The importance of this gene in regulation of intracellular and extracellular dopamine is clear. [Gainetdinov et al. \(1999\)](#) also knocked out the plasma membrane transporter gene and similarly produced hyperactive mice who also had impaired spatial cognitive function. These mice had decreased locomotion in response to psychostimulants. However, the authors postulate that this response depended on serotonergic transmission. Thus, the interaction of dopamine and serotonin systems is suggested.

[Xu et al. \(1994\)](#) and [Accili et al. \(1996\)](#) knocked out the D1 and D3 receptor genes, respectively, and produced hyperactive mice, whereas [Baik et al. \(1995\)](#) and [Rubenstein et al. \(1996\)](#) knocked out the D2 and D4 receptor genes and produced hypoactive mice. However, D4 knockout mice were better coordinated and had increased synthesis of dopamine in the caudate and putamen.

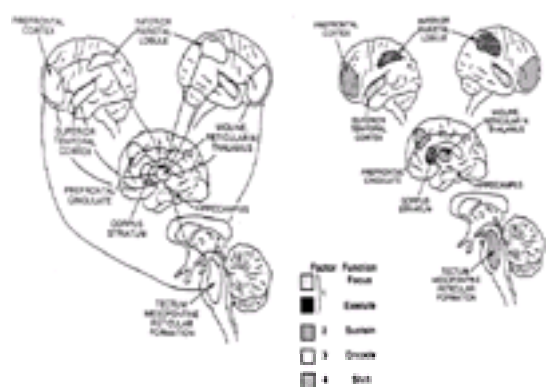
These studies clearly show that the dopamine genes are part of an integrated system wherein dysfunction in one area results in compensatory mechanisms in other areas. The dopamine genes also undoubtedly are linked and integrated with genes involving other neurotransmitters. More detailed and extensive studies involving both animal models and human subjects will be needed to identify these complex interrelationships.

#### Summary of Molecular Genetic Studies

Most of the molecular genetic studies to date have focused on dopamine-related genes (e.g., DRD2, DAT1, DRD4, DBH, DRD5, DRD1). Replications of positive associations with ADHD have been seen most frequently with the DAT1 gene and the DRD4 seven-repeat allele. However, often these positive associations are not limited to ADHD but occur with other conditions as well, and so are not specific for ADHD. Furthermore, for each of these genes, there are some studies that have shown negative results that do not support the association. Clearly, more research is needed to clarify the role of these genes in ADHD.

#### Neuroanatomic Aspects

The complexity of trying to pinpoint particular neuroanatomic areas that might be involved in symptoms such as hyperactivity, impulsivity, and inattention can best be illustrated in the following example. [Mirsky \(1987\)](#), in his review entitled “Behavioral and Psychophysiological Markers of Disordered Attention,” makes the case that attention is not a single function but has various distinct and separate aspects such as focusing, executing, sustaining, and shifting attention. He further suggests that these different functions involve different brain regions that are interconnected and organized into a system. This attentional system is so widespread that it is very vulnerable to damage and dysfunction. Depending on where the damage or dysfunction occurs, different aspects of attention may be affected ([Fig. 28.2](#)).



**Figure 28.2.** Semischematic representation of brain regions involved in attention. **Left:** Some interconnections among the regions are shown. The connections are conceivably sufficient to support the concept of an attention system. **right:** Tentative assignment of functional specializations. (Reproduced with permission from Mirsky A: Behavioral and psychophysiological markers of disordered attention. *Environ Health Perspect* 74:191–199, 1987.)

Specifically, [Mirsky \(1987\)](#) suggests

the functions of focusing on environmental events are shared by superior temporal and inferior parietal cortices, as well as by structures that comprise the corpus striatum, including caudate, putamen, and globus pallidus. The inferior parietal and corpus striatal regions have strong motor executive functions. Considerable amounts of encoding of stimuli are accomplished by the hippocampus, an essential mnemonic function that seems to be required for some aspects of attention. The capacity to shift from one salient aspect of the environment to another is supported by the prefrontal cortex. Sustaining a focus on some environmental event is the major responsibility of rostral structures, including the tectum, mesopontine reticular formation and midline, and reticular thalamic nuclei (p. 197).

In a similar recent outline, [Faraone and Biederman \(1999\)](#) suggested

It is likely that a network of interrelated brain areas is involved in the attentional-executive impairments of ADHD children. The cingulate cortex plays a role in motivational aspects of attention and in response selection and inhibition. A system mainly involving right prefrontal and parietal cortex is activated during sustained and directed attention across sensory modalities. The inferior parietal lobule and superior temporal sulcus are polymodal sensory convergence areas that provide a representation of extrapersonal space which plays an important role in focusing on and selecting a target stimulus. The brainstem reticular activating system and reticular thalamic nuclei regulate attentional tone and filter interference, respectively. Working memory deficits implicate a distributed network including anterior hippocampus, ventral anterior and dorsolateral thalamus, anterior cingulate, parietal cortex, and dorsolateral prefrontal cortex (p. 795).

Thus, there is a consistent suggestion that problems of attention, impulsivity, and hyperactivity involve abnormalities in the frontocortical and subcortical systems, particularly prefrontal and basal ganglia areas ([Casey et al., 1997](#); [Castellanos, 1997](#); [Faraone and Biederman, 1999](#); [Swanson et al., 1998a](#); [Zametkin et al., 1998](#)).

It therefore is not surprising that, in their early review of the literature, [Zametkin and Rapoport \(1987\)](#) showed that over the years a wide variety of brain areas have been implicated in these symptoms by various authors.

More recently, neuroimaging studies have provided more direct evidence of possible areas of brain abnormalities in subjects with symptoms of inattention, hyperactivity, and impulsivity. These studies involve either structured or functional imaging. CT and MRI provide structured images, whereas SPECT and PET



produce functional images. These latter studies require ionizing radiation and have thus been associated with ethical concerns regarding the study of children and normal control subjects. Functional MRI (fMRI) studies hold promise because they do not require ionizing radiation and combine both types, allowing structural and functional views by measuring blood flow.

Table 28.3 summarizes various types of neuroimaging studies involving subjects with problems of attention, impulsivity, and hyperactivity. As can be seen from the table, studies vary in methods used (e.g., CT, MRI, fMRI, PET, SPECT), subjects studied (e.g., male and female; children, adolescents, adults; medicated, unmedicated; control subjects used—normal control subjects, matched on various parameters, sibling controls), and areas of the brain investigated (e.g., cortical, subcortical). Therefore, it is not surprising that findings of the various studies vary considerably. However, a certain consistency does emerge. Many studies (Amen et al., 1993; Casey et al., 1997; Castellanos et al., 1996; Ernst et al., 1994; Filipek et al., 1997; Hynd et al., 1990; Lou et al., 1984; Rubia et al., 1999; Semrud-Clikeman et al., 2000; Sunshine et al., 1997; Zametkin et al., 1990, 1993) have found a decrease in function (blood flow or glucose metabolism) in the frontal lobes, particularly the prefrontal cortex. Some found this abnormality only on the right (Castellanos et al., 1996; Filipek et al., 1997), whereas others did not. Another consistent finding involving the cerebral cortex is a smaller cerebellum (Berquin et al., 1998; Castellanos et al., 1996; Mostofsky et al., 1998; Nasrallah et al., 1986). Mostofsky et al. (1998) and Berquin et al. (1998) indicated that the decrease was specifically in the posterior vermis of the cerebellum. The anterior cingulate cortex has been shown to have decreased activity in subjects with ADHD (Bush et al., 1999; Zametkin et al., 1990), as has the temporal lobe (Ernst et al., 1994).

**Table 28.3. Neuroimaging Studies**

Several studies have shown a smaller corpus callosum in subjects with ADHD (Giedd et al., 1994; Hynd et al., 1991; Semrud-Clikeman et al., 1994). The findings in other cortical areas appear less consistent. The parietal lobe was found to be smaller in subjects with ADHD by Filipek et al. (1997), but had more activation according to Sunshine et al. (1997) and was found to be hyperperfused by Lou et al. (1989). Similarly, the occipital lobe was found to be smaller in subjects with ADHD by Filipek et al. (1997) but hyperperfused by Lou et al. (1984, 1989, 1990) and Schweitzer et al. (2000). The suggestion is that the increased activity in the parietal and occipital areas during tasks may, in fact, contribute to the distraction and inattention of the subjects with ADHD.

The other area of the brain that has been consistently implicated in ADHD is the striatal area, consisting of the basal ganglia with its caudate nuclei, nucleus accumbens, globus pallidus, and putamen. Specifically, decreased activation and right hyperperfusion were noted by Vaidya et al. (1998) and Lou et al. (1989), respectively.

A smaller globus pallidus was noted in three studies, although Singer et al. (1993) and Aylward et al. (1996) both stipulated a smaller left globus pallidus, whereas Castellanos et al. (1996) pinpointed a smaller right one.

Similarly, a smaller caudate nucleus was suggested in several studies, with a smaller left caudate nucleus identified by Filipek et al. (1997), Casey et al. (1997), and Semrud-Clikeman et al. (2000), whereas Castellanos et al. (1996) and Zametkin et al. (1990) both pointed to a smaller right caudate nucleus. These differences may be associated with the larger number of female subjects in the studies by Castellanos et al. (1996) and Zametkin et al. (1993). Decreased functioning of the caudate nucleus was documented through hypoperfusion by Teicher et al. (1996), and decreased response by Rubia et al. (1999). Only one study (Mataro et al. 1997), involving adolescents with ADHD, showed a larger right caudate nucleus and suggested this was evidence of immature development in this area with decreased reabsorption.

Although few studies have documented any abnormalities in the putamen, Ernst et al. (1994) suggested decreased activity in the right and left posterior putamen in girls with ADHD.

Substriatal structures such as the thalamus and the hippocampus also have been shown to be less active in subjects with ADHD. Specifically, Zametkin et al. (1990) showed the right thalamus to be less active. Subsequently, Zametkin et al. (1993) and Ernst et al. (1994) showed that the left thalamus was smaller. These later studies had more female subjects. Decreased activity of the hippocampus was consistently shown to be on the right by Zametkin et al. (1990, 1993) and Ernst et al. (1994).

In summary, neuroimaging studies suggest decreased size and activity in the frontal lobes, particularly the prefrontal area. A smaller corpus callosum and cerebellum and decreased activity in the anterior cingulate also have been consistently found.

Decreased activity in the striatal area also has been shown. A smaller and less active globus pallidus and caudate nucleus have both been documented, although in some studies this is on the right, in others on the left. Decreased activity in substriatal structures (e.g., thalamus and hippocampus) also has been documented.

These findings have led to the hypothesized interconnecting cortico-thalamic-cortical system with a reciprocal excitatory and inhibition network of these areas as illustrated by Swanson et al. (1998a) and described by Castellanos (1997).

## NEUROCHEMICAL ASPECTS OF ADHD

### Neuroanatomic Studies of Neurotransmitters

Even though some areas of the brain have been clearly associated with certain neurotransmitters, such as the caudate nucleus and corpus striatum with dopamine (Mattes, 1980) and the median raphe area with serotonin (Wirtshafter et al., 1986), neuroanatomic studies of neurotransmitters have proven very complex. The complexity comes from the fact that any particular area can be involved with several different neurotransmitters or receive projections from various neurotransmitter pathways or nuclei. Thus, there rarely is a one-to-one correspondence between a particular area and a sole neurotransmitter that exerts exclusive influence on this area.

Hence, we see that the frontosubcortical systems (identified previously) that control attention and motor behavior are rich in catecholamines. The catecholamine hypothesis therefore has been popular as an explanation for the abnormalities of attention, hyperactivity, and impulsivity seen in subjects with ADHD (Oades, 1987; Pliszka et al., 1996). This hypothesis emphasizes the central role of dopamine (Castellanos, 1997) and norepinephrine (Arnsten et al., 1996; Mefford and Potter, 1989).

### NONPHARMACOLOGIC STUDIES OF NEUROTRANSMITTERS AND THEIR METABOLITES

These types of studies have compared subjects with symptoms of hyperactivity, impulsivity, and inattention and normal control subjects with respect to monoamines and their metabolites in urine, plasma, platelets, and, rarely, cerebrospinal fluid (CSF). The limitations of such peripheral measures in reflecting an accurate CNS neurotransmitter picture are well outlined by Zametkin and Rapoport (1986). A comprehensive review and outline of these types of studies was carried out by Zametkin and Rapoport (1987) and is summarized in Table 28.4. In general, no consistent differences in any of the peripheral measures of monoamines and their metabolites were found between children with these symptoms and normal control subjects. However, in a series of studies conducted at the National Institute of

Mental Health, Castellanos et al. (1994, 1996) showed a positive correlation between levels of hyperactivity and the CSF concentration of homovanillic acid (HVA; the peripheral dopamine metabolite) in boys diagnosed with ADHD. Furthermore, the HVA concentration in CSF was a good predictor of the therapeutic response to methylphenidate, dextroamphetamine, and pemoline. The boy with the greatest CSF HVA concentration had the best responses to treatment. These findings were consistent with an earlier study (Shetty, 1973) showing that postamphetamine decreases in CSF HVA were highly correlated with behavioral improvement.

Investigator	ADHD	Control	Age	Method	Findings
Boyd and Chubb, 1975	28	18	10-12	CSF	No differences in HVA
Boyd et al., 1977	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1981	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1982	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1983	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1984	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1985	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1986	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1987	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1988	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1989	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1990	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1991	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1992	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1993	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1994	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1995	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1996	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1997	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1998	15	15	10-12	CSF	No differences in HVA
Boyd et al., 1999	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2000	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2001	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2002	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2003	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2004	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2005	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2006	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2007	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2008	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2009	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2010	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2011	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2012	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2013	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2014	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2015	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2016	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2017	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2018	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2019	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2020	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2021	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2022	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2023	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2024	15	15	10-12	CSF	No differences in HVA
Boyd et al., 2025	15	15	10-12	CSF	No differences in HVA

Table 28.4. Blood, Urine, and Cerebrospinal Fluid Comparisons of Control Children and Children with Attention-Deficit/Hyperactivity Disorder

### PSYCHOPHARMACOLOGIC STUDIES OF NEUROTRANSMITTERS

These types of studies look at the particular psychopharmacologic agent, its possible relationship to one or more neurotransmitters, and its clinical effect. From such analysis, it is then postulated how the drug may work and what the possible underlying problem in the neurotransmitter system may be.

Again, [Zametkin and Rapoport \(1987\)](#) provide an excellent review and summary of such studies. More recently, [Biederman and Spencer \(1999\)](#) reviewed various types of psychopharmacologic agents, including stimulants; noradrenergic agents (e.g., tricyclic antidepressants, bupropion, monoamine oxidase inhibitors, tomoxetine);  $\alpha_2$ -noradrenergic agonists (clonidine and guanfacine); as well as serotonergic and mixed serotonergic-noradrenergic agents.

The authors concluded that agents effective in ADHD tend to have adrenergic/dopaminergic activity. Thus, stimulants are thought to be involved in blocking the reuptake of dopamine and norepinephrine into the presynaptic neuron and increasing the release of these monoamines into the extraneuronal space by altering monoaminergic transmission. [Wang et al. \(1995\)](#) used PET scans with [ $^{11}\text{C}$ ]-methylphenidate to show its binding to dopamine transporter after intravenous administration to normal adults. Methylphenidate binding localized to the basal ganglia (putamen, caudate, and ventrostriatum) and the cortex (temporal insula and cingulate gyri). [Volkow et al. \(1998\)](#), using similar techniques, showed that the transport binding and clearance times corresponded to clinical effect, and the degree of blocking of dopamine transport was dose dependent. [Ding et al. \(1997\)](#) showed that the D-threo isomer and not the L-threo isomer, which comprises 50% of commercially available methylphenidate, binds selectively to the dopamine transporter.

In general, the tricyclic antidepressants are thought to be less effective than the stimulants. The tricyclic antidepressants, particularly the secondary amines (desipramine, nortriptyline, and protriptyline), are thought to be involved in the transport and reuptake of norepinephrine. Bupropion, also shown to be effective in ADHD, is thought to possess both indirect dopamine agonist and noradrenergic effects. Tomoxetine, an experimental noradrenergic-specific compound, has shown some promising results for adults ([Spencer et al. 1998](#)) and children ([Spencer et al., 1999](#)) with ADHD. Controlled studies with  $\alpha_2$ -adrenergic agonists are limited.

Serotonergic and mixed serotonergic/noradrenergic agents appear to be less effective in ADHD than stimulants, suggesting a lesser role of serotonin in ADHD. Clinically, selective serotonin reuptake inhibitors have not been found to be effective for ADHD symptoms ( [National Institute of Mental Health, 1996](#)). [Ernst et al. \(1997\)](#) evaluated the effect of selegiline, a selective monoamine oxidase type B inhibitor, which is thought to affect serotonin metabolism; selegiline affected monoamine metabolites and dopamine plasma levels, as well as producing clinical changes, suggesting the involvement of several neurotransmitter systems.

However, findings for the mixed serotonergic/noradrenergic atypical antidepressant venlafaxine are unclear. Although response rates of 77% in adults with ADHD ([Adler et al., 1995](#); [Findling et al., 1996](#); [Hornig-Rohen and Amsterdam, 1995](#); [Reinherr et al., 1995](#).) and 50% in children with ADHD ([Luh et al., 1995](#)) have been reported, dropout rates due to side effects (particularly increased hyperactivity) have been high at 21% and 25%, respectively.

In general, pharmacologic studies support the importance of the catecholamines, dopamine, and norepinephrine in ADHD, with a less prominent role for serotonin.

#### Dopamine

There is much evidence to support the importance of dopamine in ADHD. The neuroanatomic areas implicated in this condition (cortical-striatal-thalamic-cortical network) are known to be areas of dopamine concentration. Molecular genetic studies have suggested the involvement of a dopamine transport gene and various dopamine receptor genes, particularly the DRD4 and DRD2 genes. Stimulant agents, which are very effective for ADHD symptoms, are known to be involved in binding the dopamine transporter and thus preventing reuptake of dopamine into presynaptic nuclei.

[Ernst et al. \(1999\)](#), using PET and the tracer [ $^{18}\text{F}$ ]-fluorodopa, showed that the accumulation of [ $^{18}\text{F}$ ]-fluorodopa in the right midbrain was 48% higher in 10 children with ADHD compared with 10 normal control subjects and was correlated with symptom severity. Finally, various studies using animal models ([Boix et al., 1998](#); [Fahlke and Hansen, 1999](#); [Russell et al., 1998](#)) also have implicated dopamine and its associated structures.

#### Noradrenergic Hypothesis

The noradrenergic system also has been implicated in a number of ways. [Mefford and Pothier \(1989\)](#) hypothesized that an imbalance in tonic epinephrine formation would disrupt the normal inhibition of locus ceruleus neurons, resulting in inattention, distractibility, sleeping difficulties, and some cognitive deficits. The authors suggested that this may be the underlying problem in ADHD.

[Oades et al. \(1998\)](#) showed that plasma levels of norepinephrine and epinephrine were slightly elevated, but urinary levels of norepinephrine and serotonin metabolites were markedly elevated in children with ADHD versus healthy control subjects. Similarly, [Halperin et al. \(1997\)](#) showed that plasma levels of the noradrenergic metabolite 3-methoxy-4 hydroxyphenylglycol were higher in children with ADHD and reading disabilities compared with such children without reading disabilities. [Pliszka et al. \(1996\)](#) reviewed a large number of studies involving plasma and urinary levels of epinephrine and concluded that children with ADHD may show higher levels of norepinephrine activity and lower levels of epinephrine activity.

[Biederman and Spencer \(1999\)](#) clearly reviewed the studies showing the efficacy of noradrenergic agents, such as tricyclic antidepressants, bupropion, monoamine oxidase inhibitors, novel adrenergic agents, tomoxetine, and a  $\alpha_2$ -noradrenergic agonist (clonidine and guanfacine) in subjects with ADHD. The efficacy of these noradrenergic agents suggests the importance of the noradrenergic system in this condition.

#### Serotonergic Hypothesis

This hypothesis is based on the fact that serotonin-depleted animals show increased aggression and hyperactivity. In addition, hyperactive subjects have shown inconsistent changes in platelet and blood 5-hydroxytryptamine (serotonin). The hypothesis was supported by results of drug studies involving tricyclic antidepressants and monoamine oxidase inhibitors, which affect serotonin metabolism and are moderately effective for the symptoms of hyperactivity, impulsivity, and inattention. However, pharmacologic studies involving L-tryptophan, a serotonin precursor ([Nemzer et al., 1986](#)), and fenfluramine, which acutely increases and then depletes brain serotonin, showed no consistent results.



Similarly, in a small open study, [Barrickman et al. \(1991\)](#) suggested fluoxetine (a selective serotonin reuptake inhibitor) may be beneficial in treating children with ADHD. Most clinical experience ([National Institute of Mental Health, 1996](#)) did not support this finding. However, [Spivak et al. \(1999\)](#) showed that children with severe ADHD tended to have lower blood levels of serotonin compared with children with mild ADHD.

[Roffman and Raskin \(1997\)](#) also implicated the serotonergic system when they showed in an animal study that the serotonin synthesis inhibitor p-chlorophenylalanine significantly affected stimulant-induced stereotypic behavior in these animals. However, it can be concluded that if serotonin plays a role in ADHD, it is not a central but rather an adjunctive role to one or more of the other neurotransmitters.

#### *Nonspecific Catecholamine Hypothesis*

It is clear from the foregoing discussion of specific neurotransmitter systems that not one but several neurotransmitter systems are involved in ADHD. In an early review, [Zametkin and Rapoport \(1987\)](#) concluded that there is no basis to believe that a single neurotransmitter abnormality is responsible for symptoms of ADHD. Subsequently, [Rogeness et al. \(1992\)](#) reviewed the three main neurotransmitters that may influence behavior problems in children: dopamine, norepinephrine, and serotonin. They suggested that ADHD is best understood by the *interaction* of multiple neurotransmitters. Specifically, they stressed that the *balance* between the norepinephrine and dopamine systems is critical, rather than variations in the individual systems. Furthermore, the authors stated that the development of individual systems from infancy to adulthood is influential on behavior because this process affects the relative activity of the systems based on neuronal maturation. Hence, results of past studies may have been confusing because multiple neurotransmitters were not studied at once and narrow age ranges were not used.

[Pliszka et al. \(1996\)](#) also presented a multistage hypothesis, which emphasized the interaction of norepinephrine, epinephrine, and dopamine in the modulation of attention and impulsivity.

[McCracken \(1991\)](#) was more specific in his hypothesis and stated that for a drug to be effective in treating ADHD, it needed to increase both dopamine release and adrenergic inhibition in the locus ceruleus.

Similarly, [Mercugliano \(1995\)](#) stressed that the most effective drugs in the treatment of ADHD are those that increase transmission of dopamine and norepinephrine.

[Arnsten et al. \(1999\)](#) also stressed the importance of optimal levels of norepinephrine and dopamine receptor stimulation. The authors suggest that too little or too much receptor stimulation in response to stress impairs neuronal activity in the prefrontal cortex and subsequent cognitive function, such as working memory. Further support for the interrelation and importance of norepinephrine and dopamine has been provided by [Lanau et al. \(1997\)](#), who showed that both transmitters are potent agonists at the dopamine D4 receptor.

#### *Other Neurotransmitters*

Other neurotransmitters, such as g-aminobutyric acid (GABA), which is thought to be a predominantly inhibiting neurotransmitter in the CNS, and histamine, which acts centrally and peripherally, have not been studied in relation to the symptoms outlined, but may be implicated in the future.

[Mrzljak et al. \(1996\)](#), however, pointed out that dopamine D4 receptors are abundant in GABAergic interneurons in prefrontal cortex. Furthermore, [Prosser et al. \(1997\)](#) showed that plasma GABA levels were elevated in nonmedicated behaviorally disordered children. These levels were seen to be lower after medical treatment.

[Passani et al. \(2000\)](#), in an extensive review of the neurotransmitter histamine, suggest that although the neurotransmitter is found in the tuberomammillary nuclei of the hypothalamus, these cells' widespread projections to the cerebral cortex, thalamus, and pontomesencephalic tegmentum extend its effect to many functions. The histaminergic system is thought to act as a regulatory center for whole-brain activity. It also is thought to be involved in the regulation of physiologic functions and behavior, including learning and memory. Histamine's effects on cognition might be explained by its modulation of the cholinergic system.

Clearly, little research has involved these other neurotransmitter systems that also may play a role in the symptoms of inattention, hyperactivity, and impulsivity.

In summary, much work remains to be done in trying to untangle the role and relative importance of the various neurotransmitter systems, their interaction, the regulation of an optimal balance, and all the factors that may disrupt this interaction and balance.

#### *NEUROENDOCRINE ASPECTS*

The interest in neuroendocrine aspects in children with symptoms of hyperactivity, impulsivity, and inattention comes from two main but interrelated areas. The preceding discussion suggested that norepinephrine, dopamine, and serotonin may be involved in the symptoms described. However, these same neurotransmitters are known to affect hormone production and release in the anterior pituitary and thus affect the hypothalamic–pituitary–adrenal axis and the hypothalamic–pituitary–growth hormone axis. Reports of possible growth suppression in children given stimulants to treat symptoms of hyperactivity, impulsivity, and inattention have resulted in considerable interest in the growth hormone axis in these children. A number of studies investigated growth hormone levels in children with these symptoms on and off stimulant medication and in matched, normal control subjects. In a series of studies, [Greenhill and colleagues \(1981\)](#) showed no change in the sleep-related pattern of human growth hormone secretion before or after treatment with dextroamphetamine. An insulin tolerance test also showed no differences in growth hormone response. However, boys who received long-term methylphenidate treatment for their symptoms showed elevations of sleep-related growth hormone compared with normal control subjects ([Greenhill et al., 1984](#)). [Shaywitz et al. \(1982\)](#) studied hyperactive children treated with methylphenidate and found that growth hormone levels increased with plasma levels of methylphenidate, both peaking at the same time. [Garfinkel et al. \(1986\)](#), exploring the effect of dextroamphetamine in hyperactive adolescents, found that the growth hormone response in these subjects was greater than that of normal control subjects and that it peaked at the point of maximal absorption and clinical effect of the amphetamine. [Weizman et al. \(1987\)](#) studied the effects of acute and chronic methylphenidate administration on b-endorphin, growth hormone, prolactin, and cortisol levels in children with these symptoms. They found that with chronic use of methylphenidate, the results of growth hormone challenge tests were augmented. [Hunt and colleagues \(1984\)](#) demonstrated growth hormone response to clonidine administration in a similar group of children.

The complexity of the neuroendocrine issue has been well illustrated by [Jensen and Garfinkel \(1988\)](#), who looked at prepubescent and pubertal hyperactive and control children and measured their growth hormone response to L-dopa and clonidine challenges. They found that the growth hormone response was lower in the prepubescent children, and also lower to the L-dopa than to the clonidine challenge. In addition, the response in the hyperactive children was lower than that in control subjects. This clearly suggests that both the dopamine and norepinephrine systems are involved in growth hormone release and that this process is affected by pubertal development and stimulant medication.

The interest in thyroid hormone and its association with symptoms of hyperactivity and impulsivity was first sparked by [Hauser et al. \(1993\)](#) when they reported high rates of these symptoms in people affected by a genetic disorder that causes generalized resistance to thyroid hormone. However, this is a very rare condition, and subsequent studies (e.g., [Toren et al., 1997](#)) that have explored thyroid function in children with ADHD showed no thyroid abnormalities in these children. [Sawada et al. \(2000\)](#) suggested that given the significant sex difference in ADHD prevalence, estrogen may exert a neuroprotective role and thus prevent the development of the condition. Possible mechanisms for the neuroprotective effects of estrogen suggested by the authors included protection of estrogens as transcription factors and protection against oxidative stress by estrogens acting as antioxidants.

Conversely, [King et al. \(2000\)](#) found that in an animal model involving spontaneously hypertensive rats (SHR), which have been used as an animal model for ADHD, early testosterone treatment of SHR resulted in additional deficits in spatial memory in the water maze. In addition, the SHR had high basal adrenocorticotropic hormone and low corticosterone levels, suggesting a dysfunctional stress axis similar to that in humans with persistent ADHD (for review, see [Ryan, 1998](#)). Finally, there also was a further suppression of tyrosine hydroxylase immunoreactivity in the frontal cortex of androgen-treated SHR. The authors thus suggest that early androgen treatment may enhance the expression of hyperactivity and inattention in genetically predisposed animals and subjects.

In summary, neuroendocrine systems, particularly the hypothalamic–pituitary–adrenal axis, may be involved in ADHD. Involvement of growth hormone, cortical estrogen, and androgen have all been suggested. However, to date, the few studies that have been done had small, diverse populations. Clearly, more well-controlled

research is needed on young, prepubertal and pubertal populations to help clarify the role of neuroendocrine systems in this condition.

## NEUROPHYSIOLOGIC ASPECTS

In an effort to determine whether neuronal responses differ significantly in children with symptoms of hyperactivity, impulsivity, and inattention, researchers have examined the central and peripheral neural conducting responses in children with these symptoms. Centrally, these studies have involved EEGs and evoked potential recordings, and peripherally they have focused on motoneuron excitability. In reviewing many of these neurophysiologic studies ( [Weiss and Hechtman, 1993](#)), one is struck by the variability of the findings and the difficulty in coming to firm conclusions.

It has been estimated ( [Cantwell, 1980](#)) that 35% to 50% of children with ADHD symptoms may have abnormal EEGs. The most common abnormality reported involves diffuse, nonspecific changes and extensive slow EEG activity ( [Grunwald-Zuberbier et al., 1975](#); [Satterfield, 1973](#)). [Shetty \(1973\)](#), in his series of 75 hyperactive subjects, indicated that the most consistent abnormality was the absence of an appropriate amount of well-organized alpha waves for age.

Another EEG abnormality that has been described in hyperactive children involves difficulties with attenuation of alpha waves, which presumably reflects the decreased capacity of these children to stop attending to redundant events ( [Fuller, 1977](#)).

All of the aforementioned abnormalities have been described as immature patterns and interpreted as representing delayed CNS maturation in these children. Some support for this hypothesis was provided by [Hechtman and colleagues \(1978\)](#) when they compared sequential EEGs of hyperactive children, adolescents, and young adults and found increasing normalization of EEGs with increasing age, particularly in late adolescence.

Evoked potential studies have been inconclusive, giving conflicting results. [Satterfield \(1973\)](#) reported that the auditory evoked potentials in hyperactive subjects had longer latencies and lower amplitudes compared with age-matched control subjects. On the other hand, [Buchsbau and Wender \(1973\)](#), studying visual and auditory average evoked responses, showed increased latencies and higher amplitudes with increases in intensity of visual stimuli. Age- and sex-matched control subjects showed lesser increases in amplitude and decreases in latency. [Hall et al. \(1976\)](#), recording evoked potentials in response in four stimulus intensities under conditions of attention and inattention, found no differences in stability, amplitude, or latency between hyperactive children and control children. Most of these studies recorded evoked potentials at one or, at most, a few electrode sites.

[Satterfield et al. \(1988b\)](#) conducted a study on 20 6-year-old children with attentional problems and 20 matched, normal control subjects, using 19 recording sites and 2 choice discrimination tasks. They concluded that the children with attentional problems had abnormalities in the mismatch process, resulting in poor discrimination of salient stimuli. This is an automatic process not under voluntary control. It is thought to be a component of a basic orienting response that is mediated by the reticular activating system in unanesthetized animals ( [Aston-Jones, 1985](#)). A second abnormality found by [Satterfield et al. \(1988a\)](#) involved frontal processing negativity, reflecting additional processing (e.g., rehearsing of the standard in memory) of the attended stimulus. This activity is thought to be under voluntary control. Thus, the multisite recording studies illustrate the great complexity of these functions.

More recently, [Baving et al. \(1999\)](#) obtained baseline EEG recordings in 117 children (66 aged 4 years, 51 aged 8 years) with ADHD. The recordings were subjected to power spectral analysis and laterality scores were obtained. Boys with ADHD exhibited less right-lateralized frontal activity pattern than normal control boys. Girls with ADHD displayed more right-lateralized frontal activity pattern than normal control girls. This finding applied to children in both the age groups. The authors point out that this pattern of frontal brain activation is consistent with MRI studies of ADHD.

[Halliday and colleagues \(1983\)](#) investigated the effect of methylphenidate on visual evoked potentials in hyperactive children and concluded that the evoked response potential measures were more sensitive to age and attention conditions and less to methylphenidate dose. Peripheral studies that have looked at motoneuron excitability also give conflicting results. Thus, [Pivik and colleagues \(1986\)](#) showed that some hyperactive children had significantly elevated, whereas others had significantly depressed responses compared with matched control subjects.

The divergent, inconsistent, and inconclusive findings of these neurophysiologic studies may be related to the fact that we are attempting to understand complex, multifaceted central processes using fairly gross and perhaps nonspecific peripheral measures. Recent technological advances, which include measures of cerebral blood flow and glucose utilization, and positron-emitting liquids that can label and quantitate specific neurotransmitter receptors, will enable researchers to examine the functions of hyperactivity, impulsivity, and inattention with much greater precision centrally and peripherally, and may shed light on the many questions that remain.

## CONTRIBUTING PSYCHOSOCIAL FACTORS

In this section, we discuss possible psychosocial factors that can give rise to or complicate symptoms of hyperactivity, impulsivity, and inattention. Furthermore, these symptoms can significantly affect the person's emotional, social, and academic development. It is clear that relationships with parents, teachers, and peers, academic success, and self-esteem are all directly or indirectly influenced by these symptoms. Needless to say, these social and emotional parameters may in turn affect self-esteem and motivation and further augment the handicaps associated with the primary symptoms. Thus, the biological, social, academic, and emotional spheres all are interrelated and significantly and continually influence each other.

### Environmental Factors

It has been suggested that the physical environment may significantly contribute to the development or worsening of symptoms of hyperactivity, impulsivity, and inattention. Some authors (e.g., [Needleman, 1983](#)) have suggested that children living in environments where they are exposed to high lead levels are more likely to develop such symptoms. Lead, however, cannot account for the bulk of ADHD cases. Many children with ADHD symptoms show no lead contamination, and many children with high lead exposure do not acquire ADHD.

The idea that certain foods may contribute to the development of ADHD received attention when it was claimed that ADHD could be cured by eliminating food additives from the diet ( [Feingold, 1975](#)). However, systematic studies showed that diet was not effective and concluded that food additives do not cause ADHD ( [Conners, 1980](#)). Another dietary theory suggested that excessive sugar intake can lead to symptoms of hyperactivity, impulsivity, and inattention. Although some studies seemed to support this hypothesis, most systematic, controlled studies did not ( [Wolraich et al., 1995](#)).

The association between ADHD symptoms and pregnancy and delivery complications is not consistently positive, but on balance, the literature supports the idea that pregnancy and delivery complications can predispose children to ADHD ( [Sprich-Buckminster et al., 1993](#)). The specific pregnancy and delivery complications that have been implicated include toxemia or eclampsia, poor maternal health, maternal age, fetal postmaturity, long duration of labor, fetal distress, low birth weight, and antepartum hemorrhage.

Maternal smoking during pregnancy has been shown to increase the risk of antepartum hemorrhage, low maternal weight, low fetal weight, and abruptio placentae ( [Kline et al., 1989](#)). Maternal smoking also increases carboxyhemoglobin levels in both maternal and fetal blood, thus increasing the risk of fetal hypoxia ( [Fielding, 1985](#)). Both low birth weight and fetal hypoxia have been implicated in the etiology of ADHD. Maternal smoking also exposes the fetus to nicotine, which can damage the brain at critical times in the developmental process. Animal studies of pregnant mice and rats have shown a positive association between chronic exposure to nicotine and hyperactive offspring ( [Johns et al., 1982](#); [Slotkin et al., 1993](#); [Van de Kamp and Collins, 1994](#)).

It is unclear how significant a physically chaotic environment may be in contributing to the development of symptoms of hyperactivity, impulsivity, and inattention, although such an environment certainly is important in maintaining and augmenting these difficulties.

The prevalence of children with hyperactivity, impulsivity, and inattention is greater in disadvantaged, larger inner city environments. However, such settings also have more poverty, malnutrition, poor prenatal, perinatal, and neonatal health care, and more family violence. Any combination of these factors also may play a significant contributory role in the development and perpetuation of these symptoms.

Similarly, a school or classroom setting that is very unstructured or disorganized has a detrimental effect on children with these symptoms, causing their behavior to



deteriorate and their learning and academic achievement to be significantly impaired.

[Taylor \(1985\)](#) has pointed out the importance of cultural, social, and family variables in learning to focus attention and control impulsivity and hyperactivity. This process of socialization may be influenced by characteristics of the parents, the child, and the reciprocal interactions that develop between them. Increased psychopathology has been reported in parents of children with hyperactivity, impulsivity, and inattention ([Morrison, 1980](#); [Stewart et al., 1980](#)). This may suggest the genetic vulnerability of these children, but it also may result in problematic parenting, which can contribute to the development or perpetuation of these symptoms in the child. Parenting styles of parents whose children have symptoms of hyperactivity, impulsivity, and inattention have been looked at by a number of authors ([Campbell, 1973](#); [Humphries et al., 1978](#); [Mash and Johnston, 1982](#)). In general, these researchers found that parenting styles tended to more intrusive, controlling, and disapproving (particularly in structured tasks as opposed to free play) compared with parents of age-matched, normal control subjects. This negative parenting style improved significantly when the child's behavior improved because of stimulant medication treatment ([Humphries et al., 1978](#)). It therefore was concluded by [Humphries et al. \(1978\)](#) that the parents were responding to the child's behavior and not causing it. [Mash and Johnston \(1983\)](#) showed that having a child with these difficulties affects the parents' confidence in their parenting skills and results in more reported stress, social isolation, self-blame, and depression in the parents. We can, thus, clearly see the effects of this negative parenting style on the child's emotional development, competence, and self-esteem. Similarly, the child's problems affect the parents' sense of effectiveness and emotional well-being, which, in turn, reverberates in their interactions and relationship with their child. The same vicious cycle of negative interactions, stress, and sense of failure for both child and teacher can become established in the school, with similar detrimental effects.

It has been suggested that the symptoms of hyperactivity, impulsivity, and inattention may be expressions of underlying anxiety or depression. [Tizard and Hodges \(1978\)](#) showed that overactivity and inattention were prominent in children growing up in institutions. It is hypothesized that the underlying anxiety resulting from a lack of secure and stable relationships is expressed through these symptoms. Similarly, children raised in a disturbed family environment may experience similar insecurities and anxieties and express these feelings in a similar way. Children can show symptoms of hyperactivity, impulsivity, and inattention during acute periods of stress. However, these periods usually are of short duration (a few days or weeks), and the stress usually is readily identifiable (e.g., parental separation, a new sibling).

For children who live in chronically stressful, multiproblem circumstances, it is difficult to determine whether the symptoms reflect expression of underlying anxiety or depression, a problem in parenting and socialization, a genetically influenced biological problem, or some interaction of a number of these factors.

Relatively little research has addressed the psychosocial versus the biological aspects of these symptoms. The combination and interrelationship of both of these aspects has received even less attention. It is hoped that with current technological advances and our greater ability to look at such problems in a multifaceted way, we will gain a clearer, more comprehensive understanding of the possible etiology, development, and treatment of symptoms of hyperactivity, impulsivity, and inattention.

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## 29 DEVELOPMENT OF THE SYMPTOMS OF VIOLENCE

Dorothy Otnow Lewis, M.D.

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What do we know about human violence? Are human beings aggressive by nature? Must we be tamed to treat each other properly? And, if so, how should we go about that task? If we spare the rod, will we spoil the child? Was Wordsworth right—do we come into the world in a state of innocence, “trailing clouds of glory,” only to be corrupted by society? Rousseau thought so. Was Locke correct? Do we enter this world blank slates on which our environments and experiences will scrawl our course? Are “all men created equal,” or are some of us innately more aggressive than others? These questions, which for millennia have dominated literature, religion, and philosophy, are finally being tackled by science. And, as will become clear, there is something to be said for each of the foregoing points of view. We enter the world with certain innate but not inevitable potentials for behavior. What happens to us makes the difference.

The nature of human behavior is so complex that it is hard to agree on the very meaning of human aggression. Are we talking about the battles between Microsoft and Netscape or Serbia and Bosnia? Are we considering the behaviors of Lucretia Borgia or Lizzie Borden? John D. Rockefeller or Jack the Ripper?

The behaviors of other animals are more stereotyped than those of human beings and are therefore more easily classified. For example, [Moyer \(1971\)](#) has categorized animal aggressive behaviors as follows: predatory, intramale, fear induced, irritable, territorial, maternal, and instrumental. [Flynn and colleagues \(1970\)](#) have condensed these aspects of aggression into two categories, affective and predatory. Affective aggression, which includes intramale, fear-induced, irritable, territorial, and maternal types, involves intense activation of the sympathetic nervous system. There is an increase in blood pressure, piloerection, pupillary dilation, and threatening behaviors. Predatory aggression, on the other hand, evokes little autonomic activation and is accomplished by silently stalking the prey and expeditiously subduing it in the interest of providing a meal. Others have divided animal behaviors into offensive and defensive ([Blanchard and Blanchard, 1977](#)) and spontaneous versus conditioned aggression ([Thompson, 1969](#)). For the purposes of this chapter, the term *human aggression* is used simply to denote behaviors by one person intended to cause pain, damage, or destruction to another. It is not used as a synonym for assertiveness or ambitiousness.

[Volavka \(1999\)](#) suggests that most human violence can be categorized as impulsive or premeditated. Others have conceptualized two kinds of aggression: defensive, characterized by fear and high plasma cortisol levels, and offensive, characterized by impulsiveness and low serotonergic brain activity, high testosterone levels, and low cortisol levels ([Kalin, 1999](#)). The Federal Bureau of Investigation classifies murderers as organized and disorganized ([Douglas and Olshaker, 1995](#)), categories roughly similar to Volavka's. Although some investigators report apparent physiologic and cognitive differences between impulsive offenders and those who premeditate their acts ([Barratt, et al., 1997a, 1997b](#)), it would be a mistake to assume that those who premeditate their violence are necessarily healthier or more neuropsychiatrically intact than impulsive offenders (current insanity statutes to the contrary notwithstanding). Some of the most violent, diabolical, psychotic acts are planned over months and even years. Copious writings of the Unibomber attest to this truth.

But what about wars? Surely, the attacks on Pearl Harbor and Hiroshima cannot be discounted as the products of madmen. Some of the canniest (if not best) minds were behind them. Clearly, violence does not always bespeak “insanity.”

### EVOLUTION AND VIOLENCE

Before “attacking” the question of whether within the human species some of us are innately more aggressive than others, it would be wise to take distance and examine ourselves as a species. Does it matter that we *Homo sapiens sapiens* are, in essence, a variety of ape? It does. [Wrangham and Peterson \(1996\)](#) note that of 4,000 mammals, only chimpanzees and humans live in patrilineal, male-bonded communities in which males form “aggressive coalitions with each other in mutual support against others” (p. 24). For centuries, it was assumed that humans were the only animals that actually made war on each other. Jane Goodall's observation of raiding parties of male chimpanzees and of their gratuitous violence toward already subjugated members of other chimpanzee communities put that misconception to rest, while taking us humans down a peg ([Goodall, 1986, 1991](#)).

Since Darwin, we have had some inkling of our primate origins. However, not until the discovery of the structure of deoxyribonucleic acid (DNA) and the ability to type its variations could we appreciate our evolutionary closeness to the “other” great apes, especially to the chimpanzee. In 1904, [Nuttall \(1904\)](#) reported that human blood did not react with rodent antisera, precipitated moderately in the presence of monkey antisera, and formed the thickest precipitant of all when mixed with ape antisera. Thus, began the biochemical documentation of our closeness to apes. Subsequent studies comparing the structure of gorilla, orangutan, chimpanzee, and human DNA indicated not only that humans are genetically more closely related to chimpanzees than we are to any other known species, but that the DNA of chimpanzees themselves is more similar to our DNA than it is to that of the other great apes (e.g., gorillas) ([Sibley, et al., 1990](#)). Given our biological and behavioral similarities to the great apes, particularly to the chimpanzee, it is not unreasonable to assume that at least some of our human aggressiveness can be explained in terms of evolution. Such aggressiveness must, at some time, have had survival value. Whether this large brain of ours, with its potential for destruction as well as reflection, will continue to serve us well remains to be seen.

### SEX AND AGGRESSION

Are all humans equally aggressive? Clearly not. The males of our species, like male chimpanzees, are more violent than females—no doubt about it. Whether measured in childhood in terms of roughness of play ([Maccoby and Jacklin 1974](#)) or in adulthood in terms of violent crime ([Federal Bureau of Investigation, 1998](#)), the male human behaves in demonstrably more aggressive ways than the female. These differences are not peculiar to a particular society but have been observed cross-culturally in societies as different from each other as Ethiopia and Switzerland ([Omark et al., 1973; Whiting and Edwards, 1973](#)). In the United States, men are approximately eight times as likely to commit murder, nine times as likely to commit armed robbery, and four to five times as likely to commit aggravated assault as women ([Federal Bureau of Investigation, 1998](#)). Men also are many times more likely than women to commit nonviolent crimes such as burglary, car theft, drunken driving, and even fraud.

The greater inherent aggressiveness of the male human also is reflected in the patriarchal nature of almost all human societies ([Wrangham and Peterson, 1996](#), p. 118). In fact, a 1971 survey of 93 societies around the world revealed that men retained all important political positions in 88% of them and that in most societies men tended to hold all significant leadership roles in kinship groups as well. Most surprising is the finding that maltreatment of women by men (i.e., wife beating) is more the rule than the exception in societies the world over ([Heise et al., 1994; Levinson, 1989](#)). Among mammals, rape reportedly is common in but two nonhuman mammals (orangutans and elephant seals), and occurs occasionally in chimpanzees, gorillas, and howler monkeys ([Galdikas, 1995; Palmer, 1989; Wrangham and Peterson, 1996](#)). Thus, in addition to human beings, four of the five mammals known to rape are primates; three are apes. Needless to say, men are many times as likely to commit rape as women. The human male is, in short, the predominant maker and breaker of the rules in human society.

Given the overwhelming evidence that human males are more violent than females in nearly all societies, before considering the cultural or environmental contributions to human violence, it makes sense to examine what we know about our own biology. What factors seem to contribute to greater male violence?

Much as we may wish to attribute most, if not all, of the differences between human male and female aggressiveness to cultural influences, overwhelming evidence exists that, long before birth, the die is cast; male and female brains develop differently *in utero*. The architectural and functional differences between male and female brains are, to this day, “areas” of controversy. Whereas some investigators have noted marked differences in what are referred to as the *sexually dimorphic* areas of the hypothalamus and corpus callosum (Allen et al., 1989, 1991; Arai et al., 1985), others have highlighted the similarities between male and female brains. Nopoulos and colleagues (2000), in their magnetic resonance imaging (MRI) study, stressed the similarities between male and female brains. However, they found that human males have larger brains than females and, what is more, the greatest size discrepancies are in the cerebrum, that alleged repository of reason and impulse control. The male cerebrum is reportedly 8% larger than the female.

Gonadal hormones play important roles in the development of aggressive behaviors. Gonadal hormones are especially critical during fetal and neonatal development (Goy and McEwen, 1980). These hormones have been shown to affect sexually dimorphic areas of the brain, and their early presence has been reported to be important to the normal development of aggressive behaviors in many animals. The importance of androgens to fighting behaviors or dominance has been demonstrated in such diverse species as fish, lizards, birds, and chimpanzees (Floody and Pfaff, 1972). However, when severely threatened, even castrated animals have been able to fight fiercely (Barfield, 1984). Estrogens have been observed to have varying behavioral effects, depending on the state of the recipient. On the other hand, in some species, when given prenatally, estrogen has been associated with masculine brain development and aggressive behavior (Gorski, 1986; Paup et al., 1972; Ruppert and Clemens, 1981). Androgens such as testosterone are metabolized in the brain to form estrogens (Reddy et al., 1974).

It is now well recognized that estrogens influence the sexual differentiation of the male and female brain and thus influence behavior (Hutchison et al., 1999). Estrogens have been found to influence dendrite formation and the formation of synapses (Garcia-Segura et al., 1986; Woolley and McEwen, 1994). They have permanent organizing effects on the structure and function of the brain during critical prenatal periods. The transformation of testosterone to the estradiol essential to brain differentiation requires activation of the cytochrome P-450 aromatase enzyme (Kelly et al., 1999; Reznikov et al., 1999). *In vitro* studies of neurons indicate that neurons from the male fetal hypothalamus have higher aromatase activity than neurons from the female fetal hypothalamus. Furthermore, a greater number of neurons from male brains contain aromatase than do neurons from female brains (Hutchison et al., 1999). Testosterone, in turn, increases aromatase activity and greatly increases estradiol formation. However, cells containing aromatase reportedly respond only during critical developmental phases. The actual molecular mechanisms that are activated in response to estradiol and masculinize the fetal brain are not yet understood. What we do know is that testosterone activates the aromatase in the brain; it is converted to estradiol; estradiol further masculinizes the brain; and these changes, in turn, affect postnatal behavior. The relationship of the masculinization of the fetal brain to the production of neurotransmitters and receptors in the brain is not yet well understood (Reznikov et al., 1999), however, differences in the amount, turnover, and localization of catecholamines have been documented in the brains of male and female rats. The effects on behavior of sex differences in the central nervous system (CNS) are extremely complex. “Steroid derivatives progesterone, testosterone, and glucocorticoids, referred to as neuroactive steroids or neurosteroids, can also alter behavioral responses through nongenomic actions, and supports their potential role in mediating sex-related differences” (Kelly et al., 1999, p. 659). The behavioral effects of neurosteroids are numerous and include sedative, anesthetic, and anxiety-reducing properties. They are thought to play important roles in mediating sex-related differences in behavior responses (Kelly et al., 1999). Exactly how biochemical events are translated into behaviors is not known.

In summary, a plethora of data from such diverse sources as archeology, anthropology, genetics, and biochemistry tell us that we are a potentially aggressive species, and that the males among us are, by nature, more aggressive than the females. But is that the whole story? Is biology destiny? Not necessarily. After all, most men are not murderously violent, and not all women are “shrinking violets.” Why, then, within our species, are some of us more violent than others?

## FROM THE SOCIOLOGIC TO THE BIOCHEMICAL—A TREND

In the middle of the 20th century, at least in part in reaction to Lombroso's earlier theories that criminals represented a degenerate biological phenomenon (Alexander and Selesnick, 1966), investigators looked almost exclusively to society for explanations of criminality. Merton (1938, 1957), one of the most influential social scientists of the time, noting that criminality and violence were considerably more prevalent in the disadvantaged sectors of society than the well-to-do, theorized that antisocial behavior was a manifestation of the efforts of the socioeconomically deprived to attain, by hook or by crook, the wealth and status that the more successful and dominant sectors of society could obtain legitimately. Other social scientists hypothesized that deviance was basically a reflection of society, and that subcultures of violence encouraged criminality (Cohen, 1955). Children became delinquent and violent because of their exposure to deviant behaviors (Cloward and Ohlin, 1960). Criminality was, in essence, an alternative lifestyle.

The past quarter century has seen an almost 180-degree about-face in the study of violence and criminality, as investigators have turned back to biology to try to explain deviance, especially to explain violence. Advances in the so-called hard sciences (e.g., genetics, biochemistry, cellular biology, neuroanatomy, neurophysiology), increasing our ability to identify, measure, and even visualize biological similarities and differences among us macroscopically and microscopically, have undoubtedly encouraged this research direction. Studies of the relationship of such neurochemicals as serotonin (Sahakian, 1981), norepinephrine (Stolk et al., 1984), dopamine (McKenzie, 1971), vasopressin (Coccaro et al., 1998), and nitric oxide (Nelson et al., 1995) to aggressive behavior in animals (Alpert et al., 1981; Lal et al., 1968; Lycke et al., 1969; Randrup and Munkvad, 1966; Reis, 1972) encouraged similar studies in humans. Initially, the finding of a relationship between low cerebrospinal fluid (CSF) levels of 5-hydroxyindole acetic acid (5-HIAA; a metabolite of serotonin) and impulsiveness and aggressiveness in humans (Brown et al., 1979; Brunner, 1996; Linnoila et al., 1983; Virkkunen et al., 1989) seemed to shed light on the question of why some individuals were more aggressive than others. Studies of men depleted of tryptophan, the amino acid precursor of serotonin, indicated that they become more aggressive in a laboratory setting than do men not so deprived (Bjork et al., 1999). Of note, those men with a tendency toward hostility as measured on an aggression questionnaire reportedly responded more aggressively to tryptophan depletion than did nonhostile men.

However, the relationship of serotonin levels and serotonin system responsiveness to degrees of aggression is not simple. A study of young (7- to 11-year-old) aggressive boys with attention deficit/hyperactivity disorder revealed a *greater* prolactin response to fenfluramine (i.e., greater serotonergic responsivity) in aggressive than in nonaggressive boys (Halperin et al., 1996). A study of blood serotonin [5-hydroxytryptamine (5-HT)] levels in adolescents diagnosed with conduct disorder reported that those with early-onset maladaptive behaviors had *higher* blood 5-HT levels than later offenders and that blood 5-HT correlated positively with ratings of aggressiveness (Unis et al., 1997).

In a sophisticated study of the relationship of brain 5-HIAA levels in infants to subsequent behavioral characteristics, Clarke and colleagues (1999) followed a sample of children from their first weeks of life to 30 months of age. They found but a modest correlation between low CSF 5-HIAA levels and aggressive behaviors. In contrast, environmental factors such as poverty, as reflected in Medicaid insurance, and poor family cohesiveness were strongly associated with behavior problems at 30 months of age. Another recent study documented a relationship between low socioeconomic status, low prolactin response, and increased aggression (Matthews et al., 2000). Finally, a metaanalysis of 39 studies linking 5-HIAA levels to violence in humans found that psychiatric patients as a group, whether violent or nonviolent, had similar 5-HIAA levels. The mean 5-HIAA level in both violent and nonviolent patient groups was lower than the mean level in groups of normal, healthy volunteers (Balaban et al., 1996). Thus, serotonin levels may prove to be indicators of overall psychopathology that only at times is manifested by aggressiveness.

## MENTAL ILLNESS AND AGGRESSION

Does mental illness itself predispose an individual to violence? For years, psychiatrists promoted the notion that mental illness is no different from any other kind of physical illness in terms of aggressiveness. That is not so. Studies from around the globe, including studies from more and less developed nations, indicate that violence is more common among seriously mentally ill individuals than it is among the general population. In a study of patients with schizophrenia from 10 different countries around the world, the occurrence rate of assault was extremely high (20.6%), especially in “developing countries” (31.5%) compared with “developed countries” (10.5%) (Volavka, 1997). The study of a Danish birth cohort revealed that hospitalized patients diagnosed with schizophrenia had higher arrest rates for violent crime than did nonpatients (Brennan et al., 2000). Monahan (1997) reported that, in a study of psychiatric patients from three U.S. cities, those diagnosed with affective disorders had the highest rates of violence postdischarge. In a study of 812 psychiatric patients, within 6 months after discharge, almost half of the patients behaved violently. Of those who became violent, 16% of the men and 31% of the women had an affective disorder (Newhill et al., 1995). In a much smaller follow-up study of male psychiatric patients, those with major affective disorder were significantly more likely to commit violent crimes than were those diagnosed with schizophrenia (Hodgins et al., 1999). Hodgins et al. distinguished between those with early antisocial behaviors and those whose antisocial behaviors started concomitant with the onset of affective disorder (Hodgins et al., 1998). We have found in our own clinical work that many manic-depressive, delinquent adolescents, whose hypomanic, problematic behaviors started in childhood, were dismissed then as having conduct disorders and only later identified as suffering from bipolar



disorders. When a careful history of the periodicity of antisocial behaviors was obtained, as well as a family psychiatric history, it became evident that many of the adolescents had experienced extremes and fluctuations of mood since childhood that contributed to their social maladaptation. It is vital that some child psychiatrists, confronted with a child's repeated misbehavior, consider the possibility that the child may be manic or hypomanic.

Another psychiatric disorder associated with episodic extreme violence is posttraumatic stress disorder (PTSD) ([McFall et al., 1999](#)). The more serious the symptomatology, the greater the violence. Child abuse seems to intensify PTSD symptoms in subsequently traumatized individuals ([Bremner et al., 1993](#)). Homicidal violence has been perpetrated by individuals with severe dissociative disorders, especially by men with dissociative identity disorder ([Lewis et al., 1997](#); [Loewenstein and Putnam, 1990](#)). This disorder comes into being as a result of early, ongoing, extreme maltreatment, the psychobiological effects of which are discussed later.

One of the rarest and therefore most overlooked causes of episodic recurrent violence in children is the acute confusional state that is sometimes associated with the development of migraines. During these attacks, children may appear confused, agitated, hyperactive, and even violent. These states, considered to be auras associated with migraines, can last from minutes to hours. They may occur before, during, after or independent of the migraine headaches themselves ([Eggers, 2001](#)). During these episodes, children may destroy objects and even attack others who attempt to restrain them. Children may experience trance-like or dreamlike states, macropsias, or micropsias, olfactory, visual, and auditory hallucinations, déjà vu, and just about all of the symptoms associated with complex partial seizures ([Ardila and Sanchez, 1978](#)). Sometimes the episodic, agitated, aggressive auras precede by years the development of migraine headaches, making the diagnosis difficult. Often the diagnosis must be made retrospectively, after onset of the headaches ([Ehyai and Fenichel, 1978](#); [Steifel and Ruttner, 1995](#)).

Granted, mental illness is associated with violence. Nevertheless, most mentally ill individuals are not violent. They are, rather, more vulnerable to the stressors in their immediate and extended environments.

## GENES AND AGGRESSION

Scientists have identified specific genes controlling the synthesis and breakdown of specific neurotransmitters in animals ([Cases et al., 1995](#); [Nelson et al., 1995](#)) and humans ([Brunner et al., 1993](#); [New et al., 1998](#)). The genes controlling the breakdown of monoamine oxidase ([Belfrage et al., 1992](#); [Cases et al., 1995](#)) and catechol-O-methyltransferase ([Gogos et al., 1998](#)) have been identified, as has the gene for tryptophan hydroxylase, important in serotonin synthesis ([Nelson et al., 1994, 1997](#)). Scientists now are able to manipulate genes in animals and thereby modify behaviors. Some scientists have gone so far as to suggest that such manipulations (or machinations) might have applications for the management of human aggression ([Tecott and Barondes, 1996](#)). Twin studies as well as studies of adopted-away children of criminal biological parents have been interpreted as showing a genetic contribution to violent, antisocial behavior ([Cadoret et al., 1997](#)). However, no study to date has demonstrated a genetic predisposition specifically to violence. [Gjone and Stevenson \(1997\)](#), in a study of a large sample of twin pairs, using parent report data, showed an association between early temperament and subsequent aggression. Most closely associated with subsequent aggression were the characteristics of emotionality and high activity, especially in boys.

Estimates of the genetic contribution to aggression depend in great measure on the methods used to measure aggression. Observational measurements of aggressive behavior yield lower heritability statistics than do report and questionnaire studies ([Cadoret et al., 1997](#)). Whatever the study, scientists agree that genetic predispositions *alone* do not account for complex human behaviors. It would seem, rather, that adverse home environments, and their interactions with biological temperamental characteristics, may increase the likelihood of a maladaptive, aggressive behavioral outcome ([Cadoret et al., 1995](#)).

The bottom line is that the ways we are treated *in utero*, at birth, during infancy, and in childhood and adolescence are the most important influences on our adaptation and, hence, on our aggressiveness.

## ENVIRONMENTAL INFLUENCES ON AGGRESSION

### Intrauterine and Perinatal Factors

The very position *in utero* of animals in a litter has been found to influence behavior. Female mice that develop *in utero* between two males are more aggressive after birth than those positioned between other females ([vom Saal, 1984](#)). Thus, animals from the same litter who have shared the same uterus at the same time have not had identical prenatal environments. Maternal stress during pregnancy also has been shown to affect the behavior of offspring long after delivery ([vom Saal, 1984](#)).

In humans, there is strong evidence that different kinds of noxious prenatal influences, ranging from minor viral infections to maternal anxiety and psychological stress, have been associated with childhood maladaptation ([Pasamanick, 1956](#); [Rutter, 1970](#); [Stott, 1973](#); [Stott and Latchford, 1976](#)). The adverse effects of maternal alcoholism and other substances on fetal development and subsequent postnatal social and intellectual functioning are well documented. The exposure of the fetus to abnormal levels of certain gonadal hormones also has been associated with behavioral sequelae ([Ehrhardt and Money, 1967](#); [Ehrhardt et al., 1968](#)). Our own studies ([Lewis and Shanok, 1977](#); [Lewis et al., 1979, 1985](#)) and those of others ([Levine et al., 1985](#)) have documented the higher prevalence rate of histories of perinatal problems in the delinquent versus nondelinquent populations.

Given the vicissitudes of life *in utero* and at birth, we should not conclude that even temperamental styles such as "emotionality" and early activity levels necessarily reflect genetic endowment. Similarly, it would be an error to take for granted that the relationship between an adoptee's aggressiveness and a biological parent's antisocial history is necessarily genetic. Parents who have been in and out of prisons are not likely to have been well nourished nor are they likely to have received the best medical care. The lives of mothers who release their children for adoption cannot have been easy. There is now evidence that heightened maternal cortisol as a result of stress affects the fetus and increases aggressiveness postnatally. Furthermore, the partners with whom offenders link up frequently suffer from mental illness and have been psychiatrically hospitalized ([Lewis et al., 1976, 1981](#)). Thus, the adopted-away children of criminal parents in all likelihood had stressful *in utero* experiences. Moreover, the genes they inherited may well have predisposed them to a variety of different kinds of mental disorders other than antisocial personality disorder that compromised normal adaptation. In other words, behaviors that at first appear to be genetic may not be. As Buttercup in Gilbert and Sullivan's *H.M.S. Pinafore* so aptly put it, "Things are seldom what they seem."

### Nurturing

Nurturing affects temperament. Animals bred to have an especially gentle nature, if cross-fostered by adult females of a violent strain, will become more aggressive than is their usual nature ([Southwick, 1968](#)). There is evidence that mice reared in the company of their fathers as well as their mothers grow up to be more aggressive than those raised only by mothers ([Mugford and Nowell, 1972](#)). On the other hand, the effects of cross-fostering aggressive mice by less aggressive females are more variable. Certain strains retain their aggressiveness ([Southwick, 1968](#)), whereas others become less aggressive ([McCarthy and Southwick, 1979](#)). In another study, mice from an aggressive strain reared by males and females of their own strain were more aggressive than those raised either by two females or a male and a female of a less aggressive strain ([Smith and Simmel, 1977](#)). Isolation during critical developmental periods can engender aggression in otherwise gentle animals ([Brain and Nowell, 1971](#); [Goldsmith et al., 1976](#); [Luciano and Lore, 1976](#)). Thus, there is evidence from animal studies that innate temperament can be modified and that the quality of parenting and environment influences adult behaviors.

Very early experiences have especially powerful and enduring effects on physiologic and psychological development and, therefore, on behavior. In animals, qualities of parenting during the first days and weeks of life are particularly important to subsequent social adaptation. Although many reptiles are equipped to defend themselves shortly after birth, most mammals require parental protection for varying periods.

In humans, the first months and years of life are periods of extreme vulnerability, emotionally and biologically. Although the auspicious neuropsychiatric and emotional cards a person is dealt at birth may seem to predict a successful game, experience can reverse what under other circumstances would have been a winning hand. Adversity during the earliest periods of development can change the relationship of a child to the environment from a gentle give-and-take to a rough-and-tumble struggle. For example, research on attachment has shown that the ways that mothers and infants interact during the earliest weeks and months of life affect subsequent aggression in the child ([Lyons-Ruth, 1996](#)). Attachment behaviors, which are evoked in the infant by stress, especially by separation, have the goal of reducing anxiety and restoring a sense of security. At times of stress, securely attached infants turn unambivalently to their mothers for comfort. Adequate mothers sense their children's needs and respond appropriately. Mothers who lack tenderness, are inappropriately intrusive, and harbor suppressed anger toward their babies seem to engender insecure, avoidant attachments. Avoidant attachment has, in turn, been associated with subsequent aggressive behavior in boys ([Renken et al., 1989](#)).

Researchers have noted more seriously pathologic, "disorganized" patterns of attachment in certain infants. The mothers of these infants are especially disturbed and



distressed, and are more likely than the mothers of securely attached infants to have experienced abuse or abandonment themselves. They also are more likely to have been psychiatrically hospitalized ([Lyons-Ruth et al., 1991](#)). Their children seem to lack consistent strategies for reducing stress. These children's behaviors are hard to categorize but tend to include apprehension, helpless or depressed behaviors, unpredictable alterations between approach and avoidance, and apparent "freezing" or stilled, slowed movements ([Main and Soloman, 1990](#)). Several studies have shown a relationship between disorganized attachment and early childhood aggression ([Lyons-Ruth and Block, 1993](#)). Looking retrospectively, preschoolers manifesting extremely hostile behaviors were found to be six times more likely than normal children to have been classified in infancy as having had disorganized attachments to their mothers.

Poor mothering takes its toll intellectually as well as behaviorally. Infants with disorganized attachments lag in their mental development regardless of maternal IQ ([Lyons-Ruth, 1991](#)). [Lyons-Ruth and colleagues \(1996\)](#) found that the combination of early disorganized attachment and lags in cognitive development were associated with aggressive behaviors at 7 years of age. Thus, it would appear that poor mothering affects brain function itself.

The period during which attachment forms is crucial developmentally. During the first year of life, the human brain increases in weight from an average of 400 g to an average of 1,000 g, and this growth spurt continues through the second year of life ([Glaser, 2000](#); [Schore, 1994](#)). The brain develops in a genetically determined sequence from brain stem to cortex ([Nelson and Bloom, 1997](#)), and myelination of the frontal lobes is not complete until early in the third decade of life ([Benes, 1998](#); [Pincus, 2000](#)). However, with few exceptions ([Gould et al., 1997, 1999](#)), the brain's neurons form and migrate to their genetically determined positions during embryonic and early postnatal life. The degree to which new neurons are created later in development is an active area of research. During the first 2 years of life, an enormous proliferation of brain cells, axons, dendrites, and synapses occurs, with more synapses created than eventually will be used. Which brain cells and synapses will survive and which, by virtue of disuse, will be pruned and ultimately disappear depends to a great extent on experience ([Singer, 1995](#)). In short, the ways in which infants are treated, the kinds of attachments they are fortunate or unfortunate enough to make, affect the very structure and function of their brains and thus influence their behaviors. Studies of animals ([Hubel and Wiesel, 1979](#)) and humans ([Scarr, 1993](#)) support the concept of sensitive periods in brain development during which particular kinds of stimulation must occur if development is to proceed normally. Human beings have evolved in such a way that certain types of mothering experiences are requisite for brain and behavior to develop normally. Good parenting has survival value. In the second year of life, the frontal lobes are still growing. These are the areas of the cerebrum involved not only in reasoning and judgment but in the regulation of goal-directed behavior and the control of affects and impulses ([de Haan et al., 1994](#); [Glaser, 2000](#); [Thatcher, 1994](#)). During this crucial period of brain growth, infants cannot modulate their own arousal. The caregiver therefore must respond to the infant's stress and restore a sense of safety and well-being. There is good reason to believe that these early lessons in affect and impulse control, which seem to leave indelible impressions on the infant brain and mind, affect adaptation throughout life ([Sroufe et al., 1999](#)).

Although the earliest experiences may have special adaptational consequences because of the young child's extreme vulnerability, behavioral adaptation is the result of the ongoing interaction of the child with his or her environment. Attachment itself is not fixed and can change in response to levels of maternal stress ([Vaughn et al., 1979](#)). The usual adverse behavioral consequences of compromised early attachment also may be modified by improved social supports for the caregiver ([Erickson et al., 1985](#)). There is something about early, nurturant mothering, fostering secure bonds, however, that enables securely attached children to withstand subsequent adverse environments better than children who have not experienced secure attachment. School-age experiences and the quality of care during early adolescence do affect adaptation. However, all things considered, a major, if not *the* major, influence on behavioral outcome seems to be the nature of early mothering.

## Abuse

A copious literature attests to the association of maltreatment in childhood and aggression in adolescence and adulthood ([Cicchetti and Carlson, 1989](#)); the extent to which the category of "abused" children overlaps with that of children who have suffered attachment disorders and the independent influences of each are difficult if not impossible to measure. Whereas complete emotional deprivation of infants leads to severe depression and even to death ([Bowlby, 1969, 1975](#); [Spitz, 1946](#)), lesser degrees of neglect have been associated with poor peer relationships and aggressive behaviors ([Mueller and Silverman, 1989](#)). [Widom's \(1989\)](#) research suggests that neglect can be as powerful an influence on the development of aggression as physical abuse. Severe physical abuse has been associated with subsequent extremely violent behaviors ([Feldman et al., 1986](#); [Lewis et al., 1986, 1988a, 1988b, 1989, 1997](#)).

Early, repeated sexual abuse seems to have especially devastating consequences. There is evidence that a disproportionate percentage of sexually assaultive adults have histories of having been sexually abused as children ([Groth, 1979](#); [Rubenstein et al., 1993](#)). In the author's experience, the most grotesque sexual crimes have been committed by men who, as children, experienced extraordinary sexual abuse.

How do severe abuse and neglect engender aggression? The answer is not simple and must be gleaned from a variety of different kinds of studies and observations. What is clear is that the human organism is so constructed that maltreatment affects every aspect of functioning—cognitive, emotional, and physiologic.

## NEUROCHEMICAL EFFECTS OF ABUSE

From a biological standpoint, we do know that stressors of various kinds affect us physiologically, activating the hypothalamic–pituitary–adrenal axis and causing the release of catecholamines from the brain and the adrenal medulla and the release of cortisol from the adrenal cortex ([Le Doux, 1996](#)). Animal studies have shown a relationship among stress, hypersecretion of cortisol, and damage to the brain ([Sapolsky et al., 1990](#); [Uno et al., 1989, 1990](#)). Studies indicate that excessive exposure to glucocorticoids results in a loss of neurons in the hippocampus as well as a decrease in dendritic branching of the remaining neurons ([Sapolsky et al., 1985](#); [Wooley et al., 1990](#)). It is thought that damage occurs because of cellular vulnerability to the neurotoxic effects of the excessive release of excitatory amino acids such as glutamates ([Armanini et al., 1990](#); [Virgin et al., 1991](#)). Glucocorticoids also reportedly inhibit neurogenesis. Neurogenesis has been reported to occur even in mature animals ([Gould et al., 1999](#); [Reagen and McEwen, 1997](#)). Support for the toxic effects of glucocorticoids on the human brain come from computed tomography and MRI studies of individuals treated with glucocorticoids ([Bentson et al., 1978](#); [Okuno et al., 1980](#)) and of patients with Cushing's syndrome ([Starkman et al., 1992](#)). MRI studies of subjects with PTSD after combat trauma or child abuse ([Bremner et al., 1995, 1997](#); [Stein et al., 1997](#)) reported diminished hippocampal volume ranging from 5% to 26%. Although it has been assumed that these volume changes reflect the damaging effects of excessive cortisol secretion resulting from stress, other neurobiological mechanisms also may be operating. In his critical review of the literature on glucocorticoids and hippocampal atrophy, [Sapolsky \(2000\)](#) observes, "stress decreases the levels of brain-derived neurotrophic factor messenger ribonucleic acid (RNA) in the hippocampus" (p. 392), and some of this inhibition is thought to be independent of glucocorticoids ([Smith et al., 1995](#)). Whatever the underlying mechanism, abusive treatment and severe emotional stressors actually affect brain structure and function.

## NEUROANATOMIC CONSEQUENCES OF ABUSE

More recently, [De Bellis and colleagues \(1999\)](#), using MRI scans, compared the brains of 44 maltreated children with PTSD with a healthy, matched comparison sample. The cerebral volumes of the abused children were reportedly 7% smaller than in the control subjects; the corpus callosum was smaller, and the volumes of CSF in the lateral ventricles and cortical and prefrontal cortical areas were larger in the abused sample. Whether these cerebral abnormalities were reflections of the effects of increased catecholamine concentrations, high cortisol levels, lack of early stimulation, or other developmental catastrophes could not be ascertained. Whatever the psychobiological explanations, this degree of brain pathology cannot help but impair intellectual and behavioral functioning. As [Glaser \(2000\)](#) so cogently observes in his review of the literature on child abuse and neglect and the brain, "The neurobiological findings shed some light on the many emotional and behavioral difficulties which children who have been abused and neglected show. Hyperarousal, aggressive responses, dissociative reactions, difficulties with aspects of executive functions, and educational underachievement thus begin to be better understood" (p. 110).

Another common result of abuse or neglect is direct injury to the CNS. Almost any CNS trauma may be associated with hyperactivity, emotional lability, diminished intelligence, poor judgment, and impulsiveness. Children with these kinds of deficits and vulnerabilities are less able to think ahead and understand the consequences of their behavior. They are more likely than normal children to respond aggressively to provocative stimuli, be they in the form of violent television programs and movies or interpersonal conflicts. In our own studies of violent juveniles and adults, medical histories were so replete with evidence of CNS insults that it usually was impossible to ascertain the exact cause of brain dysfunction. It is known that injuries to the CNS, even apparently mild concussions, can have a cumulative effect neurologically and cognitively ([McCrea et al., 1997](#)). Extremely violent juveniles often have experienced multiple accidents and injuries, many of them inflicted by those entrusted with their care ([Ewing-Cobb et al., 1998](#); [Hatzitaskos et al., 1994](#); [Lewis et al., 1979, 1988b, 1989](#)).

The temporal and frontal poles of the brain, especially the undersurfaces of the frontal lobes, are the most vulnerable to contusions after injury ([Pincus, 2000](#)). Numerous studies have documented a relationship between frontal lobe dysfunction and aggressiveness. Several investigators, using different neuropsychological test protocols, have demonstrated evidence of greater frontal lobe dysfunction in delinquents than nondelinquents ([Berman and Siegal, 1976](#); [Wolff et al., 1982](#); [Yeudall et al., 1982](#)). Frontal lobe dysfunction has been associated with especially aggressive behaviors, as measured neuropsychologically ([Brickman et al., 1984](#)). Studies also have documented the association of aggression with frontal lobe injury ([Grafman et al., 1996](#)) and frontal lobe dysfunction ([Raine et al., 1994, 1997](#)).



These findings are not surprising, given the importance of the frontal lobes in modulating emotions and impulses.

Our overall adaptation as a species, our ability to overcome obstacles, defend ourselves when threatened, procreate and cooperate to raise our young, and by and large not kill each other when angered, is a reflection of the balance in our brains between appetites and drives and reflection and control. Animal studies have shown us that the part of the CNS most involved in sexual and aggressive feelings lies deep in our brains, in a complex central area commonly referred to as the *limbic system*. The most important areas of the limbic system associated with aggression are thought to be the hypothalamus, the septal area, and the amygdala. In animals, activating and inhibiting areas are very near each other. Ablation and stimulation of these parts of the brain results in an increase in or a diminution of aggressive behaviors, depending on the areas stimulated and the kinds of animals studied.

Some of the most fascinating animal research regarding limbic function and aggression involves the amygdala, the area of the brain located medially, deep within the temporal lobes. Experiments on animals suggest that the amygdala is especially involved in aggression in response to fear ( [Egger and Flynn, 1963](#); [Siegel and Flynn, 1968](#)). Damage to the amygdala has been reported to result in a diminution of aggressive behaviors in response to novel stimuli.

Studies in which electrodes have been implanted in the brains of human subjects indicate that the limbic system also is involved in human aggression ( [Mark and Ervin, 1970](#)). Abnormal electrical activity in parts of the limbic system, as occurs in some psychomotor/complex partial seizures, has been reported to be associated with aggressive behaviors ( [Mark and Ervin, 1970](#)), although debate continues regarding whether directed aggression can occur during a seizure itself ( [Delgado-Escueta et al., 1981](#)). Reports of aggressive behavioral changes in patients with brain tumors localized to parts of the limbic system also are consistent with findings of an association between limbic activity and aggression ( [Malamud, 1957, 1965](#)).

Damage to the medial portion of the temporal lobes has been associated with poor impulse control and aggression ( [Poston et al., 1994](#)). Our own studies ( [Lewis et al., 1982](#)) and those of others ( [Devinsky and Bear, 1984](#); [Fenwick, 1989](#); [Spiers et al., 1985](#)) suggest that certain patients with temporal lobe/complex partial seizures may behave in episodically aggressive ways. Although directed violence toward another person during a seizure is relatively rare, such violence does occur postictally, and several investigators have suggested that personality changes occur in some patients with seizures that include periods of intense anger ( [Devinsky and Bear, 1984](#); [Fenwick, 1989](#)). However, most patients with epilepsy are not violent. Usually the frontal lobes, those sentinels of social adaptation, inhibit violent impulses arising in the limbic system. When, however, there is abnormal limbic activity and defective frontal lobe control (as is the case in some extremely aggressive juvenile and adult offenders), the stage is set for violence to break through. One of the most repetitively homicidal serial killers we have seen had a cyst in the right anterior temporal lobe, scarring in both frontal lobes, and spiking on electroencephalography in the frontal and temporal regions. In his case, violence was preceded by an aura and followed by deep sleep. On awakening, his memory of his violent acts was clouded. This kind of case is rare, but it illustrates the difficulties in adaptation when the limbic system and frontal lobes are both severely damaged. This patient also had been the victim of early, ongoing sexual abuse, a fact that raised the question of whether the disequilibrium in his brain would have expressed itself as violence had his upbringing been less deviant and more nurturing.

#### PSYCHOLOGICAL AND COGNITIVE EFFECTS OF ABUSE

The effects of abuse are not simply physical. Maltreatment has psychological consequences as well. Abuse actually changes the way in which the environment is perceived. Studies of animals as well as children have shown that one of the consequences of maltreatment is the development of hypervigilance. It has been demonstrated that defeat increases defensiveness in laboratory mice ( [Flannelly et al., 1984](#)). What is more, this conditioned defensiveness is generalized to other situations and other opponents ( [Leshner, 1981](#); [Seward, 1946](#)). Likewise, abused children seem to become hypervigilant, repeatedly misinterpreting their surroundings, and perceiving ambiguous stimuli as threatening ( [Dodge et al., 1984](#); [Rieder and Cicchetti, 1989](#)). These observations regarding young children are consistent with the author's clinical observations of violent juvenile delinquents. In fact, the symptom that the author and others (e.g., [Myers et al., 1995, 1997](#); [Ulzen and Hamilton, 1998](#)) have found distinguishes violent delinquents from their less aggressive peers most clearly is episodic paranoid ideation and misperceptions ( [Lewis et al., 1989](#)). Similarly, paranoia is the most important symptom shown to distinguish violent psychiatric patients from other patients (Yesavage, 1983a, 1983b).

Paranoia is a symptom common to numerous mental disorders (e.g., mania, depression, schizophrenia, Alzheimer's disease) and seems to have a certain survival value. Fearfulness keeps us on our toes. Fear in numerous species, from crocodiles to humans, is processed in the amygdala. Stimulation of the amygdala elicits defensive responses in such diverse species as lizards, dogs, and monkeys ( [Le Doux, 1996](#)). In human beings, when the amygdala is stimulated in the course of surgical procedures, the most commonly reported experience is a sense of fear and foreboding ( [Halgren, 1992](#)). Unlike lizards and other animals, human beings, by virtue of their large cerebral cortex, usually can process the experience intellectually and act rather than simply react. The prefrontal cortex exercises some control over the kinds of automatic defensive reactions engendered in the amygdala. However, as noted previously, recent data suggest that violence often is associated with frontal lobe dysfunction. It is reasonable to suspect that the paranoid misperceptions and consequent violent responses of many repeatedly violent juveniles and adults result, at least in part, from the reactions of a fear-primed amygdala, absent the controls of normally functioning frontal lobes.

We know that once a response has been established or conditioned (e.g., a hormonal or neurophysiologic response to fear-inducing stimuli), it is easily reevoked by exposure to similar stressors. Such recurrent, maladaptive physiologic responses probably play important roles in the hypervigilance and retaliatory aggression of many severely abused, violent children and adults.

Child abuse also contributes to violence by diminishing a child's expressive skills. Severely abused children manifest specific kinds of deficits in verbalization. For example, toddlers who have been abused are less able than normal children to use words that reflect their inner states or feelings ( [Cicchetti and Beeghly, 1987](#)). Ironically, these truly tortured souls speak less about their negative feelings than do children from nonabusive backgrounds. The abused child's inability to identify, much less verbalize his or her own feelings of distress is paralleled by an inability to appreciate the distress of others. Toddlers who have been physically abused show less empathy than their peers to the distress of other children ( [Main and George, 1985](#)). The extent to which this empathic deficiency is primarily psychodynamic or is related to frontal lobe dysfunction has not been determined. Whatever its cause, it undoubtedly contributes to the abused, violent child's imperviousness to the pain of others.

The tendency of abused children to repress or deny their own painful feelings as well as the pain of others, their inability to articulate emotions, their emotional lability and impulsiveness, their tendency to distort reality and perceive threats where they do not exist, and their resultant tendency to put angry feelings into actions rather than words can become a relatively enduring adaptational style. This constellation of characteristics accounts in great measure for the tendency of clinicians to dismiss severely abused, aggressive children, adolescents, and adults as simply conduct disordered or sociopathic.

#### Modeling of Abusive Behaviors

Aggression also is learned. There is sound experimental evidence that modeling plays an important role in the development of aggressive behaviors in animals ( [Hamburg, 1971](#)), as well as children ( [Bandura, 1973](#)). We also know that aggressive behaviors (like more adaptive behaviors) can be learned through reinforcement. One of the most important contributions to our understanding of the role of reinforcement in the genesis of aggression is [Patterson's \(1977\)](#) observation that when children's aggressive behaviors are punished severely by parents, they tend to continue. [Farrington \(1978\)](#) also found severe physical punishment to be a major antecedent of aggressive delinquency. Probably the most powerful generator of aggression in animals and possibly in humans is the repeated infliction of pain ( [Berkowitz, 1984](#)). So strong is this response in animals that a conventional experimental method for inducing murderous behaviors in mice and rats involves administering painful shocks to their feet (an ethically questionable practice). Physical torment also is an effective means of engendering viciousness in fighting dogs (e.g., pit bulls). The consequences of maltreatment in animals include the development of hypervigilance. Defeat in animals tends to engender defensiveness ( [Flannelly et al., 1984](#)), which, in turn, is generalized to other situations and other opponents ( [Leshner, 1981](#); [Seward, 1946](#)). It therefore is no surprise that children who have been painfully physically abused tend to behave in more aggressive ways than their nonabused peers ( [Cicchetti and Carlson, 1989](#); [Widom, 1989](#)).

To summarize, child abuse contributes to violence in several ways:

1. It provides a model of behavior.
2. It teaches aggression through reinforcement.
3. It inflicts pain, a known powerful stimulus to violence.
4. It creates neurophysiologic responses in the brain that both damage the brain and affect its functioning.
5. It damages parts of the brain related to impulses and their control.
6. It increases paranoia.
7. It impairs cognition and the ability to make judgments and foresee consequences.
8. It diminishes the ability to verbalize feelings.



9. It interferes with the child's ability to empathize with the feelings of others.

In short, child abuse is the most powerful generator of child, adolescent, and adult violence that we know. The earlier, the more pervasive, and the more enduring the abuse, the more likely it will engender aggressive adaptational styles. Furthermore, the more neuropsychiatrically impaired the child is to begin with, the more likely the abuse will result in a repetitively violent individual. Boys are especially vulnerable. Psychotic symptoms, especially paranoia, brain damage or dysfunction, and abuse are a deadly combination ([Lewis et al., 1988a, 1988b, 1989](#)).

## SOCIAL FACTORS AND VIOLENCE

### Cultural and Socioeconomic Influences

It is obvious from the differences in the rates of violent crime in different societies and in different sectors within societies that social factors influence aggressive behaviors. The mortality rates alone of young men in different parts of the world attest to the importance of social forces. For example, in 1995, only 38 young men between 15 and 24 years of age in both England and Wales combined died of homicide, compared with 6,224 such deaths in the United States ([Currie, 2000](#)). Although murder rates throughout much of the world increased markedly in the 1990s, the rate in the United States stands out in comparison with most European countries, Japan, and India. ([Grossman, 1996](#)). The rate of serious juvenile violence in the United States rose dramatically between 1987 and 1994 and since then (i.e., between 1994 and 1999) has decreased ([Farrington and Loeber, 2000](#)). True, the overall homicide rate in the late 1990s had diminished to 1960s levels; however, as [Currie \(2000\)](#) observed, the decline in overall violent deaths was more a reflection of lower mortality rates for adults older than 25 years of age. Whereas in 1998 the homicide death rate of Americans 25 years of age and older was approximately half what it was in 1970, the death rate by homicide of children 14 to 17 years of age was almost 50% higher than in 1970, and for young adults between 18 and 24 years of age it had almost doubled. To quote [Currie \(2000\)](#), "Lethal violence in the United States, in short, has increasingly become a young person's game" (p. 749). Some have hypothesized that the dramatic rise in juvenile violence in the early 1990s was a reflection of increases in gang membership, gun use, drug dealing, and drug use, especially the use of crack cocaine ([Farrington and Loeber, 2000](#)). Whatever the causes of the previous increase and the more recent decrease in violent crime, the fact remains that the United States still stands out among other developed countries in terms of its violence, especially its high rate of juvenile homicide. Juvenile violence also has increased in other advanced countries, but, in Western European countries, the increase has been predominantly in less serious forms of assault ([Currie, 2000](#)).

[Courtwright \(1998\)](#), in his trenchant analysis of the sociocultural characteristics of our country that contribute to its excessive violence, observes, "...other things being equal, the total amount of violence and disorder in society is negatively related to the percentage of males in intact families of origin or procreation..." (p. 247). He goes on to say, "Families rather than governments are the first best defense against violent and disordered behavior" (p. 279). He points out that the particular mixture of demographic, cultural, and social characteristics of our frontier, its surplus of young, unparented, unmarried men, its code of honor, its racist attitudes, its heavy alcohol consumption, its lack of religious convictions or practices, its devotion to guns, and its lack of an effective system of law enforcement formed a matrix for violence. Moreover, the closing of the frontier did not eliminate the "national self-image" in that, to this day, Americans continue "to think a manly man is someone with a gun and an attitude" (p. 4). Courtwright points out that, after World War II, when marriage rates went up and families focused on childrearing, social stability, and financial security, church attendance rose and violent crime diminished. However, between the 1960s and the 1980s, with the sexual revolution and the increasing divorce rate, the overall number of American men living alone roughly doubled and, in spite of increased use of contraception and abortion, the American population saw "a huge increase in the percentage of children who were illegitimate and raised in fatherless families" (p. 5). Courtwright stresses the civilizing influences of early upbringing in stable families and draws a cogent analogy between the violence-inducing frontier society and today's urban ghettos. He states, "Indeed, it is possible to think of the urban ghettos as artificial, unusually violent frontier societies—vice-ridden combat zones in which groups of armed, unparented, and reputation-conscious young bachelors, high on alcohol, cocaine, and other drugs, menace one another and the local citizenry, undeterred, if not untouched, by an entropic justice system" (p. 272). He concludes that in environments in which stable, intact families and adequate parental guidance are lacking (e.g., frontier towns, ghettos, urban areas during the early morning hours), violence will flourish. Therefore, it is not surprising that at this time our country is more violent than most Western European countries.

Juvenile violence is not spread uniformly across our country. Juvenile violence is far more prevalent in urban than rural settings. According to [Farrington and Loeber \(2000\)](#), in 1977, there were 56 known juvenile homicide offenders per million juveniles in the United States, and more than one-fourth of these homicidal juveniles came from just 8 of the 3,139 counties in the United States. The cities in these counties were Chicago, Illinois; Los Angeles, California; Houston, Texas; New York, New York; Baltimore, Maryland; Detroit, Michigan; Philadelphia, Pennsylvania; and Dallas, Texas. Most homicides were committed with firearms. Much juvenile and adult violence would not be lethal were access to guns more difficult.

In addition to the availability of guns, what promotes violence? [Currie \(2000\)](#) reminds us that violence in general, and youth violence in particular, is linked to "the experience of growing up toward the bottom of the U.S. social and economic ladder" (p. 752). He notes that material poverty and extreme economic inequality are associated with high homicide rates; this is true if one compares violence in states, cities, or neighborhoods within cities. He points out that efforts to determine whether the major contributing factor is socioeconomic inequality or racial subordination are academic because all three, violence, poverty, and racial subordination, cluster together in the same populations. Being relatively low on the socioeconomic totem pole is not associated with violence so much as is living in grinding poverty. In their study of violent crime in Columbus, Ohio, [Krivov and Peterson \(1996\)](#) found an especially high rate of violent crime in the poorest of poor neighborhoods. According to Currie, "It is this strong link between extreme deprivation and violence that accounts for the tragic association between violence and race in America." Bolstering this contention was the finding that in Columbus, Ohio, extremely deprived white neighborhoods had nearly as high violent crime rates as extremely deprived African-American neighborhoods.

Poverty alone does not seem to account for violence. Sociologists believe that a strong influence on criminality springs from "social disorganization," the lack of extended families to support youngsters whose families lack the stability to nurture them properly ([Currie, 1998](#)). Children in these communities often are shifted from caretaker to caretaker. Proper nutrition and safety, much less the establishment of secure attachment, fall by the wayside. The challenge often is just staying alive. One of the most convincing and thought-provoking theories of the linkage of violence and social deprivation was proposed by [Landau \(1984\)](#). He hypothesized that violence and aggression would increase in societies when social support systems malfunctioned or totally failed. Using inflation rates and the ratio between marriage rates and divorce rates as measures of social stress and family instability, Landau found an association to exist between these indices of societal malfunction and rates of violent crime in 11 of 12 countries studied in the 1960s and 1970s. The only country in which this association was not found was Japan, a country, as Landau noted, in which there are strong behavioral controls outside the family (e.g., in schools and workplaces). The Japanese culture itself instills strong feelings of shame for moral transgressions. The societal stressors associated with violence in other countries are associated with suicide rates rather than murder rates in Japan.

In impoverished societies, the determination of which children will succumb to violence as an adaptational style depends on a multiplicity of other variables, including biological, family, peer, school, and community factors ([Farrington and Loeber, 2000](#)). Among short-term precipitants of violence, Farrington and Loeber cite "being bored, angry, drunk, or frustrated" as well as "situational opportunities, including the availability of potential victims" (p. 744). Among the individual characteristics conducive to violence, [Farrington and Loeber \(2000\)](#) stress impulsivity or low IQ. Numerous studies from various parts of the world have documented an association between low or low-normal IQ and violence ([Teichner et al., 2000](#)). It is important to stress that neither impulsivity nor low IQ is necessarily inherited. The wealth of data cited in the foregoing discussion attests to the potentially adverse effects on intelligence, temperament, and behavior of impaired attachment, child abuse, neglect, and neurologic insults. In summary, the kinds of external stressors and intrinsic vulnerabilities that play roles in individual violence (e.g., physical discomfort, lack of nurturance and stability, CNS dysfunction, increased rates of mental illness, child abuse) are more pervasive in disadvantaged communities and contribute significantly to the disproportional rate of violence in the most impoverished sectors of society. Whether mental illness, cognitive impairment, and intellectual limitations cause individuals to sink into these impoverished environments, or whether the environments themselves give rise to these kinds of disabilities is not easily determined. It is reasonable to infer that interactions among vulnerabilities and adverse environments create matrices for violence.

### Media Influences

Evidence regarding the influence of violent entertainment on children and adolescents is not conclusive. Nevertheless, many studies of violence and the media suggest a link between the two, especially in the case of very vulnerable children ([Lagerspetz, 1989](#)). Clearly, the media affect different individuals differently, and violent children tend to gravitate more toward aggressive films than do better-functioning children. Children from violent homes are reportedly affected differently from those growing up in nurturing households ([Brown and Pennell, 2000](#)). Thus, causality is hard to prove. Some studies suggest that exposure to violent material increases violent fantasy (and, perhaps, by extension, violent behavior), especially in men ([Hess et al., 1999](#)). A study of the effects of violent video games found a relationship between playing or viewing violent video games and subsequent aggression, especially in men. The authors reported that men in general had a more hostile view of the world than women. Although academic achievement reportedly is inversely proportional to time spent playing video games, causality would be difficult to prove ([Anderson and Dill, 2000](#)).

A major vulnerability to the effects of exposure to violent materials is youth. Small children, to begin with, react differently from older children to the effects of aggression on others. For example, small children often continue to act aggressively in spite of a victim's expressions of pain, whereas older children and adults are more likely to be inhibited by the victim's suffering ( [Patterson et al., 1967](#)). In an experiment in which children of different ages were shown the beginning of a series of aggressive films and asked to choose among endings for them, younger children tended to choose violent outcomes for violent films, whereas older children were more likely to take into consideration whether the violence observed was justified or unjustified when choosing an outcome ( [Leifer and Roberts, 1972](#)). It would seem that emotional and cognitive maturity are important factors influencing choice.

A study of the content of a random sample of music videos aired at selected times of high adolescent viewership revealed that 14.7% of videos contained overt interpersonal violence, with an average of 6.1 violent acts per violent video ( [Rich et al., 1998](#)). Attractive role models were aggressors in over 80% of these videos. African Americans were overrepresented as aggressors and white women were the most frequently depicted victims. The authors suggested that such depictions reinforced false stereotypes. Ways in which media may affect behavior include modeling, disinhibition, desensitization, the arousal of aggressive feelings, and the encouragement of risk-taking ( [Derksen and Strasberger, 1996](#); [Huesman et al., 1997](#)).

The extent to which media contribute to violence not only by producing violent films but by transmitting news of violence from all over the globe deserves attention. [Groebel and Hinde \(1989\)](#) capture the interaction among biological and psychological vulnerabilities and the influence of the media in their statement, "The most likely link between the media and aggression is a reciprocal one. News media reflect societal aggressiveness but also create the image of a violent world. Viewers with aggressive predispositions prefer violent programs which in turn reinforce their aggressive tendencies" (p. 139). Thus, in a free society, in which all news can be reported and there are few restrictions on the aggressive content of programming, so that for the majority of individuals have unlimited access to information, we accept the risks inherent in exposing more vulnerable individuals, young and old, to violent stimuli.

### Gangs and Crowds

A word should be said about the tendency of relatively nonviolent youngsters to act aggressively when in gangs or crowds. People have a tendency to look with suspicion on others they perceive to be different from themselves ( [Grossman, 1996](#)). What is more, it is easier to stereotype and ultimately dehumanize a group we do not know than it is an individual with whom we have a relationship. This tendency to regard people who are different from us with suspicion accounts to some extent for the greater amount of violence in a heterogeneous society such as the United States, compared with a more homogeneous society like Japan. In a crowd, we also tend to lose our own individuality. With anonymity we act as if we are less responsible for our own behaviors and thus permit ourselves a greater expression of aggression than we would were we acting alone.

### REEXAMINING HUMAN NATURE

This chapter opened with a question: What kind of animal are we? From the data presented, it might be inferred that we are essentially violent. That is not so—not even in the United States. After all, in 1997, in the United States, there were only 4.1 arrests per 1,000 juveniles for seriously violent crimes (e.g., homicide, rape, robbery, aggravated assault). The rate ranged from a high of 10.2 per 1,000 in Illinois to 0.8 per 1,000 in West Virginia ( [Snyder and Sickmund, 1999](#)). Thus, we are a relatively nonviolent beast, considering our evolutionary origins.

What other evidence have we of our essentially peaceful nature? When the World War II historian S.L.A. Marshall and his team of interviewers spoke with thousands of soldiers in more than 400 infantry companies in Europe and the Pacific right after combat, the researchers found that only 15% to 20% of American riflemen had fired on the enemy ( [Marshall, 1978](#)). Soldiers in World War II were not unique in their reluctance to kill their fellow men. Studies of Civil War battles and of European battles of the late 19th century suggest that our forebears were similarly reluctant to kill each other, even in battle ( [Ardant du Picq, 1946](#); [Griffith, 1989](#)). There also is evidence that intense interpersonal combat takes an enormous psychological toll on soldiers. We do not like to kill each other. Historians have estimated that a third of all Israeli and Egyptian casualties in the 1973 Arab-Israeli war were psychiatric in nature ( [Shalit, 1988](#)). The overwhelming majority of soldiers kept in continuous battle for extended periods (e.g., 2 months) succumb to a variety of different psychiatric conditions that impair their ability to function in the field ( [Swank and Marchand, 1946](#)). War—killing each other—does that to us. [Grossman \(1996\)](#), in his summary of the literature on the effects of mortal combat, concludes, "There can be no doubt that this resistance to killing one's fellow man is there and that it exists as a result of a powerful combination of instinctive, rational, environmental, hereditary, cultural and social factors" (p. 39).

Given our apparently innate reluctance to kill each other, what is it that enables or even causes some of us to override these inhibitions? The Army has learned that to get ordinary human beings to kill each other, they must be trained to do so. Normal people must be conditioned to kill each other. Whereas during World War II, soldiers were trained to fire at targets in a field, modern training simulates battle, with human-shaped targets popping up and, if hit, immediately falling down. This instantaneous and repeated reinforcement of behavior has been credited in part with American soldiers' rise in firing rates from 15% to 95% between World War II and the Vietnam War. To get soldiers to overcome their inhibition to kill, every effort is made to dehumanize the enemy and desensitize the soldier to the act of killing ( [Grossman, 1996](#)). Soldiers also are taught to obey orders of their superior officers without question. In short, it takes a lot to get a normal human being to kill another human being.

One must ask, therefore, What about those four juveniles per thousand who are arrested for seriously violent acts? Some would dismiss the recurrently violent among us as simply inherently antisocial or sociopathic ( [Grossman, 1996](#)). To bolster such theories, they turn to studies reporting apparently inherent autonomic hyporeactivity in antisocial youngsters ( [Hare, 1970](#); [Mednick, 1981](#); [Raine et al., 1990](#)). Others point to the twin and adoption studies as evidence of innate predispositions to violence ( [Cadoret et al., 1995, 1997](#)).

Clearly, temperaments differ, and they differ from birth ( [Chess and Thomas, 1984](#)). But temperament is not synonymous with behavior. The ways in which temperamental predispositions manifest themselves depends on experience. Violent individuals are made, not born. Even autonomic arousal, a characteristic that may at first seem inherent, can be modified by experience (e.g., prefrontal cortical damage diminishes autonomic responsiveness to social stimuli) ( [Damasio, 1998](#)). Some of the factors that influence aggression are the timing, duration, and intensity of stimuli, with early stimuli having an especially strong impact. These influences cannot really be separated into psychological and biological, environmental and intrinsic because, as we have seen, there is a constantly changing dynamic interaction between the two. In human beings, we know that when intrinsic vulnerabilities such as brain dysfunction or a predisposition to mental illness are coupled with an abusive upbringing, violence often follows. A useful way to conceptualize the influences on aggression is as follows: Anything that increases irritability, discomfort, fearfulness, suspiciousness, and impulsivity lowers the threshold for aggression and increases the likelihood of violent behavior. Anything that impairs reality testing, judgment, foresight, self-esteem, empathy, self-control, and the ability to verbalize feelings rather than act on them also lowers the threshold for violence and enhances aggressiveness.

In contrast, anything that increases self-control, enhances judgment and foresight, increases self-esteem and a sense of security, increases the ability to recognize one's own feelings and the feelings of others, and increases the ability to express these feelings verbally rather than behaviorally raises the threshold for aggression and diminishes the likelihood of violent behavior.

Our understanding of human nature, of our aggressive evolutionary origins and yet innate disinclination to murder each other, suggests that much human violence can be prevented. The simple act of nurturing our young adequately would be a good place to start. Once a child has been damaged psychologically, emotionally, and neurophysiologically and has adopted an aggressive adaptational style, the degree to which that course can be reversed remains a question. Although the first 2 years of life are critical to normal brain development, "The process of neural plasticity in response to learning and the acquisition of new memories continues throughout childhood and into adulthood" ( [Glaser, 2000](#), p. 101) There is reason to believe that even in late childhood and adolescence, with appropriate intervention, change for the better can occur ( [Kazdin, 2000](#)).

We may not know exactly what works to diminish violence once it has developed, but we have excellent indicators of what does not work. As this review of violence illustrates, we know that isolation and neglect often lead to aggressive behaviors. We know that responding punitively to aggressive behaviors reinforces these behaviors. We know that physical discomfort and frustration increase violence. We know that physical brutality engenders violence. And we know that witnessing the violence of others is conducive to the development of an aggressive adaptational style. Given the fact that at this time in our history our correctional systems incorporate all of the factors known to enhance violent behaviors, and that younger and younger children are being incarcerated, it is not surprising that the rate of recidivism for violent offenders, children and adults, is so high. However, knowing what we already know about human nature, with a little ingenuity we could use this knowledge to devise effective prevention and treatment programs.



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## 30 NEUROPSYCHIATRIC SIGNS, SYMPTOMS, AND SYNDROMES

Daniel T. Williams, M.D.

[Delirium](#)  
[Predisposing Factors and Clinical Features](#)  
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In recent years, psychiatrists have been rediscovering the clinical importance of their historical roots in medicine in general and in neurology in particular. The proliferating volume of clinical research data has generated a growing awareness of important biological influences in psychiatric disorders, which in some circles, for some time, had been viewed almost exclusively in terms of their psychological and environmental determinants. This contemporary reintegrated neuropsychiatric perspective has, in turn, been associated with an increased concern by psychiatrists with the psychiatric manifestations of and complications of primary neurologic disorders ([Brumback and Coffey, 1998](#)).

In this context, clinicians evaluating children and adolescents with neurologic dysfunction must also be prepared to integrate developmental considerations in evaluating and treating these patients ([Kagan, 1998](#)). Syndromes that affect the central nervous system (CNS) tend to have particularly augmented cognitive, emotional, and behavioral effects if they begin and persist during these early periods of development. In this regard, a syndrome that is static in its neurologic sequelae (e.g., cerebral palsy) is quite different in its psychiatric implications from a progressive disorder, such as one of the neurodegenerative disorders, commonly diagnosed initially in childhood. Furthermore, the area and extent of neurologic involvement is crucial to determining the impact on development. Thus, a CNS lesion that generates intellectual impairment in the form of learning disabilities or frank mental retardation will make a substantial difference in the patient's coping capacities, quite apart from other focal neurologic or psychiatric impairments.

The Isle of Wight study demonstrated that children with brain injury suffer a significantly greater rate of psychiatric impairment than children with comparable degrees of physical disability not involving the CNS ([Goodman, 1994](#); [Rutter, 1989](#)). Beyond the psychological sequelae of impaired physical functioning, the clinician must consider the potential direct neurophysiologic impact of CNS dysfunction on emotional, cognitive, and behavioral capacities ([Howe et al., 1993](#); [Pine et al., 1993](#)).

Not all CNS dysfunction meriting differential diagnostic consideration by child psychiatrists is as evident as that following a well documented brain injury. Indeed, one of the crucial tasks of the clinician in evaluating a youngster manifesting signs or symptoms of dysfunctional behavior or emotion is to assess the patient multidimensionally. This precludes the simplistic assumption that the dysfunction can be understood as deriving exclusively from either neurogenic or psychogenic origins. The multi-axial diagnostic format of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV and DSM-IV-TR) encourages clinician consideration of psychopathology in this multidimensional manner ([American Psychiatric Association, 2000](#)).

With a growing level of sophistication, we have come to appreciate important neurogenic substrates of the CNS dysfunction central to a growing number of traditional psychiatric disorders, including the schizophrenias, pervasive developmental disorders, tic and Tourette's disorders, attention deficit/hyperactivity disorder, affective disorders, and anxiety disorders, to name just a few. Because we now recognize that every behavior and subjective experience has a neurophysiologic correlate (which most often currently is not identifiable), the neurogenic versus psychogenic distinction is ultimately a semantic one, determined by our current level of neurophysiologic sophistication.

For purposes of this chapter, the neuropsychiatric signs, symptoms, and syndromes considered are those relevant to definable or presumed brain lesions. *Signs* are objective, observable deviations from normal behavior, performance, or neurophysiology. The mental status examination and more formal psychological testing assess both spontaneous behaviors and responses to standard cognitive queries. These often are usefully supplemented by formal neurologic evaluation, with its associated assessment of other areas of CNS function. *Symptoms* are the subjective observations by the patient of some deviation of experience or behavior from the norm. It is important in the case of children and adolescents to ascertain independently, from parents, to what extent such subjective complaints by the patient represent a change from the youngster's preexisting state and over what time frame.

[Table 30.1](#) delineates a variety of diagnostic parameters, together with relevant signs and symptoms important to the diagnosis of neuropsychiatric syndromes ([Horvath et al., 1989](#)). It must be emphasized that, in assessing children and adolescents, the clinician needs to have a sense of the relative range of normal vis-à-vis an individual patient's age. This makes more complicated an assessment that, for adults, by contrast, can be gauged against a relatively unimodal standard normal range.

Consciousness and arousal	Is there a reduced state of awareness (clouding)? Is there a disturbance in levels of the sleep-wake cycle? Is there a marked change in customary activity level? Is the capacity to shift focus and sustain attention impaired?
Motion	Are there any inappropriateness, flaccid, or hyperactive?
Reaction	Is the ability to process information reduced?
Cognitive processing	Is there an impairment of content? (e.g., concrete versus poor abstract/concept-forming ability?) Is there a loss of general intellectual content?
Cognitive content	Are there any delusions or paranoid ideas, hallucinations?
High cortical functions	Is there evidence of aphasia, hemiparesis, or incoherent speech, apraxia, or constructional difficulty, apraxia?
Memory	Is there difficulty to learn new information and to transfer it from short- to long-term memory? Is there difficulty to remember if familiar event in the past? Is there difficulty with recall or recognition? Is there difficulty with verbal or visual memory?
Mood	Is the mood consistently depressed, elated, irritable, aggressive, or anxious? Is the mood labile, apathetic, indifferent?
Judgment	Is social judgment impaired? Is there any "magical" thought?
Time course	Do the symptoms fluctuate? Do the symptoms progress steadily or in a stepwise manner? Is there a discrete decline in function or a discrete increase in symptoms?

**Table 30.1. Diagnostic Parameter Signs and Symptoms**

Many of these signs and symptoms can be adequately evaluated by a clinician in the course of a routine office evaluation. Nevertheless, the general clinical impression thus generated often can be usefully supplemented by formal psychological and neuropsychological testing. Although specific discussion of such testing is beyond the scope of this chapter, it behooves the clinician to be aware of the special techniques available for the assessment of the infant and young child ([Neuper et al., 1998](#)); some of the tools available to aid in clinical evaluation of cognitive and behavioral function ([Weinberg et al., 1998](#)); and the potential benefits of formal neuropsychological testing ([Boll et al., 1998](#)).

This chapter follows the DSM-IV and DSM-IV-TR format of replacing the DSM-III-R category of "Organic Mental Syndromes and Disorders" because this designation incorrectly implies that "nonorganic" mental disorders do not have a biological basis ([American Psychiatric Association 2000](#)). Instead, DSM-IV and DSM-IV-TR use two categories for what have traditionally been regarded as neuropsychiatric syndromes:

1. Delirium, Dementia, and Amnesic and Other Cognitive Disorders
2. Mental Disorders Due to a General Medical Condition



The essential ingredient in both of these broad diagnostic groups is an experiential or behavioral abnormality associated with transient or permanent documentable dysfunction of the brain. In this chapter, several of the aforementioned DSM-IV and DSM-IV-TR categories of mental disorder are addressed as they pertain to children and adolescents. Mental disorders associated with intoxication and withdrawal are discussed in [Chapter 73](#). Mental disorders associated with acquired brain injury are addressed in [Chapter 33](#). Mental disorders associated with brain tumors, neurodegenerative disorders, epilepsy, and cerebral palsy are addressed in [Chapter 61](#).

## DELIRIUM

Delirium may be defined as a transient and usually reversible dysfunction in cerebral metabolism that has an acute or subacute onset, that is caused by the direct physiologic consequences of a general medical condition, and that is manifest clinically by a wide array of neuropsychiatric abnormalities ( [American Psychiatric Association, 2000](#)). Although the term has been used with differing connotations over the years and has many synonyms in the neurologic and psychiatric literature, it seems best for current purposes to use the preceding definition of delirium corresponding to that outlined in DSM-IV and DSM-IV-TR. Because, as noted earlier, disorders associated with intoxication and withdrawal are discussed in another chapter, [Table 30.2](#) outlines diagnostic criteria for delirium due to general medical conditions.

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- A. Disturbance of consciousness (i.e., reduced clarity of awareness of the environment) with reduced ability to focus, sustain, or shift attention.
- B. A change in cognition (e.g., memory deficit, disorientation, language disturbance) or the development of a perceptual disturbance that is not better accounted for by a preexisting, established, or evolving dementia.
- C. The disturbance develops over a short time (usually hours to days) and tends to fluctuate during the course of the day.
- D. There is evidence from the history, physical examination, or laboratory findings that the disturbance is caused by the direct physiologic consequences of a general medical condition.
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Adapted with permission from American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., text revision. Washington, DC: American Psychiatric Association; 2000.

**Table 30.2. Diagnostic Criteria for Delirium Due to General Medical Conditions**

### Predisposing Factors and Clinical Features

Aside from the factor of age (children and the elderly reportedly have a higher risk), polypharmacy and multiple medical problems are probably the most common risk factors to delirium ([Trzepacz and Wise, 1997](#)). In addition, the following factors predispose to the development of delirium: postcardiotomy status, severe burns, preexisting brain damage, drug addiction, and acquired immunodeficiency syndrome ([Pontrelli et al., 1999](#)). Although formal epidemiologic studies are lacking, there is a consensus of reported clinical impressions regarding the vulnerability of children to delirium ([Amit, 1988](#); [Platt et al., 1994](#); [Prugh et al., 1980](#)). Because of the general acceptance of a tendency for children to regress under stressful circumstances, milder forms of delirium may be mistaken for simply regressive or provocative behavior. As with adults, however, undetected delirium may proceed to the point of self-injury or serious interference with medical treatment.

A clinical consensus suggests that, as the severity of a pathophysiologic stress increases, so does the probability of development of a delirium ([Wise and Brandt, 1992](#)). This is particularly true for burn patients, as well as for postoperative patients. Additional factors that are considered to foster the development of delirium include sleep deprivation, sensory deprivation, and sensory overload.

In addition to those clinical features outlined in [Table 30.2](#), the clinician also should note the possible presence of a prodromal period, characterized by restlessness, anxiety, sleep disturbance, and irritability. Also noteworthy and frequently present is a rapidly fluctuating and usually reversible course, which may include either hypoactivity or hyperactivity. Emotional lability often is observed, as is disorientation, dysgraphia (difficulty writing), constructional apraxia (difficulty drawing), and dysnomic aphasia (difficulty naming objects). Motor abnormalities that may be observed include tremor, asterixis (hand flap on extension), myoclonus (clonic muscle spasm), and symmetric reflex and muscle tone changes ([Lipowski, 1990](#)).

Although [Trzepacz \(1994\)](#) has reviewed the relative merits of 12 different assessment instruments that have been used for delirium with adults, there do not appear to be comparable instruments available for children. This probably reflects the greater difficulty in standardizing such instruments for children at various developmental levels. Therefore, currently, the clinician must integrate the diagnostic criteria for delirium with a cogent sense of age-appropriate developmental norms.

### Differential Diagnosis

Insofar as the basic etiology of delirium is conceptualized as a derangement in the functional metabolism of the brain, it is clear that a vast array of medical conditions originating within as well as outside the CNS can engender such a derangement. Because these numerous medical conditions cannot be reviewed here, an outline of more common causes is provided in [Table 30.3](#). As [Wise and Brandt \(1992\)](#) note, the catchy mnemonic “I WATCH DEATH” serves to remind the clinician of the potentially severe morbidity and mortality that can ensue from untreated or undertreated delirium. A more comprehensive list for consideration is outlined by [Trzepacz and Wise \(1997\)](#).

Infection	Encephalitis, meningitis, abscess, acquired immunodeficiency syndrome
Withdrawal	Alcohol, barbiturates, sedative-hypnotics
Acute metabolic	Acidosis, alkalosis, electrolyte disturbance, hepatic failure, renal failure
Trauma	Head trauma, postoperative stress, burns
Drug pathology	Alcohol, benzodiazepines, narcotics, hydrocephalus, seizures, stroke, tumor, vasculitis
Hypoxia	Anemia, carbon monoxide poisoning, hypotension, pulmonary embolism, cyanosis
Deficiencies	Vitamin B <sub>12</sub> , hypovitaminosis, niacin, thiamine
Endocrinopathies	Hypothyroidism, hyperthyroidism, hyperparathyroidism, hypoparathyroidism
Acute vascular	Hypertensive encephalopathy, stroke, migraine
Toxic/drug	Medications, pesticides, solvents
Heavy metals	Lead, manganese, mercury

DE, central nervous system.  
Adapted from Wise VG, Brandt DT. Delirium. In: *Handbook of Clinical Child Psychology*, 2nd ed. Washington, DC: American Psychological Press; 1992. pp. 291-316.

**Table 30.3. Causes of Delirium (“I WATCH DEATH”)**

Ordinarily, a psychiatrist confronted by a delirious patient promptly involves a neurologic colleague in clarifying the differential diagnosis. It behooves the child psychiatrist, nevertheless, to be familiar with the neuropsychiatric evaluation of the patient with delirium. A summary of this process is outlined in [Table 30.4](#). It is most important to recall that more than one etiologic factor may be simultaneously at work in any patient.

1. Mental status  
 History: presence of consciousness, orientation, affect, mood, insight, thought, thought process, performance (memory, concentration, writing, motor and construction, apraxia, agnosia, speech)

2. Physical status  
 Neurologic: examination (head, eye, strength, lateral eye, cerebellum, meningeal signs, gait)  
 Review of systems: EKG, pulse, temperature, blood pressure, respiratory rate  
 Review of laboratory tests: serum electrolytes, renal and liver function tests, hematology, urinalysis, toxicology, thyroid function tests, serum ammonia, serum lactate, serum glucose, serum calcium, serum magnesium, serum phosphorus, serum uric acid, serum ferritin, serum vitamin B<sub>12</sub>, serum folate, serum vitamin D, serum parathyroid hormone-related protein, serum prolactin, serum cortisol, serum growth hormone, serum insulin, serum C-peptide, serum insulin-like growth factor-1, serum insulin-like growth factor-1 binding protein-3, serum insulin-like growth factor-1/2, serum insulin-like growth factor-1/2 binding protein-3, serum insulin-like growth factor-1/2 binding protein-3, serum insulin-like growth factor-1/2 binding protein-3

3. Laboratory examination: toxic  
 Blood chemistry: electrolytes, glucose, Ca, albumin, blood urea nitrogen, creatinine, liver function, blood count (hemoglobin, white count and differential, platelet count), serum ammonia, serum lactate, serum glucose, serum calcium, serum magnesium, serum phosphorus, serum uric acid, serum ferritin, serum vitamin B<sub>12</sub>, serum folate, serum vitamin D, serum parathyroid hormone-related protein, serum prolactin, serum cortisol, serum growth hormone, serum insulin, serum C-peptide, serum insulin-like growth factor-1, serum insulin-like growth factor-1 binding protein-3, serum insulin-like growth factor-1/2, serum insulin-like growth factor-1/2 binding protein-3, serum insulin-like growth factor-1/2 binding protein-3

4. Laboratory: based on clinical judgment  
 Electroencephalogram (EEG): focal abnormalities (Corbin defined by voltage slowing or low voltage fast activity) Computerized tomography (CT scan) Magnetic resonance imaging (MRI) (with contrast) Lumbar puncture (indicator of infection or increased intracranial pressure)

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**Table 30.4. Neuropsychiatric Evaluation of the Patient with Delirium**

Clearly, the prognosis for a given patient with delirium varies with the etiology and, most important, with whether irreversible damage to the CNS is sustained. Hence, the outcome, depending on the timeliness and effectiveness of treatment, may vary from full recovery through progression to stupor and coma, to seizures, to chronic brain syndromes, or to death.

**Treatment**

If the clinician's differential diagnostic assessment has elucidated an etiology for the delirium, then specific treatments that can lead to resolution of the delirium often may be initiated. Specific treatments for identified causes of delirium are beyond the scope of this chapter and would be dictated by the tenets of accepted medical practice. In those situations where the etiology is unknown, even after a thorough initial evaluation, some interim supportive measures may well be helpful while diagnostic efforts continue. These are outlined under the following headings.

*MEDICAL MEASURES*

A patient with delirium should not only be in a hospital setting, but should be under close observation by nursing staff who are familiar with the management of delirium. This should involve frequent monitoring of not only vital signs but mental status, especially with a view to preventing self-injurious behavior.

*PHARMACOLOGIC MEASURES*

In the absence of double-blind trials, clinical experience has led to the advocacy of haloperidol administration in patients with delirium, based on numerous clinical reports of efficacy. Haloperidol also is favored in delirium because of its negligible anticholinergic and hypotensive properties and because it can be given parenterally ([Lipowski, 1990](#)). Although the use of haloperidol in pediatric patients with delirium is not well documented, its efficacy in children with a broad spectrum of more commonly encountered psychiatric disorders ([Teicher and Gold, 1990](#)) suggests that it may have a useful role in pediatric delirium ([Trzepacz and Wise, 1997](#)). Clinical titration with close monitoring, beginning with small doses and repeating every 30 to 60 minutes until the patient is calm, is common clinical practice. As with other psychiatric disorders, pediatric haloperidol doses may range from 0.25 to 10 mg/day, or from 0.05 to 0.15 mg/kg/day.

Benzodiazepines have the disadvantage of engendering sedation, which may further impair the delirious patient's cognition and may exacerbate the delirium. Small doses of intravenous lorazepam have been reported to be of some benefit, however, for patients who have not responded to high doses of haloperidol alone ([Lipowski, 1990](#)).

The use of risperidone in delirium has, thus far, been the subject of only isolated case reports in adults and adolescents ([Sipahimalani and Masand, 1997](#)). The use of this and other atypical neuroleptics in this domain awaits further systematic reports of clinical experience.

*PSYCHOSOCIAL MEASURES*

The presence of a calm family member or nursing attendant to provide reality testing, orientation, and reassurance can be very helpful in diminishing anxiety and allaying fears.

**DEMENTIA**

Dementia is a disorder of significant decline in multiple cognitive functions from the individual's previous intellectual level. In terms of the DSM-IV-TR definition, the essential feature of dementia is impairment of memory, associated with at least one of the following cognitive disturbances: (a) aphasia (language disturbance); (b) apraxia (impaired ability to carry out motor activities despite intact motor function); (c) agnosia (failure to recognize or identify objects despite intact sensory function); and (d) disturbance in executive functioning (i.e., planning, organizing, sequencing, abstracting) ([American Psychiatric Association, 2000](#)). The disturbance is severe enough to interfere with work and school, usual social activities, and relationships with others. The diagnosis of dementia is not made if these symptoms occur only in the presence of reduced ability to maintain or shift attention to external stimuli, as in delirium; however, delirium and dementia may coexist.

Although dementia is found predominantly in the elderly, some neuropsychiatric disorders described later in this volume, such as traumatic brain injury or degenerative brain disorders, may cause dementia in childhood and adolescence. In terms of DSM-IV-TR criteria, the diagnosis of dementia may be made at any time after the IQ is fairly stable (usually by age 4 to 6 years). Examples of medical conditions that may induce dementia in children or adolescents would include head injury, brain tumors, human immunodeficiency virus infection, cerebrovascular accidents, or neurodegenerative disorders ([Lerner et al., 1997](#)). Dementia in children may present as a deterioration in functioning, such as in school performance, or as a significant delay or deviation in normal development.

The mode of onset, subsequent course, and clinical management of dementia depend substantially on the underlying etiology. For this reason, such clinical features are addressed with reference to both dementia and other organic mental syndromes in the subsequent chapters of this volume dealing with acquired brain disorders and neurologic syndromes (see [Chapter 33](#) and [Chapter 61](#)).

**AMNESTIC DISORDERS**

Amnesic disorders are characterized by an impairment of memory that is attributable to a specific organic factor, such as a general medical condition (e.g., brain injury, vitamin deficiency), a substance of abuse, a toxin, or a medication side effect. The diagnosis requires the absence of the clouded sensorium associated with delirium or the more pervasive cognitive impairments associated with dementia. The syndrome, more commonly encountered as the Wernicke-Korsakoff syndrome among adult chronic alcoholics and attributed to thiamine deficiency, is relatively uncommon in children. It may be associated in children, however, with head trauma, hypoxia, lead or carbon monoxide poisoning, and herpes simplex encephalitis ([Mikkelsen, 1991](#)). Temporal lobectomy for intractable complex partial seizures may result in memory deficits, but this usually is of major significance only when surgery is bilateral ([Shimanura and Gershberg, 1992](#)).

**OTHER ALTERED STATES OF CONSCIOUSNESS**

Not all altered states of consciousness encountered clinically by child psychiatrists are subsumed by the neuropsychiatric syndromes outlined previously. Indeed, on a broad basis, most of the diagnostic categories encountered clinically, ranging from anxiety disorders and affective disorders to the schizophrenias, involve subtle or not so subtle alterations of attention, perception, cognitive processing, consciousness, and the like, subsuming all the diagnostic parameters delineated in [Table 30.1](#). A critical diagnostic task therefore is that of distinguishing those constellations of signs and symptoms associated with "traditional" psychiatric disorders from those associated with "neuropsychiatric" syndromes. The general current presumption is that the traditional psychiatric disorders are byproducts of particular constitutional or genetically based vulnerabilities, interacting with the individual's life experiences. In neuropsychiatric disorders, by contrast, the CNS abnormality is viewed as the overriding determinant of the presenting psychopathologic process. As reflected in our previous discussion, there are many instances where such distinctions are not



absolute. Thus, a *de novo* organic brain injury is likely to generate new psychopathology that interacts with a patient's preexisting vulnerabilities and personality structure. Alternatively, for example, a patient with a long-standing epileptic seizure disorder may develop secondary psychogenic seizures as a result of developmental adversities or intervening life stresses. The clinician's task, therefore, is to draw on all available sources of information to formulate as comprehensive a diagnostic assessment as possible, as the prerequisite for an effective treatment plan. Implicit in the foregoing is the premise of the multivariate etiology of any psychopathologic picture and an intent to give adequate consideration to both psychogenic and neurogenic factors. With these considerations in mind, three additional syndromes presenting as altered states of consciousness and presenting potential challenges to neuropsychiatric assessment are briefly noted; two others are discussed in some detail.

Altered states of consciousness associated with alcohol and drug use are addressed in [Chapter 73](#), those associated with dissociative disorders are addressed in [Chapter 70](#), and those associated with seizure disorders are addressed in [Chapter 61](#).

## STUPOR AND CATATONIA

Stupor has been defined differently by different authors. For most neurologists, stupor connotes a condition of behavioral nonresponsiveness, including loss of speech (mutism), markedly reduced movement (hypokinesia), and a reduced level of consciousness, from which the subject can be aroused only by vigorous and repeated stimulation. Stuporous patients are, thus, seen by most neurologists as having diffuse organic cerebral dysfunction of greater severity than that encountered in delirium, representing usually the next stage along a neurophysiologic continuum between clear consciousness and coma ( [Cutting, 1992](#); [Plum and Posner, 1980](#)). For many psychiatrists, on the other hand, stupor descriptively has connoted a relative preservation of consciousness in conjunction with mutism and hypokinesia ([Roberts, 1984](#); [Rogers, 1991](#)). As such, stupor has been associated in the psychiatric literature with traditional psychiatric conditions characterized by catatonia.

One of the most reasonable ways of reconciling this disparity in perspectives regarding stupor is to observe that both neurologists and psychiatrists see patients to whom they apply the symptomatic designation of stupor, but that the samples of patients they see, although overlapping somewhat, are significantly divergent. This becomes important in the differential diagnosis of stupor, regarding the need to separate organic from psychogenic stupors. Thus, [Plum and Posner \(1980\)](#) found psychogenic unresponsiveness to occur in only four of 386 unresponsive patients they saw on a neurology service. The predominant "psychogenic" diagnoses in their series of patients were catatonic schizophrenia and hysterical conversion, the latter usually associated with depression. By contrast, [Joyston-Bechal \(1966\)](#) found that in a psychiatric population, 69% of 100 "stuporous" patients had nonorganic disorders only. Of these 100 patients, 25% had depression, 34% schizophrenia, 23% organic disorders (mainly dementia), and 10% hysterical features. Nearly all the patients in both of the aforementioned and other studies ( [Cutting, 1992](#)) were adults, with no comparable series being available for children and adolescents.

[Cutting \(1992\)](#) proposes a classification in which catatonia is designated as a subtype of stupor, referring primarily to psychiatric symptoms traditionally associated with mood disorders, schizophrenia, and, less commonly, somatoform disorder. By contrast, the DSM-IV and DSM-IV-TR ( [American Psychiatric Association, 2000](#)), in addition to the previously noted traditional categories for catatonia, have added the category of "catatonic disorder due to a general medical condition," presumably as a way of reminding the psychiatrist to consider possible underlying organic factors rather than jumping to a premature diagnostic conclusion premised on the more familiar, traditional psychiatric diagnoses. In this regard, DSM-IV-TR defines catatonia as being characterized by any of the following: motoric immobility (hypokinesia or akinesia), excessive motor activity, extreme negativism or mutism, peculiarities of voluntary movement (e.g., waxy flexibility), echolalia, or echopraxia. The diagnosis of catatonic disorder due to a general medical condition is not given if the catatonia occurs exclusively during the course of a delirium.

Clarification of the cause of stupor or catatonia clearly is crucial in light of the potentially serious morbidity and mortality involved if the underlying condition is not effectively treated. Furthermore, inappropriate treatment, such as the use of additional neuroleptics in patients with neuroleptic-induced catatonic stupor ( [Philbrick and Rummans, 1994](#)) or other mismanagement of organic stupor, can cause further deterioration. Because by definition the patient is unable to give a history, securing other reliable informants becomes important. The history may disclose previous physical or mental illness or recent insults that may help clarify etiology. An illustrative example is a report of seven cases of posttraumatic mutism in children after severe head injury ( [Dayer et al., 1998](#)).

One report ( [Cohen et al., 1999](#)) reviewed all published reports on catatonia in adolescents from 1977 to 1997 (42 cases) and reviewed the authors' experience with 9 additional cases seen over 6 years at the authors' medical center. The authors note that, in contrast to its more common occurrence in adult affective disorder and schizophrenia, catatonia appears to occur rarely in adolescence (0.6% of psychiatric inpatients). The diagnoses of affective disorder and schizophrenia still predominated in the adolescent group, with representation also of pervasive developmental disorders and primary neurologic conditions. While maintaining that therapeutic management should be individually and diagnostically based, the authors emphasized (a) the frequency of neuroleptic-induced adverse effects, (b) the potential effectiveness of sedative drugs on motor signs, (c) the potential merits of electroconvulsive therapy for life-threatening or pharmacologically unresponsive conditions, and (d) the necessity to manage supportively family reactions and fears.

[Wing and Shah \(2000\)](#) lend additional evidence for viewing catatonia as a developmental complication emerging in later adolescence and young adulthood in neuropsychiatrically vulnerable patients. They surveyed 506 referrals to a center for autistic spectrum disorders and found that 17% of referrals aged 15 years and older had severe exacerbation of catatonic features. Further, they were significantly more likely than the comparison group to have had, before the exacerbation of these features, impaired language and passivity in social interaction.

On physical examination, a psychogenic catatonic disorder is suggested by closed eyes that resist opening and that close rapidly when the force used to open them is released. Although fixation of the eyes on objects in the patient's field of vision suggests alertness and awareness of the environment, this also may be seen in some organic disorders. Random roving eye movements, however, strongly suggest an organic disorder. On the other hand, voluntary deviation of eyes to the ground, no matter which side the patient is lying on, points to a psychogenic stupor ( [Roberts, 1984](#)).

The alert patient in psychogenic stupor usually attempts to protect himself or herself from injury ( [Rogers, 1991](#)). Thus, if the physician lifts the patient's arm and allows it to fall toward the patient's face, the patient usually will modify its path to avoid its striking him or her. Alertness also is suggested by a blink response to visual threat and purposeful, directed attempts by the patient to deflect a painful stimulus. Autonomic hyperarousal usually is present, with tachycardia, hypertension, and pupils that are equal, dilated, and responsive briskly and symmetrically to light.

The use of intravenous orazepam may be helpful when there is a strong index of suspicion that a patient's stupor or catatonia may be of psychogenic etiology ( [Clark and Rickards, 1999](#)). Careful neurologic evaluation of the patient should precede the use of this procedure, however, because the drug may confuse the monitoring of the patient's level of consciousness, which is vital when a progressive neurologic lesion is suspected. Similarly, it may interfere with interpretation of the electroencephalogram. In organic brain disorders, amobarbital sodium tends to cause cognitive deterioration: If this occurs, its use must be immediately stopped and the patient examined for any neurologic signs that may have been revealed. In the psychogenic patient, underlying psychopathology, such as positive symptoms of psychosis, may be revealed. The unresponsiveness of psychogenic patients tends to diminish with amobarbital sodium, but this may require a high dose of the drug. This is in contrast to the relatively low dose that often causes sedation and confusion in patients with organic brain disorders.

Further aspects of differential diagnosis and treatment would follow similar channels of consideration as those outlined previously in the section on [Delirium](#). Certainly, involvement of a neurologic colleague in the assessment to rule out pertinent neurologic etiology is essential before proceeding to treatment intervention for presumed psychogenic etiology. Furthermore, positive evidence for an underlying psychiatric diagnosis should be delineated before a specific psychiatric treatment plan can reasonably be initiated.

## CONCLUSIONS

Being cognizant of the diagnostic significance of neuropsychiatric signs, symptoms, and syndromes is essential for the well trained child and adolescent psychiatrist. Implicit in this is the psychiatrist's developing a multidimensional perspective that carefully attends to both the neurogenic and psychogenic influences that each patient brings to his or her interactions with the outside world. Only with such an adequately broad-based perspective on diagnosis can effective treatment planning for patients ensue.

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# 31 THE DEVELOPMENT OF PAIN PERCEPTION AND PRINCIPLE OF PAIN CONTROL

Neil L. Schechter, M.D.

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[History of Undertreatment](#)  
[Definition](#)  
[Neurophysiology of Pain](#)  
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There has been a dramatic change in the approach to pain in children since the mid-1980s. New research has provided a better understanding of the neurobiology and psychology of pain, as well as techniques to treat it more effectively. This new information has effectively counteracted the prevailing myths that had implied that children did not experience pain, or if they did, its impact was relatively insignificant. This chapter reviews some of the newer insights regarding the treatment of pain, focusing on our better neurologic understanding, on improved assessment techniques, and on the basic principles of pain management that apply to both acute and chronic pain situations.

## HISTORY OF UNDERTREATMENT

In 1974, Jo Ann Eland ([Eland, 1977](#)) examined the charts of 25 children who had undergone surgery at a large teaching hospital and found in total only 24 doses of analgesics had been prescribed for them throughout their entire hospital stay. Half of these doses were opioids and half were nonopioids. The average child who received any analgesia got approximately two doses. Children who had traumatic amputations, nephrectomies, and other painful surgeries were given essentially no analgesia. A number of other studies in this period documented this phenomenon. Work by [Mather and Mackie \(1983\)](#), [Beyer et al. \(1983\)](#), and [Schechter et al. \(1986\)](#) all revealed that children's pain in general was minimally treated, that PRN orders would typically be interpreted to mean "as little as possible," and that adults with similar pain problems received significantly more doses of analgesia than did children. The seminal and simultaneously most horrifying research of this era was the work of Anand, who examined the impact of surgery on infants for patent ductus without anesthesia, which was the standard of care in many institutions. Anand reported that preterm and term infants operated on without anesthesia had an enormous outpouring of stress hormones and clinically did much more poorly than did infants who received adequate anesthesia ([Anand et al., 1987](#)). The combination of these studies led to a significant change in the approach to postoperative pain management. By the late 1980s, editorials in most major journals demanded adequate anesthesia and postoperative analgesia ([Berry and Gregory, 1987](#); [Fletcher, 1987](#); [Hatch, 1987](#)). More research emerged on the use of patient-controlled analgesia for children ([Rodgers et al., 1988](#); [Tyler, 1990](#)), as well as on other approaches, and clinical practice guidelines were developed by a number of scientific and governmental agencies to help medical professionals treat pain more effectively ([Acute Pain Management Guideline Panel, 1992](#); [American Pain Society, 1989](#)). These responses clearly had an impact on pain management, and more recent studies of postoperative pain suggest a change both in practice and attitude.

In addition to postoperative pain management, another area that has seen a dramatic change is the management of procedure pain. Throughout the 1980s, it was not uncommon for children to undergo painful procedures such as bone marrow aspirations and laceration repairs with minimal or no sedation ([Bernstein et al., 1991](#); [Hockenberry and Bologna-Vaughn, 1985](#)). Often, when sedation was used, inappropriate agents were administered. Most poignantly, for many children with cancer, the painful procedures that they were forced to undergo as part of their diagnosis and treatment often were rated as worse than the disease from which they were suffering ([McGrath et al., 1990](#)). Frustratingly little research was focused on procedure pain management, and most oncologic research tended to focus on cure and not on symptom control. Again, in the past few years, with the growth of the palliative care movement in adults ([World Health Organization Expert Committee, 1990](#)), a simultaneous focus has occurred in children, and guidelines for procedure pain management in children have been developed by a number of professional societies ([American Academy of Pediatrics, Committee on Drugs, 1992](#); [American Society of Anesthesiologists, 1996](#)). Although there remain differences among the guidelines, it is quite clear that subjecting children to painful procedures without sedation no longer is considered acceptable. Pediatricians had come to accept children's fearfulness about pending procedures as implicit in pediatric care. Such assumptions are no longer valid.

Other pain problems have finally been subjected to examination, as well. Cancer pain ([Schechter et al., 1990](#)), sickle cell pain ([Shapiro, 1993](#)), pain associated with human immunodeficiency virus infection and acquired immunodeficiency syndrome ([Wishnie and Weisman, 1997](#)), and cystic fibrosis pain ([Ravilly et al., 1996](#)) have all received significant attention in the pediatric literature. Common pain problems also have begun to be scrutinized ([Schechter, 1995](#)). The pain associated with pharyngitis, otitis media, and immunizations, which historically had been ignored and thought to be an unfortunate but necessary component of pediatric medicine, is now being studied.

There continue to be significant areas where improvement is necessary, however. Children with chronic diseases whose pain problems are intermittent and unpredictable clearly are less well treated than those who have acute pain episodes that can be predicted, such as after surgery ([Johnston et al., 1992](#)). Even postoperative pain, however, continues to be problematic. The enormous increase in day-case surgery has left most pain management to be done at home by parents who may be anxious and inexperienced about using analgesia. Recent surveys have suggested a considerable amount of untreated pain after ambulatory surgery ([Nikanne et al., 1999](#)). Newborn pain also remains an area that requires attention. Although there have been great strides made in many settings, procedures such as circumcisions often are done without adequate analgesia or anesthesia, and postoperative care remains far less optimal than in older children and adults ([Anand et al., 1996](#); [Porter et al., 1997](#)). Finally, pain problems associated with no obvious pathologic process (recurrent abdominal pain, "growing pains," headache, fibromyalgia) continue to be challenging and require new thinking and approaches.

In summary, then, we have seen essentially a revolution in attitude and approach to pain in children. In the 1970s and early 1980s, there was almost no literature on pain management except for those documenting its inadequacy. Since that time, there has been an outpouring of research in major journals and of reviews available in the key textbooks, which allows practitioners easy access to this information. Assessment techniques have been standardized and subjected to more rigorous psychometric scrutiny. Children and their families are demanding more humane and compassionate care, and these factors all have combined to improve dramatically the management of pain in children.

## DEFINITION

Pain is defined by the International Association for the Study of Pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" ([Merskey et al., 1979](#)). The definition goes on to state that pain is always subjective and is learned through experiences related to injury early in life.

Although this well accepted definition represents a significant advance in our understanding of pain, it also creates a number of potential problems. The definition

states clearly that pain results from sensory stimulation as interpreted through the broader lens of the individual's experience. Thus, the same painful stimulus can be interpreted in very different ways depending on modifying factors that may serve to magnify or dampen its magnitude. Modifying factors are discussed subsequently, but include age, sex, culture, context or meaning of the pain, as well as a child's temperament and affective state. The combination of the neurophysiologic stimulus and a composite of these variables serves to define the pain experience caused by a specific stimulus in an individual.

This definition has come under increasing scrutiny, however, by people who work with children in pain. It states that pain is learned through experiences with pain early in life, which implies therefore that newborns *cannot* experience pain because they have not had previous experience with injury. The definition also implies that the person must be capable of describing or reporting his or her pain, thus eliminating individuals such as newborns or developmentally disabled children and adults who are not capable of describing their discomfort. [Anand and Craig \(1996\)](#) have expressed concerns about this long-accepted definition and have proposed instead that pain perception is an inherent quality of life that appears early in development and serves as a signaling system for tissue damage. The signaling may occur not only through verbal description, but through a variety of behavioral and physiologic responses that are described later. They have called for a change in the accepted definition, and this issue is under review.

## NEUROPHYSIOLOGY OF PAIN

The basic concept of nociception is that pain messages emerge in the peripheral nervous system and are transmitted to the central nervous system, where they are interpreted.

Mechanical, thermal, or chemical stimuli excite various types of nociceptors in the periphery that give rise to either unmyelinated C fibers (small and slow conducting) or thinly myelinated A-delta fibers (larger and fast conducting). These primary afferents have their cell bodies in the dorsal root ganglion and project to the dorsal horn of spinal cord. Tissue injury in the periphery can result in the release of mediators (bradykinin, cytokines, prostaglandins) that sensitize A-delta and C fibers and increase pain sensation. Repeated injury to these fibers in the newborn period may lead to a surge in nerve growth factor and sprouting of nerves in inappropriate locations, leading to aberrant innervation, lowered pain thresholds, and a permanent effect on development of nerves in the injured region. Therefore, tissue injury in the newborn period may have permanent effects on the development of the pain system.

Nociceptive impulses are transmitted by these afferents to the dorsal horn of the spinal cord, where messages are amplified or attenuated in the complex layers of the dorsal horn and by interaction with surrounding neurons peripherally or centrally. In the spinal cord, a variety of neurotransmitters further modify nociceptive information. Substance P and calcitonin magnify transmission, whereas other neurotransmitters such as endogenous opioids may dampen transmission. The pain stimulus then proceeds up the nervous system through spinothalamic, spinoreticular, and spinomesencephalic tracts. Thalamic projections distribute the pain message throughout the brain because there is no single pain center in the brain. Projections to the cortex account for the impact of past experience on pain, whereas projections to the limbic system allow for the impact of emotions.

In addition, there are a host of descending projections that might inhibit and dampen the pain message. Descending inhibitory pathways arise from a number of different regions, such as the periaqueductal gray matter, the inferior olivary nucleus, and a number of other foci. These tend to become functional later in life, and therefore it is believed that excitatory mechanisms are in place significantly before inhibitory mechanisms. The complex interplay of all of these systems and the involvement of multiple dimensions creates each individual's unique response to painful stimuli.

It has become clear that the pain systems are in place relatively early in the life of the individual and, as a result, infants have the ability to perceive pain before birth. [Anand and Hickey \(1987\)](#) have made the case, in fact, that infants are hyperalgesic because distances traveled by nerve impulses are shorter, there are more nociceptors per surface area than at any time in life, there are no inhibitory mechanisms in place, the cutaneous flexor reflex has a lower threshold in younger infants, and it takes higher levels of analgesia to achieve pain relief.

Finally, as has been mentioned, it is clear that untreated pain may have long-term consequences. Data from rat pups and human newborns who undergo repeated procedures suggest a change in their nervous system, yielding hyperalgesia ([Fitzgerald et al., 1989](#)). Studies of boys undergoing circumcision without anesthesia suggest a different response to immunizations at 4 and 6 months ([Taddio et al., 1997](#)). [Grunau et al. \(1994\)](#) and [Johnston and Stevens \(1996\)](#) and others have looked at the impact of multiple procedures that occur in the neonatal intensive care unit and found predictable effects later in infancy. [Anand et al. \(1999\)](#) even have suggested that pain and stress in the newborn period stemming from unanesthetized ventilation or inadequate analgesia for painful procedures may be responsible for an increase in intraventricular hemorrhage.

## MODIFYING FACTORS

As is evident from the previous discussion, the amount of pain that a person experiences from a specific amount of trauma depends not solely on the extent of the tissue damage, but on a host of modifying factors that potentiate or diminish suffering. Different individuals clearly experience different amounts of pain from the same noxious stimulus, and the same noxious stimulus can cause varying amounts of pain in the same individual at different times in his or her life. Although many factors have been identified that have a role in the pain experience, some of the key modifiers for children are the context or meaning of the pain, the age of the child, the sex of the child, culture, and, finally, affect state. Extensive work has been done in each of these areas, and a thorough discussion is beyond the scope of this review.

### Meaning of the Pain

Early studies of pain suggested a fixed relationship between the intensity of the stimulus and the magnitude of the pain. This theory, known as *specificity theory*, was eventually rejected because it was not in concert with clinical observation.

One of the first investigators to notice a difference in pain responsiveness was Beecher, an anesthesiologist. Beecher's work highlighted the differences in pain perception between soldiers and civilians ([Beecher, 1956](#)). He found that only 25% of the 215 wounded soldiers he cared for requested opioids, whereas 82% of civilians with similar injuries requested pain relief. Beecher identified the fact that for the soldiers, the pain had a meaning that was very different from that of the civilians. For them, it was an expected part of their responsibility and often was seen as a sign of valor. For the civilians, however, the pain was not expected and represented a personal catastrophe and an uncertain future. Since Beecher's initial work on the context and meaning of pain, many other studies have identified similar disparities. For example, cancer surgery usually is reported as more painful than similar surgeries in which concern about cancer is not an issue. Cesarean sections reportedly hurt less than similar uterine surgery ([Bruegel, 1971](#)). In general, when the pain seems to have little benefit and to represent potential disfigurement or disability, it is perceived as more painful than in situations where this is not the case.

### Age

A number of studies have examined the relationship of children's response to pain as a function of their age. This literature seems to imply quite strongly that younger children experience more pain from a given stimulus than do older children. This has been a consistent finding in the literature. It is possible it represents a reporting bias of the child, a cognitive lack of understanding of the potential value of the procedure, or an actual difference in the experience. Regardless, in the overwhelming majority of research which has investigated bone marrow aspiration, venous cannulation, and blood sampling, younger age has been associated with greater pain.

### Culture

It is self-evident that both culture and ethnicity may have a role in the pain experience. These factors provide the framework through which a child sees the world and form the basis of shared attitudes, values, goals, and practices. How much pain expression is acceptable, the level of pain to which one should respond, strategies used to soothe and comfort, and how one responds to pain, all have strong cultural roots.

Many studies have attempted to identify unique patterns of response to pain among different cultures. [Zborowski's \(1962\)](#) seminal work in this field in the 1950s and 1960s continues to be cited today, primarily because of a lack of subsequent research. When comparing Italian, Jewish, "old American," and Irish people's response to a standardized pain stimulus, Zborowski found significant differences that could be explained by the individuals' cultural beliefs. Subsequent work has been done on many other ethnic groups ([Aun et al., 1986](#); [Gaston-Johansson et al., 1990](#)), as well on as the impact of acculturation ([Pfefferbaum et al., 1990](#)), that is, as one group becomes more and more like the mainstream culture.



Despite this conventional wisdom, few studies of adults and even fewer studies of children have examined the cultural contribution to pain perception. It is important to recognize that although there may well be intergroup differences in pain response, there are enormous intragroup differences, as well. Treating children's pain in a "culturally sensitive" way emphasizes the avoidance of stereotypes, while simultaneously considering culturally unique beliefs about causes of illness and implications of pain, the use of folk cures and alternative remedies, and the awareness of interactional styles.

## Psychological Variables

### ANXIETY

It is quite clear that a number of psychological factors can affect pain perception. There is extensive literature on the role of anxiety in promoting pain. This seems to occur in both individuals who are chronically anxious, as well as in those in whom anxiety is situationally induced. Newer studies have examined the benefit of preparation and education in reducing anxiety. In patients who are prepared in a developmentally appropriate way and who can anticipate the type of pain that they will experience, pain often is reported as decreased ([O'Byrne et al., 1997](#)).

Other factors, however, affect the value of preparation. There clearly are patients who are information seeking, and there are those who are information avoidant. These personality characteristics have a role in the value and style of preparation.

### DEPRESSION

Depression, likewise, appears to promote pain. In people who are depressed, pain is experienced more intensely. For example, patients who have recently sustained the loss of a loved one have, on average, four times the number of physical symptoms than in age-matched control subjects who have not had a similar loss ([Klerman and Izen, 1977](#)). Depression also may be a response to chronic pain. [Heiligenstein and Jacobsen \(1988\)](#), in evaluating children with cancer pain who were thought to be depressed, found that alleviation of pain often eliminated the depression without the need for antidepressants or other types of therapy.

Regardless, it is clear that the person's emotional state plays a role in the interpretation of a noxious stimulus.

## Personality Characteristics and Individual Differences

It has long been postulated that individuals with different personality styles react to or experience pain differently. There have been many studies that have looked at personality inventories and identified the fact that people with certain particular personality patterns were more prone to experience acute and chronic pain. Certainly, the adult literature is replete with suggestions that particular personality patterns on the Minnesota Multiphasic Personality Inventory tend to be associated with chronic pain states ([Blumetti and Modesti, 1976](#)). Typically, the studies report that people with elevations on the hypochondriasis, hysteria, and depression subscales are more likely to have chronic pain. Studies using other inventories such as the Maudsley suggest that patients classified as "introverts" have lower tolerance for laboratory-induced pain ([Haslam, 1967](#)). Specific pain problems, such as complex regional pain syndrome 1 (formerly, reflex sympathetic dystrophy), fibromyalgia, and chronic fatigue syndrome, all have been vaguely associated with specific emotional states and personality styles, and recent work by [Varni et al. \(1996\)](#) has suggested that decreased self-esteem may be associated with chronic pain conditions.

This literature is not nearly as clear as it once was thought to be, however. Other studies suggest these associations are more limited than initially thought and may even be artifactual. The traditional notion, for example, of the child with recurrent abdominal pain being shy, perfectionist, and compliant has been seriously questioned by investigators such as [McGrath et al. \(1983\)](#), who found no relationship between recurrent abdominal pain and a host of psychological variables or personality styles.

Another aspect of personality that has been investigated is the style of sensory processing. [Petrie \(1967\)](#), in a classic study, blindfolded subjects and asked them to estimate the size of wooden blocks. He found that some individuals tended to increase the size of what they perceived, others tended to decrease the size, and others report on it more accurately. He reported that in laboratory-induced pain, subjects who increased the size of the block tended to have the lowest tolerance for pain and therefore experienced the most pain, whereas those who reduced the size of the block tended to have the most tolerance for pain.

Another formulation about personality uses the construct of temperament, theoretically a biologically based, often inherited, set of behavioral characteristics (the "how" of behavior). Temperament is present early, and although it evolves over time, certain temperamental characteristics remain relatively stable throughout childhood. Temperamental characteristics include attention span, adaptability, threshold of responsiveness, intensity, and approach/withdrawal ([Chess and Thomas, 1996](#)). These dimensions have been clustered into three types—easy, slow to warm up, and difficult. In a variety of studies, it has been shown that the child's temperamental characteristics clearly have a role in response to pain. Adaptability, the temperamental characteristic that describes how an individual responds to new situations, has been shown to be the strongest predictor of how an individual responds to an injection ([Schechter et al., 1991](#)). [Oberklaid et al. \(1997\)](#) identified the fact that children with growing pains were more likely to be more intense and to be rated as more difficult temperamentally than control subjects. Across the studies on temperament and pain, characteristics associated with increased "difficultness" (increased reactivity, decreased adaptability, lower threshold, more negative mood, and greater intensity) were clearly correlated with increased pain behavior.

Many of these personality studies reported are problematic. They often suffer from inadequate controls, and the construct validity of some of the instruments used remains in question. It is by no means clear that pain problems will automatically develop in individuals with particular personality characteristics. It is likely, however, that psychological styles are important modifiers in the interpretation of painful stimuli.

## Summary

In summary, then, it is clear that pain is a composite of the noxious stimulus and its interpretation. That interpretation, or "lens" through which that noxious stimulus is viewed, is affected by the child's cognitive state, age, sex, culture, personality style, temperament, and emotional state, as well as family modeling and culture. The relative contribution that each of these factors makes varies from individual to individual, but it should be evident that the same noxious stimulus does not cause the same pain in different people.

## ASSESSMENT OF PAIN IN CHILDREN

Developmentally appropriate assessment is the cornerstone of pain treatment. Lack of adequate assessment techniques has significantly handicapped progress in pain management in children. Without adequate assessment and documentation, appropriate treatment is impossible and the impact of an intervention cannot be adequately determined. It also is possible to dismiss and ignore pain if its extent and intensity are not clearly documented.

Unfortunately, for younger children, direct reporting of pain is not always feasible, nor will they alert their care providers when they are in pain. As a result, this group was most vulnerable to undertreatment before adequate assessment techniques were developed. At this juncture, however, many unidimensional as well as multidimensional pain assessment techniques are available for children of all ages. It now is imperative that they be used, and the Joint Commission on the Accreditation of Healthcare Organizations (JCAHO) has incorporated documentation of pain management into their survey of hospital adequacy.

Because pain is an inherently subjective experience, the individual's report of his or her discomfort has been assumed to be the gold standard by which pain is assessed. In adults and children older than 8 years of age, pain intensity traditionally is assessed by use of a visual analog scale, typically a 10-cm line with anchors of "no pain" on one end and "the most pain imaginable" on the other end. The child or adult is then asked to rate pain on that line. Variations of this approach involve a line with numbered increments. The child is then asked to select the number that correlates with his or her discomfort. Most children older than 8 years of age have an internal symbolic representation of their discomfort and, thus, the ability to quantify their pain, which they can translate into a specific number. Children younger than 8 years of age, however, although able to report pain, often cannot offer specific numerical ratings. As a result, since the mid-1980s, a variety of modified self-report scales have been developed for children older than 3 years of age. These may involve the use of colors ([Eland, 1982](#)), the use of cartoon faces expressing varying degrees of discomfort ([McGrath et al., 1985](#)), the use of photographs of other children who are experiencing various degrees of discomfort ([Beyer and Aradine, 1988](#)), the use of a "pain thermometer," and the use of poker chips or other manipulatives ([Hester et al., 1990](#)). What they have in common is that children are asked to quantify in a ways that are developmentally appropriate the intensity of their discomfort. Although children do have a tendency to select higher numbers

than adults might select for the same pain problems, in general, many of these instruments have reasonable psychometric properties, and they allow the pediatric provider at least a gross estimate of the magnitude of the discomfort the child is experiencing. These instruments have been a major advance in caring for children in pain. Most institutions select a specific modified self-report scale, which should be available on all flow charts near the child's bedside table. In that way, children in all domains of the hospital and across all shifts receive a similar assessment technique whose results should be reasonably reliable.

For children younger than 3 years of age, or for children or adults who are developmentally delayed, these measures are not appropriate. As a result, nonspecific proxy measures of pain (pain behaviors and physiologic variations) must be used. Composite measures incorporating both of these types of markers have been developed that appear to have clinical utility.

Traditionally, a number of behaviors are associated with discomfort. These behaviors include position, crying, lack of appetite, as well as facial expression. The area of facial expression has received significant investigation, particularly in infant pain ([Grunau and Craig, 1987](#)). Specific patterns that involve closed eyes, furrowed brows, nasolabial flattening, and a taut tongue have been associated with pain. A specific technique for evaluating facial expression (the Neonatal Facial Action Coding System) has been developed and offers results that are thought to be reliable and reproducible ([Grunau et al., 1998](#)). Other research has examined the use of primarily physiologic parameters to assess pain. Increased heart rate, respiratory rate, and blood pressure have all been associated with acute pain ([Franck et al., 2000](#)). Increased cortisol (although not an instantaneous marker), likewise is associated with distress ([Sweet and McGrath, 1998](#)). Increased palmar sweating ([Harpin and Rutter, 1982](#)), decreased vagal tone ([Porges, 1992](#)), as well as decreased oxygen concentration in the blood have all been associated with increased pain.

A number of investigators have developed composite measures, using facial expression, other behaviors, and physiologic parameters. There are many measures that have been developed, including the Children's Hospital of Eastern Ontario Pain Scale (CHEOPS), the Observational Pain Scale (OPS), the Neonatal Infant Pain Scale (NIPS), and the Premature Infant Pain Profile (PIPP). All of these have in common some composite of behaviors and physiologic parameters that yield a theoretically clinically usable score to make judgments about the child's level of pain. These are reviewed extensively elsewhere ([Finley and McGrath, 1998](#); [Stevens, 1997](#)).

Although no one instrument is universally accepted, at this juncture it does appear that there are adequate scales available to clinicians. What is most important is that each institution select scales that make sense within the logistical constraints of that institution, and use them in a routine, standardized, and uniform manner.

## **PAIN MANAGEMENT**

### **General Principles**

Although there clearly are significant differences in the way that acute and chronic pain in children is approached, there are many principles that apply to pain management in general. First and foremost is the use of a preventative approach. When pain can be anticipated, such as after surgery or in the midst of a chronic illness that is associated with pain, it should be treated prophylactically. This is the seminal principle that has emerged from the study of pain over the past 20 years. It makes no pharmacologic or humanitarian sense to require an individual to experience discomfort before treating it if that pain can be predicted. Lower doses of analgesics are necessary to prevent pain from occurring than to eliminate it once it has occurred. This principle is responsible for the general change in analgesic prescribing from PRN, which was the standard of care 20 years ago, to scheduled analgesics for predictable pain problems, which is the standard of care today.

Another key element of the war on pain is the use of adequate assessment. As previously mentioned, assessment is the cornerstone of pain therapy. It is necessary to develop a system that supports and expects the routine assessment of pain and in which that information yields appropriate interventions. Without assessment, adequate interventions cannot take place. Pain should be reassessed after the intervention has taken place to determine if the intervention was adequate and to determine whether additional interventions are necessary. Pain documentation should be done in a uniform manner and specific protocols and algorithms that flow from the documentation should be decided on in each institution. Hospital-wide quality assurance mechanisms should monitor the adequacy of pain documentation, and its importance has been recognized by its incorporation into recent JCAHO expectations.

Another key principle in pain management in children is the importance of the simultaneous use of pharmacologic and nonpharmacologic strategies. No matter what the level of pain, both of these general approaches should be part of the overall treatment plan. Preparation, parental presence, self-control, distraction, massage, hypnosis, transcutaneous electrical nerve stimulation, all potentiate the efficacy of analgesic therapy.

Parents should be involved in all decisions about their child's treatment, as well as their child's pain management. When pain can be expected after a procedure, parents should be queried regarding their opinion as to how the child will respond. That information should be factored into any treatment plan. In studies, parent opinion has been shown to be the variable that is most predictive of children's response to noxious stimuli ([Schechter et al., 1991](#)). Parents should be present during painful procedures, if at all possible, to help provide comfort ([Bauchner et al., 1996](#); [Shaw and Routh, 1983](#)). They can be taught specific strategies or use other methods, such as stroking, swaddling, and cuddling the child. All of these approaches, as is discussed later, have demonstrated effects on pain.

Another principle of pain management that is somewhat unique to children is the notion that analgesia should be provided in as pain-free a way as possible. Children are fearful of needles, and if pain relief is offered through the use of a needle, children often will not report subsequent discomfort for fear of receiving another injection. If at all possible, nonnoxious routes, such as oral and intravenous routes, should be used for analgesic administration.

Pain associated with diagnostic procedures is for many children the worst part of having a chronic disease. This pain should be aggressively treated, especially early on, so that it does not set in motion a cascade of fear and anxiety about subsequent procedures that colors the child's hospital experience and negatively affects all of his or her encounters with health care providers.

Finally, as much as possible, normative development should be promoted despite the difficulties the child is encountering. School attendance should be emphasized as well as maintaining the child's involvement in social and athletic activities. Pain should be treated aggressively, but secondary gain from it should be minimized. Children should be encouraged to participate in school and social events, so that increased vulnerability and social isolation do not occur.

### **Pharmacologic Treatment of Pain**

A number of drugs from many different categories have a role in the treatment of pain. In general, the administration of pharmacologic agents is part of an overall plan for pain management. The choice of agents is determined by the type and severity of the pain. Some of the agents described are in and of themselves analgesic, whereas others have properties that potentiate analgesia. Categories that are discussed include local anesthetics, which should be used for all needle procedures; nonsteroidal antiinflammatory drugs (NSAIDs), which are used for mild to moderate pain; opioids, which are used for moderate to severe pain; and a variety of adjuvant agents that potentiate analgesia and are more effective for opioid-resistant pain.

#### **LOCAL ANESTHETICS**

Local anesthetics should be used for all needle procedures. There have been significant advances in the availability of local anesthetics and a number of new agents hold enormous promise. A eutectic mixture of local anesthetics (EMLA) combines lidocaine and prilocaine in a cream that when placed under an occlusive dressing produces anesthesia through intact skin. EMLA causes some vasoconstriction and requires approximately an hour to work. It provides anesthesia to a depth of 2 to 4 mm, and is therefore beneficial for venous cannulation, phlebotomy, and other injection-related pain when adequate time is available ([Halperin et al., 1989](#); [Robieux et al., 1991](#)). Lidocaine can be administered through injection or through iontophoresis, in which an electric current drives it through the skin. Because lidocaine burns when injected, it can be buffered with sodium bicarbonate to reduce burning significantly. Amethocaine can be applied as a gel or cream. It works far more rapidly than EMLA and penetrates intact skin in approximately one-half hour. It causes vasodilation instead of vasoconstriction, which may offer advantages over EMLA ([Lawson et al., 1995](#)). Finally, vapocoolant spray also has been used to reduce injection pain. This has the advantage of rapid onset, but the disadvantage of lasting only approximately 10 seconds, with minimal depth of penetration ([Reis and Holobukov, 1997](#)).

Other local anesthetics that are longer acting are beyond the scope of this discussion. Also, discussion regarding drugs used for laceration repair can be found in alternative sources ([Ernst et al., 1995](#)). On the horizon is a long-acting local anesthetic that may last up to 1 week. Such an agent would significantly reduce the need for systemic analgesia after surgery.



## NONOPIOID ANALGESICS

This category contains two drug families, acetaminophen and the NSAIDs. Both categories of drugs have a ceiling effect, that is, a dose beyond which no additional analgesic efficacy is obtained.

Acetaminophen has certain advantages over the NSAIDs, and as a result, it is used more commonly. It seems to have limited effect on the gastrointestinal tract, platelet aggregation, or on the genitourinary tract, all of which are affected by many of the NSAIDs. Unfortunately, acetaminophen overdose has a significant impact on the liver. Its major disadvantage is its lack of antiinflammatory activity compared with NSAIDs.

The NSAIDs appear to work by inhibiting cyclooxygenase (COX), which blocks the synthesis and release of prostaglandins and leukotrienes. Prostaglandins mediate the inflammatory response, which can lead to additional pain in traumatized tissue. NSAIDs have a ceiling effect and their chronic use is limited by their effect on gastric mucosa, platelets, and renal parenchyma. Despite this, drugs such as ibuprofen and naproxen appear to have opioid-sparing and antiinflammatory effects and are an excellent choice for mild to moderate pain. They often are used individually or in conjunction with a “weak” opioid.

Recently, NSAIDs have been developed that have more selective effects. The COX-2 inhibitors work by inhibiting the COX-2 that is released in association with tissue in injury, while sparing COX-1, which has a protective effect on the gastric mucosa ([Peterson and Cryer, 1999](#)). As a result, these agents have antiinflammatory activity without some of the side effects that hamper many of the other NSAIDs.

## OPIOIDS

Opioids are centrally acting analgesics that function by mimicking the actions of endogenous opioids in the central nervous system. These agents are the drugs of choice for moderate to severe pain, and they have a long history of safe use and efficacy in children.

Opioids can be categorized in a number of different ways. One method of categorization is by the potency of analgesia they provide. Traditionally, there have been agents that have been considered “weak” (codeine and oxycodone) that are used for more moderate pain, often in conjunction with a NSAID or acetaminophen. There also are opioids that are considered “strong,” such as morphine and fentanyl. Opioids also can be categorized by their half-life, with the shorter-acting agents being fentanyl, morphine, and meperidine and the long-acting opioids being methadone, morphine sulfate (MS Contin; Purdue Frederick, Norwalk, CT), and oxycodone (OxyContin; Purdue Frederick). They also can be categorized as to their chemical derivation. Morphine, codeine, and hydromorphone are phenanthrene derivatives, whereas fentanyl and meperidine are phenylpiperidine derivatives. This has particular relevance when dealing with opioid side effects or allergy. Finally, they can be categorized by receptor type. Morphine, fentanyl, and codeine are mu receptor agonists, whereas drugs like nalbuphine and butorphanol are mixed agonist–antagonist drugs.

Regardless of which agent is used, a number of basic principles apply:

1. As with other agents, whenever possible, opioids should be used in a scheduled manner as opposed to an as-needed manner.
2. Opioids have no ceiling effect. The right dose is the one that eliminates pain. The doses typically suggested in most tables are the starting doses in opioid-naïve patients.
3. Opioids have a number of predictable dose-related side effects. Constipation and itching are quite frequent. Sedation, respiratory slowing, and respiratory arrest can occur as well. Therefore, when these drugs are used, appropriate monitoring should be put in place.
4. The blood–brain barrier is immature at birth and as a result the central nervous system is more permeable to morphine. Certain enzyme systems that metabolize opioids also are immature at birth. Newborns have a greater unbound fraction of morphine, slower clearance, and more vulnerability to its effects. Therefore, in children younger than 3 months of age, starting doses of morphine should be significantly reduced (one-fourth of the traditional dose for older children) and used only in a carefully monitored setting.

### Specific Opioids

Morphine is the gold standard and the classic mu receptor agonist. Morphine may be administered intravenously, rectally, intrathecally, subcutaneously, as well as by a number of other routes.

Fentanyl is 100 times more potent than morphine. It can be administered intravenously, intramuscularly, subcutaneously, transmucosally, and transdermally. It is used most commonly for short, painful procedures, but can be used for postsurgical pain as well as in continuous infusions or by frequent boluses. When used transdermally, it requires multiple half-lives to achieve efficacy and has prolonged activity after removal of the patch owing to fat storage.

Meperidine is a synthetic opioid that at one time was thought to offer advantages over morphine because it was thought to have less effect on the sphincter of Oddi and on bowel motility. This has been disputed in recent literature, however. More significantly, meperidine has a metabolite, normeperidine, that can cause hallucinations, agitation, and even seizures ([Armstrong and Berston, 1986](#)). As a result, meperidine has become far less popular and is no longer a drug of choice for acute or chronic pain.

Methadone is a long-acting opioid with a half-life of 19 hours, which has significant variability among individuals. It can be used as an alternative to continuous-infusion opioids because of its long half-life. Methadone has historically been used as a drug to maintain opioid-addicted patients and, as a result, carries an unfortunate stigma, but it remains a valuable drug in many acute and chronic pain situations.

Codeine usually is administered by the oral route, typically for mild to moderate pain, and often in conjunction with acetaminophen or NSAIDs. Although equal potency to morphine can be obtained with large doses of codeine, there is a concomitant increase in side effects that make it an inappropriate choice for severe pain. A number of other similar agents (hydrocodone and oxycodone) also are available. It is important when using codeine preparations to be aware of the possibility of acetaminophen toxicity. For example, patients taking 10 acetaminophen plus codeine tablets daily are ingesting over 3 g of acetaminophen, which is above the recommended dose and may cause toxicity.

As previously mentioned, opioids can be administered through a variety of routes, including oral, subcutaneous, intrathecal, epidural, intravenous, subcutaneous, and transmucosal, as well as by patient-controlled analgesia with or without a continuous infusion. They are an excellent choice for severe pain. Although there is significant concern about side effects, in reality, when monitored appropriately, these agents can be used safely. Issues of addiction and dependency have historically constrained opioid use ([Weissman and Haddox, 1989](#)). Most experts agree, however, that the treatment of pain in patients with medical illnesses rarely is associated with subsequent addiction, and that the likelihood of addiction in children is essentially negligible ([Kanner and Foley, 1981](#); [Porter and Jick, 1980](#)). Addiction itself is a psychological disorder reflected in obsessive attention to the drug and the paraphernalia associated with it. Dependency, however, is a physiologic phenomenon that occurs in any individual who uses these agents for over 7 days and can be addressed merely by weaning the child at a rate of 10% to 20% per day.

## ADJUVANT MEDICATIONS

A number of other agents have gained currency in the treatment of pain. In particular, certain antidepressants and anticonvulsants may have activity in opioid-resistant pain, such as neuropathic pain associated with nerve injury. Gabapentin has shown significant efficacy for diabetic neuropathy ([Rowbotham et al., 1998](#)). Tricyclic antidepressants such as nortriptyline and amitriptyline also have shown efficacy in this traditionally opioid-resistant type of pain ([Heiligenstein and Steif, 1990](#)).

Another category of adjuvant medication is the stimulants. These agents by themselves may not have analgesic properties, but they seem to counteract some of the sedation associated with opioids, and seem to have antidepressant qualities as well. They are increasingly used in chronic pain and palliative care medicine ([Bruera et al., 1992](#); [Yee and Berde, 1991](#)).

## Nonpharmacologic Approaches

A number of strategies that do not involve drugs are critical to the comprehensive management of pain. In particular, in chronic pain situations where children may have to cope with some degree of pain for the foreseeable future, strategies that facilitate coping are extremely beneficial. In addition, during painful procedures, a number of strategies in conjunction with pharmacologic approaches have emerged that help children through these trying times

### *PSYCHOLOGICAL/BEHAVIORAL STRATEGIES*

Because children tend to be more suggestible and more trusting than adults, they often respond well to psychological strategies that divert attention from their pain or reframe it.

Preparation of the child for pending procedures has been shown clearly significantly to ameliorate anxiety, which decreases discomfort. Preparation must be done in a developmentally appropriate way, and include both descriptions of the procedure itself, as well as how the procedure will feel ( [Zeltzer et al., 1989](#)). Different children with different personality styles react differently to preparation, and child health professionals need to individualize the approach based on the child's unique temperament and personality. Age-appropriate discussion about procedures has been shown clearly to lessen pain of procedures.

Another nonpharmacologic approach involves the active participation of parents during medical procedures. Although this now seems intuitive, it was not always so, and parents in other eras often were banished from treatment rooms because they were thought to increase the child's anxiety, to become anxious themselves during the procedure, and to increase anxiety in the operator. Overwhelmingly, however, studies that have looked at the role of parents during painful procedures have suggested strongly that children value their parents' presence in the treatment room during procedures ( [Bauchner et al., 1996](#)). Parents themselves, when involved as "coaches" providing comfort to their children during the procedure, feel less helpless and have more control during times that often are threatening and anxiety filled for both parents and children ( [Broome and Endsley, 1989](#)). Parental presence provides comfort for the child because there is a familiar person in the room, which often allows the procedure to be accomplished more effectively. Parents can quickly be taught a number of simple techniques that can help divert a child's attention from the procedure, or they can use other strategies that they have developed while helping their child through difficult times in the past.

Breathing techniques, distraction, and hypnosis are all strategies that help reduce anxiety during procedures. Distraction often involves the use of a breathing technique, counting, or using party blowers ( [Manne et al., 1994](#)) and bubble solutions. Other techniques involve having the parent read with the child or tell the child a favorite story ( [Kuttner, 1989](#)). Visual imagery techniques, where a child imagines that he or she is in a more pleasant place than the treatment room, also have been shown to be quite effective. Hypnosis is the more intense use of fantasy and suggestion ( [Katz et al., 1987](#); [Zeltzer and LeBaron, 1982](#)). Here, the parent involves the child in a fantasy, such as taking a magic carpet ride. Other hypnotic techniques may involve reframing the situation using techniques such as a "pain switch" or a "magic glove," in which children are told that using a particular technique will change the way a painful procedure feels. Although distraction techniques require essentially no training, hypnosis is slightly more complex. There are a number of courses available administered by professional societies to teach hypnosis for pain management in children.

Another nonpharmacologic approach to pain management is through the use of rehearsal. With this technique, children may watch the procedure they are about to undergo on a videotape, speak to someone who has undergone the procedure, and perform some version of the procedure on a stuffed animal ( [Jay et al., 1985](#)). This increased familiarity with what will happen to them gives children some sense of mastery over the upcoming event, and has been clearly associated with reduced pain after the procedure. Finally, there is literature that suggests that increasing one's control over the illness and its treatment can reduce pain ( [Kavanagh, 1983](#)). The ultimate example of this is the use of patient-controlled analgesia, where the child can administer his or her own medication with the use of a specially designed pump. Children older than 7 years of age can use this device, but other strategies to enhance self-control, such as giving the children some sense of control over the site for venous sampling, having them help with bandage removal, or selecting the time of day that something will occur, can be beneficial. All of these techniques clearly reduce significantly the pain associated with procedures and the effects of chronic hospitalization as well.

### *PHYSICAL APPROACHES*

There are a number of physical approaches to pain reduction. They work essentially at the level of the dorsal horn by stimulating larger afferents that carry less noxious messages. Cold, heat, and vibration can be used to reduce pain. Transcutaneous electrical nerve stimulation is a technique that uses a specifically designed instrument that generates various patterns of electrical sensations ( [Lander and Fowler-Kerry, 1993](#)). When a person has pain in an isolated area, such as a limb, electrodes can be placed in an appropriate location and a variety of different patterns of electrical impulses can be tried to dampen the pain messages. This technique has demonstrated efficacy in highly localized pain problems such as complex regional pain syndrome 1 and certain types of postoperative pain. It has less efficacy in more generalized pain problems, such as the pain associated with sickle cell disease.

## **SPECIFIC PAIN PROBLEMS**

### **Postoperative Pain**

Although postoperative pain management is not in the purview of most readers of this text, it is important to discuss it because in many ways it epitomizes the evolution of both attitudes and thinking about pediatric pain. As mentioned earlier in this chapter, inadequate postoperative pain management was the standard of care in the 1970s and 1980s. More recent surveys clearly suggest that there has been a dramatic improvement in pain management in children, although other investigators continue to question the actual degree of change ( [Hamers et al., 1998](#)). Earlier on, essentially no institutions had protocols in place to provide a uniform standard of care for children, but now most institutions that care for children have developed standardized approaches. In surveys of hospitalized patients, postoperative pain management was the one area in which there was general agreement that significant improvement had occurred ( [Johnston et al., 1992](#)). The predictability of postoperative pain has helped to foster the development of plans to ameliorate it. Unfortunately, with the escalation of day-case surgery, postoperative pain management once again is a problem because most of it must be done at home by parents, many of whom are uncomfortable medicating their child ( [Finley et al., 1996](#)). Another change that has occurred in the area of postoperative pain management is the increased use of regional techniques, such as epidurals. These approaches allow for better postoperative pain control without the use of systemic opioids.

In general, however, when regional techniques are not used, most clinicians order either continuous infusions or boluses of morphine every 2 hours for postoperative pain. For children older than 7 years of age, patient-controlled analgesia is available and often a desirable option. For infants younger than 6 months of age, a significant reduction in opioid dose is required, and administration needs to occur in a carefully monitored setting such as an intensive care unit.

What is most critical is that the general principles that have been mentioned are put in place. Good assessment is absolutely essential. It should naturally be assumed that what would hurt an adult would hurt a child, and therefore children who undergo painful surgeries should have scheduled postoperative analgesics given that predictable pain will ensue. Noxious routes should be avoided and the doses traditionally listed should be viewed only as suggested starting doses.

### **Procedure Pain**

For many children, the necessary medical procedures associated with diagnosis and treatment of disease are by far the worst part of their illness. For example, work by [McGrath et al. \(1990\)](#) and [Miser et al. \(1987\)](#) clearly suggests that for children with cancer, bone marrow aspirations and lumbar punctures are perceived by the children as worse than the disease itself. If inadequate analgesia is provided during the initial procedures, a cycle of fear and dread of future procedures may occur and subsequent procedures may be considered more painful than if adequate anesthesia was initially offered ( [Weisman et al., 1998](#)). Therefore, significant attention must be paid to pain management during procedures, especially at the outset of an illness.

A number of general principles have evolved that attempt to minimize pain and distress associated with procedures. Obviously, the intervention should be tailored to the child's own personality characteristics. Some children may require general anesthesia because of overwhelming anxiety and worry, whereas others may be far less responsive to the procedure. If at all possible, procedures should be performed in a treatment room and not in the child's room, which should function as a refuge from painful events. The people performing the procedures should be as skilled as possible. Because of the potential for negative imprinting that can occur, trainees should perform procedures only if a child has had a significant anesthesia/analgesia. Behavioral and pharmacologic techniques should be used jointly, as has been suggested for all pain problems. Local anesthetics should be used for all needle procedures, and parents should be present and involved as coaches. If the pain associated with the procedure is considered moderate or severe, sedation should be used. If sedation is used, many professional societies have developed specific guidelines that involve monitoring criteria, eating before the anesthesia, availability of resuscitative drugs and equipment, and the presence of appropriately trained personnel ( [American Academy of Pediatrics, Committee on Drugs, 1992](#); [American Society of Anesthesiologists, 1996](#)). Behavioral-cognitive strategies, such as



distraction and visual imagery, should be used even if pharmacologic sedation is scheduled. As was previously mentioned, if a series of painful procedures is necessary, aggressive pharmacologic approaches should be used during the first procedure so that a negative cycle of fear and anxiety does not occur.

### **Pain Associated with Chronic Disease**

Chronic diseases that affect children often are associated with pain. The pain may be secondary to the disease itself, to the procedures that are necessary to evaluate the disease, or to the treatment associated with the disease, such as radiation or chemotherapy for children with cancer. Because it is less predictable than other types of pain, such as postoperative pain, pain associated with chronic disease often is less well treated. In a survey of hospitalized children done in the mid-1990s, most of the children with less well treated pain problems were those with chronic disease, compared with those who were postoperative ( [Johnston et al., 1992](#)).

A number of general principles have emerged in the treatment of chronic pain in children. Because there can be multiple sources of pain, a pain problem list often is helpful so that the clinician can monitor pain from various sources and be sure that each is adequately addressed. Often, asking children to keep a pain diary is a valuable way of gathering this information, as well as empowering children by involving them in the diagnostic and treatment process. Analgesics should be administered in a scheduled way when pain is predictable. It often is helpful to use a long-acting medication with a short-acting medication for breakthrough pain. If frequent use of the short-acting medication is necessary, the dose of the long-acting medication should be increased. Once children are on stable therapy, if a dramatic dose escalation is necessary for pain relief, initially the clinician should consider disease extension before tolerance is considered and a thorough search for it should be undertaken.

There is a growing literature on the use of opioids for nonmalignant pain ( [Portenoy, 1996](#); [Zenz et al., 1992](#)). In the past, obsessive preoccupation with diversion and addiction was the norm, but now it is believed that these concerns should not restrict the humane use of opioids for individuals with chronic pain that interferes with their life. As much as possible, normal behavior should be encouraged and pain should not be allowed to become the major focus of the child's life. Normal development should be encouraged; school attendance should be demanded whenever possible. Physical activity has multiple benefits, and should be dictated by the child's level of impairment. Whatever that level is, however, some physical therapy or physical activity should be built into the child's schedule. Not only does this help with normative development in maintaining adequate muscle tone, but it helps with increased appetite and promotes restorative sleep at night.

Children with pain associated with chronic disease often benefit from nonpharmacologic strategies because they will have to live with pain for the foreseeable future and strategies that help them cope are extremely beneficial. Relaxation, self-hypnosis, and breathing techniques allow them to have some role in controlling their own pain. Depression often accompanies chronic pain and should be dealt with psychotherapeutically and pharmacologically, if necessary.

### **CHRONIC PAIN WITHOUT PATHOLOGY**

A number of chronic pain conditions exist in which there is no obvious alteration of physiology or anatomy. Recurrent abdominal pain, headache, "growing pains," fibromyalgia, and chronic fatigue syndrome are all situations in which a diagnostic process often yields no specific answers. Such conditions, however, should be handled using approaches similar to those used in pain associated with chronic disease. These include encouraging normative behavior and development, active involvement of the child in assessment through use of pain diaries, emphasis on nonpharmacologic techniques, pharmacologic control of pain, and therapy for depression or other psychological contributions to the child's discomfort.

### **SUMMARY**

The management of pain in children has undergone remarkable changes in a short time. The available literature has escalated exponentially, and attitudes have undergone significant revision as well. Practice patterns also have changed in many areas. Certainly, for predictable pain problems, we have made dramatic strides, but for less predictable pain patterns, we still have a way to go.

As emphasized throughout this chapter, the treatment of pain is essential to the humane care of children. Assessment is the cornerstone of that treatment, and needs to be undertaken in a uniform, developmentally appropriate manner. A number of pharmacologic and nonpharmacologic strategies are available, and most should be used jointly. Most important, pain should be anticipated and treated in a preventative manner if at all possible.

The multidisciplinary management of pain is one of the genuine advances in medical science in the past 20 years and should be considered an essential part of the compassionate medical care of children.

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## 32 GENETIC INFLUENCES ON CHILD PSYCHIATRIC CONDITIONS

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In child psychiatry, interest in genetic factors is at an all-time high. Psychiatric disorders often are transmitted within families, and research has implicated genetic factors in a variety of mental, developmental, and behavioral disorders of childhood onset ( [Lombroso et al., 1994](#)). Specific genetic mutations have been characterized in some developmental neuropsychiatric disorders (e.g., fragile X syndrome and Prader-Willi syndrome) although, thus far, identification of etiologic gene mutations in psychiatric illness has been unsuccessful.

Psychiatric disorders are examples of complex traits. As such, the transmission within families does not follow classic mendelian inheritance patterns, and it is presumed that a number of factors contribute to the ultimate expression of clinical psychiatric symptoms. These factors may include environmental variables, such as gestational and psychosocial conditions, biological variables such as comorbid medical conditions, and genetic factors. The genetic factors could be single loci, or as is more likely, psychiatric disorders could result from the expression of more than one gene (oligogenic or polygenic) in a larger context of potentially numerous environmental influences. It is quite likely that none of these etiologic factors is necessary or sufficient for the expression of any psychiatric disorder.

Although vital contributions come from research on environmental influences in psychiatric disorders, genetic study of disease yields evidence about the underlying biology and transmission of the illness, two important areas related to treatment and prevention. It is expected that early identification of vulnerability to psychiatric illness would allow for the development of early intervention and prevention strategies.

Considerable work has been done since the early 1990s to develop valid and reliable assessment instruments for the diagnosis and measurement of child psychiatric disorders. These instruments have made it more feasible to undertake genetic studies of childhood disorders. Concurrently, the development of more sophisticated genetic methods and successful genetic studies of other complex human diseases (e.g., colon cancer, diabetes, hypercholesterolemia) has led to an overall increased interest in the importance of genetic factors for the manifestation and expression of human disease.

Interest in the genetics of child psychiatric disorders is a part of a substantial increase in basic research in child psychiatry since the early 1980s. The purpose of this chapter is to summarize the methods suitable for the genetic study of child psychiatric conditions and to provide a brief review of the current understanding of the genetics of several of these disorders.

### METHODS SUITABLE FOR THE GENETIC STUDY OF CHILDHOOD DISORDERS

Historically, the approach taken in human genetics to determine if an illness has a genetic basis was to search for an aberrant or missing protein that would result in some observable structural or biochemical change. Emphasis has now shifted to the direct detection of altered deoxyribonucleic acid (DNA) sequences. Although this approach has been successful for many human diseases, it has not been effective in the study of psychiatric/behavioral disorders of childhood. In the absence of such data, other methods are useful to help establish the importance of genetic factors in the expression of an illness. Those methods include (a) studies of twins, (b) studies of adopted children, and (c) studies of families.

#### Twin Studies

Twin studies are quite useful in establishing that genetic factors are important in the manifestation and expression of a disorder ( [LaBuda et al., 1993](#); [Spector et al., 2000](#)). Historically, this approach came into use to estimate the relative contribution of genetic and environmental influences. The underlying genetic theory is straightforward. Monozygotic (MZ) twins are genetically identical. If a trait is completely determined by genetic factors, then MZ twins should have exactly the same phenotype. In contrast to MZ twins, dizygotic (DZ) twins share, on average, only 50% of their genetic material. Thus, if there is no unique contribution of twin environments, DZ twins should be no more similar than singleton siblings. For example, if a trait is determined by a fully penetrant autosomal dominant gene, then the concordance rate for DZ twins should be 0.50, compared with 1.0 for MZ twins. If a trait is determined by an autosomal recessive gene, then the expected concordance for DZ twins would be 0.25.

When inheritance is complex, mendelian patterns are not observed, and the concordance rates for MZ and DZ twins are not expected to approximate mendelian segregation ratios. In these circumstances, a significantly greater concordance for MZ twins compared with DZ twins is taken as evidence for the involvement of genetic factors in the phenotype being studied.

One of the advantages of the twin methodology is that it is possible to study the contribution of genetic factors to traits that may not present a stable phenotype throughout the course of development. A major complication of research designs that require assessment of children and adults in families is the determination of the "childhood affected status" of adults in the family units under study. Although various attempts have been made both to establish methods to assess adult phenotypes of childhood disorders and to develop methods to make retrospective diagnoses of childhood conditions, major questions remain concerning their reliability and validity. Twin studies can surmount these difficulties because twin pairs can be studied in the appropriate development epoch, so that reliable assessments can be made.

As has been pointed out ( [Pauls, 1985](#)), one of the most interesting applications of the twin study paradigm is the examination of discordant MZ twin pairs. Assuming a genetic etiology for a behavioral disturbance, MZ twins should show full concordance rates. The fact that they do not implies that environmental factors also are of etiologic importance. A closer examination of discordant twins should help to identify important nonheritable factors that mediate the expression of the underlying genetic vulnerability.

Another potentially important use of twin studies has been illustrated by [Plomin and Daniels \(1987\)](#). These investigators have demonstrated the ability for twin studies to estimate the nonshared environmental variance important for the manifestation of a phenotype. As these investigators have pointed out, nonshared environment may be the most influential factor in contributing to the phenotypes of children within families.

The greatest weakness mentioned by critics of the twin studies is the assumption that the environments for MZ and DZ twins are comparable. The assumption has been examined ( [Cohen et al., 1975](#)), and no relevant differences have been found. In addition, as [Rowe \(1983\)](#) has indicated, it is not enough to show that DZ twins differ from MZ twins or that singletons differ from twins and use this to condemn twin studies in general. It must be demonstrated that such differences in biology or environment are etiologically important for the particular disturbance under investigation.



## Adoption/Separation Studies

If there is a substantial environmental component important for the manifestation of a disorder, the twin study design cannot completely separate its effect from the genetic factors that also may influence the trait. Adoption or separation studies provide a method to examine more closely the extent to which environmental factors influence the phenotype being studied. If a trait is genetic, then children should resemble their biological relatives to a greater degree than they resemble their adoptive relatives. Thus, the extent to which adopted children resemble their biological parents is taken as evidence of a genetic contribution. If children resemble their unrelated adoptive relatives to any degree, then that resemblance is, at least in theory, due to shared environments. Separation studies therefore provide a potentially powerful method of separating the effects of genes and environment.

Although quite useful, adoption studies have some significant drawbacks in the study of childhood disorders. In fact, it is noteworthy that few adoption studies of childhood psychiatric disorders have been reported. One reason for this could be that it is critical to obtain reliable diagnostic assessments of the parents as children. Because much of the information about the biological parents comes from obstetric records, it often is impossible to retrieve the necessary information for reliable and valid assessment of the parents. A further complication is that data about the biological father often are completely missing. Therefore, it is not possible to evaluate the phenotype of the child in light of the phenotype of the father. As pointed out ( [Pauls, 1985](#); [Rutter et al., 1990](#) ), missing or incomplete data regarding one or both biological parents can seriously compromise the usefulness of adoption studies. One way to overcome this limitation is through the use of prospective longitudinal study designs ( [Plomin and DeFries, 1985](#); [Plomin et al., 1988](#) ).

## Family Studies

As discussed, twin and adoption/separation studies provide data useful in establishing that genetic factors are important for the expression of a disorder. However, both approaches are limited in that it is not possible to test hypotheses about specific genetic mechanisms of transmission. Studies of biological families yield data that allow examination of specific genetic hypotheses. However, as with twin studies, if there is a major environmental component important for the manifestation of the phenotype under investigation, then data from family studies will not prove the existence of genetic factors. Genetic and family environmental factors can interact in determining similarities and differences among relatives and cannot be unambiguously quantified without prior specific identification. Thus, it is important to distinguish between shared and nonshared familial and nonfamilial environmental factors. It also is important to keep in mind the possibility of confounding familial environmental similarity and genetic similarity within a given family.

Family studies have been helpful in establishing that most major adult psychiatric disorders are inherited. A major limitation of family studies for childhood psychiatric disorders is related to assessment and diagnosis, the same limitation as was discussed for adoption studies. It is essential that all relatives in a family study be reliably and validly assessed. Because there always are at least two adults (i.e., the parents) in every nuclear family, it is essential that methods be available to obtain the necessary information to assess the childhood phenotype of those adults. Retrospective data may be unreliable or unavailable. Information regarding current symptomatology is useful only if there is a one-to-one correspondence between the childhood phenotype and adult phenotype. Clearly, more longitudinal studies need to be completed to learn more about the adult phenotypic outcomes of child psychiatric disorders.

## Molecular Genetic Studies

Although results from twin, adoption/separation, and family studies can provide convincing evidence that genetic factors contribute significantly to the expression of childhood psychiatric disorders, none of these methods can prove the existence of specific genetic etiologic factors. One of the strongest forms of evidence for a genetic etiology is the demonstration of a molecular abnormality or the existence of a linked genetic marker. The search for specific molecular abnormalities or biochemical markers so far has not been fruitful in child psychiatry. A potentially more promising approach is the use of genetic linkage information.

Genetic linkage occurs when two different loci are sufficiently close on the same chromosome that they are transmitted together from parent to child. The demonstration of linkage in humans requires family studies to establish that alleles at the two loci cosegregate in families. Family data are used to estimate how frequently alleles at two loci are transmitted in combinations different from those occurring in the parents. If new combinations occur with equal frequency to parental combinations, then the two loci are not linked. If the frequency of parental combinations is relatively greater than that of new recombinants, the two loci are linked, and the frequency of new recombinants can be used to estimate the strength of linkage. The degree of linkage is measured as the recombination fraction and can range from 0.0 (complete linkage) to 0.5 (independent assortment). The minimum recombination frequency of 0.0 is found when alleles at two separate loci always are transmitted together. The maximum recombination frequency of 0.5 is found for alleles that have the same likelihood of being transmitted in new combinations as in the old combinations, from generation to generation. The maximum recombination occurs for loci far apart on the same chromosome and for loci on different chromosomes.

The statistical significance for a particular recombination fraction is determined by measuring the difference in the probability of observing linkage and comparing it with the probability of observing independent assortment in a given family. Finding statistically significant deviation from independent assortment for alleles at a known marker locus and at a hypothesized locus for a complex trait provides convincing evidence that there is a major gene contributing to the expression of the disorder. It is highly unlikely that any other explanation could mimic linkage with a marker locus.

Some problems in detecting linkage in human data include small family size, the inability to control matings, and the small prior probability that two loci are linked. The linkage method has had limited applicability until recently because of the paucity of available marker loci in humans. With the advent of recombinant DNA technology, this has changed dramatically. As part of the advance in genetics brought about by recombinant DNA techniques, there are now tens of thousands of DNA polymorphisms available for use in genetic linkage and association studies.

The current inventory of marker loci now spans the entire human genome at a remarkably fine level. In fact, the completion of the human genome sequence ( [International Human Genome Sequencing Consortium, 2001](#); [Venter et al., 2001](#) ) gives promise that in the not-distant future all human genes will be mapped and characterized. In parallel with that accomplishment, efforts are underway to identify and validate all segments of the genome that comprise functional genes. As the total human catalog of approximately 35,000 genes is annotated, it will be necessary to elucidate the sequence variations that are associated with functional differences.

Microsatellite polymorphisms (also referred to as *short tandem repeats*, or STRs) were the preferred markers of variation throughout the 1990s ( [Weber and May, 1989](#) ), and many thousands of these were identified. Because these markers are characterized by high levels of heterozygosity and large numbers of alleles, they provide a very useful tool for pedigree-based linkage analysis. The use of these markers has led to the mapping of a respectable number of monogenic and a few polygenic diseases. More recently, however, a new generation of markers has emerged based on a class of polymorphisms that results from single nucleotide changes. Such single-nucleotide polymorphisms (SNPs) will be extremely important in cataloging the variation within genes that is responsible for increased risk of or protection from disease. SNPs usually are biallelic and consequently less polymorphic than microsatellites, but their great abundance and accessibility to high-throughput, low-cost automated genotyping technologies eventually may lead to the replacement of microsatellites in gene mapping and positional cloning.

Given the phenotypic and apparent genetic complexity of child psychiatric disorders, traditional linkage methodologies may not be well suited for identifying genes of etiologic significance. In fact, most of the current studies involve approaches that do not require specification of the underlying genetic mechanism. In addition, given the availability of tens of thousands of polymorphic markers, an alternative approach that involves looking for population associations between known genes or markers and disorders has been suggested ( [Risch and Merikangas, 1996](#) ), although many difficulties remain ( [Chakravarti, 1998](#) ).

It is clear that a great deal of effort remains to identify rigorously susceptibility genes for child psychiatric disorders. One of the most difficult problems with linkage and association studies of psychiatric disorders has to do with understanding the precise nature of the inherited phenotype. All mental disorders have variable expression, and the data from twin studies usually have demonstrated the importance of nongenetic factors in their etiology. Nongenetic (or environmental) factors, along with variable expressivity and variable age of onset, reduce the power of the statistical methods available for the detection of linkage and association. Although an increase in the size of the sample being studied often helps to increase statistical power, a better understanding of the inherited phenotype is critical to the eventual identification of susceptibility genes for child psychiatric disorders. It is becoming increasingly clear that *Diagnostic and Statistical Manual of Mental Disorders* (DSM) diagnostic categories may not be optimal phenotypes for genetic studies that are designed to find and characterize genes for psychiatric disorders.

In the absence of a well understood etiology, demonstration of the presence of genetic factors usually does not resolve questions about the nature of the genes involved and how they interact with environmental factors. Is a particular gene necessary for the disease to develop? Will a certain genotype always lead to illness?

Are there ameliorating environments that prevent illness in people otherwise genetically susceptible? These questions need to be addressed. The full scope of gene–environment interactions can be understood only after the full disease process is understood.

Establishing that a disease locus is linked to a specific marker will be a major step in understanding the pathophysiology of child psychiatric disorders. However, before this methodology can be used successfully, there needs to be fairly strong evidence that at least some forms of a specific illness have an underlying genetic mechanism that is relatively simple (i.e., one or two major genes contributing significantly to the expression of the phenotype). As is discussed in the following section, there is considerable evidence that childhood psychiatric/behavioral disorders are influenced by genetic factors. However, the specific mode of transmission is not understood for most disorders of childhood; thus, the pursuit of genetic linkages needs to occur in the context of other studies examining specific genetic hypotheses of transmission.

## REVIEW OF THE GENETICS OF CHILDHOOD NEUROPSYCHIATRIC DISORDERS

In this section, the evidence that genetic factors are important for some of the more extensively studied childhood disorders is reviewed. A comprehensive review of all childhood disorders is beyond the scope of this chapter; for the current presentation, disorders were chosen that provide examples of the application of the approaches discussed previously.

### Attention Deficit/Hyperactivity Disorder

Attention deficit/hyperactivity disorder (ADHD) is a common neuropsychiatric and behavioral disorder of childhood onset ( [Barkley, 1998](#)). The prevalence of ADHD is high; it is the most frequently observed neurobehavioral problem in the pediatric age group. Prevalence rates have been estimated to range between 2% and 15% ( [Rutter, 1983](#)), and boys are affected approximately four times as often as girls ( [Shaywitz et al., 1983](#)). Although a variety of etiologies have been identified for some cases (infection—[Shaywitz and Shaywitz, 1982](#); head trauma—[Rutter, 1981](#); pregnancy and birth complications—[Whittaker et al., 1997](#); and intrauterine exposure to toxins—[Shaywitz et al., 1983](#)); in most individuals, no single etiologic factor is evident.

Cumulative results from several lines of investigation yield evidence that genetic factors play an important etiologic role in the expression of hyperactive and inattentive behaviors and ADHD. Results of several twin studies suggest that genetic factors are important for the expression of normal activity levels ( [Scarr, 1966](#); [Vandenberg, 1962](#); [Willerman, 1973](#)). However, many ADHD studies are weakened by uncertain validity of diagnosis and rating scales, small sample sizes, and ascertainment bias.

In an early study, [Lopez \(1965\)](#) examined 10 twin pairs (4 MZ, 6 DZ) in which one twin had “clinical hyperactivity.” He found 100% concordance in the MZ pairs, compared with 17% concordance in the DZ pairs. However, in light of several methodologic weaknesses in this study (four of the DZ twins were opposite-sex pairs), these results could not be considered definitive. In a large study of 13-year-old twin pairs (102 MZ and 111 same-sex DZ twin pairs), [Goodman and Stevenson \(1989\)](#) found that MZ twins had higher concordance on objective measures of inattentiveness and hyperactivity than same-sex DZ twins. Family environment measures and perinatal adversity did not significantly relate to hyperactivity measures. The researchers concluded that heritability accounted for 30% to 50% of the explainable variance in inattention and hyperactivity in this twin population. The common environmental effects accounted for less than 30% of the variance. [Stevenson \(1992\)](#) performed multiple regression analysis on data from a sample of 91 MZ twins and 105 same-sex DZ twins. These results also showed a significant genetic contribution to individual differences in activity levels (those measured through maternal ratings but not teacher ratings) and attention abilities. An important consideration is that the degree of heritability for hyperactivity and inattention is higher when estimated in a sample of relatives of severely affected ADHD probands, suggesting that the more deviant ADHD symptoms are linked to higher amounts of genetic influence (e.g., [Edelbrock et al., 1995](#); [Gillis et al., 1992](#)). More recent twin studies have supported this conclusion, indicating that most of the phenotypic hyperactivity-impulsivity variance (70% to 91%) is attributable to the genetic variance and that the amount of variance increases with the increased severity of the phenotype (e.g., [Faraone, 1996](#); [Gjone et al., 1996](#); [Levy et al., 1997](#); [Sherman et al., 1997](#); [van der Oord et al., 1996](#)).

Family history studies also provide support for the hypothesis that hyperactivity (and, by implication, ADHD) is familial. [Morrison and Stewart \(1971\)](#) surveyed first- and second-degree relatives of 59 hyperactive children and 41 control children. Twenty percent of parents of children with ADHD were retrospectively diagnosed as hyperactive, compared with 5% of parents of the control group. When data from first- and second-degree relatives were combined, the rates of hyperactivity were significantly higher in the relatives of the hyperactive children. [Cantwell \(1972\)](#) also showed that the frequency of hyperactivity was higher among relatives of hyperactive children than among relatives of control children. In a clinic sample of children (n = 72), [Pauls et al. \(1983\)](#) demonstrated that ADHD is familial and that it is vertically transmitted in families. Logistic regression analyses of family history data incorporating sex of the proband, parental affected status, and family history most adequately explained the patterns of ADHD in these families.

In addition, families of ADHD probands are characterized by higher overall rates of psychopathology, including higher rates of ADHD, conduct problems, substance abuse, and depression (e.g., [Barkley et al., 1990](#)). Conduct problems, substance abuse, and depression in the parents, however, is related more to the presence of conduct disorder in the children with ADHD than to ADHD itself ( [Barkley, 1998](#)).

Separation studies also suggest a genetic component in ADHD. [Morrison and Stewart \(1973\)](#) compiled self-report data from the adoptive parents of 35 hyperactive children. The adoptive parents reported lower levels of childhood hyperactivity among their biological relatives than the control group of biological parents raising their own hyperactive children. Because no information was obtained from the biological parents of the adopted children, no direct comparisons could be made between the adoptive and biological parents of the 35 adopted hyperactive children. In a separate study of adopted children, [Cantwell \(1975\)](#) reported results similar to those of [Morrison and Stewart \(1973\)](#). A weakness of both studies is bias resulting from the selection process permitting families to adopt and the inability to compare the families of the biological parents of adopted hyperactive children with the families of the adoptive parents of these same children. Nevertheless, these studies show that children may more closely resemble the families of their biological parents than those of their adoptive parents. One large study, however, studied biological parents of adoptees and found that if one of the biological parents had been judged delinquent or had an adult criminal conviction, the adopted-away son had a higher likelihood of having ADHD ( [Cadoret and Stewart, 1991](#)). A study of international adoptees revealed a significant genetic contribution (47%) to the variability on the Attention Problems Scale of the Child Behavior Checklist that is highly correlated with the ADHD diagnosis ( [van den Oord et al., 1994](#)). Hence, these results suggest that some genetic component is important for the manifestation of hyperactivity and inattention.

Another line of evidence for a genetic component of ADHD comes from a study comparing full- and half-sibling pairs ( [Safer, 1973](#)). In a comparison of the concordance of such sibling pairs for the diagnosis of minimal brain dysfunction, significantly higher concordance levels were found among the full-sibling pairs than among the half-sibling pairs.

In more recent family studies of patients with ADHD, [Biederman and coworkers \(1986, 1987a, 1987b\)](#) reported a higher frequency of ADHD among first-degree relatives of affected probands. These investigators interviewed all family members of 22 male subjects with ADHD and 20 unaffected control subjects. The morbidity ratio for ADHD was 31.5% among relatives of subjects with ADHD, compared with 5.7% for relatives of control subjects. In addition, rates of major affective disorder, conduct disorder, and oppositional disorder were significantly increased in the relatives of the ADHD probands. This study was the first to assess directly all first-degree relatives using structured interviews. The results give strong support to the hypothesis that ADHD is familial. However, because of comorbidity in the probands, it is not clear whether ADHD is a primary or secondary phenomenon. It appears that the increased rates of conduct and oppositional disorders occurred only in those families where the ADHD proband also had conduct disorder ( [Biederman et al., 1987a](#)). Although this suggests some specificity of transmission, the sample was too small to obtain definitive results. In addition, the considerable comorbidity (affective disorders and anxiety disorders) in first-degree relatives of children with ADHD complicates genetic interpretations of these findings ( [Biederman et al., 1991a, 1991b](#)). More recent analyses of ADHD probands (n = 140) and first-degree relatives explored the validity of subgrouping probands by comorbid status and demonstrated evidence for family-genetic risk factors in ADHD ( [Biederman et al., 1992](#)). Between 10% to 35% of the immediate family members of children with ADHD also were likely to have the disorder, with the risk of ADHD to siblings of affected children at approximately 32% ( [Biederman et al., 1992](#)), and the risk to children of parents with ADHD at approximately 57% ( [Biederman et al., 1995](#)).

Although all of these studies lend support to a hypothesis that some heritable component is important in the expression of ADHD, no clear mode of transmission has been determined. Because of the male preponderance, [Omenn \(1973\)](#) examined a sex-linked hypothesis. He concluded that it was unlikely because of the frequency of father-to-son transmission. [Morrison and Stewart \(1974\)](#) suggested a polygenic mode of transmission, but the report was based on a small number of families of hyperactive children. In a genetic latent structure analysis of dysmorphology in children with ADHD, an autosomal dominant model emerged when dysmorphology and ADHD were compiled into a single factor ( [Deutsch et al., 1990](#)). However, the validity of this model has not yet been tested.

Quantitative genetic analyses of the Boston familial dataset suggests a single-gene mode of ADHD inheritance ( [Faraone et al., 1992](#)). This finding started a string of



studies looking for genes involved in the expression of the disorder. The first candidate gene was the dopamine type 2 receptor (DRD2), with some researchers indicating the association between DRD2 and ADHD ([Blum et al., 1996](#); [Comings et al., 1991](#)) and others failing to replicate the association ([Gelernter et al., 1991](#)). The second candidate gene is the dopamine transporter (DAT1); the evidence for the involvement of DAT1 also is controversial, both supporting ([Cook et al., 1995, 1998](#); [Gill et al., 1997](#)) and dismissing ([Swanson et al., 1998](#)) the association. Yet another interesting candidate gene is DRD4. Similar to other candidate genes, some researchers reported the association ([LaHoste et al., 1996](#); [Muglia et al., 2000](#)), whereas others could not replicate its presence (e.g., [Hawi et al., 2000](#); [Kotler et al., 2000](#)).

In an interesting study of 18 families by [Hauser et al. \(1993\)](#), ADHD was strongly associated with a generalized resistance to thyroid hormone (RTH). This thyroid disorder is caused by mutations in the thyroid receptor beta gene and characterized by reduced responsiveness of peripheral and pituitary tissues to the actions of thyroid hormone. This study showed that, among the affected children, 70% met criteria for ADHD, whereas 20% of those who were unaffected met ADHD criteria. Among the affected adults, 50% met criteria for ADHD as children, compared with 2% of the unaffected subjects. Although there is no (converse) increased incidence of RTH in children with ADHD ([Weiss et al., 1993](#)), this finding should help guide future genetic and biological/hormonal studies of ADHD. For example, in a later study, researchers found approximately 50% comorbidity between RTH and ADHD in a group of RTH probands ([Stein et al., 1995](#)). However, given that RTH is rare in children with ADHD (approximately 1:2,500) ([Elia et al., 1994](#)), thyroid dysfunction appears unlikely to be a major cause of ADHD in the general population.

Thus far, no clear understanding of the genetics or the pattern of transmission of ADHD has emerged. Early studies were limited because (a) only families of male subjects were examined, (b) only parents of subjects were included, or (c) sample sizes were too small. More recent studies have used diverse ascertainment schemes and are characterized by larger sample sizes. In summary, this research has indicated an etiologic complexity underlying ADHD, with a number of areas where advances might enhance our understanding of the nature of the disorder. Specifically, the phenotype of ADHD needs clarification; a scientific unification of the nosologic classification of ADHD (e.g., inattention and hyperactivity-impulsivity subtypes) needs to be developed; and the effect of comorbid illness on ADHD classification needs to be explored.

## Autism

Evidence for a genetic contribution to the syndrome of autism comes from twin studies and family studies. Several reports of twins appeared in the literature before 1977. [Rutter \(1967\)](#) reviewed all twin studies reported before 1967 and concluded that no valid inferences about a genetic contribution to autism could be drawn from them. Additional reports appeared during the next 10 years ([Kean, 1975](#); [Kotsopoulos, 1976](#); [McQuaid, 1975](#)), but the conclusion remained unchanged ([Hanson and Gottesman, 1976](#)).

The results of another twin study were reported by [Ritvo et al. \(1985a\)](#). Their sample included 40 twin pairs ascertained through a registry established to identify high-density families and twin pairs. Individuals were included in the registry as a result of response to an advertisement published in a newsletter of the National Society for Autistic Children. The sample, thus, does not represent a random ascertainment of twin pairs. There were 23 MZ and 17 DZ twin pairs. The concordance rate was 95.7% for the MZ twins, compared with 23.5% for the DZ twins. These rates were significantly different and seem to present striking evidence for genetic factors. It is difficult to interpret these results, however, because the sample relied on voluntary participation. It also included opposite-sex twins in the DZ sample, which, as explained later, skews results. Thus, although the concordance rate evidence is impressive, it is of limited value because it is not clear what biases were operating in the selection of the sample.

The twin studies reported before 1977 and the study by [Ritvo et al. \(1985a\)](#) are difficult to interpret because of their methods of ascertainment. Most of the studies before 1977 appeared as case reports in the literature, and as such represented a biased sample of twins. [Folstein and Rutter \(1977\)](#), in summarizing the reports of 32 twin pairs that appeared before 1977, suggested two problems in the interpretation of the existing data. First, the sample of 32 pairs contained approximately twice as many MZ as DZ twin pairs (22 vs. 10). The frequency of DZ twins is approximately twofold that of MZ twins in the general population, and therefore, these 32 pairs are not a representative sample of twins. The same can be said for the [Ritvo et al. sample \(1985a\)](#), which contained 23 MZ and 17 DZ twin pairs. Second, in all of these studies, many of the twin pairs were opposite-sex pairs. With the reported sex difference in the frequency of autism, opposite-sex twin pairs are of limited value because their inclusion would tend to decrease the DZ concordance rate. In addition, few reports of same-sex twin pairs contained adequate clinical description and evidence of zygosity to allow confident interpretation of the data.

In response to the problems of ascertainment bias, [Folstein and Rutter \(1977\)](#) sought to obtain a complete and unbiased sample of same-sex twin pairs that included at least one autistic child. They reported on a study of 21 pairs of same-sex twins that were systematically collected through schools, hospital twin registries, and the National Society for Autistic Children in England. Of the 21 pairs, 11 were MZ twins. The diagnosis of autism was based on the criteria developed by [Kanner \(1943\)](#) and [Rutter \(1971\)](#). In all, the 21 twin pairs gave rise to 25 autistic children. Four of the MZ co-twins were diagnosed as having autism, whereas none of the DZ co-twins met the criteria for the syndrome. This difference is significant ( $p = 0.055$ ).

Another twin study using a similar ascertainment strategy was reported by [Steffenburg et al. \(1989\)](#). These investigators screened all Nordic countries for the occurrence of cases of autism with a same-sex twin younger than 25 years of age. Twenty-one pairs (11 MZ and 10 DZ) of twins and one set of MZ triplets were ascertained. Zygosity testing was completed for 18 of the twin pairs. In the remaining four pairs, zygosity was determined on the basis of placental evidence and physical appearance. The pairwise concordance for autism was 91% for MZ twins and 0% for DZ twins.

Both of these studies suggest that genetic factors are important in the expression of autism. However, because the MZ twins were not always fully concordant, they also suggest that nongenetic factors exist that may be of etiologic importance. Discordance for autism among twins could be explained if autism represents only the most severe expression of some other cognitive disorder. Presumably, several factors could interact to produce the severe phenotype, but the underlying genetic liability would be for a milder cognitive impairment. [Folstein and Rutter \(1977\)](#) developed this hypothesis. They proposed that if it was correct, the co-twins might exhibit some cognitive impairment without necessarily manifesting the full syndrome of autism. When Folstein and Rutter examined the co-twins in their study for some cognitive deficit, they found that in addition to the four autistic MZ co-twins, five other MZ co-twins showed some cognitive impairment. Hence, 9 of 11 MZ twin pairs were concordant for a cognitive deficit, whereas only 1 of 10 DZ twin pairs was concordant for a cognitive impairment. This difference is highly significant ( $p = 0.0015$ ). Further evidence for this hypothesis comes from a subsequent twin study ([Bailey et al., 1995](#)) that found 60% of MZ twins concordant for autism versus 0% for DZ pairs. Most of the nonautistic MZ twins were beset by social difficulties when examined at follow-up; using a combined broad phenotype of cognitive/social difficulties, the concordance rate in MZ pairs was 92%, versus 10% in DZ pairs.

These results suggest that there are genetic factors that may contribute to the expression of autism in some individuals. In addition, they support the hypothesis that these genetic factors also may be responsible for a different, possibly milder, manifestation in the relatives of autistic individuals. If this hypothesis is true, there should be a higher-than-expected frequency of this milder disorder in the relatives of autistic patients compared with the frequency in the general population.

[August et al. \(1981\)](#) attempted to test the hypothesis proposed by [Folstein and Rutter \(1977\)](#) by examining the siblings of autistic children. They used a case-control study design. The experimental group consisted of the siblings of 41 autistic individuals ascertained from the records of the autism program at the University of Iowa child psychiatry service. These 41 probands had a total of 71 siblings. The control group consisted of the 38 siblings of 15 individuals with Down's syndrome. Only Down's syndrome with the standard trisomy 21 was included in the study, to ensure that this type of cognitive deficit would not be transmitted within these families. The families of patients with Down's syndrome were chosen because August and coworkers wanted to compare the rates of cognitive impairment in two types of families in which at least one individual was impaired. The rationale was that if having an impaired child in the family was detrimental for nongenetic reasons, the rates of cognitive impairment should be similar in the two types of families. If, on the other hand, there were some factors specific to autism that could cause other cognitive deficits, the rates of disabilities should be higher in the siblings of autistic children. The subjects with Down's syndrome were selected, so that the ages of the siblings were in the same range as the ages of the siblings of the autistic children.

Only two siblings (2.8%) of autistic children in this study ([August et al., 1981](#)) met [Rutter's \(1971\)](#) criteria for autism. None of the siblings of the Down's syndrome probands was autistic. This rate is similar to the rates of 2% for sibling of autistic individuals reported by [Rutter \(1968\)](#) and the rate of 2.8% for the siblings of twins reported by [Folstein and Rutter \(1977\)](#). Although this is a very small recurrence risk, Rutter has pointed out that it represents at least a 50-fold increase over the general population prevalence.

Additional evidence for the familiarity of autism comes from a report by [Ritvo et al. \(1989b\)](#) of the recurrence risk estimates obtained from families identified through an epidemiologic study of autism in Utah. Two hundred seven families of autistic individuals were ascertained. The recurrence risk for autism in these families was estimated to be 8.6%. There was a suggestion of a sex difference in that if the first autistic child was male, the rate among siblings was 7%, compared with 14.5% when the first autistic child was female, although these differences were not significantly different. The overall risk of 8.6% represents a 215-fold increase over the

population prevalence estimate in the state ([Ritvo et al., 1989a](#)).

Although these findings considerably strengthen the evidence that autism is inherited, data for the familial nature of autism are even more convincing when the information on cognitive impairment is examined. In the study of [August and coworkers \(1981\)](#) discussed previously, a separate diagnosis of cognitive disability was made if at least one of the following criteria was met: (a) delay in spoken language persisting beyond the age of 30 months; (b) gross abnormality in language (either expressive or receptive); (c) verbal, or full-scale IQ score less than 80; (d) specific learning disability in the areas of reading, spelling, or arithmetic as assessed by a standard score of less than 80 on the Wide-Range Achievement Test; and (e) scholastic performance deficits of such severity as to require special educational remediation. One criterion omitted by August and coworkers but included in the Folstein and Rutter twin study was the symptom of grossly abnormal articulation persisting to 5 years of age. Thus, although the two sets of criteria were not exactly the same, there was a substantial overlap, so that a meaningful comparison could be made. Moreover, most of the nonautistic MZ co-twins were socially reticent, and this disability was even more striking when the twins were reexamined in adulthood ([Bailey et al., 1995](#)).

[August and colleagues \(1981\)](#) showed that the rate of cognitive impairment in the siblings of autistic children was considerably higher than the rate among the siblings of patients with Down's syndrome. Of the siblings of autistic patients, 11 (15.5%) showed some cognitive impairment, whereas only one (2.6%) of the siblings of patients with Down's syndrome was cognitively impaired. The difference is statistically significant. These results are consistent with those of [Folstein and Rutter \(1977\)](#) and suggest that autism may be etiologically related to these disorders of cognition, at least within families of autistic individuals. Autism, thus, may be the most severe expression of a disorder of cognition, and there may be genetic factors important for the manifestation of this spectrum of disabilities.

In a more recent study, [Freeman et al. \(1989\)](#) present data that they conclude do not support this hypothesis. Wechsler Intelligence Scales, Wide-Range Achievement Tests, and the Shipley-Hartford Tests were administered to 122 parents and 153 siblings of 62 autistic probands who were part of the Utah study discussed earlier ([Ritvo et al., 1989a, 1989b](#)). All of these studies of cognitive difficulties need to be interpreted with care. In all studies, learning problems were defined as being below some score on some cognitive or achievement test. Learning problems usually are defined as a discrepancy between IQ and achievement, so it is difficult to determine just what the frequency of learning problems was in the families of autistic probands.

The hypothesis of the etiologic relationship between autism and specific cognitive and social developmental disorders (e.g., language impairment and social deficit) has been tested in a number of family studies of broader autism phenotypes ([Bolton et al., 1994](#); [Landa et al., 1992](#); [Pickles et al., 1995](#); [Piven et al., 1991, 1994](#); [Santangelo and Folstein, 1999](#)). Indeed, parent and adult siblings of probands with autism described themselves more often, compared with families of control individuals, as aloof, unctuous, and emotionally detached ([Piven et al., 1994](#)). Moreover, relatives of individuals with autism reported significantly fewer friendships and informal social contacts than did relatives of control subjects ([Santangelo and Folstein, 1999](#)) and were less skilled in carrying out socially oriented narrative discourse ([Landa et al., 1992](#)). In addition, there were higher rates of anxiety disorders and major depression in autism relatives ([Piven et al., 1991](#)). However, relatives of autistic individuals did not show consistent elevation of mental retardation or reading and spelling problems ([Santangelo and Folstein, 1999](#)).

Although most of the autism studies presented here suggest the importance of genetic factors, most of the data do not allow specific genetic hypotheses about mode of inheritance to be tested. Genetic hypotheses were examined in a study by [Ritvo et al. \(1985b\)](#). The authors limited themselves to families in which at least two children met DSM-III criteria for autism. The families were ascertained through several sources, including medical records, referral from other clinicians, and response to an advertisement for "high-density" families. After attempting to correct for the bias introduced by this ascertainment scheme, the researchers used two methods to test several genetic hypotheses. Using a method developed by [Gladstein et al. \(1978\)](#), they tested the multifactorial-polygenic hypothesis and were able to reject it. They also tested the autosomal dominant and autosomal recessive hypotheses with classic segregation analysis. They were able to reject the dominant hypothesis but not the recessive hypothesis.

These results need to be interpreted with caution. First, as Ritvo and colleagues point out, their findings do not generalize to all patients with autism. These multiple-incidence families are a highly selected group and are not representative of all families. Second, the particular ascertainment scheme used makes it difficult to estimate accurately the ascertainment probability. As the authors point out, the estimates of the segregation frequency are fairly robust if the estimate of the ascertainment probability is reasonably close. However, it is not clear how misspecification of the ascertainment probability affected the estimate of the segregation ratio in these families. Thus, these results should be viewed as preliminary. Additional work is needed with a sample selected through conventional ascertainment procedures.

An important point to keep in mind when studying the genetics of autism is that it is likely to be a heterogeneous disorder. It is known that the syndrome has multiple etiologies ([Rutter, 1974](#)). It can develop in association with (and presumably as a result of) conditions as pathologically diverse as congenital rubella ([Chess et al., 1971](#)) and infantile spasms ([Taft and Cohen, 1971](#)). In addition, [Folstein and Rutter \(1977\)](#) suggested other possible nongenetic etiologic factors. They found that the autistic twin in a discordant twin pair was more likely to have experienced events that could have resulted in brain damage than were the autistic twins in a concordant twin pair. In all of the concordant MZ twin pairs, neither twin had experienced any trauma that could be interpreted as contributing to the development of their autistic behavior. These findings suggest etiologic heterogeneity.

Certainly, autism can (and does) develop after an environmental insult. However, it also can develop in the absence of any identifiable environmental agents. Any genetic factors that might exist for autism almost certainly will be more easily identifiable in the group of patients who do not experience any major environmental trauma. Thus, in future studies, a sufficient number of patients should be included, so that the data can be divided into groups based on presumed environmental insult to the proband. If the recurrence risks differ between the groups, this would provide additional evidence for specific etiologic subtypes and suggest which types were most likely to be genetic.

However, even the subset of patients with no known environmental risk factors could be heterogeneous. Nearly all inherited disorders in which the genetic mechanism is clearly understood have been shown to be genetically heterogeneous. The mucopolysaccharide storage diseases provide an excellent example. Initially, all affected children were considered to have "gargoylism." The clinical characteristics were quite uniform, and it was thought to represent a single disorder that showed a familial tendency. It has been shown subsequently that the mucopolysaccharidoses encompass defects of many different enzymes, each with a different underlying genetic mechanism.

Often, the multiple etiologies of a "single disease" become easily identifiable as new phenotypic levels are defined: What is apparently homogeneous at the level of gross symptomatology becomes obviously heterogeneous as physiologic or biochemical aberrations are used to redefine the phenotype. In future studies of the genetics of autism, care needs to be taken to obtain a homogeneous sample of autistic patients. A level of homogeneity can be achieved by using very strict diagnostic rules or by studying only those patients with a specific biochemical abnormality or genetic marker. For example, it has been shown that some autistic people also have the marker X syndrome ([August, 1983](#); [Brown et al., 1982](#); [Gillberg, 1983](#); [Meryash et al., 1982](#)). Certainly not all autistic patients have the mar(X) chromosome ([Watson et al., 1984](#)), but those who do may represent a subtype of autism with unique etiologic factors. By studying the families of a group of patients positive for the mar(X) chromosome, it might be possible to identify unique factors associated with this subset of autistic patients. Explorations of the link between the X chromosome and autism continues. (For review, see [Folstein and Piven, 1991](#).)

In addition to the X chromosome, current research on the genetic etiology of autism is focusing on regions of chromosome 15 ([Cook et al., 1998](#); [Maestrini et al., 1999](#); [Smith et al., 2000](#); [Wolpert et al., 2000](#)), chromosome 7 ([Ashley-Koch et al., 1999](#); [Ingram et al., 2000](#)), and several other genomic locations ([Auranen et al., 2000](#); [Philippe et al., 1999](#); [Risch et al., 1999](#)). Similar to the situation with ADHD research, the results are controversial, characterized by both replications and failures to replicate.

Although the results from these types of studies will not generalize to all autistic individuals, this should not be a deterrent to special ascertainment strategies. If it is possible to identify a specific etiology for a homogeneous group selected with a unique ascertainment, the residual cases will then be more homogeneous than the original population. Thus, the chances of determining the important etiologic factors for them are enhanced.

### Specific Reading Disability

Specific reading disability (RD), or dyslexia, is characterized by the presence of a discrepancy between predicted reading ability and actual reading achievement. Poor reading skills are present despite conventional instruction and adequate intelligence and sociocultural opportunity. The prevalence of RD among school-age children has been estimated to be between 6% and 9% ([Shaywitz et al., 1990](#)). These investigators found an equal sex ratio for RD in their sample. This is in contrast to many other identified samples, in which the rates of RD are two to four times higher in boys than in girls. These differences could be due to differing ascertainment



strategies or possibly different diagnostic criteria for RD.

Over the years, the definition and diagnosis of RD have not been uniform. It is not always clear that the results from various studies can be easily compared. Attempts have been made to identify more homogenous subtypes. [Johnson and Myklebust \(1967\)](#) and [Boder \(1971\)](#) observed that the spelling errors made by dyslexic persons could be categorized into several types: (a) auditory predominant, (b) visual predominant, and (c) a mixed subtype. [Mattis et al. \(1975\)](#) also suggested three distinct subtypes of RD. They used neuropsychological tests to define each of the three categories: (a) language disorder, (b) articulatory and graphomotor dyscoordination, and (c) visual-spatial perceptual disorder. The Colorado Family Reading Study (CFRS) ([Decker and DeFries, 1980](#); [DeFries and Decker, 1981](#); [DeFries et al., 1978](#); [Foch et al., 1977](#); [Lewitter et al., 1980](#)) identified four dyslexia subtypes in probands: (a) a spatial/reasoning deficit; (b) a coding/speed deficit; (c) a relatively specific reading disability subtype, with a deficit only in reading; and (d) a mixed or global subtype. These subtypes accounted for 91% of the CFRS proband sample. It is difficult to say whether the relatively specific reading-disabled subtype found by these investigators has been identified by others. Unfortunately, most classifications used slightly different diagnostic schemes for patient identification, and few attempts have been made at cross-validation.

In the 1990s, componential models of dyslexia were developed. These models are based on a number of general and specific linguistic cognitive processes (often referred to as reading- or dyslexia-spectrum processes). Despite considerable differences in clinical definitions of dyslexia, there is significant agreement among researchers regarding its cognitive spectrum. (For review, see [Sternberg and Spear-Swerling, 1998](#).) There is general consensus that single-word reading (ability to pronounce printed real words) is the best single criterion for the type of deficit typically referred to as specific reading disability. There also is general agreement that the spectrum of disability includes at least two major procedural deficits: one in phonologic skills and one in automatized lexical retrieval. Finally, the acquisition of reading is related to the acquisition of vocabulary; therefore, some investigators include vocabulary measures in their analyses of cognitive profiles of individuals with reading disability.

The phonologic processing deficiency usually is described as a two-component process: (a) an inability to segment words into phonemes (the so-called phonologic awareness deficit) and (b) an inability to decode written symbols (the so-called phonologic decoding deficit). Even though these two processes are substantially correlated, their developmental and etiological relationship are not fully understood. The automatized retrieval deficiency is operationalized as a rapid automatized naming deficit, wherein an individual is required to name from a printed page colors, objects, digits, or letters as quickly as possible. The relationship between deficits in rapid naming and in phonologic processing, and the contributions of each deficiency to dyslexia, are not straightforward. Current opinions span a continuum, from the concept that these processes are distinct entities to the concept that they are different manifestations of the same underlying latent process. Finally, a number of investigators have used measures of vocabulary in their investigation of normal and disabled reading. The nature of the relationship between vocabulary and single-word reading is complex and, most likely, reciprocal, with reading ability/disability both being influenced by and influencing vocabulary development.

Specific reading disability is familial. As early as 1905, studies were reported in which children with dyslexia often had affected relatives ([Fisher, 1905](#); [Hinshelwood, 1907, 1911](#); [Stephenson, 1907](#); [Thomas, 1905](#)). [Later, Orton \(1937\)](#) and [Eustis \(1947\)](#) also reported the familial nature of RD and, in addition, noted that affected individuals usually had normal intelligence. In 1950, Hallgren published the results of one of the largest family studies of RD, with 116 probands and 391 first-degree relatives. Hallgren reported a male excess among affected probands and affected first-degree relatives. Overall, 160 of the 391 (41%) first-degree relatives had RD. Hallgren calculated segregation ratios based on several mendelian models and proposed that RD was an autosomal dominant trait with sex-modified expression. Other studies confirm the familial nature of RD and that boys have higher prevalence rates than girls, but no specific genetic model were tested ([DeFries et al., 1978](#); [Finucci et al., 1976](#); [McGlannon, 1968](#); [Owen et al., 1971](#); [Walker and Cole, 1965](#)).

Further attempts to examine specific genetic hypotheses have resulted in inconsistent findings. [Symmes and Rapaport \(1972\)](#) proposed sex-linked recessive inheritance after studying the families of 54 children with RD. [Zahalkova and coworkers \(1972\)](#) studied the families of 65 dyslexic children, calculated a segregation ratio of  $0.42 \pm 0.06$ , and concluded that inheritance was autosomal dominant, with partial sex limitation. [Lewitter et al. \(1980\)](#) performed segregation analyses on families collected through 133 children with RD to test the hypotheses proposed by previous investigators. They also analyzed three subsets of the total data: families of male probands, families of female probands, and families of severely affected probands. No one hypothesis was supported in all the analyses. The researchers concluded that the results suggested that RD was genetically heterogeneous; however, when the analysis was restricted to children only, they were unable to reject the hypothesis of a single major locus. In 1991, Pennington et al. examined four independently ascertained samples (204 RD families) and found evidence for the hypothesis of major locus transmission in three samples and polygenic transmission in one sample.

Although RD clusters in families and appears to be transmitted, the data do not prove that the transmission is genetic. Additional evidence for a genetic contribution to the manifestation of RD has come from at least two other sources. The first is twin studies. In a 1967 literature review, Zerbini-Rudin found that of 17 MZ twin pairs, 100% were concordant for RD, compared with 35% (12/34) of DZ twin pairs. [Bakwin \(1973\)](#) examined 338 pairs of same-sex twins and found 14% (97/676) affected with RD. In the MZ twin pairs in which at least one twin had RD, 84% of the co-twins were also affected. In contrast, only 29% of the DZ twin pairs were concordant.

More recent twin studies continue to support a genetic hypothesis for RD. [DeFries et al. \(1987\)](#) estimated heritability for RD to be 30%. [Stevenson et al. \(1987\)](#) also found a low heritability for reading accuracy, but a substantially higher estimate (73%) for spelling. [Ho et al. \(1988\)](#), using a small sample of twins with the Bannatyne Cognitive Profile, reported that the MZ concordance for the profile was 67%, compared with 30% for DZ twins. However, these results may be compromised by the fact that reading problems are significantly more common in twins than in singletons ([Hay et al., 1984](#)). This finding suggests that there may be a major environmental component important for the expression of RD in twins and therefore, the estimates of heritabilities from twin data could be grossly inaccurate.

A second source of evidence for a genetic contribution to RD comes from linkage studies. As discussed earlier, the demonstration of linkage of a purported locus for a trait with a known genetic marker locus is strong evidence that a genetic component is important in the etiology of the trait.

Using current molecular techniques of linkage analysis, investigators have carefully studied selected pedigrees of dyslexic individuals in which developmental dyslexia recurs in different generations. The results of one early study suggested that a major gene for dyslexia was located on the short arm of chromosome 15 ([Pennington et al., 1991](#); [Smith et al., 1983, 1990](#)). [Fulker and colleagues \(1991\)](#) followed up these findings by selecting from the original extended-family study a sample of siblings who represented lower (i.e., more extreme) levels of reading ability. They applied multiple regression techniques, and their results also pointed to chromosome 15. However, subsequent molecular linkage studies, which included the same dyslexia pedigrees, refuted the original findings ([Cardon et al., 1994](#); [Lubs et al., 1991](#); [Rabin et al., 1993](#)). Furthermore, independent investigators who examined Danish families with an autosomal dominant pattern of transmission for dyslexia also were unable to replicate the chromosome 15 finding ([Bisgaard et al., 1987](#)). Later, however, [Grigorenko et al. \(1997\)](#) also found linkage evidence for deficits in word recognition on the long arm of chromosome 15q, and [Schulte-Körne et al. \(1997\)](#) and [Morris et al. \(2000\)](#) reported an association between a marker and reading disability in the same area.

Subsequent linkage studies were based on a possible association between dyslexia and immune system function. Of interest is that the region identified on chromosome 15 by [Smith et al. \(1983\)](#) as linked to dyslexia is near the b<sub>2</sub>-microglobulin gene, which has been implicated in the human autoimmune system ([Lazarus and Owen, 1994](#)). Although the causal basis of the association between autoimmune disturbances and dyslexia has not yet been demonstrated, the evidence of association has suggested a number of chromosomal regions as candidates.

In 1987, Pennington et al. advanced human leukocyte antigen (HLA) as a candidate region for dyslexia susceptibility loci. [Later, Cardon et al. \(1994\)](#) reported findings from an affected sibling-pair study that provided evidence for linkage between reading disability and DNA markers in the HLA region on chromosome 6. These investigators used a quantitative multitrait phenotype of reading by combining several reading and phonologic test scores and weighting them according to a system derived from previous research. A number of research groups have replicated these findings successfully ([Fisher et al., 1999](#); [Gayán et al., 1999](#); [Grigorenko et al., 1997, 2000](#)), although one has not ([Field and Kaplan 1998](#); [Petryshen et al., 2000](#)). Differences in phenotype definitions may account for these variations in replicability ([Grigorenko et al., 2000](#)).

Another region suggested by the association with immunologic functioning is the area around the Rh locus on chromosome 1. [Rabin et al. \(1993\)](#) reported suggestive evidence for the linkage of dyslexia to Rh and other markers on the short arm of chromosome 1. Linkage analyses with the Rh protein marker and two other DNA markers in the region (1p34-p36) yielded a lod score of 1.95 at  $q \leq 0.2$  for all families. Coincidentally, a German family was identified in which dyslexia and delayed speech development cosegregated with a balanced translocation involving chromosomes 1 and 2—t(1;2)(p22;q31) ([Froster et al., 1993](#)). Both [Smith et al. \(1991\)](#) and [Grigorenko et al. \(1998\)](#) have provided supporting evidence for a dyslexia locus on chromosome 1.

[Lubs and colleagues \(1991\)](#) identified a family with a translocation and fusion of chromosomes 13 and 14. Six of the seven family members with the translocation also have dyslexia; however, there is one dyslexic family member who does not have the translocation. Thus, this family provides a clue suggesting that there might be another gene associated with dyslexia on chromosome 13 or 14. These researchers also conducted a random genome testing that included selected markers on



chromosomes 1 to 4, 6, 8, 9, 11, 13, 14 to 16, and 18 to 21 ([Lubs et al., 1991](#)). No significant results were obtained.

Using a large extended Norwegian family in which developmental dyslexia was inherited as an autosomal dominant trait, [Fagerheim et al. \(1999\)](#) conducted a genome-wide search for linkage. This search resulted in the identification of a region on the short arm of chromosome 2 as a dyslexia susceptibility region. These results, however, have not been confirmed by any other group.

To summarize the linkage studies, multiple regions of interest have been identified throughout the human genome. None of these findings can be considered definite, and more research in those same regions is needed. However, a number of observations can be made on the basis of these results. First, various groups of researchers have identified overlapping but differential linkages to different reading-related processes. These semidifferential linkages of different phenotypes to different chromosomal regions might be either indicative of the genetic heterogeneity of developmental dyslexia or, if there are many genes involved in the manifestation of each separate process, suggestive of partial, but not complete overlap of these process-specific sets of genes. Second, because that clustering of different reading-related deficiencies often is observed in a single family, similar remedial efforts within a given family might have differential success. These individual differences (even within a single family) in reception of and response to remedial help are questions for special investigation. Third, it is possible that the observed variation in linkage results is related to the degree of severity of developmental dyslexia, even within one family. Fourth, it is possible that the observed variation within a family is due to the developmental differentiation of what dyslexia is—what is viewed as indicative of dyslexia in an adult in the family might be quite different from what is viewed as indicative of dyslexia in a child in the same family. Thus, the search for genes might be carried out not under the assumption that there is a single phenotype that breeds through multiple generations (e.g., the phenotype of single-word reading), but under the assumption that there are developmentally appropriate forms of the transmitted phenotype (e.g., rhyming in early childhood, phonemic segmentation during early stages of reading mastery, phonologic decoding during adolescence).

Specific reading disability is a complex disorder that is clinically heterogeneous and probably heterogeneous in etiology and pathogenesis. As noted earlier, several attempts have been made to identify different subtypes. Two studies ([Omenn and Weber, 1978](#); [Pennington and Smith, 1983](#)) have used the classification schemes of [Boder \(1971\)](#) to determine if the defined subtypes would “breed true” within families.

[Omenn and Weber \(1978\)](#) studied 21 families with multiple members with RD. Following criteria proposed by [Boder \(1971\)](#), individuals were classified as auditory-predominant subtypes if their history and test results showed dysphonetic spelling errors, striking mispronunciation, and greater difficulty in learning from oral presentation; individuals were classified as visual-predominant subtypes if their history and test results showed phonetic spelling errors, no mispronunciation, and greater difficulty in learning from written or visually presented material. Individuals were classified as mixed if they had characteristics of both previous subtypes. Eleven probands were classified visual predominant, seven as auditory predominant, and three as mixed. Most of the affected relatives who could be diagnosed had the same subtype as the proband, giving support for the hypothesis that the phenotypic subtypes reflect independent familial predispositions.

Of the four groups identified by [Decker and DeFries \(1980\)](#), the relatively specific reading-disabled subtype showed the most significant concordance between probands and other family members, specifically siblings. Thus, this subtype gave strong evidence for genetic factors contributing to a specific form of RD. However, as indicated previously, it is difficult to know whether this group identifies the same group as the Boder scheme.

[Pennington and Smith \(1983\)](#) indicated that there appears to be some evidence for specific subtypes to cluster in the same families in the study reported by [Smith and colleagues \(1983\)](#). The families giving evidence for linkage tended to have dyslexia due to subtle language processing deficits but appeared to have good visual-spatial skills. However, individuals in the family that did not give evidence for linkage appeared to have a visual-spatial dyslexia.

Although all of the aforementioned reports suggest some specificity, there have been no reports of specific tests of genetic hypotheses for these subtypes. The analyses done by [Lewitter et al. \(1980\)](#) divided the sample into age- and sex-defined groups but not into groups of clinically defined subtypes.

As noted, many studies show a male preponderance for RD. Using these data, several authors have suggested that RD may be a trait with a sex-modified threshold important for its expression ([Childs and Finucci, 1979](#); [Herschel, 1978](#)). If the genetic mechanism for the trait also is related to its differential expression in the sexes, then the risk to relatives should differ depending on the sex of the proband. That is, because girls are affected less frequently, the risk to the relatives of female probands should be greater than the risk to relatives of male probands. This is not the case for RD. [Childs and Finucci \(1979\)](#) state that the risk to the relatives of female probands is not significantly greater than the risk to the relatives of male probands. [Omenn and Weber \(1978\)](#) show that the risk is greater for relatives of female probands, but not significantly so. When the data presented by [Finucci et al. \(1976\)](#) are separated by sex of proband, the risks are not different for the two classes of relatives ([Pauls and Kidd, 1981](#)). These results could mean that (a) in each of these studies, the sample size is too small to obtain significance or (b) the sex difference observed is not related to the transmission of the trait. These ideas are supported by the work of [Shaywitz and colleagues \(1990\)](#) that distinguishes between school-identified RD sex ratios and research-identified sex ratios.

If the sex difference is not related to the transmission of RD, then it could be explained in several ways. First, the social or educational environment could be sufficiently different—favorable to girls, so that they would express the trait less often, even when they were genetically predisposed. If that were the case, one would expect a slightly different concordance rate for male and female MZ twins. If, in fact, boys were more apt to experience or be identified as having RD because of a detrimental, and biased, social or educational environment, then their concordance rate should be somewhat lower than that for girls. [Bakwin \(1973\)](#) reported concordance rates for male and female MZ twins to be 84% and 83%, respectively.

Another explanation for the observed difference could be that the male and female developmental processes interact differently with the genetic predisposition for RD and that the interaction produces more severe effects in boys than it does in girls. If that is the case, one also would expect boys to be more severely affected than girls.

In summary, it is clear that much work remains to be done to gain a better understanding of RD and its etiology. It is known that RD is familial and that part of the reason for the familial clustering is an underlying genetic mechanism. At present, it is not known what that genetic mechanism is, but a number of interesting candidate genes are being closely investigated. A variety of genetic models have been proposed; the most frequently mentioned one is a quantitative trait locus model. However, few if any of the alternative hypotheses have been rigorously excluded. Diagnostic uncertainty and probable etiologic and phenotypic heterogeneity cloud the picture. Work is needed to establish useful and unified criteria for all age ranges and to determine if there are phenotypic differences that tend to cluster in families. Yet, in spite of these complications, the field of RD is arguably the one where both theoretical models of the disability and its underlying genetic mechanisms are comparatively better understood than those in other areas of childhood neuropsychiatry.

### Gilles de la Tourette's Syndrome

Gilles de la Tourette's syndrome (TS) is a familial neuropsychiatric disorder with onset in childhood, characterized by chronic intermittent motor and vocal tics. The familial nature of TS was first described by Gilles de la Tourette in 1885. However, it was not until the 1970s ([Eldridge et al., 1977](#); [Shapiro et al., 1978](#)) that studies demonstrated an increased frequency of positive family history for tics in families of patients with TS. The family history study of [Kidd et al. \(1980\)](#) was the first to present frequencies of TS and chronic tics (CT) among relatives. That early study combined TS and CT into a single category and showed that the risk to relatives was significantly elevated over what would be expected by chance. In 1981, Pauls and colleagues analyzed the data collected by Kidd and coworkers, as well as family history data collected from TS clinic patients, to demonstrate that (a) the increased risk for TS and CT was consistent across the two samples and higher than expected by chance, (b) CT appeared to represent a variant expression of the syndrome, and (c) the patterns of occurrence of TS and CT were consistent with vertical transmission in families.

Subsequent to these studies, six groups ([Baron et al., 1981](#); [Comings et al., 1984](#); [Curtis et al., 1992](#); [Devor, 1984](#); [Kidd and Pauls, 1982](#); [Price et al., 1987](#)) reported results of genetic analyses of family history data in which specific transmission hypotheses were examined. All groups concluded that the pattern of transmission in families was consistent with a genetic hypothesis that postulated a single major locus to be responsible for susceptibility to TS or CT. However, this was not the only hypothesis supported. [Kidd and Pauls \(1982\)](#) were unable to reject the hypothesis that transmission of the syndrome was consistent with the contribution of many genes, each with equal and additive effect to the expression of the disorder (the so-called multifactorial-polygenic model). Likewise, [Comings et al. \(1984\)](#) could not reject the multifactorial-polygenic model unless extended relatives were included and the population prevalence for TS and CT was restricted to be less than 0.0075. However, the consistent conclusion of authors in all of the studies was that the mode of inheritance that gave the best fit to the data was one that postulated an underlying single major locus with two alleles. Although all studies supported a single-locus model, not all studies suggested an identical mode of inheritance. Three of the studies ([Baron et al., 1981](#); [Comings et al., 1984](#); [Price et al., 1987](#)) concluded that the most likely mode of inheritance for TS or CT was autosomal dominant with sex-specific penetrances. [Curtis et al. \(1992\)](#) did not detect the excess of male cases of TS that is commonly described. Results from the remaining two studies



suggested an additive model.

As indicated, all of these studies relied on family history data for the analyses. That is, the diagnoses of relatives were based on information given by one or at most two informants per family. It has been demonstrated in studies of other neuropsychiatric disorders that family history data underestimate the "true" rates of illness as determined by direct assessment ([Andreason et al., 1977](#); [Orvaschel et al., 1982](#)). This was demonstrated to be true for TS and CT as well ([Pauls et al., 1984](#)). Thus, the results of the genetic analyses summarized previously should be interpreted with caution. This is particularly true if the underestimation of rates does not occur uniformly across all classes of relatives. For example, if the rate of illness is consistently underestimated in the parents of the patients, then the pattern of illness will more closely resemble the pattern for a recessive or partially dominant condition. If there is a consistent underestimate of the illness in the relatives of a particular type of proband (e.g., relatives of male probands), the resulting patterns could lead to spurious conclusions.

[Pauls and coworkers \(1984\)](#) demonstrated that there appeared to be consistent underestimation of rates in the parents of TS probands in the family history studies of [Kidd et al. \(1980\)](#) and [Pauls et al. \(1981\)](#). Thus, the penetrances reported by [Kidd and Pauls \(1982\)](#) would be expected to be underestimates. This also could be true for the results reported by [Devor \(1984\)](#) because he analyzed only pedigrees reported in the literature.

Biases resulting from consistent underreporting of illness in relatives of a specific type of proband could also occur when it is generally believed by the informant that the illness is more common in one type of relative than another. This appears to have happened in the data collected by [Kidd et al. \(1980\)](#) and [Pauls et al. \(1981\)](#). These investigators reported an apparent increased risk to relatives of female probands in the family history they collected. However, data collected in the Yale Family Study of TS ([Pauls and Leckman, 1988](#)) suggest that the sex of the proband has no effect on the rates among relatives. Therefore, the analyses of Kidd and Pauls that incorporated a sex difference need to be reevaluated.

The results presented by [Baron et al. \(1981\)](#), although appearing to be consistent with a dominant mode of inheritance, predicted that approximately 70% of male and 50% of female patients would be phenocopies (individuals with the disorder but without the gene.) This high phenocopy rate could have resulted from a combination of underreporting for parents or underreporting for female relatives. Because fewer parents and female relatives would have been identified as affected, there would have been more families with isolated cases and, thus, a higher estimate of phenocopies.

Therefore, because of a variety of reporting biases, each of the aforementioned studies has some potential difficulties. Because these reporting biases can affect patterns within pedigrees, it is not surprising that the specific estimates of gene frequency and penetrances vary considerably from study to study. Accurate estimates of genetic model parameters are critical for genetic counseling and for genetic linkage analyses.

A twin study completed in the Child Study Center and Department of Human Genetics at Yale University also demonstrated the effect of relying on family history data for diagnostic purposes. [Price et al. \(1985\)](#) reported the concordance rate for TS to be 0.53 for MZ twin pairs and 0.08 for DZ twin pairs. The data for this study were obtained by questionnaires sent to the mothers of twins and from follow-up telephone interviews with those mothers.

The results from the twin study are comparable with those reported in some of the studies discussed earlier. Concordance rates for MZ twins can be interpreted as an estimate of penetrance if the underlying genetic mechanism is a single locus. Thus, the concordance rate of 0.77, when co-twins with either TS or CT are considered to be affected, can be thought of as a penetrance of 0.77. The estimate compares favorably with the estimates of penetrance reported by [Baron et al. \(1981\)](#), [Comings et al. \(1984\)](#), and [Price et al. \(1987\)](#). However, just as a number of possible reporting biases could affect the results of genetic analyses based on family history data, reporting biases also could affect the results in a twin study.

As a follow-up to the study by [Price et al. \(1987\)](#), investigators at the Yale Child Study Center contacted all twin pairs and conducted personal interviews with each twin. The resulting estimates of concordance indicate that even for twins, the rates of illness can be underestimated if the investigator relies on historical data provided by an informant rather than on data collected by direct personal interview of the subject. The concordance rates estimated from personal interview data increased to 1.0 for MZ twins when co-twins with either TS or CT were included as affected ([Walkup et al., 1987](#)).

Another major focus of the TS family studies has been the delineation of the behavioral phenotype associated with the underlying genetic diathesis. Although a number of psychiatric and behavioral disorders have been hypothesized ([Comings and Comings, 1987](#)), the strongest data concern obsessive-compulsive disorder (OCD). Evidence in support of this association comes from uncontrolled clinical studies ([Cummings and Frankel, 1985](#); [Fernando, 1967](#); [Jagger et al., 1982](#); [Kelman, 1965](#); [Montgomery et al., 1982](#); [Morphew and Sim, 1969](#); [Nee et al., 1980, 1982](#); [Steffl, 1984](#); [Yaryura-Tobias et al., 1981](#)). In addition, family study data indicate that OCD alone (no current or past history of tics) could represent a variant expression of the disorder ([Pauls et al., 1986](#)). Results from several more recent studies have supported these earlier findings ([Curtis et al., 1992](#); [Frankel et al., 1986](#); [Green and Pitman, 1986](#); [Robertson et al., 1988](#); [Walkup et al., 1987](#)), and it is generally accepted that in families of individuals with TS, the two disorders are etiologically related. In addition, [Pauls and Leckman \(1986\)](#) reported that in relatives of TS probands, girls were at greater risk for development of OCD without TS or CT than were boys. These data suggest that rather than a sex-specific frequency of the illness (limited to only TS or CT), there could be a sex-specific expression of the illness, with OCD representing the part of the TS spectrum more frequently expressed in girls.

To determine whether the occurrence of OCD in these families was consistent with genetic transmission of TS, CT, or OCD together, [Pauls and Leckman \(1986\)](#) performed segregation analyses to test specific genetic hypotheses. They concluded that the syndrome is inherited as an autosomal dominant condition. What is noteworthy is that regardless of who was included as affected, whether (a) only relatives with TS, (b) relatives with TS or CT, or (c) relatives with TS, CT, or OCD, the results all were consistent with an autosomal dominant hypothesis. The fact that the inclusion of OCD resulted in a significantly better fit to the observed data within families suggests that OCD is part of a genetically mediated TS spectrum. Penetrances estimated when relatives with OCD are included suggest that there may not be a large difference in the probability of expression of some form of the illness but that the expression may be more specific to a particular sex.

These findings have been replicated in two independent samples ([Eapen et al., 1993](#); [van de Wetering, 1993](#)). Eapen and coworkers studied the families of 40 consecutive TS probands seen in a London clinic and van de Wetering and colleagues studied the families of approximately 45 patients with TS seen in Rotterdam. Both studies found strong statistical evidence that the pattern of transmission of TS, CT with OCD, and OCD without tics was consistent with autosomal dominant inheritance. The penetrances obtained for these two samples were remarkably similar to those reported earlier in this section, ranging from 0.5 to 0.9 for male patients and 0.2 to 0.8 for female patients, depending on the diagnostic scheme used in the analyses. These consistent findings provide a strong rationale for the ongoing linkage studies.

An additional segregation analysis painted a more complex picture of TS inheritance. [Hasstedt et al. \(1999\)](#) reanalyzed 182 family members in a single large TS pedigree for which prior analyses had rejected mendelian inheritance. Those investigators did not use typical diagnostic criteria; rather, they assigned affected status based on a point system that incorporated the TS symptom types, the number of tics reported or observed, and any obsessive-compulsive symptoms. Also, the analyses incorporated an assortative mating correlation in the genetic model because the rate of TS in spouses of the pedigree descendants was greater than that seen in the general population. The new results showed an "intermediate" mode of inheritance, with a penetrance of 0.28 in heterozygotes and 0.98 in homozygotes.

The reanalysis by Hasstedt et al. is especially interesting in light of a significant finding of "bilineal transmission" of TS by [Kurlan et al. \(1994\)](#). The frequency of transmission of TS from both maternal and paternal sides of probands' families was determined by examining 39 high-density families (defined as five or more affected relatives within three generations of the proband) and the families of 39 consecutively evaluated TS clinic probands. Relatives within three generations of the proband were assessed; the presence of tics in relatives was confirmed by direct clinical examination of at least one member on both the maternal and paternal sides. Their results indicated that 33% of the high-density families and 15% of the consecutive-evaluation families showed bilineal transmission of TS. When obsessive-compulsive behaviors were included in the affected status, the proportions were 41% bilineal transmission in high-density families and 26% bilineal transmission in the consecutive-evaluation families. No consanguinity was evident in any of the families. In addition, this study demonstrated that the frequency of bilineal transmission is related to the severity of TS in the proband. An increased severity of symptoms among the affected offspring of two TS-affected parents also was reported by [McMahon et al. \(1996\)](#). Further support for significant bilineal transmission of TS also was provided by [Hanna et al. \(1999\)](#). In a sample of 51 families ascertained through a TS-affected proband, 57% exhibited unilineal transmission and almost 26% showed bilineal transmission.

Taken as a whole, these findings indicate a dosage-effect model of TS, wherein a TS proband who is homozygous for a TS susceptibility allele is likely to be more severely affected than if the same person were heterozygous. Because linkage studies are critically dependent on proper specification of the genetic model, it may be fruitful in future analyses of TS linkage data to include model parameters that reflect an additive gene-dosage effect.

Not all analyses, however, have indicated that the transmission of TS can best be modeled by Mendelian inheritance. In a report of the results of complex segregation



analysis of TS using regression-based methods, [Seuchter et al. \(2000\)](#) found no evidence to support Mendelian inheritance. Their analyses of 108 families, ascertained through single TS-affected probands, incorporated age-of-onset effects, as well as TS-spectrum disorders and major gene effects.

Most reports of TS genetic analyses, however, do support the concept of TS-spectrum behaviors as an inherited disorder and so genetic linkage and association studies are warranted to complete our understanding of the inheritance of this syndrome. Several early linkage studies of TS ( [Barr et al., 1999](#); [Heutink et al., 1990](#); [Pauls et al., 1990](#)) reported no significant evidence for linkage. [Pakstis and colleagues \(1991\)](#) pointed out that an important assumption of those analyses was that the major locus for TS was the same in different pedigrees under study; genetic heterogeneity could obscure a positive finding.

Some association studies in the early half of the 1990s gave evidence for an association between the dopamine D2 and D3 receptor loci and TS ( [Comings et al., 1991, 1993](#)), as did a small twin study published by Wolf et al. (1996), but these results have been refuted in studies using well defined, large samples of patients with TS. [Brett et al. \(1993\)](#) and [Nothen et al. \(1994\)](#) used robust analytic methods (haplotype relative risk) to control for undetected ethnic differences and any resultant skewing of allele frequencies observed in the sample. In fact, most of the genes in the dopamine system have been excluded, including the dopamine receptors D1, D2, D3, D4, and D5, dopamine b-hydroxylase, tyrosinase, and tyrosine hydroxylase ( [Brett et al., 1995](#)).

Additional studies giving evidence of exclusion of linkage of specific genes to TS include [Gelernter et al. \(1993\)](#), in which the dopamine receptor D1 was excluded by genotyping 124 individuals in a single pedigree segregating TS, and [Brett et al. \(1995\)](#), in which the serotonin receptor subtype 5HT-1A and the tryptophan oxygenase genes were excluded by linkage analysis of genotypes from 116 individuals in a single pedigree segregating TS.

Some positive, but not statistically definitive, evidence for TS linkage was reported by [Barr et al. \(1999\)](#). A panel of 386 markers were used to scan the genome at an average resolution of 10 centimorgans. No significant findings were observed using parametric, model-based linkage analyses that specified autosomal dominant inheritance with reduced penetrance. However, using the nonparametric, model-free affected pedigree member analytic method (APM), the authors reported eight markers that were statistically significant. That disparity in results, even though the same families were used, is not surprising because the model-based method has high false-negative rates when the model is misspecified, whereas the model-free method has comparatively high false-positive rates.

An important step in mapping genes for TS was the genome-wide scan using the affected sib-pair (ASP) method, performed by an international consortium of TS investigators ([Tourette Syndrome Association International Consortium for Genetics, 1999](#)). This study reported several chromosomal "hot spots" that may be important in the etiology of TS. Seventy-six families with two or more TS-affected siblings were genotyped on a panel of approximately 400 markers that spanned the entire human genome. None of the "hot spots" reached the level of clear-cut statistical significance, but several regions reached significance levels that justified intensive follow-up with more powerful analytic techniques and additional family samples. Although the ASP approach has been available for some time, only recently has it become evident that its application is increasingly important in the genetic study of disorders where there may be genetic heterogeneity and where the mode of inheritance is complex.

The singular advantage of the ASP method is that no prior assumptions regarding specific genetic mechanisms of a disease are required: It is essentially a model-free procedure but does not have the false-positive rate of the model-free APM method. The analytic approach compares the sharing of alleles at a given locus shared by two affected siblings. If that sharing is significantly higher than expected by chance, it suggests that a gene of etiologic importance for the trait in question is close to the marker being examined. There is a loss of statistical power when performing ASP analyses compared with family pedigree analyses; however, the ASP approach can provide important preliminary evidence for linkage, forming the basis for further investigations using more powerful techniques, as was seen in the TS ASP study.

Close on the heels of the sib-pair genome scan, a positive linkage finding for TS was reported ( [Mérlette et al., 2000](#)) in one large French Canadian family. In that kindred, 40 of the 127 family members examined were affected with TS-spectrum disorders. In a multipoint parametric linkage analysis based on the specification of specific genetic models, a LOD score of 3.24 was reported on chromosome 11 (11q23). Importantly, those investigators corrected for a false-positive signal due to multiple testing (multiple phenotypes and inheritance models).

Turning from linkage studies to cytogenetic evidence, [Comings and colleagues \(1986\)](#) reported a 46,t(7:18)(q22;q22.1) balanced reciprocal translocation in six relatives with TS. This report suggested that a gene for TS could be near the 18q22.1 breakpoint. [Subsequently, Donnai \(1987\)](#) reported a female patient with a mildly hypoplastic midface, ticlike movements, mild OCD, panic attacks, and visual hallucinations who carried a deletion of the long arm of chromosome 18 at 18q22.1. Given the relationship between OCD and tics, this report provided additional support for the hypothesis that a gene for TS might be located in this region. These two reports led to the still-tentative assignment of the TS gene to chromosome 18q22.1 by the chromosome 18 committee at the Human Gene Mapping Conference IX. A paper by [Boghossian-Sell et al. \(1996\)](#) reports the mapping of the breakpoint on chromosome 18; two markers (D18S61 and D18S488) are the closest proximal and distal (respectively) markers to the breakpoint. Two yeast artificial chromosome clones that span the translocation were isolated and should prove useful for the discovery and characterization of genes at the breakpoint and their potential involvement in TS.

Another cytogenetic abnormality [a *de novo* deletion, del(9)(qter@p2304:)] was reported in a Latin American male patient with TS by [Taylor and colleagues \(1991\)](#). This patient was mildly dysmorphic with microcephaly, prominent supraorbital ridges, and slight midfacial hypoplasia. In addition, he exhibited a number of characteristics of the 9p deletion syndrome in the oral cavity and the fingers. A study by [Gericke et al. \(1995\)](#) found that 12 male subjects with TS exhibited a chromosomal fragility at 22q12-13 that was not observed in 10 normal control male subjects. Unfortunately, linkage analyses using probes specific for these chromosomal regions have not confirmed linkage of a gene for TS to any of those regions ( [Heutink et al., 1990](#)).

Most recently, a family was reported with a balanced chromosomal translocation [t(1;8)(q21.1;q22.1)] in family members with TS and in relatives relating affected members. In a follow-up of that study ([Matsumoto et al., 2000](#)), the translocation breakpoints were cloned and sequenced. The CBFA2T1 gene was identified in close proximity to, but not spanning, the breakpoint. Sequencing of the CBFA2T1 exons in 37 unrelated patients with TS did not identify any mutations. Although changes in gene coding regions that result in altered amino acid sequences are the most obvious type of mutation, it is very possible that this translocation instead alters regulatory sequences controlling either the CBFA2T1 gene or other nearby genes.

In summary, our understanding of the genetics of TS has advanced steadily since the early 1980s. By combining analyses of the TS phenotypic spectrum with genotypic data, the field now stands at the threshold of identifying genes that have a major impact on the etiology of TS, and through those genes, gaining an understanding of the causative biology.

## Obsessive-Compulsive Disorder

Obsessive-compulsive disorder (OCD) has a lifetime prevalence of approximately 2% in the United States ( [Robins et al., 1984](#)). Previously thought to be a rare disorder, it is now estimated that OCD affects nearly 5 million U.S. citizens. OCD is characterized by the presence of distressful, time-consuming obsessions or compulsions ([American Psychiatric Association, Committee on Nomenclature and Statistics, 1994](#)). Evidence for a genetic basis of OCD is found in family-genetic studies of OCD and TS, and in twin studies of OCD.

The notion that OCD has a familial form is well supported by the current literature. Family-genetic studies ( [Bellodi et al., 1992](#); [Black et al., 1992](#); [Lenane et al., 1990](#); [Leonard et al., 1992](#)) report an increased prevalence of OCD and related obsessional traits among first-degree relatives of OCD-affected probands. [Lenane et al. \(1990\)](#) show 30% of probands with at least one first-degree relative affected with OCD; including a diagnosis of subclinical OCD as affected, they find an overall rate of 22% among all first-degree relatives, 10-fold greater than either the control group or the general population prevalence (subclinical OCD was defined as meeting all DMS-III-R criteria except one of the following: symptoms occur less than 1 hour per day, lack of ego dystonicity/insight, or lack of interference/distress). In a similar study, [Black et al. \(1992\)](#) found that the risk of broadly defined OCD was increased in parents of probands but not in control subjects (16% vs. 3%). A study by [Leonard et al. \(1992\)](#) showed the age-corrected risk of OCD in all first-degree relatives of 54 childhood-onset OCD probands to be 17%, compared with the population prevalence of 2%; similarly, [Bellodi et al. \(1992\)](#) found an OCD rate of 9% among first-degree relatives of 21 childhood-onset probands. Few of the OCD family studies reported in the literature have failed to find an increased risk of OCD among first-degree relatives of probands; in those cases, methodologic questions concerning data collection techniques (e.g., reliance on family history information obtained from only one informant in each family) and diagnostic categories (e.g., subclinical OCD classified as an anxiety disorder rather than a form of OCD) may have contributed to an underestimation of the rate of OCD in relatives.

An OCD family study that addressed those shortcomings by combining a large sample size with a control group, use of sensitive family history methods, and blind best-estimate diagnoses was reported by [Pauls et al. \(1995\)](#). Results from that study showed the rate of OCD is significantly higher in the relatives of OCD probands compared with control subjects (10.3% vs. 1.9%). This also is true for subclinical OCD; 7.9% of the first-degree relatives of OCD probands are affected, compared with only 2.0% of the control subjects. The overall rate of OCD and subclinical OCD among relatives of OCD probands was 18.2%, compared with 4.0% among



control subjects. The rate of tics (TS and CT) among relatives of OCD probands was 4.6%, significantly higher than among control subjects (1%). The sex effect seen in previous studies of families of TS probands (with female relatives being more likely to have OCD without tics) was marginally supported in these data. There is a trend for a higher rate of tics among relatives of female probands compared with relatives of male probands. Interestingly, these data also suggest that not all OCD is related to TS because it is more likely that a relative will have tics if the proband also has tics: If all OCD was related to TS, the rate of TS and CT should be the same among relatives of OCD probands with and without tics. An important effect of the proband's age at OCD onset on the rates of illness among the first-degree relatives also was seen. If a child has onset of disease between the ages of 5 to 9 years, the risk to relatives increases until it is twice that of relatives of probands with onset between 9 and 18 years of age.

The most recent family study of OCD was that of [Nestadt et al. \(2000\)](#). In that study, 80 probands and 326 first-degree relatives were examined. Using a generalized regression model that incorporated age, sex, and type of interview, and also accounted for the potential effects of intrafamily correlations (resulting from assessment of multiple relatives for a disorder transmitted within a family), Nestadt and colleagues found a lifetime prevalence rate of OCD among first-degree relatives that was 11.7% versus 2.7% in the control relatives. Thus, the concept of OCD as a familial disorder continued to be supported, with a nearly fivefold increased risk of OCD among first-degree relatives of affected individuals. In an interesting examination of the obsessive-compulsive phenotype, the Nestadt et al. study showed that relatives of OCD probands are at a greater risk for obsessions than for compulsions. This indicated that the familial nature of OCD may be centered more on obsessive rather than compulsive symptoms, or that there may be a greater symptomatic heterogeneity underlying the category of compulsions. Finally, this study also replicated the previous finding that the familiarity of OCD is associated with early ages of onset (younger than 18 years of age). An interesting result seen in both the Pauls et al. and Nestadt et al. studies was that adults with OCD have fewer children than control subjects.

Family-genetic studies of TS (see the earlier section on TS) provide another line of evidence for a genetic basis of some form of OCD. Interestingly, in the study by Leonard et al. of OCD probands, TS was an exclusionary criterion for participation in the study; at follow up 2 to 7 years later, 12% of probands had onset with TS.

Twin studies of OCD also reveal a strong hereditary component. [Inouye \(1965\)](#) reported 80% concordance between MZ twins and 25% between DZ twins for obsessional neuroses, and 15 years later [Carey and Gottesman \(1981\)](#) reported 87% concordance between MZ twins and 47% between DZ twins. Both studies indicate a large heritability for OCD, although they are constrained by small sample sizes and diagnostic categories that no longer are used.

There are a handful of published segregation analyses using OCD probands. [Nicolini and colleagues \(1991\)](#) performed segregation analyses on data collected from 24 OCD families to examine whether transmission patterns were consistent with simple mendelian models of inheritance. The probands were ascertained through the UCLA Child Psychiatry Clinic; the mean age of onset was 9.1 years. Eleven of the 24 probands had a positive family history of OCD; no further characterization of the sample is given. All available first-degree relatives were directly interviewed, with family history information used for unavailable relatives. Segregation analyses were performed including as affected all individuals with a diagnosis of OCD, CT, or TS. These investigators were unable statistically to reject either an autosomal dominant or autosomal recessive model.

Later segregation analyses were consistent with the Nicolini study results ([Cavallini et al., 1995](#)). Using more comprehensive analytic methods, Cavallini and colleagues found evidence for a major gene effect in a sample of 92 families ascertained through an OCD proband. Although the most parsimonious result was an autosomal dominant model, other major gene solutions (i.e., additive) also could adequately explain the observed familial patterns with no significant difference among them.

[Alsobrook et al. \(1999\)](#) reported the results of complex segregation analyses of 100 families ascertained through adults with OCD. Using the entire dataset and a classification of OCD as affected for the relatives, the best-fitting model was the "mixed model" of a major genetic locus operating against a significant polygenic background. The lack of definitive results may have been because approximately half of the families did not have any relatives with OCD. This pattern of partial familiarity in family samples also has been observed in at least two other studies. [Nicolini et al. \(1991\)](#) also reported that approximately half of the 24 probands in their study did not have a family history of OCD. In addition, in an effort to incorporate phenotypic subtypes in the analyses, Alsobrook et al. used a four-factor symptom dimension [four symptom groups indicated as statistically significant from a factor analysis in the probands ([Leckman et al., 1997](#))]. Analyses limited to families of probands with high scores on symmetry and ordering symptoms indicated the involvement of a major genetic locus; however, these authors also were unable to discern the specific mendelian model.

[Cavallini et al. \(1999\)](#) also reported results from a complex segregation analysis of OCD families. Their approach included information from first- and second-degree relatives, although only a limited portion of the second-degree relatives was directly assessed. In a sample of 107 families ascertained through a proband with OCD, the transmission of OCD was best modeled by a major genetic locus. However, the analyses could not significantly differentiate among dominant, additive, or recessive models. When the affected phenotype was expanded to include TS and tic disorders, only the general unrestricted model of a major genetic locus and a multifactorial background fit the data. These results still support the concept of OCD as an inherited disorder, although the precise nature of the genetic transmission remains elusive. In addition, the analyses by Cavallini et al. strengthen the idea that OCD is genetically heterogeneous. Interestingly, approximately half of their sample of families (54 of 107) also consisted of sporadic cases.

Turning to molecular studies, there currently are no reports of a concerted effort to perform a genome-wide scan for OCD as was done in autism, RD, and TS. There are many reports analyzing individual candidate genes or suites of genes, but no clear evidence has emerged to implicate a specific gene strongly and reproducibly ([Billet et al., 1998](#); [Karayiorgou et al., 1999](#); [McDougle et al., 1998](#)). In many cases, the reports of positive gene associations are based on suboptimal experimental designs, whereas many reports are conflicting or unreplicated.

Our understanding of the genetics of OCD has grown significantly, particularly since the early 1990s. Because of the phenotypic heterogeneity, progress toward mapping genes involved in OCD will likely follow the same course as seen for RD, with efforts to delineate significant phenotypic subtypes occurring in parallel with further genetic and molecular genetic studies.

## SUMMARY

Although the notion that genetic factors are important in the manifestation of childhood psychiatric disorders is not new, most of the work done to examine genetic hypotheses is relatively recent. The evidence reviewed in this chapter supports the concept that genetic factors are important in the expression of some of the more serious neuropsychiatric disorders of childhood. Given the rapid pace of development of new methods for studying the inheritance of human disorders, coupled with progress in the nosology of child psychiatric conditions, we can anticipate major advances in our understanding of the genetic and biological mechanisms underlying childhood psychiatric disorders. This will ultimately lead to improved and individualized therapies, both pharmaceutical and behavioral. All of these efforts will be well served by the accomplishments of the Human Genome Project.

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## 33 TRAUMATIC AND INFECTIOUS BRAIN INJURY IN CHILDREN: PSYCHIATRIC SEQUELAE

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Traumatic and infectious brain injuries affect large numbers of children worldwide. In industrialized countries, traumatic brain injury (TBI), primarily from motor vehicles, is an everyday risk of childhood. In the United States alone, an estimated 88,000 children per year sustain a TBI ( [Kraus, 1995](#)). In nonindustrialized countries, infectious brain injury (IBI) in the form of *Haemophilus influenzae* type b meningitis affects over 200,000 children annually ( [Salisbury, 1998](#)). Worldwide, more than 1 million children are known to be infected with the human immunodeficiency virus (HIV) ( [UNAIDS, 1998](#)), and of these children, almost one-fourth will develop an encephalopathy as part of the acquired immunodeficiency syndrome (AIDS) ( [Lobato et al., 1995](#)).

Acquired brain injury from trauma or infection concerns child psychiatrists for several reasons. First, children who survive severe TBI or IBI are at increased risk for overt neurologic impairments (e.g., cerebral palsy, mental retardation, epilepsy) ( [Menkes and Ellenbogen, 2000](#); [Weil et al., 2000](#)), which are associated with a markedly increased risk for psychopathology of all types ( [Rutter et al., 1970](#)). Second, accumulating evidence suggests that even those children who survive TBI or IBI without major neurologic impairment may be at increased risk for psychopathology ( [Brown et al., 1981](#); [Chadwick et al., 1981a](#); [Max et al., 1997c](#)). Third, because of individual differences in psychiatric outcomes after TBI or IBI, it is important for the clinician to be knowledgeable about the sources of this variability to interpret clinical information and plan interventions for individual children ( [Fletcher et al., 1995a](#)).

Potential sources of variability in psychiatric outcome after TBI or IBI include *brain injury characteristics*, such as the type severity, location, and age of the injury; *child characteristics*, such as age at the time of injury, age at the time of the follow-up, sex, and premorbid cognitive and behavioral characteristics; *family characteristics*, such as socioeconomic circumstances, premorbid family functioning, and family history of psychopathology; and *other possible outcomes* of brain injury, such as motor and sensory deficits, general and specific cognitive impairments, academic failure, impairment of adaptive functioning, and postmorbid family dysfunction ( [Fletcher et al., 1995a](#); [Shaffer, 1995](#)).

This chapter has three sections. The first provides a brief historical overview of important methodologic issues in research on the relation of TBI and IBI to childhood psychopathology. The second and third sections pertain to TBI and IBI, respectively. For each type of acquired brain injury, relevant background information is reviewed first, followed by a review of broadly consistent findings across studies with respect to factors that influence psychiatric outcomes.

### HISTORICAL OVERVIEW

Based on available methods for measuring brain injury and psychopathology in children, 20th century research on the contribution of TBI and IBI to childhood psychopathology can be divided into three overlapping but quite distinct periods.

During the first period, which dates from approximately 1900 to 1970, study of the psychiatric sequelae of acquired brain injury was limited to clinical observation. Especially influential were a series of case reports ( [Bond and Partidge, 1926](#); [Ebaugh, 1923](#); [Hohman, 1922](#)) describing postencephalitic children who exhibited hyperactivity, antisocial behavior, and emotional instability. A similar pattern of behavior was described in children after TBI ( [Blau, 1936](#); [Strecker and Ebaugh, 1924](#)). However, absent a comparison group similar to the affected children in all respects but the brain injury and absent information on the premorbid behavior of the affected children, no firm conclusion could be drawn that this pattern of behavior was due to the injury.

The second period, which dates from approximately 1970 to 1985, marks the beginning of controlled studies of psychiatric outcomes as measured with standardized instruments after TBI ( [Brown et al., 1981](#); [Shaffer et al., 1975b](#)) and IBI ( [Taylor et al., 1984](#)). During much of this period, reliable and sensitive methods of brain imaging were not available, and investigators for the most part relied on clinical signs of acute neurologic impairment [e.g., loss of consciousness (LOC), posttraumatic amnesia (PTA), or sensory or motor deficits] to determine the presence and severity of the brain injury. Psychopathology was measured with standardized instruments that had been developed in the general population of children. Children with TBI or IBI typically were compared with children with orthopedic injuries or with siblings, respectively. In the best studies, information about premorbid behavior was obtained prospectively, that is, as close to the injury or illness as possible to determine the temporal sequence needed to establish causality.

The third period dates from approximately 1985 through the present, and has witnessed an explosion of research effort that exploits advances in measurement of brain injury and psychopathology. Brain imaging methods such as computed tomography (CT), both structural and functional magnetic resonance imaging (MRI), and single-photon emission CT have permitted new ways of identifying brain injury and measuring its severity (e.g., [Kesler et al., 2000](#); [Marshall et al., 1991](#)). The assessment of psychopathology has been augmented by the development of new instruments that are sensitive to the types of behavior that are thought to occur characteristically, if not uniquely, after acquired brain injury ( [Barry et al., 1996](#); [Max et al., 2000](#)). Many of these behaviors are descriptively similar to those noted in case reports from the first period and include measures of Personality Change Due to a Medical Condition (PC) ( [American Psychiatric Association, 1994](#); [Max et al., 1997a, 1997c](#)) and the somatic, cognitive, and behavioral complaints termed the *postconcussional syndrome* ( [Roberts et al., 1996](#)). At the same time, other possible outcomes of brain injury, such as attentional ( [Dennis et al., 1995](#); [Ewing-Cobbs et al., 1998b](#); [Konrad et al., 2000](#)), memory ( [Anderson et al., 2000](#); [Proctor et al., 2000](#)), and language deficits ( [Chapman, 1995](#); [Dennis and Barnes, 1990](#); [Docking et al., 2000](#)), have been studied, as well as other indicators of outcome such as academic performance ( [Ewing-Cobbs et al., 1998a](#)), adaptive functioning ( [Cattalani et al., 1998](#)), social functioning ( [Andrews et al., 1998](#)), and family function ( [Andrews et al., 1998](#); [Max et al., 1998b](#); [Taylor et al., 1995](#)). Findings from these studies are being interpreted within more sophisticated models that view psychopathology as both an outcome and as a mediator or moderator of other outcomes, so that intervention can be applied at the most effective point ( [Taylor et al., 1995](#)).

From a methodologic standpoint, an ideal study of psychiatric outcomes after acquired brain injury (a) uses a representative sample of affected children (to avoid referral bias); (b) uses an appropriate control group; (c) enrolls subjects prospectively rather than retrospectively (to avoid recall bias and to determine the temporal sequence needed to establish causality); (d) uses widely accepted definitions of brain injury severity and other characteristics (to enable comparison with other studies); (e) uses both traditional psychiatric diagnostic instruments, as well as psychiatric outcome measures that are sensitive specifically to the effects of brain injury; (f) assesses other variables that may confound, mediate, or moderate the effects of brain injury on behavior, such as preinjury behavior and family

characteristics; and (g) assesses other indicators of outcome, such as school performance and family functioning.

Few studies meet all of these criteria, in part because our appreciation of these requirements has evolved over time. As a result, across studies, some of the variability in outcome after acquired brain injury is related to differences in methodology; this is noted where relevant. However, this chapter strives to emphasize broadly consistent findings across methodologically sound studies with respect to predictors of psychiatric outcomes, with particular attention to studies from the last decade. Because predictors of psychiatric outcome have been studied more systematically with respect to TBI, this topic is covered first.

## TRAUMATIC BRAIN INJURY

Traumatic brain injury (TBI) is defined as physical damage or impairment in function of the brain secondary to the exchange of acute mechanical energy. This definition typically excludes other head injuries such as nondepressed and linear skull fractures, as well as other brain injuries resulting from birth trauma, poisoning and asphyxia, cerebrovascular events, or tumors ([Fife, 1986](#)).

### Classification, Pathophysiology, and Epidemiology of Traumatic Brain Injury

#### CLASSIFICATION BY CAUSE

At a first level, TBI is categorized as caused by either open or closed head injury. Open head injury occurs when the skull is penetrated, as by a bullet or by a depressed skull fracture. Closed head injury occurs when a mechanical load, either dynamic or static, is applied to the intact skull ([Rao and Lyketsos, 2000](#)). Dynamic loading, exemplified by high falls and motor vehicle accidents in which the child is a passenger, occurs when there is a sudden rapid movement of the head for less than 50 milliseconds, resulting in rapid acceleration and deceleration of the brain in the skull ([Duhaime et al., 1995](#); [Gennarelli, 1993](#)). Static loading, exemplified by crush injuries from heavy objects such as television sets, occurs when static forces are applied for longer than 200 milliseconds over a relatively large area, slowly deforming the cranium ([Gennarelli and Meaney, 1996](#); [Presad et al., 1999](#)). In children, more than 90% of cases of TBI are due to closed head injury ([Menkes and Ellenbogen, 2000](#)), and most involve a dynamic load ([Presad et al., 1999](#)). In the past, open and closed head injury were thought to result in “focal” and “diffuse” brain injury, respectively. However, recent neuropathologic and brain imaging studies, discussed later in the section on Pathophysiology, suggest that open and closed head injury are rarely pure in terms of focal and diffuse effects on the brain ([Fletcher et al., 1995b](#)).

#### CLASSIFICATION BY SEVERITY

Traditionally, severity has been assessed on the basis of initial level of consciousness, duration of coma, or extent of PTA ([Adams et al., 1991](#)).

Level of consciousness can range from the fully conscious state to a confusional state to stupor and coma ([Plum and Posner, 1980](#)). Coma is a state in which the patient lies with eyes closed and evidences no awareness or responsiveness to external and internal stimuli. The comatose patient fails to react to noxious stimuli with defensive localized movements, and does not speak ([Salzman, 1990](#)). The Glasgow Coma Scale (GCS) ([Teasdale and Jennett, 1974](#)) has become the standard index to assess initial level of consciousness in adults and measure duration of LOC. It assesses function in three domains: eye opening, verbal response, and motor response, yielding a range of scores from 3 to 15. The Glasgow Children's Coma Scale has been adapted for infants and toddlers; it emphasizes brain stem functions and does not assess higher integrative cortical functions ([Raimondi and Hirschauer, 1984](#)).

PTA, another index of severity, refers to the period after recovery from coma in which continuous memories are not stored ([Russell and Smith, 1961](#)). The period of PTA is on average four times longer than the coma stage ([Guthkelch, 1979](#)). For adults, the point at which they can correctly report what has happened to them and the circumstances of their hospitalization and accurately state the month, year, day of month, and day of week has been taken as the resolution of PTA ([Cronwall, 1989](#)). The Children's Orientation and Amnesia Test ([Ewing-Cobbs et al., 1990](#)) evaluates PTA in children 3 to 15 years of age and is considered valid ([McDonald et al., 1994](#)).

The most recent and widely accepted classification system for severity depends on the degree of the initial impairment, combining the initial GCS, the duration of LOC, and the duration of PTA ([Kraus, 1999](#)). *Mild* injury is defined as a GCS of 13 to 15, LOC of less than 30 minutes, or PTA of less than 1 hour; *moderate* injury is defined as a GCS of 9 to 12, LOC of 1 to 24 hours, or PTA of more 30 minutes to 24 hours; and *severe* injury is defined as a GCS of 8 or less, LOC of more than 24 hours, or PTA of more than 1 day. In the past, some studies have used only the GCS to classify the severity, whereas others have used LOC or PTA.

#### PATHOPHYSIOLOGY

Conventionally, a distinction is made between primary brain injury and two types of secondary brain injury. One type of secondary brain injury arises from the brain's normal response to the primary injury and the other from noncerebral insults such as hypoxemia or hypotension ([Adelson and Kochanek, 1998](#)).

Primary brain injury begins within the first few milliseconds of an injury to the brain, although it may take hours to days to evolve. Primary brain injury includes vascular injuries (e.g., subdural hematoma and subarachnoid hemorrhage), as well as three types of injuries to the brain substance: (a) cerebral laceration, (b) diffuse axonal injury, and (c) cerebral contusion ([Kraus and Sorenson, 1994](#)).

Cerebral laceration is relatively rare and usually is seen in open head injury as a result of a penetrating missile or depressed skull fractures. However, in infants, lacerations of white matter can occur without fracture owing to the soft consistency of the incompletely myelinated cerebrum and the pliancy of the infant's skull. Lacerations frequently involve the frontal and temporal poles and are associated with tears in the dura and major vessels and secondary thromboses, hemorrhages, and cerebral ischemia ([Menkes and Ellenbogen, 2000](#)).

Diffuse axonal injury, the most common lesion identified on MRI after a closed head injury, is caused by severe angular acceleration–deceleration forces that occur with dynamic loading ([Adams et al., 1991](#)). The most characteristic sign for diffuse axonal injury is LOC. The duration of unconsciousness is related to the degree of axonal injury, with widespread severe damage leading to deep or persistent coma ([Teasdale and Mendelow, 1984](#)).

Diffuse axonal injury is thought to be a major mechanism of damage in severe closed head injury. Children are more susceptible to diffuse axonal injury than adults because of their head-to-body ratio, weak neck musculature, and incomplete myelination ([Adelson and Kochanek, 1998](#)). Shearing forces sustained during injury are thought to stretch the axons, opening membrane channels at the moment of injury, with an influx of calcium that increases the activity of proteolytic enzymes that disrupt axonal architecture and result in axonal swelling ([Maxwell et al., 1997](#)). When axonal swelling exceeds a certain level, effective transection of the axon has occurred, without evident tearing or damage to adjacent blood vessels. The neuropathologic picture of diffuse axonal injury correlates well with findings on MRI, where it appears as small, oval, focal abnormalities in white matter tracts, usually adjacent to cortical gray matter, but sometimes in the splenium of the corpus callosum ([Gentry et al., 1988](#)). In very severe TBI, the white matter between the cortex and subcortical centers is transected, leading to death ([Weil et al., 2000](#)).

In mild TBI, lesser degrees of diffuse axonal injury and loss of cerebral autoregulation are thought to be responsible for concussion, defined as a transient but instant widespread loss of neuronal function ([Menkes and Ellenbogen, 2000](#)). The concept of concussion as a fully reversible injury with no structural alteration of the brain has been challenged on the basis of several studies using both CT scanning and MRI ([Levin et al., 1987a, 1987c](#)). These studies have demonstrated other occult intracranial lesions such as cerebral contusions (see later), intracranial hematomas, and subarachnoid hemorrhage in a significant number of patients designated as having mild head injury.

Cortical contusion is the second most common type of brain injury visualized by MRI after closed head injury ([Menkes and Ellenbogen, 2000](#)). Contusions consist mainly of petechial hemorrhages in the superficial cortical layers, occurring at the site of penetration or the impact (coup) area, or at the contrecoup area. After closed head injury, contusions are most likely to occur on the crests of gyri on the orbital aspects of the frontal lobes and the inferolateral aspect of the temporal lobes ([Courville, 1964](#); [Gentry et al., 1988](#)) because of bony protuberances on the anterior fossa and sphenoid region ([Miller and Becker, 1982](#)). The severity of brain damage caused by contusion depends on the extent of the vascular injury, but the injury tends to be focal rather than diffuse. Brain imaging studies have shown that the frontal lobes are particularly vulnerable to contusion in closed head injury, leading to a new appreciation of focal effects in closed head injury ([Ewing-Cobbs et al., 1995](#); [Fletcher et al., 1995a](#)).

The brain's normal reaction to primary brain injury is a cascade of physiologic and biochemical events within the brain. In children, where most cases are closed head



injury, diffuse injury to the brain may lead to cerebral swelling, which in turn leads to intracranial hypertension, ischemia, and vasospasm ( [Fishman, 1992](#)). Endogenous biochemical cascades of excitatory amino acids (glutamate, aspartate); cytokines; and complement may aggravate diffuse brain swelling. Children can be more prone than adults to sustain a hypoxic or hypotensive episode posttrauma, and these noncerebral events also contribute to diffuse brain swelling ( [Cruz et al., 1992](#)). The acute treatment of primary TBI in children is the province of neurology and neurosurgery and is primarily directed toward interrupting the brain's normal secondary response to injury, as well as avoiding and treating other noncerebral conditions, such as hypotension, that may contribute to brain swelling ( [Adelson and Kochanek, 1998](#)).

Seizures are a complication of TBI, occurring in 6% of mild and 35% of severe TBIs ( [Bruce, 1993](#)). Seizures associated with mild TBI are unique to childhood. Early seizures are more common in children younger than age 5 years, usually occur within 24 hours, and do not predict outcome ( [Hauser, 1983](#)). Early seizures should be distinguished from later-onset epilepsy, which develops in 5% of patients with TBI. Epilepsy occurs more commonly after open head injury and is more likely to occur as the level of severity increases. Partial seizures are more common than generalized seizures after head injury ( [Bruce, 1993](#); [Jennett and Teasdale, 1981](#)).

## EPIDEMIOLOGY

Approximately 15,000 children annually in the United States sustain a severe TBI ( [Di Scala et al., 1991](#)). According to the U.S. National Pediatric Trauma Registry, 73% or 210 of 286 children who were discharged from a major trauma unit with multiple functional impairments also had significant behavior problems ( [Di Scala et al., 1991](#)).

The rate, severity, and mechanism of TBI vary by age. The rate increases steadily up to 14 years of age, with a dramatic peak occurring at age 15 years. Ninety-three percent of all TBI in children ( [Kraus et al., 1986](#)), compared with 80% in adults ( [Kraus and Sorenson, 1994](#)), has been classified as mild. Mild TBI is most common in 10- to 14-year-olds, whereas the peak incidence of severe and moderate injury occur at age 15 years, but a relatively high proportion of moderate to severe injuries also occurs in infants younger than 1 year of age ( [Kraus et al., 1986](#)). In infancy, the male-to-female ratio is 1:1, but beginning at 5 years of age, boys begin significantly to outnumber girls ( [Courville, 1964](#)).

The predominant mechanism of injury also differs by age. In infancy, child abuse accounts for most cases of TBI. Among preschoolers, falls are the predominant mechanism, whereas among early elementary school-age children, pedestrian accidents are the most common source of TBI. In 10- to 14-year-olds, there is a dramatic increase in injuries from sport and bicycle accidents. Beginning at age 15 years, motor vehicle accidents in which the victim is a passenger becomes the most common cause of TBI ( [Kraus et al., 1986](#); [Rivera and Mueller, 1986](#)).

Demographic risk factors for TBI include poverty; single-parent households; and parental history of psychiatric disorder, drug and alcohol abuse, or physical illness. Child characteristics, such as risk-taking behavior, also are important ( [Bijur and Haslum, 1995](#)), although others ( [Donders, 1992](#); [Pelco et al., 1992](#)) have not found this relationship.

## Measurement of Psychopathology After Traumatic Brain Injury

Measures used to study psychopathology after TBI include traditional measures developed to assess psychopathology in the general population, including psychiatric interviews such as the Kiddie-Schedule for Affective Disorders and Schizophrenia ( [Chambers et al., 1985](#)) and others ( [Rutter et al., 1970](#)), as well as behavior problem checklists such as the Achenbach Child Behavior Checklist (CBCL) ( [Achenbach and Edelbrock, 1983](#)). Recently, more targeted measures have been developed to assess symptoms thought to be characteristic of TBI.

[Max et al. \(1998a\)](#) developed the Neuropsychiatric Rating Schedule, an interview designed to elicit the symptoms of Organic Personality Syndrome as per the third edition, revised, of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III-R) ( [American Psychiatric Association, 1987](#)) and PC as per DSM-IV ( [American Psychiatric Association, 1994](#)). It consists of 22 items that cover affective instability, aggression and rage, markedly impaired social judgment (e.g., disinhibition), apathy, and paranoia.

The postconcussional syndrome, which is not a DSM-IV diagnosis, refers to a constellation of somatic and psychological symptoms, including headache, dizziness, fatigue, diminished concentration, memory deficit, irritability, anxiety, insomnia, hypochondriacally concern, hypersensitivity to noise, photophobia ( [Levin et al., 1982](#)), and seizure-like symptoms ( [Roberts et al., 1996](#)), frequently reported in patients with brief periods of coma or otherwise disturbed consciousness after TBI. Measures developed to assess postconcussional symptoms include the Iowa Interview of Partial Seizure-Like Symptoms ( [Roberts et al., 1996](#); Varney et al.) as adapted for children ( [Roberts et al., 1996](#)), the Pediatric Inventory of Neurobehavioral Symptoms ( [Roberts, 1990](#)), the Neurobehavioral Rating Scale ( [Levin et al., 1987b](#); [Vanier et al., 2000](#)), and the Post-Injury Symptom Checklist ( [Barry et al., 1996](#)).

## Predictors of Psychopathology After Traumatic Brain Injury

### BRAIN INJURY CHARACTERISTICS

#### Severity of Brain Injury

Severity as measured by initial neurologic impairment has been the most studied predictor of psychiatric outcome after TBI. Despite varying criteria for severe versus mild TBI, a fairly consistent finding across studies is that, after accounting for premorbid psychopathology, severe but not mild TBI increases the risk for psychopathology. However, as reviewed later, controversy continues over the psychiatric significance of mild TBI.

**Severe Brain Injury.** The first prospective study of closed head injury to use standardized psychiatric diagnostic assessments remains a benchmark in the field. Rutter and colleagues ( [Brown et al., 1981](#); [Rutter et al., 1980](#)) obtained an account of premorbid behavior as soon as possible after the injury and examined behavior change over time with a longitudinal design. The possibility of a threshold effect for severity of injury was examined by including a severe injury group, defined as having PTA of at least 7 days, and a mild injury group having PTA or more than 1 hour but less than 1 week. The comparison group consisted of 28 children who had received hospital care for orthopedic injuries.

The rate of newly appearing psychiatric disorders was much increased in the severe head injury group (60%), whereas new psychiatric disturbance in the mild head injury group (24%) was no different from that seen in the orthopedic control subjects (24%). This was true at 4 months, 1 year, and 2 years and 3 months after the accident. There appeared to be a threshold effect and a modified dose-response effect of severity: Among the mild injury group, children with less than 7 days of PTA had rates similar to those with less than 24 hours of PTA. In the severe injury group, those who had PTA for longer than 21 days had a higher rate of new disorder than those who had PTA of 7 to 21 days.

[Brown et al. \(1981\)](#) considered whether the psychiatric disorders that appeared for the first time after head injury were distinctive in pattern. At 1 year, among children in the severe brain injury group who had new-onset disorders, 14% had emotional disorders, 10% mixed emotional and conduct disorders, 5% conduct disorder, and 5% psychosis. Another 24% exhibited a disinhibited state. Typical behaviors included being unduly outspoken, making frequent personal remarks, or asking embarrassing questions and getting undressed in inappropriate social situations. Other behaviors included forgetfulness, loquaciousness, carelessness in personal hygiene, and impulsivity ( [Brown et al., 1981](#); [Shaffer, 1995](#)). The disinhibited children all had a PTA exceeding 3 weeks, and three showed persistent intellectual impairment, suggesting that brain damage was directly implicated in the etiology. The clinical picture in two of the children evolved to a picture of attention deficit/hyperactivity disorder (ADHD) by the second year ( [Shaffer, 1995](#)).

The findings of [Brown et al. \(1981\)](#) with respect to the excess risk for psychiatric disorder among children with severe, but not mild injury, are supported by other retrospective ( [Lemkuhl and Thoma, 1990](#); [Shaffer et al., 1975b](#)) and prospective studies ( [Max et al., 1997a, 1997b, 1997c](#)) of TBI. Moreover, ongoing work by Max et al. ( [Childers et al., 1998](#); [Max et al., 2000](#)) has extended the findings of [Brown et al. \(1981\)](#) with respect to the "disinhibited state" as a distinctive novel pattern of behavior after severe brain injury. In a both retrospective and prospective study, [Max et al. \(2000\)](#) studied the prevalence and correlates of the DSM-IV equivalent of the "disinhibited state," namely, the diagnosis PC Due to a Medical Condition (Head Injury). They found approximately 40% of consecutively hospitalized children with severe TBI had ongoing persistent PC an average of 2 years postinjury. Another 20% had a history of remitted and more transient PC. PC occurred in 5% of mild to moderate TBI but was always transient. The PC most often was a combination of the PC subtypes included in DSM-IV, with the labile, aggressive, or disinhibited subtypes being the most common, the apathetic subtype being transient, and the paranoid subtype rare. In subjects with severe TBI, persistent PC was significantly associated with severity of injury, particularly impaired consciousness over 100 hours, adaptive and intellectual functioning decrements, and concurrent diagnosis of

secondary ADHD. However, persistent PC was not related to psychosocial adversity.

As reviewed by [Max et al. \(2000\)](#), PC is the most recent name given to a syndrome or syndromes of behavioral change attributed directly to brain injury from various causes. The behavioral changes are distinct from disorders resembling affective, anxious, psychotic, and disruptive behavior disorders that may or may not be the direct result of acquired brain injury. In the past, terms given to this group of behaviors include *organic personality syndrome* ([American Psychiatric Association, 1987](#)); *frontal lobe syndrome* ([Lishman, 1978](#)); *compartmental learning disabilities* ([Price et al., 1990](#)); *posttraumatic chronic behavior disorder* ([Blau, 1936](#)); and the *regional prefrontal syndromes*, including the dysexecutive type (dorsal convexity system) disinhibited type (orbitofrontal system), and apathetic type (mesofrontal system) ([Duffy and Campbell, 1994](#)).

**Mild Brain Injury.** As reviewed previously, prospective studies using psychiatric diagnoses as outcomes do not find that mild TBI increases the risk for psychopathology. Moreover, two well designed prospective studies that focused on mild TBI and used behavior problem checklists developed in the general population also failed to find effects of mild TBI on psychopathology.

The first of these studies is the UCLA Study of Mild Closed Head Injury in Children and Adolescents ([Asarnow et al., 1995](#)). This study was a prospective cohort follow-up of a representative sample of children diagnosed with mild closed head injury in area emergency departments. Comparison groups consisted of children with other nonhead/brain injuries admitted to the same emergency departments as the cases and noninjured children in area schools who were demographically similar to the cases. Mild TBI was defined as an uncomplicated closed brain injury, which operationally meant that each child had a minimum of two concussive symptoms documented in the emergency department records and GCS score greater than 12. Within 1 month of the injury, parents were asked to complete the CBCL for the 6 months before injury and were asked to complete it again at 12 months postinjury. The mild TBI group did not show an increased rate of behavior problems at 12 months postinjury compared with the 6 months before injury. The investigators stressed that the mild TBI group would have had elevated rates of behavior problems had they not measured preinjury level of functioning. The children in the mild TBI group actually had higher levels of behavior problems before the injury, and the rates of behavior problems actually declined in the 12 months after the injury, consistent with the view that mild TBI did not directly cause an increased rate of behavior problems, at least as measured by the CBCL.

In the second study, [Bijur and Haslum \(1995\)](#) studied 12,000 children enrolled in the British Cohort Study and assessed at ages 5 and 10 years. Mild injury was defined as parental report of a head injury that resulted in concussion or LOC and treatment as an outpatient or admission overnight to the hospital. Using behavior problem checklists, the head-injured children were compared with a random sample of children who had no injuries and with children who had limb fractures, children with lacerations, and children with burns. Behavior problems did not differ significantly across groups. Although parental ratings of hyperactivity were highest in the brain-injured group, even after control for preinjury hyperactivity, hyperactivity scores in the groups with other injuries also were higher than in control subjects. This suggested that differences between the brain-injured group and control subjects were due to factors that elevate the risk for any injury. Moreover, children with multiple mild head injuries did not show adverse behavioral sequelae. Other controlled studies of mild TBI in children have been consistent with these findings (e.g., [Fay et al., 1993](#)). Although it is clear that most children with mild TBI do not experience psychiatric morbidity, a "miserable minority" ([Ruff et al., 1994](#)) do experience problems, often described as the postconcussional syndrome. [Roberts et al. \(1990\)](#) have argued that behavior problem checklists developed in the general population, such as the CBCL ([Achenbach and Edelbrock, 1983](#)), are not sensitive to postconcussional symptoms, which include episodic or spell-like symptoms ([Roberts et al., 1990, 1995, 1996](#)). Using an adaptation of an interview to assess partial seizure-like symptoms, they found that children with mild and severe injury were equally likely to endorse symptoms of staring spells; there was no dose-response curve relating frequency or severity of episodic symptoms to injury severity. Other investigators ([Barry et al., 1996](#)), however, have found the frequency of postconcussional symptoms as assessed with the Post Injury Neurobehavioral Checklist was related to severity of injury in children with moderate to severe injury, suggesting that similar symptoms may be useful in identifying sequelae associated with mild TBI. In adults with mild TBI, postconcussional symptoms have been linked to abnormalities in neuropsychological assessment and in functional neuroimaging ([Barry et al., 1996](#)).

#### *Site of Brain Injury*

The only study to date of open head injury ([Shaffer et al., 1975a](#)) found no significant effects for site of injury after controlling for psychosocial correlates. Although closed head injury in general has been considered to be diffuse, brain imaging studies suggest that contusional injuries of the frontal lobe may be more common than previously thought ([Filley et al., 1987](#); [Fletcher et al., 1995b](#); [Levin et al., 1993](#)). In a small sample of 53 children with closed head injury, [Filley et al. \(1987\)](#) found that children who had persistent behavior problems at long-term follow-up (average, approximately 3 years) displayed either a profile of overarousal, with inattentiveness, irritability and hyperactivity, often accompanied by impulsivity, inappropriate behavior, and aggressiveness, or a profile of underarousal with apathy, poor motivation, and social withdrawal. All of these patients had either discrete frontal lesions on CT or prolonged coma with slow recovery of attention, thought to be due to diffuse axonal injury and consistent with the disinhibition noted in clinical observations and more recent research. Although [Max et al. \(2000\)](#) found no relation between site of lesion (frontal, temporal, parietal, occipital) as recorded from the initial CT scan and labile, aggressive, or disinhibited subtypes of PC, they noted that CT is relatively insensitive in detection of diffuse injuries, subcortical lesions, and small lesions.

#### *CHILD CHARACTERISTICS*

##### *Child Age at Injury*

Two studies ([Max et al., 1997b](#); [Shaffer et al., 1975b](#)), both of severe TBI, examined the child's age at injury in relation to psychiatric outcomes, and neither found an effect. This contrasts with cognitive outcomes, where younger children usually sustain greater impairment.

##### *Premorbid Cognitive Functioning*

[Brown et al. \(1981\)](#) found that new disorders after TBI were more common among children with initial intellectual impairment.

##### *Premorbid Behavioral Functioning*

Preinjury behavior was a powerful predictor of later problems in all brain injury groups.

#### *FAMILY CHARACTERISTICS*

##### *Family Functioning*

Premorbid family adversity has consistently been found to increase risks for psychopathology after injury ([Brown et al., 1981](#); [Max et al., 1997b](#); [Shaffer et al., 1975b](#)). In a prospective study of closed head injury, the rate of appearance of new disorders was significantly greater in children who experienced the combination of a severe head injury and psychosocial adversity but was not greater than that expected from the two alone, suggesting an additive rather than a synergistic effect ([Brown et al., 1981](#)).

Both retrospective ([Perrott et al., 1991](#)) and prospective studies ([Rivara et al., 1992, 1993](#)) have found adverse effects of severe TBI on family functioning. A more recent study by [Taylor et al. \(1995\)](#) confirmed that child behavior outcomes after injury are related to postinjury family function, parental psychological distress, and the perceived impact or burden of the injury on the family. Six months after an initial postinjury assessment, parents of children with TBI report more family disagreements than do parents of children with orthopedic injuries, and parents of children with TBI tend to report more psychological distress. To the extent that these family characteristics are attributable to the injury itself (i.e., were not present before injury), the results also support the hypothesis that child outcomes depend in part on how the injury has affected the family ([Taylor et al., 1995](#)).

##### *Family Psychiatric History*

In a 2-year prospective, longitudinal study of children with TBI, [Max et al. \(1997b\)](#) found that a rating of family history of psychiatric disorder was not by itself significantly related to new-onset psychiatric disorder at 2-year follow-up.



## Other Outcomes

### NEUROLOGIC DEFICITS

In a study of closed head injury ([Brown et al., 1981](#)), psychiatric disorder was most frequent among the children showing persistent neurologic abnormalities at the 2½-year follow-up.

### ATTENTION DEFICIT/HYPERACTIVITY DISORDER

[Max et al. \(2000\)](#) found that persistent PC after severe TBI was associated with a diagnosis of secondary ADHD. [Gerring et al. \(1998\)](#) found that children in whom new-onset of ADHD developed after closed head injury had significantly greater premorbid social disadvantage and exhibited more emotional lability and aggression, other psychiatric disorders, and overall disability than children in whom new-onset ADHD did not develop. They noted that the diagnoses of ADHD and PC due to closed head injury both have deficit of inhibition as a primary feature.

In a recent study, Konrad et al. ([Eide and Tysnes, 1992](#)) compared 27 children with TBI to 31 children with developmental ADHD and 26 matched control subjects aged 8 to 12 years. Children with TBI and ADHD showed a pervasive deficit in their inhibitory control processes with respect to inhibition of both prepotent and ongoing responses. In addition, children with TBI were found to have a general slowing of their information processing that was not correlated with the inhibition deficits.

### NEUROPSYCHOLOGICAL DEFICITS

The relation between psychiatric sequelae and cognitive sequelae of TBI in children remains a theoretical and clinical conundrum. A considerable body of evidence has established that severe TBI, but not mild TBI, is associated with decrements in general intellectual functions ([Chadwick et al., 1981b, 1981c](#); [Max et al., 1999](#)), memory functions ([Anderson et al., 2000](#); [Levin et al., 1995](#)), and attention ([Chapman, 1995](#); [Dennis et al., 1995](#); [Max et al., 1999](#)).

[Max et al. \(1999\)](#) have shown that a neuropsychiatric factor that combines severity of injury measures and novel postinjury psychiatric disorders was significantly associated with cognitive and memory outcome after childhood TBI. New postinjury psychiatric disorder significantly added to indices of TBI severity in explaining some cognitive outcomes. Furthermore, cognitive outcomes were independently related to psychosocial disadvantage. Improved understanding of how neuropsychiatric and psychosocial disadvantage affect outcome is important for treatment. Treatment of novel psychiatric disorders and family therapy may be hypothesized to improve cognitive outcome. If, on the other hand, postinjury psychiatric disorders are a consequence of neurocognitive deficits rather than their cause, then cognitive rehabilitation and educational remediation after TBI may help prevent postinjury psychopathology.

## Treatment for Psychopathology After Traumatic Brain Injury

In the absence of controlled studies demonstrating clear-cut efficacy for specific treatment interventions, a broad-based, multidisciplinary approach seems most reasonable, based on accumulated clinical experience to date ([Silver and Yudofsky, 1994](#)). Insofar as many of the psychiatric disorders encountered in the wake of TBI are representative of the spectrum of child psychopathology in general, those treatment strategies that would ordinarily apply would still pertain to specific psychopathology encountered in the wake of brain injury. However, a few additional considerations particularly pertinent to the brain-injured child are addressed here.

In the psychopharmacologic domain, the greater sensitivity of brain-injured patients to the sedative and anticholinergic effects of various psychotropic medications leads to the advisory of starting with lower doses and raising them in small increments over time. Similarly, because of the greater incidence of disinhibited, impulsive, and aggressive behavior documented in children with brain injury, it is important to avoid indiscriminate use of neuroleptic drugs in such patients, in the absence of documented psychosis ([Silver et al., 1992](#)). This is true not only because of potential sedative and anticholinergic side effects and the dangers of tardive dyskinesia, but because neuroleptics may lower the threshold for seizures, to which brain-injured patients are particularly vulnerable (see the section on epilepsy in [Chapter 61](#)).

A double-blind, placebo-controlled study of methylphenidate in 15 adult patients with TBI found improvement in mood and cognition in all but one patient ([Gualtieri and Evans, 1988](#)). However, this study has not been replicated in younger patients. Promising work in adults with dopaminergic agents such as amantadine, levodopa ([Kraus and Maki, 1997](#)), and bromocriptine ([Goldberg et al., 1982](#); [Taverni et al., 1998](#)) has not been replicated in children.

The use of anticonvulsants may be indicated in children with episodic or spell-like symptoms after head injury. [Roberts et al. \(1996\)](#) found that divalproex sodium (Depakote; Abbott Laboratories, Abbott Park, IL) improved functioning in a high proportion of pediatric patients with episodic symptoms after head injury. There is some anecdotal support for the use of carbamazepine in children with disinhibited aggression, affective lability, and other psychopathologic states in the wake of brain injury ([Lewin and Sumners, 1992](#)). This would be particularly justified if the patient's electroencephalogram (EEG) demonstrated epileptiform abnormalities despite the absence of clinical seizures. Clinical experience, however, has expanded our awareness of potentially untoward effects of carbamazepine, including the possible worsening of irritability and aggressive behavior, as well as precipitation of mania ([Pleak et al., 1998](#)). There have been a number of reports regarding the potential merits of using b-adrenergic blocking medications for the treatment of uncontrolled aggressive outbursts in patients with organic brain impairment ([Connor, 1993](#); [Williamson et al., 1982](#)). This can be done in conjunction with or independent of any concomitant anticonvulsant medication. Clinical experience to date, which requires controlled replication, suggests that some patients demonstrate improved behavioral control of aggressivity and irritability after appropriate dose titration. Knowledge of appropriate monitoring and titration procedures is important and easily mastered.

Probably of equal importance to any psychopharmacologic intervention are behavioral and psychosocial measures that are widely used and of apparent benefit to a significant proportion of brain-injured youngsters, although again the results have received limited systematic evaluation. These measures include parental counseling, individual and family education and supportive psychotherapy, speech therapy, associated rehabilitative therapies, and academic tutoring. [Warschausky et al. \(1999\)](#) have reviewed the empirical support for psychological and behavioral therapies for the behavioral and psychosocial sequelae of TBI. While noting the success of operant conditioning in decreasing aggression during acute recovery and later, they noted the lack of outcome studies for treatment of internalizing disorders in particular.

Also, it has been suggested that it is possible to improve the performance of head-injured subjects by slowing down the pace at which information is presented, reducing background distractions or noise, or allowing additional time to complete tasks ([Ponsford and Kinsella, 1992](#)). Although systematic studies undoubtedly are desirable in this area, the many clinical variations in the broad spectrum of brain-injured youngsters will continue to require individual tailoring of treatment plans to meet the unique needs of individual patients.

Further complicating the assessment of minor head injury and the postconcussional syndrome are individual patient variables, including preexisting neuropsychiatric disorder, nonspecific effects of hospitalization after trauma, and secondary emotional factors, including the possible effects of potential litigation. Each of these factors may contribute disproportionately to the apparent sequelae of a head injury, quite apart from actual neurologic and neuropsychiatric sequelae.

It seems reasonable to suggest that, when a patient presents after minor head injury with persistent symptoms associated with the postconcussional syndrome, a full reassessment addressing the aforementioned differential diagnostic possibilities should be pursued. In addition to neurologic reassessment and a repeat EEG, neuroradiologic reassessment often is advisable. Thus, [Levin et al. \(1987a, 1987c\)](#) reported that, of 16 consecutive admissions with minor or moderate closed head injury, 14 had abnormalities on MRI, although their prior CT scans were normal or showed fewer abnormalities. Similarly, an apparent decline in academic performance, emotional responsiveness, or behavioral appropriateness should prompt formal neuropsychological and psychiatric consultation, with the previously outlined considerations in mind. If a patient shows no objective evidence of neurologic or cognitive impairment on the various diagnostic parameters enumerated, a supportive psychotherapeutic strategy geared to reassuring both the patient and family regarding a generally favorable prognosis and avoiding the secondary gains of invalidism is most judicious. Advising prompt settlement of any outstanding legal claims is helpful in this regard.

## Prevention of Traumatic Brain Injury

Children younger than 4 years of age who are not restrained in safety seats are 11 times more likely to be killed in motor vehicle accidents ([Silver et al., 1992](#)). Conversely, child safety seats have been found to be 80% to 90% effective in the prevention of serious injuries to children. All 50 states in the United States and the District of Columbia have mandatory laws for child safety seats. Public education to encourage compliance with these laws would contribute to primary prevention of a

large proportion of pediatric TBIs.

The potential success of prevention programs was demonstrated recently by a hospital-initiated program that targeted traffic injuries among school-age children in one section of New York City. The program included traffic safety education, expanded areas for safe play, bicycle safety education and helmet distribution, and supervised recreational activities. During the intervention, the average incidence of traffic injuries among this age group declined significantly by 36% relative to the preintervention period ([Durkin et al., 1999](#)). More effective public health measures to curb child abuse, alcoholism (especially drunk driving), and preventable accidental injuries also would be desirable.

### Conclusions Regarding Psychiatric Sequelae of Traumatic Brain Injury

First, severe but not mild brain injury markedly increases the risk for all types of psychiatric disorders as measured by traditional instruments, even after controlling for other child characteristics, such as intelligence or premorbid behavior, and family characteristics. Second, among the severely injured, those with chronic neurologic deficits have the highest risk for psychopathology; however, some severely injured children without chronic neurologic deficits also develop new psychopathology after the injury. Third, the most common type of new psychiatric disorder after severe TBI in children is PC due to TBI, and the most common symptoms are consistent with the labile, aggressive, and disinhibited subtypes ([Max et al., 2000](#)). Fourth, mild brain injury does not increase the risk for psychiatric disorders in general or for PC in particular, but may increase the risk for a constellation of cognitive, somatic, and behavioral symptoms known as the *postconcussional syndrome* ([Roberts et al., 1996](#)); further research in this area is needed. Fifth, family functioning is an extremely important predictor of outcome in both severe and mild TBI, and TBI itself may be a cause of family dysfunction. Finally, burgeoning research on the brain imaging abnormalities and neuropsychological deficits associated with postinjury psychopathology in children promises new advances in our understanding of children's outcomes after TBI.

## INFECTIOUS BRAIN INJURY

Although modern thinking about the contribution of acquired brain injury actually began with observations after IBI, psychiatric outcomes after IBI have been studied far less systematically or extensively than TBI. A detailed review of the medical diagnosis and treatment of the various forms of IBI is beyond the scope of this chapter, and the reader is referred to other texts ([American Academy of Pediatrics, 2000](#)).

### Terminology

Central nervous system (CNS) infection usually is classified by pathogen (i.e., bacterial, spirochetal, fungal, viral, protozoan). The brain responds to infection in a limited number of ways, regardless of the nature of the pathogen. CNS infections have in common a number of clinical and pathologic features, with the clinical presentation being determined by the virulence of the pathogen, the site and the extension of the infection, and the immunologic response of the individual ([Weil et al., 2000](#)). Although bacterial infections have a different presentation from viral infections, it can be difficult to distinguish bacterial from fungal infections and viral infections from chronic granulomatous, viral, spirochetal, or protozoan infections solely from the history and physical examination of the patient. The diagnosis requires laboratory studies, including the isolation and, if possible, propagation of the suspected organisms.

For the purpose of this section, we use the following definitions: *Meningitis* denotes inflammation of the meninges and is identified by an abnormal number of white blood cells (WBCs) in the cerebrospinal fluid (CSF). *Septic or purulent meningitis* denotes meningitis with evidence of a pathogen in the CSF. *Aseptic meningitis* denotes meningitis with no evidence of a bacterial pathogen detectable in the CSF by usual laboratory techniques. Most cases of aseptic meningitis have a viral etiology but also can be caused by rickettsiae, spirochetes, and other nonbacterial infections, as well as by noninfectious diseases such as systemic lupus erythematosus, multiple sclerosis, malignancies, and others. *Encephalitis* denotes inflammation of the brain. *Meningoencephalitis* denotes inflammation of the brain accompanied by meningitis.

### Bacterial Meningitis

#### EPIDEMIOLOGY

Bacterial meningitis is a serious childhood illness. Although *H. influenzae* type b (Hib) disease has been dramatically reduced in North America, northern Europe, and other countries like New Zealand ([Robbins et al., 1996](#)), it is still a leading cause of bacterial meningitis ([Salisbury, 1998](#)) in developing countries, with over 200,000 cases and more than 40,000 deaths occurring annually ([Mulholland et al., 1997](#); [Salisbury, 1998](#)). Moreover, *Neisseria meningitidis* and *Streptococcus pneumoniae* continue to be important pathogens ([Schuchat et al., 1997](#)). Recurring epidemics of meningococcal disease ([Booy and Kroll, 1998](#)), increased antibiotic resistance among pneumococci ([Goldstein et al., 1996](#)), and failure to introduce conjugate Hib vaccines in developing nations make bacterial meningitis a serious worldwide health problem.

In the United States, the most common cause of meningitis for all age groups older than 1 month is now *S. pneumoniae*, followed by *N. meningitidis*, b-hemolytic *Streptococcus*, and *Listeria monocytogenes* ([Schuchat et al., 1997](#)).

#### PATHOGENESIS

Regardless of the age of the child, most cases progress through four stages: (a) infection of the upper respiratory tract, (b) bacteremia from the respiratory focus, (c) seeding of the meninges by blood-borne bacteria, and (d) inflammation of the meninges ([Evans, 1987](#); [Weil et al., 2000](#)).

Inflammatory exudate then enters the subarachnoid space, leading to arteritis, arterial or venous thrombosis, and perineuritis, and in some cases communicating or noncommunicating hydrocephalus ([Evans, 1987](#); [Klein et al., 1986](#); [Weil et al., 2000](#)). The increased intracranial pressure or the acute hydrocephalus may reduce the perfusion of blood to the brain. The resulting changes in neuronal metabolism may cause edema or predispose to seizures. If the edema advances rapidly, it can lead to cerebral herniation and death. If the edema advances more slowly, for several days, glial proliferation occurs. The changes in nerve cells may be slight, or there may be widespread destruction of cortical cells and myelinated fibers. Dysfunction of cortical neurons, with altered mental status or seizures, occurs as much as several days before microscopic changes; this is the result not of bacteria in the substance of brain but of a metabolic or toxic encephalopathy ([Klein et al., 1986](#); [Weil et al., 2000](#)).

#### CLINICAL DIAGNOSIS

As a rule, findings in neonates and young infants are minimal and often subtle, making early diagnosis difficult to establish clinically ([Klein et al., 1986](#)). Infants may present with fever (50%), lethargy, respiratory distress, jaundice, lack of interest in feeding, vomiting, diarrhea, irritability (30%), poor muscle tone, alterations in consciousness, convulsions (40%), and a bulging fontanel (30%) ([Klein et al., 1986](#)).

In children, fever, headache, photophobia, nausea, vomiting, mental confusion, lethargy, and irritability are the usual initial complaints. These manifestations are nonspecific and are like those of nonmeningeal viral infections or other febrile illnesses. Stiff neck and other meningeal signs, seizures, and coma occur less commonly and usually later in the illness. However, when encountered, these are sufficiently characteristic of meningitis to prompt physicians to perform an immediate diagnostic lumbar puncture ([Klein et al., 1986](#); [Weil et al., 2000](#)).

The definitive diagnostic test of meningitis is the examination of the CSF by spinal tap. The routine CSF analyses are bacterial cultures, protein and glucose determinations, cell count, and differential Gram stain. If available, detection of capsular antigens using counterimmune electrophoresis or other techniques also can be helpful ([Klein et al., 1986](#); [Weil et al., 2000](#)).

#### NEUROLOGIC SEQUELAE

As much as 50% of the survivors of meningitis have some sequelae of their disease ([Evans, 1987](#); [Klein et al., 1986](#); [Weil et al., 2000](#)). Specific sequelae or complications of bacterial meningitis that have been observed include cranial nerve involvement, which often improves with time. However, 10% remain with hearing loss, hemiparesis or quadriparesis, muscular hypertonia, ataxia, mental retardation, permanent seizure disorders, or hydrocephalus. The syndrome of inappropriate antidiuretic hormone secretion develops in 30%, but this rarely is persistent or clinically significant. Subdural effusions develop in approximately 40% and occur so



frequently in young children that they can be considered as part of the general disease process rather than as a complication ( [Evans, 1987](#); [Klein et al., 1986](#); [Weil et al., 2000](#)).

### PSYCHIATRIC SEQUELAE

In general, studies that have used siblings as control subjects ( [Sell et al., 1972](#); [Taylor et al., 1984](#); [Tejani et al., 1982](#)) have found that children who survived Hib meningitis without major neurologic sequelae do not differ from the siblings on behavioral measures, although they had somewhat lower IQ scores.

[Taylor et al. \(1992\)](#) compared two groups of children with normal IQ who had recovered from complicated and noncomplicated Hib meningitis. The two groups had similar normal hearing and similar verbal IQs. The complicated group, however, had lower mean performance IQ and performed less well on perceptual neurologic tests. Parent and teacher ratings of behavior and frequencies of grade repetitions and special education placement, however, failed to distinguish the two groups.

More recently, a prospective, 7-year follow-up in New Zealand ( [Grimwood et al., 1995](#)) of children surviving bacterial meningitis and their classroom peers found that by school age, survivors had mildly decreased IQ scores and performed less well on a broad range of neuropsychological tests, with greatest impairment in verbal skills and organizational capacity ( [Anderson et al., 1997](#); [Davies, 1989](#); [Grimwood et al., 1995](#); [Taylor et al., 1990](#)). Compared with 11% of control subjects having minor disabilities, 27% of children surviving meningitis had either neurologic and behavior problems or cognitive impairments ( [Anderson et al., 1997](#); [Grimwood et al., 1996](#)). Risks for adverse outcomes were greatest in those with meningitis in infancy and in those who experienced delays in diagnosis or acute neurologic complications. Another follow-up of the same cohort and control subjects at 12 years of age found that differences in general intellectual, neuropsychological, and academic functions were maintained ( [Grimwood et al., 2000](#)). Lower-order skills improved, whereas behavioral scores as measured by the Achenbach CBCL deteriorated significantly in the meningitis group ( [Grimwood et al., 2000](#)).

Bacterial meningitis may constitute a risk factor for psychopathology in a significant minority of children; however, it should not by itself be presumed to affect negatively the behavioral outcome of any individual child. It seems that behavioral sequelae after bacterial meningitis are seen mainly in those cases with neurologic complications during the acute phase of the illness and may emerge later than cognitive sequelae ( [Grimwood et al., 2000](#); [Taylor, 1992](#)).

### Spirochetal Central Nervous System Infections

The major spirochetal infections of concern to child psychiatrists are syphilis and Lyme disease.

#### SYPHILIS

Syphilis is caused by the spirochete *Treponema pallidum*. The incidence of neurosyphilis, like that of CNS tuberculosis, declined dramatically with the introduction of antibiotics. In the United States, between 1943 and 1960, the rate of first admissions to psychiatric hospitals because of neurosyphilis fell from 4.3 per 100,000 to 0.4 per 100,000 ( [Adams and Victor, 1993](#)). Syphilis in the United States now is close to eradication. However, syphilis is worthy of review here, not only because it remains a problem in other countries, but because the clinical picture of general paresis has been considered exemplary of a chronic frontotemporal encephalitis superimposed on a chronic meningitis ( [Adams and Victor, 1977](#)).

Children may acquire syphilis through sexual contact or abuse, but most often are infected by transplacental inoculation from the mother at any time from the fourth to seventh month of gestation. Of the children of untreated syphilitic mothers, 25% to 80% contract syphilis; 2% to 16% of these children will acquire congenital neurosyphilis. Of those children in whom neurosyphilis develops, 50% to 80% will have one or more of the physical stigmata of congenital syphilis; these include interstitial keratitis; chorioretinitis; defective teeth (centrally notched mulberry molars; widely spaced, peg-shaped upper central incisors); abnormal facies (frontal bossing, saddle nose, poorly developed maxilla), bilateral knee effusions (Clutton's joints); and rhagades (linear scars at the angles of the mouth, nose, or anus) ( [Weil et al., 2000](#)).

The neuropathologic and clinical syndromes of pediatric neurosyphilis, whether congenital or acquired through sexual contact or abuse, are very similar to those seen in adults; all stem from a chronic spirochetal meningitis. The treponeme typically invades the CNS within 3 to 18 months after infecting the patient. The initial event in the neurosyphilitic infection is an asymptomatic meningitis that can be revealed only by lumbar puncture. This meningitis may remain asymptomatic until ultimately, after a period of years, it causes parenchymal damage, or it may spontaneously remit. However, as stressed by [Adams and Victor \(1977\)](#), all forms of neurosyphilis begin as a meningitis, and a more or less active meningitis invariably accompanies all the forms of neurosyphilis.

The early-appearing forms of neurosyphilis are congenital symptomatic meningitis and meningovascular syphilis. Syphilitic meningitis declares itself typically between 3 and 12 months of age with convulsions, listlessness, and other nonspecific symptoms; chronic meningitis may lead to hydrocephalus and, in some cases, retarded mental and motor development. Meningovascular syphilis declares itself somewhat later in the first months or years of life by a stroke with involvement of cerebrum, brain stem, and spinal cord, as well as diverse vascular syndromes due to arteritis and thrombosis of cerebral vessels ( [Weil et al., 2000](#)).

The later clinical forms of neurosyphilis begin 5 to 25 years after infection and include tabes dorsales, ameningoradiculitis, general paresis, and ameningoencephalitis. Tabes dorsales is rare in congenital syphilis. General paresis is the most common form of late congenital neurosyphilis. Histopathologically, it is characterized by filling of the perivascular spaces of the brain parenchyma with lymphocytes, plasma cells, and mononuclear cells and loss and degeneration of nerve cells with an increase in microglia and astroglia, deposition of iron, and presence of spirochetes in the cortex. The changes are most pronounced in the frontal and temporal lobes.

In congenital neurosyphilis, general paresis usually begins between 6 and 21 years of age, the average age of occurrence being 13 years ( [Friedland et al., 1995](#)). Girls and boys are equally affected. Approximately half the children affected by general paresis have been severely retarded since very early childhood, and in these cases, the onset of juvenile paresis is very difficult to date. In children who are mildly to moderately retarded or of normal intelligence, onset of general paresis is marked by loss of previous accomplishments. As noted by [Adams and Victor \(1977\)](#): "Silliness, forgetfulness, irascibility and inattentiveness are noteworthy behavioral abnormalities. There may be outburst of agitation and depression but rarely delusions of grandeur such as are observed in adult cases." In the late stage, the neurologic syndromes are very diverse: Approximately 25% of cases have conspicuous cerebellar deficits, 50% have limb spasticity, and up to 40% have seizures. Peculiar choreiform movements, twitches, and action tremors, pyramidal signs, and Argyll Robertson pupils are present ( [Weil et al., 2000](#)). Without treatment, the disease steadily progresses to death in 2 to 5 years ( [Friedland et al., 1995](#)).

Fortunately, neurosyphilis is highly treatable. Penicillin G is the drug of choice for treatment of all stages of neurosyphilis. Details of treatment are beyond the scope of this text. The reader is referred to the *Red Book* of the American Academy of Pediatrics ( [American Academy of Pediatrics, 2000](#)).

#### LYME BORRELIOSES

Lyme disease is caused by the spirochete, *Borrelia burgdorferi*. At least four genospecies are recognized worldwide, each with its own degree of neurovirulence ( [American Academy of Pediatrics, 2000](#)). The occurrence of cases in the United States correlates with the distribution and frequency of infected tick vectors, *Ixodes scapularis* in the East and Midwest and *Ixodes pacificus* in the West. The incubation period from tick bite to the first stage of the disease, erythema migrans, ranges from 3 to 31 days and typically is from 7 to 24 days. Late manifestations may occur months to years later. In the United States, Lyme disease has a bimodal age of distribution, with the highest incidence rates in children between ages 5 and 9 and adults older than 30 years ( [Pfister et al., 1994](#)). Lyme neuroborreliosis occurs once in every 620 infected children ( [Christen et al., 1993](#)).

The clinical manifestations of Lyme disease are divided into three stages: early localized, early disseminated, and late disease. In early localized disease, the site of recent tick bite gives rise to a unique skin lesion, erythema chronica migrans. Erythema migrans begins as a red macula or papule and enlarges over days to weeks to become a large, annular, erythematous lesion that is 5 cm or more in diameter, sometimes with a partial central clearing. The skin lesion can be accompanied by systemic systems such as fatigue (54%), myalgia (44%), arthralgia (44%), and fever, chills, or both (39%). However, the rash may not occur in approximately 10% to 20% of patients. Early disseminated disease typically manifests as multiple erythema migrans, reflecting spirochetemia with dermal dissemination. Neurologic complications of early disseminated illness occur in approximately 15% to 20% of patients, with or without the skin lesions, and include cranial nerve palsies, meningitis, and a broad range of other neuropsychiatric symptoms. In Europe, more than 80% of childhood neuroborreliosis manifests as facial palsy or an aseptic meningitis ( [Christen et al., 1993](#); [Hansen, 1994](#)), whereas in the United States, headache, stiff neck, and subtle encephalitic signs with less radiculopathy are more common ( [Reik et al., 1986](#)). A broad range of psychiatric manifestations may occur during early disseminated disease, including psychosis ( [Csaszar and Patafalvi,](#)

1994; [Stein et al., 1996](#)) and schizophreniform disorders ([Hess et al., 1999](#); [Pfister et al., 1993](#); [Roelcke et al., 1992](#); [van den Bergen et al., 1993](#)). [Fallon et al. \(1998\)](#) stress that Lyme disease may go undiagnosed in patients who present with atypical neuropsychiatric symptoms.

Late (third) stage disease is seen in untreated patients 1 to 3 years after the initial infection. It is marked by chronic arthritis that usually affects the large joints. Late disease occurs in 6% of children ([Christen et al., 1993](#)) and also is chronic. A wide spectrum of abnormalities also is seen. Although a destructive chronic meningoencephalitis can progress over many years in untreated individuals ([Bensch et al., 1987](#)), the most common neurologic complication is subacute encephalopathy characterized by cognitive deficits and disturbances of mood and sleep ([Ackermann et al., 1988](#); [Spach et al., 1993](#)). EEG changes, usually in the form of moderate generalized slowing, occur in one-third of children with late Lyme neuroborreliosis. Late disease is uncommon in children who are treated with antimicrobials in the early stage of the disease.

### Pathology

*Borrelia burgdorferi* appears in the CNS within days of the tick bite. Once in the CNS, a local inflammatory reaction may occur, resulting in meningitis or encephalitis, or the organism may remain dormant, causing illness months to years later ([Tager et al., 1999](#)).

The pathologic process of neuroborreliosis is characterized by an inflammatory thrombotic vasculopathy that can cause axonal degeneration of the peripheral nerves through involvement of the vasa vasorum, and causes ischemic lesions of the CNS ([Weil et al., 2000](#)). In fatal cases, large areas of demyelination in periventricular white matter as lymphocyte vasculitis have been found ([Oksi et al., 1996](#)). Cellular and humoral autoimmune mechanisms also are important, and the serum of infected patients contains immunoglobulin M (IgM) antibodies that cross-react with an axonal component ([Sigal and Tatum, 1988](#)).

### Neuropsychological Sequelae

Treatment within the first year of infection results in a more favorable clinical response than later therapy and probably reduces the incidence of neurologic and psychiatric complications ([Adams et al., 1994, 1999](#)). If not treated until well after the initial infection, children may be at risk for cognitive problems. [Tager et al. \(1999\)](#) found increased mood, attention, and learning problems in a subgroup of children with Lyme disease referred because of persistent cognitive complaints that developed concurrently with Lyme disease. Case studies have linked Lyme disease in children and adolescents with anorexia nervosa ([Pachner et al., 1989](#)); with obsessive-compulsive and panic disorder ([Fallon and Nields, 1994](#)); with psychotic features ([Neumarker et al., 1989](#)); and with Tourette syndrome ([Riedel et al., 1998](#)).

In adults, Lyme disease has been associated with impairments in memory, attention, mental activation, language, and motor functions ([Garcia-Monco and Benach, 1997](#); [Pollina et al., 1999](#); [Svetina et al., 1999](#)), and memory impairments may persist even after treatment ([Kaplan et al., 1999](#)). In a study of children with late Lyme disease, [Bloom et al. \(1998\)](#) found that the most prevalent neurocognitive symptoms were behavioral changes, forgetfulness, declining school performance, headache, and fatigue. General intellectual functioning was normal, but particular deficits related to auditory or visual sequencing were found.

### Diagnosis

The diagnosis of Lyme disease is based on clinical presentation supported by findings from serologic and CSF tests. For epidemiologic monitoring purposes, the [Centers for Disease Control and Prevention \(CDC\) \(1995\)](#) defined Lyme disease as the presence of erythema migrans rash 5 cm or larger in diameter, or laboratory confirmation of infection with *B. burgdorferi* and at least one objective sign of musculoskeletal, neurologic, or cardiovascular disease and a history of exposure to an endemic area. However, as stressed by Tager and Fallon (2001), these criteria were not intended nor are they adequate for clinical diagnostic purposes, for several reasons: Approximately one-third of patients do not remember a rash; encephalopathy, one of the most common signs of late neurologic Lyme disease, is not on the CDC list of objective neurologic signs; and a sizable proportion of patients do not have an adequate immune response to *B. burgdorferi* and therefore have negative results on routine serologic antibody tests. Therefore, the clinical history is particularly important and should address the main signs and symptoms as well as exposure. The differential diagnosis includes viral infections, rheumatoid arthritis, primary psychiatric disorders, connective tissue disorders, thyroid disorders, sleep disorders, other central or peripheral neurologic disorders, fibromyalgia, and chronic fatigue syndrome ([Tager and Fallon, 2001](#)).

The diagnosis is made most reliably by demonstrating the presence of IgM antibodies to *B. burgdorferi* or by polymerase chain reaction. Positive results from Western blot and enzyme-linked immunosorbent assay strongly support the diagnosis of Lyme disease ([Weil et al., 2000](#)). However, the child psychiatrist must work with local experts on Lyme disease regarding diagnosis and treatment.

### Treatment

For treatment of early localized disease, oral antibiotics such as doxycycline are recommended for children older than 8 years of age and ampicillin for children younger than 8 years of age. Early Lyme disease usually is treated for 14 to 21 days, but the optimal duration of treatment is not known. Oral treatments also are recommended for some forms of early disseminated and late disease, but CNS disease is treated with parenterally administered antibiotics such as ceftriaxone or penicillin. The reader is referred to the *Red Book* for more detailed discussion and guidelines ([American Academy of Pediatrics, 2000](#)).

If the cause of a neuropsychiatric disorder is active infection, then antibiotic therapy is necessary in addition to pharmacologic therapy for symptoms ([Logigian et al., 1999](#)). As reviewed by [Nields and Fallon \(1998\)](#), psychopharmacologic strategies include the use of selective serotonin reuptake inhibitors for irritability, carbamazepine and gabapentin for neuropathic pain, and methylphenidate, modafinil, and bupropion for fatigue and attention problems.

### Viral Central Nervous System Infections

The incidence of viral meningitis ranges, in different years, from 1.5 to 4 cases per 100,000 population. The incidence in children actually is much higher because aseptic meningitis also is a disease of the young (CDC, 1977). The incidence of viral meningoencephalitis is greater than that of bacterial meningitis in children, although the morbidity usually is less than that of bacterial meningitis. The major exception is infection due to herpes virus, the morbidity and mortality of which is very high ([Evans, 1987](#); [Weil et al., 2000](#)). The most common mode of viral CNS infection is hematogenous. The virus enters the CNS through the choroid plexus and then enters the CSF by replication in the capillary endothelium of the brain. The other route of CNS inoculation is contiguous spread through the peripheral nervous system, as in the case of herpes virus infection or rabies. Injury occurs by the process of viral replication and by vascular injury, which causes destruction of neurons and glial cells. The pathologic process of viral encephalitis is manifested by vascular mononuclear cells and neuronal destruction. In approximately 60% of cases, a specific virus can be identified by either culture or serology ([Evans, 1987](#); [Weil et al., 2000](#)).

The clinical spectrum of viral CNS infections ranges from aseptic meningitis to encephalitis to chronic infections and postinfectious neural illnesses. The clinical picture of aseptic meningitis is similar to that of bacterial meningitis. Chronic infections are discussed later, and postinfectious diseases (e.g., Guillain-Barré syndrome) are described in [Chapter 61](#).

### NEUROLOGIC AND PSYCHIATRIC SEQUELAE

Enteroviral meningitis accounts for at least 50% of cases of viral meningitis in the United States. Most of the patients are infants younger than 1 year of age, and most of the infections are mild ([Evans, 1987](#); [Weil et al., 2000](#)). Enteroviral meningitis had been reported to produce a low frequency of adverse neurologic, cognitive, or language sequelae ([Bergman et al., 1987](#); [Sells et al., 1975](#)). [Bergman et al. \(1987\)](#) followed 33 8-year-old survivors of enteroviral meningitis that occurred when the children were between the ages of 4 months and 1 year and compared them with 31 siblings. None of the survivors had a major neurologic sequela, and they performed as well as the control subjects on the cognitive and behavior tests administered.

Similarly, [Farmer et al. \(1975\)](#) followed for 6 years 15 infants with meningoencephalitis due to coxsackievirus B5 and compared them with 15 control subjects matched by age, sex, socioeconomic status, birth weight, and gestational age. There was no difference in the IQ and visual perception tests between both groups.

Some studies followed children for various periods of time after California arbovirus encephalitis ([Chun et al., 1960](#); [Gunderman and Stamler, 1973](#); [Matthews et al., 1968](#); [Rie et al., 1973](#); [Sabatino and Cramblet, 1968](#)) and found an incidence rate of between 0% and 40% for such neurologic complications as soft signs, seizures, or hemiparesis, some nonspecific behavior difficulties, and nonsignificant depressed IQs. By contrast, other studies ([Earnets et al., 1971](#); [Finley et al., 1953](#)) have reported high rates of neurologic morbidity (up to 69%) and mortality in children who had Western equine encephalitis, especially in those children younger than 1



year of age. In general, studies of morbidity after viral infections are flawed by methodologic problems that limit the generalization of their findings.

## Human Immunodeficiency Virus and Acquired Immunodeficiency Syndrome

Human immunodeficiency virus (HIV) type 1 is a retrovirus that causes AIDS, a severe condition characterized by infections with opportunistic microorganisms. The immunologic deficit is associated with viral infection of certain cell populations of the immune system, essentially T4 lymphocytes and macrophages. AIDS in children was first reported in 1983, when it manifested as an acute, lethal disease that involved the brain in up to half of cases (Epstein et al., 1986). Treatment advances have transformed HIV infection into a subacute chronic disease. As a result, psychiatric concern with respect to HIV infection must encompass not only the neuropsychiatric complications of brain involvement but coping with chronic illness and loss, often under conditions of severe psychosocial stress and behavioral aspects of prevention (Brown et al., 2000). For further discussion of some aspects of HIV, see [Chapter 98](#).

### EPIDEMIOLOGY AND ROUTES OF TRANSMISSION

Worldwide, more than 1 million children are known to be infected with HIV ([UNAIDS, 1998](#)). Best estimates suggest that three times that number are in fact infected. In the United States, there are more than 2,000 children younger than the age of 13 years with AIDS, and AIDS is now the sixth leading cause of death among 15- to 24-year-olds. Approximately 80% of pediatric AIDS cases are due to vertical (mother-to-child) transmission, 20% to transfusion of blood or blood products, and approximately 1% to sexual contact in adolescents ([Aicardi, 1992](#)).

In vertical transmission, perinatal infection probably is the most common route of infection ([Oxtoby, 1990](#)). Approximately one-fourth of infants born to an untreated HIV-infected mother also are infected ([European Collaborative Study, 1994](#)). Prenatal and perinatal administration of zidovudine has been shown to reduce transmission of HIV by over half ([Wade et al., 1998](#)). However, the pros and cons of mandatory HIV testing continue to be hotly debated ([Nakchbandi et al., 1998](#)). HIV also can be transmitted in breast milk, but many factors influence transmission risk, including the age of the child, the severity of the maternal HIV infection ([Dunn et al., 1998](#)), vitamin A deficiency ([Greenberg et al., 1997](#)), and specific blood types ([Tess et al., 1998](#)).

Among adolescents, sexual intercourse accounts for most new AIDS cases, with same-sex contact accounting for the preponderance of risk among male adolescents and heterosexual contact among female adolescents (CDC, 1997). A small percentage of cases is attributable to sexual abuse ([Stolar and Fernandez, 1997](#)).

In the United States and internationally, rates of infection are increasing most rapidly among adolescents and among heterosexual women. Despite increasing infection, advances in therapies have led to 70% of children with HIV living to the age of 6 years and 50% to the age of 9 years (Tovo et al. 1992). In children with perinatally acquired infections, the risk of encephalopathy is higher in those with AIDS (up to 23%) than in those with asymptomatic infection (approximately 10%) ([Lobato et al., 1995](#)); the estimated median survival after diagnosis of encephalopathy is 22 months.

### PATHOPHYSIOLOGY OF BRAIN INFECTION

Productive HIV-1 infection of the brain probably is limited to macrophages, microglia, and astrocytes; HIV rarely has been documented to infect neurons. The most commonly cited mechanisms of brain tissue damage have been related to mediators of the inflammatory response to HIV-1, an indirect pathophysiologic effect ([Goodkin et al., 2001](#)). HIV is carried into the brain by infected monocytes that differentiate into macrophages in brain tissue. The macrophages in effect become a storehouse of virus because, unlike in the CD4 lymphocyte, HIV-1 does not have a cytotoxic effect on these cells. Over time, in these cells, evolution toward strains associated with greater neurovirulence or cytotoxic T-lymphocyte escape may occur. These macrophages become nonspecifically activated and secrete substances, such as cytokines and chemokines, that are part of the normal inflammatory process at greater-than-normal concentrations. Among the proinflammatory cytokines are tumor necrosis factor- $\alpha$  associated with demyelination; interleukin-1 associated with neuron cell loss; and interferon- $\gamma$  associated with quinolinic acid, which is excitatory at NMDA receptors ([Goodkin et al., 2001](#)). Other indirect mechanisms include the effects of viral products on neurons and other cells of the brain ([Johnson, 1998](#)).

In children, as in adults, the encephalopathy associated with HIV-1 appears to be largely subcortical. Autopsy findings are characterized by inflammatory cell infiltrates, multinucleated cells, microglial nodules, white matter pallor, and calcification mainly in the vessels of the basal ganglia and deep cerebral white matter ([Dickson et al., 1989](#); [Falloon et al., 1989](#)). Studies of viral load and strain in the brains of adults support the concept that subcortical regions specifically are affected by HIV-1 infection ([Goodkin et al., 2001](#)).

### NEUROCOGNITIVE SEQUELAE

Among HIV-infected children, three patterns of abnormal neurocognitive development have been described ([Belman, 1992](#)). The first is a rapidly progressive encephalopathy characterized by impaired brain growth (secondary microcephaly), motor weakness with pyramidal tract signs that progresses to spastic quadriplegia, pseudobulbar signs, and loss or plateauing of developmental milestones ([Mintz et al., 1996](#)). Among HIV-infected children, progressive encephalopathy has an estimated prevalence of 13% to 23% ([Lobato et al., 1995](#)). The second is a subacute progression of encephalopathy with relatively stable periods, and the third is a static encephalopathy with a failure to achieve new milestones (Gay et al., 1995). Regular neurologic and psychometric assessments are recommended to differentiate these patterns further. Tardieu et al. (1995) found that among 133 survivors, only 67% had normal school achievement at a mean age of 9.5 years, 54% had impaired performance on visual-spatial function and time orientation tests, and 44% had speech or language delay or articulation disorders.

In a sample of 87 previously untreated children with HIV infection, Brouwers et al. ([Woods, 1998](#)) found the overall CT brain scan severity rating was highly predictive of the level of cognitive functioning. There was a higher rate of CT abnormalities (cortical atrophy, ventricular enlargement, white matter changes) in vertically infected children compared with transfusion-infected children, and intracerebral calcifications were seen only in the vertically infected group. In a study of 40 children with symptomatic HIV infection, high levels of the neurotoxin quinolinic acid in the CSF were correlated with encephalopathy, decreased performance on IQ test, and increased mortality and inflammatory neurologic disorders ([Brouwers et al., 1993](#)). In a sample of 36 HIV-infected children younger than 10 years of age, CT scan abnormalities were significantly correlated with poorer receptive and expressive language functioning, and expressive language was more severely impaired than receptive language among those with encephalopathy ([Wolters et al., 1995](#)).

Antiretroviral drugs inhibit or halt the progression of HIV and thus improve most indices of general health in pediatric AIDS. In addition, these drugs improve cognitive deficits in children with AIDS, at least for a limited period ([Pizzo et al., 1988](#); [Wolters et al., 1994](#)). [Brady et al. \(1996\)](#), in the largest study to date with children 3 months to 12 years of age, found an improvement in cognitive abilities over a 3-year period; improvement was greatest for patients 6 years of age or older. A number of smaller studies have demonstrated improved adaptive functioning with antiretroviral treatment ([Brivio et al., 1991](#); [Brouwers et al., 1994](#); [Moss et al., 1994](#)). Motor skills, however, do not appear to improve with antiretroviral therapy ([Wolters et al., 1994](#)).

### PSYCHOPATHOLOGY ASSOCIATED WITH HUMAN IMMUNODEFICIENCY VIRUS INFECTION

In the only study of psychiatric diagnoses of HIV-positive adolescents, 44% presented with current major depression, 85% had at least one Axis I DSM-IV diagnosis as determined by structured interview, and 53% had a history of psychiatric diagnosis before diagnosis of HIV infection ([Pao et al., 2001](#)). Diagnosis of a mood disorder also can be complicated by physiologic factors. For example, a child with AIDS who is withdrawing because of physical pain may be incorrectly diagnosed as depressed ([Stuber, 1992](#)). Almost 60% of children with HIV experience pain, which may negatively affect their quality of life and sleep patterns ([Yaster and Schecter, 1996](#)).

Several controlled studies suggest that HIV-infected pediatric patients experience more distress than noninfected peers. [Havens et al. \(1994\)](#) found that a group of 26 HIV-infected children with prenatal drug exposure scored significantly higher on the internalizing, somatic problems and depression/anxiety scales of the Achenbach CBCL than did uninfected children with prenatal drug exposure; this relation withstood control for age, race, and IQ. In addition to symptoms of anxiety and depression, some parents report more hyperactivity and conduct problems in their HIV-infected children ([Moss et al., 1994](#)). Another study that compared HIV-positive children with hemophilia to HIV-negative children with hemophilia and children with asthma found that anxiety disorders were more frequent in the HIV group than in the other two groups, whereas both groups of children with hemophilia were found to have lower levels of intrafamilial stress than the group with asthma ([Bussing and Burket, 1993](#)). [Brown et al. \(2000\)](#) speculate that low levels of intrafamilial stress in the hemophilia group result from the extensive support network of the National Hemophilia Program ([Cohen, 1994](#)).

## TREATMENT OF PSYCHOPATHOLOGY IN CHILDREN WITH HUMAN IMMUNODEFICIENCY VIRUS INFECTION

There are three main categories of antiretroviral medications: nucleoside analog reverse transcriptase inhibitors (NRTIs), nonnucleoside analog reverse transcriptase inhibitors (NNRTIs), and protease inhibitors (PIs). Each group has numerous side effects and special instructions on administration (CDC, 1997). The behavioral and cognitive side effects are poorly understood, and the pharmacokinetics and impact on the developing nervous system of these drugs are not known. NNRTIs and PIs are inhibitors of the cytochrome P-450 enzyme system; because this enzyme system is responsible for the metabolism of many commonly prescribed drugs, the potential for drug–drug interactions must be considered (Deeks et al., 1997). In general, these drugs are not recommended for concurrent use with antihistamines and sedative-hypnotics or amphetamines. The PI agents inhibit enzymes responsible for the metabolism of antidepressants and anticonvulsants. Drug interactions are particularly difficult to determine in complex protocols known as highly active antiretroviral therapy (HAART), in which multiple drug therapies are used in combination with a PI. The complex relationship of HAART to psychotropic medications due to shared degradative metabolic pathways is just beginning to be understood, and the clinical efficacy of multiple regimens is virtually unknown in children and adolescents (Gonzalez and Everall, 1998).

### COPING

In a metaanalysis of 38 studies investigating children's psychological and behavioral adjustment to various physical disorders, Lavigne and Faier-Routman (1993) found that parent and family risk factors were more strongly related to child adjustment than to specific disease and disability factors. The available literature on children and adolescents living with HIV infection and AIDS is consistent with this finding (Brown et al., 2000), which points to the importance of family-focused treatment. One study (Mellins and Ehrhardt, 1994) found that family-focused mental health services for children with HIV infection reduced isolation, improved family functioning, and encouraged the use of other services such as respite care.

Disclosure is an important issue confronted by all families with an HIV-infected child. The literature on chronic illnesses suggests that disclosure of the developmentally appropriate facts about illness improves psychological adaptation (Brown et al., 2000). Brown and DeMaio (Brown et al., 1992) describe two HIV-infected adolescents with hemophilia to illustrate how, if not properly managed, secrets concerning infection may interfere with optimal health care and the patient's subsequent emotional adjustment.

Many common HIV-related symptoms and physical limitations reported by young patients are emotionally based, but most of these patients are underserved (Ammann, 1994). Examples of programs that offer multidisciplinary services for HIV-positive children and adolescent are the 10 service projects of the Special Projects of National Significance Program (Woods, 1998).

### PREVENTION

The few studies on sexual risk behaviors in adolescents living with HIV suggest that both psychotherapeutic and psychoeducational approaches are appropriate (Henry, 1996; Lewis et al., 1994). A psychoeducational program was shown to improve the safety of sexual behaviors in adolescents with hemophilia (Brown et al., 1998). Although most programs targeting uninfected individuals strive to increase awareness of susceptibility to HIV and strengthen the desire for safety, more appropriate goals for infected adolescents are to increase empathy and the desire for responsible behavior (Brown et al., 2000).

### Slow Virus Infections

Slow virus infections of the CNS may be classified into those caused by unconventional agents (e.g., kuru, Jakob-Creutzfeldt disease) and those caused by conventional agents [e.g., subacute sclerosing panencephalitis (SSPE) due to measles virus, progressive rubella encephalitis, progressive multifocal leukoencephalitis (herpes simplex)]. Characteristically, slow viruses multiply in the host for prolonged periods without producing clinical symptoms. These viruses are not recognized as "foreign" by the host, and the immune response is either very late or absent. Therefore, the disease produced by a slow virus may be the result of either gradual destruction or an autoimmune response caused by termination of tolerance (Weil et al., 2000). All of these infections are characterized by progressive dementia and neurologic symptoms such as ataxia, involuntary movements, and seizures.

Subacute sclerosing panencephalitis is more likely to occur in children who had measles early in childhood. Its incidence ranges from 0.475 per million in urban populations to 1.4 to 1.8 per million in rural or suburban areas. It affects more boys than girls, in a ratio of 5:1.

Initial symptoms appear after a latency period of 4 to 15 years and are manifested by personality changes and deterioration in school performance, followed by myoclonic seizures, involuntary movements, dementia, and ataxia. The alterations in the brain consist of subacute encephalitis accompanied by demyelination lesions that usually involve the cerebral cortex, hippocampus, thalamus, brain stem, and cerebellar cortex. Characteristic EEG abnormalities may be seen as early as 4 years before the clinical appearance of myoclonus and are manifested by generalized paroxysmal bursts of high activity, with spike discharges followed by a short period of flattened activity–suppression burst pattern (Evans, 1987; Weil et al., 2000).

Rubella panencephalitis has been described in children with congenital rubella. It has an earlier onset than SSPE but has similar clinical features. Children with immunologic disorders may develop chronic enteroviral encephalitis or progressive multifocal leukoencephalitis, also manifested by dementia and movement disorders.

Kuru is a progressive degenerative disease of the CNS with predominantly cerebellar features; it is limited to a population in New Guinea that, until recently, practiced cannibalism (Evans, 1987; Weil et al., 2000).

### Congenital Infections of the Central Nervous System

Infections of the fetal CNS may cause significant damage and multiple defects. The extent and type of induction of congenital anomalies in the infected embryo depends on the time of action of the teratogen. After the second trimester, congenital malformations pursuant to congenital infections become increasingly uncommon. The main pathway of transmission of infection to the fetus is the transplacental route. Most of these congenital infections remain subclinical during the neonatal period (Menkes, 1990; Starr et al., 1970).

### RUBELLA

The frequency of congenital rubella after maternal infection with rash is more than 80% during the first 12 weeks of pregnancy, 54% at 13 to 14 weeks, and 25% at the end of the second trimester. When the CNS is involved during prenatal life, congenital malformations, including microcephaly, hydrocephalus, and spina bifida, may develop but may not be recognized until delayed motor development or speech retardation becomes obvious. Retarded language development may be due to peripheral hearing loss, focal CNS damage, or mental retardation. Most long-term survivors are deaf and have cataracts and chorioretinopathy. Severe mental retardation may occur in 24%, and spasticity is frequent, but most patients who have sustained severe neurologic damage die during the early years of life. Among the survivors, some children manifest limitations of language, cognition, and social relatedness similar to those found in autistic children (Chess, 1977; Desmond et al., 1969; Evans, 1987).

### CYTOMEGALOVIRUS

Cytomegalovirus (CMV) infection of the fetus is typically manifested at birth by persistent jaundice, hepatosplenomegaly, thrombocytopenia, anemia, and neurologic abnormalities. CMV frequently reaches the developing fetus when the mother is infected and may have important CNS sequelae. It has become clear that most newborns with CMV infections are asymptomatic at birth and remain so throughout infancy. However, Hanshaw et al. (1976) found that 10% of infected neonates had delayed psychomotor development and observed that impaired hearing may increase the number of these infants at subsequent neuropsychiatric risk. Reynolds et al. (1974) followed 18 asymptomatic CMV-infected children for 38 months and compared them blindly through IQ tests with normal control subjects. The mean IQ was 92 for the CMV carriers (seven had IQs < 90 and one > 32) and 100 for the control subjects. Hanshaw et al. (1976) followed 44 asymptomatic children with positive CMV titers in blood and compared them with a similar group of control subjects and a random sample of normal siblings. The children had psychometrics, school evaluations, and physical examinations at the ages of 3.5 to 7 years. The CMV group mean IQ was significantly lower, and they experienced failure at school 2.7 times more frequently than the matched control subjects and 8 times more frequently than the randomly selected siblings. Bilateral hearing loss was present in 1% of the CMV group. All children with IQs below 80 were positive for CMV and belonged to a low socioeconomic class. There was no school failure among the



CMV-positive children of higher socioeconomic status. The authors concluded that a child born with serologic evidence of congenital CMV infection is at higher risk for school failure if he or she is born into a family of a lower socioeconomic group.

## TOXOPLASMOSIS

Toxoplasmosis is caused by the protozoan *Toxoplasma gondii*, which is infectious to a wide range of birds and mammals. Pregnant women may acquire the infection by ingestion of oocysts in uncooked meat or from cat feces. As is the case for other congenital infections, chorioretinitis and neurologic symptoms may not be present at birth but may appear late in infancy or childhood. Rarely, toxoplasmosis may be responsible for seizures or psychomotor retardation. However, a 4-year follow-up of *Toxoplasma*-infected children with apparent neurologic disease in infancy disclosed mental retardation in 89% and seizures in 83% (Weil et al., 2000). Saxon et al. (1973) followed eight children 2 to 4 years of age who were identified at birth as having subclinical congenital toxoplasmosis and compared them blindly with eight normal children. There was no difference in assessments of behavior or motor development. The mean IQ for the untreated cases was 93, whereas for the control subjects and three children who were treated for toxoplasmosis, the mean IQ was 110.

## Conclusions Regarding Psychiatric Sequelae of Infectious Brain Injury

Children may experience permanent neuropsychiatric sequelae as a result of diverse congenital and acquired CNS infections. Children who were infected at an early age, did not receive prompt or appropriate treatment, were infected by certain virulent pathogens, or had focal seizures or residual neurologic deficits are at risk of remaining with greater degrees of mental retardation, school failure, and nonspecific behavior problems. This is especially so if they are from lower socioeconomic backgrounds, have independently inherited a predisposition for low-level intellectual functioning, or have a previous history of psychiatric disorder.

The neuropsychiatric problems emerging after CNS infections have not yet been adequately studied. Emerging emotional problems may be secondary to the CNS injury itself but may also involve mental retardation, deafness, language disorders, environmental or hereditary factors, or a combination of these. PC due to IBI has not been studied in the children. A multidisciplinary professional approach is recommended for the follow-up, remediation, and prevention of CNS infections and their neuropsychiatric complications.

## FINAL SUMMARY

It is now clear that severe forms of IBI and TBI increase the risk for psychopathology in children. In the near future, our understanding of the psychiatric sequelae of TBI and IBI is likely to be advanced rapidly by newer brain imaging techniques that may specify the circuits and neurotransmitters that have been affected. These methods will likely clarify whether specific patterns of behavior are related to site or extent of injury. As in the past, TBI and IBI are likely to contribute to our understanding of the brain substrates of psychopathology even in the absence of brain injury. In the meantime, the task for the child psychiatrist remains one of interpreting prognostic data for individual brain-injured patients and their families and planning interventions with the greatest likelihood of success. Children with prior histories of psychopathology, cognitive impairment, and family dysfunction warrant intensive support and continued monitoring, as do their families.

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# 34 TERATOLOGIC AND DEVELOPMENTAL EFFECTS OF PRENATAL DRUG EXPOSURE: ALCOHOL, HEROIN, MARIJUANA, AND COCAINE

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The study of behavioral teratology has a long-standing tradition beginning as early as Hippocrates' warning about "uterine suffocation" with maternal opiate use ([Hans, 1992](#); [Zagon and McLaughlin, 1984](#)) and the Old Testament advice to a pregnant woman to "drink not wine nor any strong drink" (Judg. 13:7, cited in Heath, 1991). These traditional and deep-seated cautions woven into the fabric of each culture's protection and valuing of a pregnancy have also fueled a long-standing scientific tradition—the investigation of the potential physical, acute, and long-term physiologic and immediate and long-term neurodevelopmental effects of prenatal exposure to drugs and other environmental substances. More than 20 agents have been shown to have teratogenic and postnatal toxic effects ([Levy and Koren, 1992](#)), whereas for many others, concern persists, but data are inconclusive. In many instances, science takes its initial cue from the prevailing public belief and fear about how a given drug or substance negatively affects its adult user and, thus, how detrimental it may be to the fetus. Because of this, commonly the first reports about the effects of prenatal exposure to a given drug or substance describe far more deleterious and severe outcomes than are true once larger and more heterogeneous samples are examined. That the first probands of any epidemic or new illness are often the most severe, most obvious, and least representative of the natural history of a condition is an observation not limited to behavioral teratology ([Day and Richardson, 1993](#)). Nonetheless, it is particularly relevant to considerations of drugs such as alcohol, opiates, or cocaine in which negative attitudes regarding adult substance abusers may also color both science and common belief regarding the developmental sequelae of maternal addiction during pregnancy ([Woods et al., 1998](#)).

The thalidomide tragedy notwithstanding, in most instances, demonstrating links in humans between prenatal drug exposure and immediate physical, neurologic, or later developmental and psychological outcomes is fraught with problems. These include determining frequency, dose, and duration of exposure and taking into account the conditions usually associated with substance abuse (e.g., poor nutrition, polydrug use, parental stress, and psychiatric illness) that also affect development. These associated conditions may either moderate the apparent direct effects of the drug on fetal neurologic development or serve as mediators for the relation between any neurologic sequelae and prenatal exposure ([Baron and Kenny, 1986](#)). Animal models provide some basis for comparison, particularly about physical effects, but they are less useful for studies of complex developmental capacities found only in higher-order primates and humans (e.g., language, complex problem-solving tasks, and neuropsychological functions such as certain domains of memory). (See, for example, [Stanton and Spear, 1990](#), for a comparison of findings in studies of animal models and prenatal cocaine exposure with findings in infants.) Moreover, although there is increasing literature for each drug about physical and behavioral teratologic effects in the infant and child related to maternal substance abuse during pregnancy (see later) ([Day, 1992](#); [Hans, 1992](#); [Mayes, 1992](#); [Streissguth, 1992](#); [Zuckerman and Frank, 1992](#)), far less systematic information is available about the effects of any one drug on parental or family functioning or the interaction between the effects of prenatal drug exposure on development and the effects of drug abuse on parenting. In the next section, we review what is known about the effects of alcohol, heroin, marijuana, and cocaine on fetal development and later behavior and developmental outcome. After that overview, we also discuss how substance abuse influences the caregiving environment.

## NEUROTOXICOLOGY OF PRENATAL SUBSTANCE ABUSE

Several crucial issues cut across all studies of prenatal drug exposure regardless of the specific drug being studied. Some of these issues are inherent to a longitudinal study design that examines the relationship between later and early events. All are particularly problematic in studies of prenatal exposure to drugs. (For more detailed methodologic reviews, see [Brooks-Gunn et al., 1994](#); [Griffith and Freier, 1992](#); [Lester et al., 1995](#); [Neuspiel, 1995](#).) These methodologic issues fall into the following broad areas: (a) definition and ascertainment of the independent exposure variable; (b) identification of crucial covariates often associated with substance abuse and exploring interactive as well as main-effects models; (c) definition of hypothesis-driven outcome variables; and (d) distinguishing between short-term and long-term effects, later effects not apparent early on, and transient effects that nonetheless may influence other developmental functions later.

### Definition of the Exposure Variable

Human models of prenatal drug exposure usually present complicated dilemmas regarding definitions of the exposure variable. This issue alone may be the single most problematic one in neurobehavioral teratologic studies. Substance abusers typically do not report consistently or reliably the frequency or amount of their drug use ([Babor, 1990](#); [Chasnoff et al., 1990](#); [Grissom, 1997](#); [Weiss et al., 1998](#)). Various strategies have been devised to improve the reliability of self-reports of substance use including use of time lines, careful training of interviewers, and narrow windows for retrospective recall ([Callahan et al., 1992](#); [Carey, 1997](#); [Rogers and Kelly, 1997](#); [Richardson and Day, 1994](#)). Even with these more sophisticated interviewing strategies, self-report of single or polydrug use typically, although not uniformly ([Richardson et al., 1993](#)), underestimates the amount of exposure particularly of illicit drugs.

Frequency of exposure obtained through self-report histories is usually expressed as a number of days per unit time (e.g., per month, use in last 30 days, use per week). Self-reports are typically, although not universally, augmented with toxicologic sampling of urine for drugs such as cocaine, marijuana, or opiates. Repeated toxicology screening throughout pregnancy may provide some confirmation or identification of users, and, not uncommonly, toxicologic screens are obtained from both infant and mother at the time of delivery. Urine toxicology testing provides a relatively narrow window on use. For example, for cocaine users, a urine toxicology test is typically positive no longer than 36 hours after use, and that window varies for other drugs. For cocaine, infants' meconium and hair (infant's or mother's) have gained some support as particularly good samples to ascertain or confirm infant exposure because they provide a longer window for ascertaining exposure. Some data suggest that meconium or hair from the newborn may be a reliable measure of exposure as far back as the middle of the first trimester of pregnancy ([Callahan et al., 1992](#); [Graham et al., 1989](#); [Kline et al., 1997](#); [Ostrea, 1995](#)). However, despite early enthusiasm for these types of longer window measures and despite their obvious utility, they do not provide a reliable quantitative estimate of exposure.

With these various problems in obtaining accurate estimates of frequency and amount of exposure, most studies of prenatal exposure to date have defined the independent exposure variable as dichotomous—exposed or not exposed. Grouping all exposed infants and children together obscures potential dose-related effects, and including those only minimally exposed in the exposed group may reduce the likelihood of detecting exposure effects. Thus, growing numbers of studies are attempting to create some metric of heavy, moderate, and light drug use to examine dose-related effects that follow either linear or nonlinear models ([Frank et al., 1998](#); [King et al., 1995](#); [Tronick et al., 1994](#)).

Route of use presents a third problem in defining the severity or amount of exposure. Although the total amount is always an important metric in defining severity of exposure, amount of time above a certain peak blood level may also be important in some models of teratogenicity. Stated another way, the teratogenic effect is carried not by total amount of exposure time but only by those times when the level of exposure is above a certain threshold. Certain aspects of fetal alcohol effects may follow this threshold rather than linear dose-related model. Blood levels peak at different levels following use depending on the preferred route of use. Intravenous use, as with heroin, and smoking crack, with rapid absorption through the pulmonary vascular bed, provide rapid and large peak blood levels to both mother and fetus. Few to no studies, particularly of cocaine, in which the routes of use may be quite varied, have examined differences in outcome depending on preferred method of use.

A fourth problem in the definition of exposure variables is polydrug use. Rarely do addicts use one drug only. Although they may consider one drug of abuse their

primary drug, polydrug use and exposure are the rule rather than the exception. For example, for cocaine users, a very typical combination is alcohol and tobacco in combination with cocaine. The same issues of defining frequency and amount of use for each drug pertain, but also there are questions of interactive effects among drugs such as alcohol with cocaine and the resulting metabolite cocethylene.

A fifth problem specific to studies of prenatal exposure is obtaining reliable estimates of frequency and amount of exposure by trimester of pregnancy. Different drugs have different effects during the three trimesters of pregnancy. For example, in the first trimester, prenatal cocaine exposure may have a direct effect on neuronal migration and brain structure formation, whereas in the third trimester, the central nervous system (CNS) effect may be on synaptogenesis in specific brain regions (Dow-Edwards et al., 1988; Frank et al., 1998; Mayes and Bornstein, 1995). Related to breaking down exposure by trimester is continued exposure postnatally. Particularly among agents that may be inhaled passively (e.g., crack, tobacco, marijuana), postnatal exposure is relatively common (for example, Bender et al., 1995; Kjarasch et al., 1991; Lustbader et al., 1998).

### Identification of Covariates and Development of Interactive Models

Substance abuse of one or multiple drugs rarely occurs isolated from other developmentally salient variables. Drug abuse is often associated with other factors that contribute to poor fetal health and infant outcome apart from or interactive with the specific teratologic effects of any one agent. Women who are chronic alcohol, heroin, marijuana, or cocaine abusers often fail to seek prenatal care and are themselves in sufficiently poor health to compromise the growth and well-being of the fetus. Thus, among pregnant women who are substance abusers, associated complications include preterm deliveries and infants who are intrauterine growth retarded or small for gestational age. The difficulties of caring for preterm infants or those who are small for gestational age are well documented because these infants often have labile states, and interaction with them is difficult (Watt, 1990; Watt and Strongman, 1985), problems that will likely be compounded if the substance-abusing environment is chaotic and inconsistent. Additionally, substance abuse often defines a postnatal environment associated with serious risks to infants' and children's development. These factors include severe poverty, virtual homelessness, early histories of abuse and neglect, chronic and acute violence, and multigenerational substance abuse with resulting parental isolation, and lack of family support systems (see also later).

It is conceptually and statistically naive to discuss all these events simply as covariates of prenatal substance exposure. Although they usually occur in association with substance use, they may or may not be related to the outcome of interest. Conversely, many of these variables probably serve as either mediators or moderators of the relation between prenatal exposure and later neurocognitive outcome (Baron and Kenny, 1986; Frank et al., 1993). For example, infants exposed prenatally to cocaine (see also later) appear more likely to have disorders of arousal or emotional regulation (e.g., Mayes et al., 1998). The strength of the relationship between prenatal cocaine exposure and the expression of this particular vulnerability may be mediated by the quality of postnatal care the infant and young child receives.

Considering mediating and moderating variables also brings up the issue of interactive as well as main-effect models. Traditionally, behavioral teratology studies have relied on main-effect conceptualizations: How much of the variance of a given neurodevelopmental outcome is explained by the prenatal exposure status? Although this is important and a first step, most developmental questions relating to prenatal exposure are probably better addressed as interactions. For example, as already cited, interactions between caregiving and prenatal exposure may more explain more of the variance in neurodevelopmental outcome than main effects alone. Interactions among a given genetic predisposition (e.g., for attentional disorders), exposure to drugs *in utero*, and postnatal neglect define another cluster of effects that are both more generalizable than single-effect exposure models and more biologically plausible.

### Definition of Outcome and Windows of Effect

Outcome variables and timing of outcome measurement vary widely in traditional behavioral teratology studies. Traditional models of teratology address both categories of outcome and window of effect (e.g., Abel, 1989; Vorhees, 1989). Typically, teratologic outcomes are expressed as CNS effects (including neurochemical, neuroanatomical, cognitive, behavioral, social-emotional); physical growth deficits (including brain growth); and malformations, and these outcomes are typically more manifest as the dose of exposure increases. At lower doses, CNS effects may still be apparent even in the absence of morphologic changes. Some effects on the CNS may only be manifest at later stages of development. These are often referred to as *latent effects* or *sleeper effects*, and examples of these are found both in studies of prenatal alcohol and marijuana exposure (e.g., Day et al., 1994). Certain developmental periods mark periods of neurologic reorganization (e.g., puberty, early school age, later half of the first year) and often prove to be important times to look for either latent or exacerbated effects of prenatal exposure. Within latent effects are those functional impairments that are apparent only under stressful, challenging, or novel conditions. In the work on cocaine in animal models, a considerable body of evidence has accumulated that this pattern may be particularly relevant to the neurotoxicity of cocaine (Spear et al., 1998).

Just as profiles of first cohorts may present a picture of more severe impairment, so are the initial outcome variables of study often more globally defined and less linked to hypotheses about pharmacologic action of the drug. Besides physical or morphologic impairment, neurobehavioral studies of prenatal drug exposure have most often focused on global measures of intelligence and general developmental functions such as memory, school performance, and incidence of maladaptive behavior. More recently, studies of *in utero* drug exposure have begun to use more functional measures such as reaction time and to focus on individual components of more general functions (e.g., visual versus auditory attention). Outcome measures such as these may be more reflective of hypotheses that are directly linked to understanding the site of action of the drug in the CNS.

Finally, it is important to make explicit that potential teratogens may exert their effects through different mechanisms. Most often assumed is a direct effects model in which the teratogen directly impairs a specific area of function in the CNS or otherwise. (Even claiming a "direct" effect on CNS is far too global a statement that does not allow for the remarkable complexity of any effect on developing neural tissue. The levels of effect in the CNS span direct toxicity to developing cells, impaired synapse or connectivity, to facilitated or impaired induction of genes that, in turn, regulate neural development.)

A second, less often explicitly discussed model is one in which the potential teratogen contributes to a domain of vulnerability that is expressed or not, depending on environmental conditions. A third model particularly relevant to conditions in which the potential teratogen is also a drug of abuse is that the drug itself may not be teratogenic for the developing child. Rather, in this third model, it may be that the context of drug abuse so alters the child's caregiving environment that any presumed teratogenicity is actually expressed through the effects of environmental chaos and deprivation.

## PRENATAL ALCOHOL EXPOSURE

Studies of the teratologic effects of prenatal alcohol exposure have been ongoing for many years since the initial reports of fetal alcohol syndrome (FAS) (Jones and Smith, 1973; Jones et al., 1973). Alcohol acts as a direct neuroteratogen affecting not only fetal facial morphology and growth but also brain growth, structure, and function through mechanisms not yet elucidated (Goodlett and West, 1992; Schenker et al., 1990). In addition to the direct effects of ethanol, prenatal alcohol exposure may also affect the fetus indirectly through maternal undernutrition and placental dysfunction and the teratogenicity of acetaldehyde, the product of ethanol oxidation (Shibley et al., 1999).

In infancy, FAS is characterized by the following: (a) intrauterine growth retardation with persistent postnatal poor growth in weight or height; (b) a pattern of specific minor physical anomalies that include a characteristic facial appearance; and (c) CNS deficits including microcephaly, delayed development, hyperactivity, attention deficits, intellectual delays, learning disabilities, and in some cases, seizures (Claren and Smith, 1978; Smith, 1982). The characteristic facial features include microphthalmia, short palpebral fissures, a thin upper lip, midface hypoplasia, and a smooth or long philtrum. Children with a history of *in utero* alcohol exposure who have either the characteristic physical appearance or CNS dysfunction are given the diagnostic label of having fetal alcohol effects (Claren and Smith, 1978). In the general population, the syndrome occurs in approximately one or two live births per 1,000, and the incidence of FAS among women with alcoholism is between 2.5% and 10% (Sokol et al., 1980). Even in the absence of FAS, infants born to mothers with *alcoholism* show an increased incidence of intellectual impairment, congenital anomalies, and decreased birth weight (Aronson et al., 1985; Day, 1992; Sokol et al., 1980). Partial expression of FAS and the issue of fetal alcohol effects have led to certain studies relating amount of exposure to the presence or absence of diagnostic criteria and to the severity of the CNS manifestations.

Nonetheless, hundreds of reports of children with FAS are now available detailing the delayed development in the first 2 to 3 years of life of children exposed to alcohol prenatally (e.g., Coles et al., 1987; Gusella and Fried, 1984; O'Connor et al., 1986). However, significantly fewer studies describe follow-up findings through school age and adolescence (Streissguth, 1992). Coles and colleagues (1991), studying children at age 70 months who were exposed to alcohol throughout gestation, reported deficits in sequential processing and on some measures of academic skills including reading and mathematics. Streissguth (1976) reported on a 7-year follow-up of 23 children of alcoholic mothers compared with 46 children of nonalcoholic mothers matched for socioeconomic status, age, education, race parity, and marital status. At 7 years of age, children of the alcoholic mothers had significantly lower intelligence quotient (IQ) scores and poorer performance on tests of reading, spelling, and arithmetic, and 44% of the children of alcoholic mothers, compared with 9% in the control group, had IQ scores in the borderline to retarded range. Significant differences in height, weight, and head circumference were also apparent. In a study of 21 children of alcoholic mothers, again compared with a



matched control sample, [Aronson and colleagues \(1985\)](#) described significantly greater problems with distractibility, hyperactivity, and short attention spans in the alcohol-exposed group. Similar behavior problems have been described for other cohorts (e.g., [Steinhausen et al., 1982](#)), and deficits in balance ([Roebuck et al., 1998](#)), as well as impairments in concentration and attention, social withdrawal, and conduct problems, continue to be described for adolescents and young adults ([Streissguth et al., 1991](#)).

Alcohol use also has an impact on the effects of other pregnancy risks such as prematurity and polydrug use. Preterm infants of women who used a high, not moderate, level of alcohol during pregnancy suffered an increased risk of developing brain hemorrhage and white-matter damage ([Holzman et al., 1995](#)). In addition to the effects of alcohol alone, more and more studies are exploring the effects of polydrug use. Numerous studies have reported that most women who use cocaine also use alcohol ([Brown et al., 1998](#)). One study compared cocaine-polydrug users who used and who did not use alcohol and found that alcohol did not account for a significant amount of the variance on birth weight, birth length, ponderal index, or irritability ([Brown et al., 1998](#)). In addition, [Brown and associates \(1998\)](#) reported that length of gestation moderated the effects of cocaine-polydrug use and alcohol-only use on fetal growth variables and irritability of infants, such that growth deficits were more pronounced in later-born infants, whereas increases in irritability were more evident in earlier-born infants.

In addition to the direct effects of prenatal ethanol exposure, the consumption of alcohol after a woman knows that she is pregnant serves as a marker for other possibly harmful behaviors. One study found that adolescent girls who continued to drink in the second and third trimesters of their pregnancies had a higher incidence of other high-risk behaviors such as smoking cigarettes and marijuana use, and they also had more prior pregnancies and less adequate prenatal care ([Cornelius et al., 1999](#)). Conversely, adolescents who stopped drinking after discovering their pregnancy had more prenatal care visits, fewer depressive symptoms, and parents with more education. These young women did not suffer the same ill effects as those mothers who continued to drink. This finding suggests that a low level of alcohol consumption for a brief period during pregnancy may carry less risk for the mother if she has access to a more advantaged environment and more psychological resources ([Cornelius et al., 1999](#)).

## PRENATAL OPIATE EXPOSURE

In contrast to those exposed to alcohol, newborns who have been exposed prenatally to opiates (heroin or methadone) are born passively addicted to the drug and exhibit withdrawal symptoms in the first days to weeks after delivery ([Desmond and Wilson, 1975](#)). Numerous studies have now also replicated the finding that prenatal opioid exposure reduces birth weight and head circumference ([Finnegan, 1976](#); [Hans, 1992](#); [Jeremy and Hans, 1985](#); [Kaltenbach et al., 1987](#); [Wilson et al., 1981](#)). Similar findings in animal models that control for exposure to other drugs such as alcohol or tobacco and for poor maternal health support the finding of an opiate effect on fetal growth ([Zagon and McLaughlin, 1984](#)). Prenatal exposure to opiates also contributes significantly to an increased incidence of sudden infant death syndrome. In some studies, the incidence of sudden infant death syndrome is eight times that reported for non-opiate-exposed infants ([Finnegan, 1979](#); [Hans, 1992](#); [Rosen and Johnson, 1988](#); [Wilson et al., 1981](#)).

On neurobehavioral assessments in the newborn period, opiate-exposed infants are more easily aroused and are more irritable ([Jeremy and Hans, 1985](#); [Marcus and Hans, 1982](#); [Strauss et al., 1976](#)). They exhibit proportionately less quiet compared with active sleep and show increased muscle tone and poor motor control (e.g., tremulousness and jerky movements). Opiate-exposed infants are less often in alert states and are more difficult to bring to an alert state. The dramatic neurobehavioral abnormalities seen in the newborn period generally diminish over the first month of life ([Jeremy and Hans, 1985](#)) for most infants and are, thus, assumed to reflect the transitory symptoms of narcotic withdrawal rather than evidence of permanent neurologic dysfunction ([Hans, 1992](#)).

Past the neonatal period, some studies have documented small, and not usually statistically significant, delays in the acquisition of developmental skills as measured by Bayley's Scale of Infant Development ([1969](#); [Hans, 1989](#); [Hans and Jeremy, 1984](#); [Rosen and Johnson, 1982](#); [Wilson et al., 1981](#)). However, much more consistent and significant across studies have been the findings of persistent problems in poor motor coordination, high activity level, and poor attention among opiate-exposed infants in the first year of life ([Hans and Marcus, 1983](#); [Hans et al., 1984](#)). These state and motor regulatory difficulties make it hard for even a well-functioning adult in a relatively nonstressed environment to care for the infant and present significant problems for an opiate-addicted adult experiencing his or her own state and attentional regulatory problems ([Hans, 1992](#)).

Follow-up studies through early childhood of opiate-exposed compared with non-opiate-exposed children have continued to report few to no differences in cognitive performance ([Kaltenbach et al., 1987](#); [Strauss et al., 1976](#); [Wilson et al., 1979](#)). However, opiate-exposed school-aged children show higher activity levels, are often impulsive with poor self-control, show poor motor coordination, and have more difficulty with tasks requiring focused attention ([Olofsson et al., 1983](#)). There is also an increased incidence of attention deficit disorder among opiate-exposed school-aged children ([Hans, 1992](#)). Two studies have described altered sex-dimorphic behavior in opiate-exposed boys ([Sandberg et al., 1990](#); [Ward et al., 1989](#)). Opiate-exposed boys showed more stereotypically feminine behavior than non-opiate-exposed boys, but there were no differences between opiate-exposed and non-opiate-exposed girls. These findings are consistent with similar observations of male rats exposed to opioid drugs *in utero* ([Ward et al., 1983](#)).

Past the years of early childhood, there are few studies of the long-term effects of prenatal opiate exposure, and those available usually lack a non-opiate-exposed control group or are not based on a longitudinal design ([Hans, 1992](#)). The data from these studies suggest that, by adolescence, opiate-exposed children exhibit an increased incidence of behavior and conduct problems including impulsivity, involvement in criminal activities or in early substance abuse, more antisocial behavior, and increased school dropout rates ([Bauman and Levine, 1986](#); [Sowder and Burt, 1980](#); [Wilson, 1989](#)). It is not altogether clear how much these problems in conduct and impulse regulation are attributable to persistent effects of prenatal opiate exposure and how much they are the consequence of cumulative exposure to the discord and dysfunction often characterizing substance-abusing households.

## PRENATAL MARIJUANA EXPOSURE

After alcohol, marijuana is the most commonly abused drug in the United States, and, like alcohol, marijuana abuse cuts across different socioeconomic groups and strata. *Marijuana*, also known as cannabis, is obtained from the flowering tops of the hemp plant from which more than 300 natural compounds including at least 61 different cannabinoids are extracted. Of these, tetrahydrocannabinol (THC), or marijuana, is the most potently psychoactive ([Levy and Koren, 1992](#)). THC readily crosses the placenta and, among heavy users, is also concentrated in breast milk ([Blackard and Tennes, 1984](#); [Perez-Reyes and Wall, 1982](#)). THC has a strong affinity for lipids and is stored in fatty tissue throughout the body ([Kruetz and Axelrod, 1973](#)). Thus, a single dose of THC in humans has a half-life of 7 days, but it may take up to 30 days to be excreted completely and accumulates throughout the body with chronic use ([Nahas, 1976](#)).

The rate of women reporting marijuana use during pregnancy varies from 5 to 34% ([Zuckerman, 1988](#)). During pregnancy, THC has documented effects in animals and humans on pituitary ovarian function, prolactin secretion, and uterine contractility ([Harclerode, 1980](#)). However, no relation has been documented between marijuana use and length of gestation or birth weight ([Fried et al., 1983](#)). Birth weight reductions associated with marijuana use have been described by others studying higher-risk, lower-income families ([Hingson et al., 1989](#)), and the results are conflicting ([Zuckerman et al., 1989](#)). Marijuana has an indirect effect on fetal oxygenation through the high levels of carbon monoxide found in marijuana smoke, levels higher than in cigarette smoke ([Wu et al., 1988](#)), which, in turn, result in fetal hypoxia. This type of effect may influence fetal growth, particularly in instances of heavy marijuana use ([Zuckerman and Frank, 1992](#)).

Few physical anomalies have been reported with marijuana exposure ([O'Connel and Fried, 1984](#); [Rose et al., 1982](#)), although several studies have suggested a link between prenatal marijuana exposure and features similar to those of FAS ([Hingson et al., 1989](#); [Qazi et al., 1982](#)). In one study, the incidence of FAS-like features was estimated to be five times higher in users of THC. It is quite likely, however, that heavy marijuana users are also abusing alcohol, and thus, the similarity to fetal alcohol effects is more likely to reflect the accompanying alcohol use ([Fried et al., 1984](#)). Several neurobehavioral findings in the newborn period point to decreased responsiveness on the Brazelton Neonatal Behavioral Assessment Scales (NBAS), particularly in visual, but not auditory, responsiveness to both animate and inanimate stimulation ([Fried, 1980](#); [Fried, 1982](#)) and a higher-pitched cry ([Fried and Makin, 1987](#)). Another characteristic of newborns exposed to heavy maternal THC use are tremors and increased startle in the first 7 to 14 days of life ([Levy and Koren, 1992](#)). Changes in sleep patterns have also been reported, including a decrease in the amount of trace alternans quiet sleep ([Scher et al., 1988](#)) and lower sleep efficiency and maintenance as measured by sleep electroencephalography by as late as 3 years of age ([Day and Richardson, 1991](#)).

Postnatally, marijuana has been identified in the urine of breast-fed infants whose mothers continue to use the drug after delivery ([Perez-Reyes and Walls, 1982](#)). However, no acute toxic effects have been identified with this level of passive exposure, although a few studies suggest possible developmental effects related to heavy postpartum exposure through breast milk ([Zuckerman and Frank, 1992](#)). In one study, marijuana exposure through breast milk in the first postpartum month was related to decreased motor development at 1 year, and there appeared to be a dose-related pattern to the level of association between exposure and motor delay ([Astley and Little, 1990](#)). Longer-term studies of the outcome of prenatal marijuana exposure are few. In one study, no association was found between prenatal marijuana use and developmental scores at 12 and 24 months ([Fried and Watkinson, 1988](#)). When these children were 4 years of age, heavy prenatal use (more than



six joints per week) was associated with lower scores on memory and verbal subscales of standard preschool intelligence tests. These findings pertained in comparison with the scores of children whose mothers had not used marijuana and after controlling for factors such as the home environment ([Fried and Watkinson, 1990](#)), although by age 5 and 6 years, these differences in IQ score had diminished ([Fried et al., 1992a](#)). In contrast, a second study ([Streissguth et al., 1989](#)) found no correlation with IQ scores at age 4 years. Behaviorally, children exposed to marijuana prenatally may show increased impulsivity and difficulties with sustained attention ([Fried et al., 1992b](#)). By early adolescence, there is a suggestion that those children exposed prenatally to marijuana may have more difficulty with complex visual processing problems ([Fried and Watkinson, 2000](#)), although further follow-up through adolescence is warranted.

## PRENATAL COCAINE EXPOSURE

Since the mid-1980s, many investigations have focused on the potential physical, neurodevelopmental, and neuropsychological effects of prenatal cocaine exposure on infants and young children. These studies have used a variety of designs including longitudinal, cross-sectional, case-control, and retrospective cohorts, have defined in multiple ways the independent variables, that is, the amount and duration of cocaine as well as other drug exposure, and have emphasized a host of different outcomes. (For a review of central methodologic issues in studies of prenatal cocaine exposure, see [Brooks-Gunn et al., 1994](#); [Frank et al., 1998](#); [Lester et al., 1995](#); [Mayes and Fahy, 2001](#); [Neuspiel, 1995](#)). Although still inconsistent or inconclusive on many crucial issues and marked by methodologic problems, published studies to date nonetheless reveal the beginnings of a profile of possible cocaine-related effects on neuropsychological functions subserving arousal and attention regulation and reactivity to stressful conditions. (For reviews, see [Frank et al., 1993](#); [Mayes and Bornstein 1995](#); [Mayes et al., 1998](#); [Richardson et al., 1993](#); [Singer et al., 1992](#).) That profile is further elaborated by findings from several animal models in which important factors such as duration and type of exposure as well as environmental conditions may be more adequately controlled ([Dow-Edwards, 1991, 1993](#); [Spear, 1995](#); [Spear, 1996](#); [Spear et al., 1998](#)). In particular, studies of prenatal and preweaning exposure in the rat point to direct effects of cocaine on the dopaminergic-mediated circuitry that is particularly important in arousal regulation and attentional reactivity ([Dow-Edwards et al., 1997](#); [Friedman and Wang 1998](#); [Friedman et al., 1996](#); [Howard et al., 1997](#); [Minabe et al., 1992](#); [Wang et al., 1995](#); [Weese-Mayer et al., 1993](#)).

Most often, infants exposed prenatally to cocaine are also exposed to other risk factors that may contribute to impaired development ([Mayes, 1992](#)). These include exposure to other substances of abuse including alcohol and tobacco, as well as opiates, marijuana, and amphetamines. Mothers who abuse cocaine often have associated health problems including a higher incidence of human immunodeficiency virus–positive titers with or without acquired immunodeficiency syndrome–related illnesses, and they have pregnancies more often complicated by preterm delivery and intrauterine growth retardation. Postnatally, infants exposed to cocaine continue to be exposed to ongoing parental substance abuse, they are more often neglected and abused, and they have parents with more frequent depression and higher overall levels of stress and anxiety ([Mayes, 1995](#)). Any one of these factors may also influence the development of early attentional and arousal regulatory functions, later language, and potentially overall developmental competence.

There are several candidate mechanisms proposed for the effect of prenatal cocaine exposure on the ontogeny of neural systems and, by extension, for cocaine-related neurobehavioral profiles ([Mayes et al., 1998](#); [Olsen, 1995](#)). These are effects on monoaminergic system development, changes in neural growth factors, alteration of ion channel and monoamine transport development, effects on other neurotransmitter systems such as neuropeptides including substance P, dynorphin, g-aminobutyric acid and glutamate sites, and alteration of immediate early gene expression ([Kosofsky et al., 1995](#); [Nestler et al., 1997](#); [Olsen, 1995](#)). Cocaine also has potent vasoconstrictive effects that may have a more generalized and less system-specific hypoxia-related deleterious effect on neural growth. The nonspecific but frequent mixture of acute and chronic stress characterizing the postnatal caregiving environments for many prenatally exposed children may also have enduring effects on attention and arousal system ontogeny perhaps through some of the same monoamine-related mechanisms.

At pharmacologically active doses in mature animals, cocaine inhibits the reuptake of monoamines (norepinephrine, dopamine, and serotonin) at the presynaptic junction. This leads to increased concentration of these neurotransmitters in the synaptic cleft and higher levels of activation in the central catecholaminergic systems ([Gawin and Ellinwood, 1988](#); [Wise, 1984](#)). Presynaptic depletion and a compensatory increase in synthesis parallel the increase in postsynaptic transmitter levels. Concomitant up-regulation of postsynaptic receptors may also result in supersensitivity to catecholamines ([Nunes and Rosecan, 1987](#)). Monoamine cell groups in the brain stem and striatum and presynaptic transporter proteins to which cocaine binds appear early in fetal development ([Choi and Ronnekleiv, 1996](#); [Lauder and Bloom 1974](#); [Pickel et al., 1980](#); [Verney et al., 1991](#)). Monoamines play critical trophic roles through all phases of CNS ontogeny—cell proliferation, neural migration, growth, and maturation, and synaptogenesis ([Lauder, 1983](#); [Lauder, 1988](#); [Mattson, 1988](#)). Additional suggestive evidence for the role of monoamines and their receptors in fetal brain development is that some receptors for serotonin and dopamine appear to be overexpressed in the immature brain and expressed in regions where they are absent in the adult ([Miller and Friedhoff, 1986](#); [Seeman et al., 1987](#); [Whitaker-Azmitia, 1991](#)). (Indeed, as a general principle, this observation of early overexpression is true of many components of the developing nervous system.) Thus, it seems reasonable to hypothesize that cocaine's effects on the developing nervous system may be mediated in part through effects on monoamine system ontogeny and monoamine function at the level of neurotransmitter and receptor synthesis, receptor sensitivity and binding, and monoamine system projections to other regions. These effects at a cellular, structural, and functional level may also be expressed behaviorally in disrupted patterns of arousal and attention regulation, given that these domains are connected intimately to monoaminergic systems.

In fetal brain development, dopamine, serotonin, and norepinephrine play critical roles in defining brain structure and neuronal formation by influencing cell proliferation, neural outgrowth, and synaptogenesis ([Lauder, 1988](#); [Mattson, 1988](#)). Cocaine readily crosses the placenta, as well as the blood–brain barrier, and brain concentrations of cocaine have been reported as high as four times those of peak plasma levels ([Farrar and Kearns, 1989](#)). Thus, cocaine may affect the formation and remodeling of brain structures through this effect on the release and metabolism of monoamines. Additionally, cocaine may influence the actual ontogeny of the neurotransmitter systems and, thus, again may modify certain critical processes in brain development. In prenatally cocaine-exposed animal models, several structures associated with mesocortical dopamine activity, including the cingulate cortex and the ventral tegmental area, as well as the ventral thalamic nucleus, show significant changes in dopaminergic activity compared with that in controls ([Dow-Edwards, 1989](#); [Dow-Edwards et al., 1988](#)). Effects on developing monoaminergic neurotransmitter systems have wide-reaching implications because they may lead to mistimed neurogenesis between the affected and unaffected areas of the brain with resultant changes in synaptic connections ([Lauder, 1991](#)). By altering monoaminergic neurotransmitter control of morphogenesis, chronic exposure to cocaine *in utero* may adversely affect autonomic function, state regulation, and regulation of attention in the developing nervous system.

The effect of cocaine on fetal development may also be expressed through the norepinephrine-related effects of cocaine on vascular tone. These consist of decreased uteroplacental blood flow, severe uteroplacental insufficiency (acute and chronic), maternal hypertension, and fetal vasoconstriction ([Moore et al., 1986](#); [Woods et al., 1987](#)), in turn, resulting in a relative state of fetal hypoxia. Moreover, in humans, cocaine use has been associated with spontaneous abortion, premature labor, and abruptions ([Bingol et al., 1987](#); [Cherukuri et al., 1988](#); [Lindenberg et al., 1991](#)). The effect of cocaine use on placental blood flow probably contributes to the relation between cocaine and fetal growth (low birth weight and microcephaly) reported by several investigators ([Fulroth et al., 1989](#); [Hadeed and Siegel, 1989](#); [MacGregor et al., 1987](#); [Mayes et al., 1993](#); [Oro and Dixon, 1987](#); [Ryan et al., 1987](#)). One report showed that crack-exposed infants were 3.6 times more likely to have intrauterine growth retardation than were infants born to non–drug-using women matched for age, socioeconomic status, and alcohol use ([Cherukuri et al., 1988](#)). Cocaine-exposed neonates may also show a greater effect on head growth, that is, the head circumference is disproportionately smaller than would be predicted by birth weight and gestational age ([Bateman and Chiriboga, 2000](#)). Additionally, because of the effect of cocaine on overall adult nutrition, compliance with prenatal care, and the usual association between cocaine use and use of other drugs such as alcohol, tobacco, and opiates ([Amaro et al., 1989](#); [Frank et al., 1988](#)), women using cocaine while they are pregnant are in an overall poorer state of health, which, in turn, increases the risk of impaired fetal outcome and fetal growth retardation. Intrauterine growth-retarded infants who are small for gestational age show persistent problems with irritability and distractibility well into the first year of life ([Watt, 1990](#); [Watt and Strongman, 1985](#)). A higher rate of congenital malformations in infants exposed to cocaine has also been reported ([Chasnoff et al., 1988](#); [Isenberg et al., 1987](#); [Teske and Trese, 1987](#)), but when various confounding factors are taken into account, only the relation between genitourinary malformations or spontaneous abortions and gestational cocaine exposure remains significant ([Lutiger et al., 1991](#)).

Behavioral and cognitive outcome measures beyond the neonatal period in studies of children exposed to cocaine prenatally have, for the most part, used general measures of developmental competence ([Mayes and Fahy, 2001](#)). On general measures of developmental competence such as the [Bayley Scales of Infant Development \(1969\)](#) that index information processing and indirectly attention, few differences are apparent between cocaine-exposed and non–cocaine-exposed infants ([Chasnoff et al., 1992](#)). The developmental profiles of a group of 106 cocaine- or alcohol-exposed 24-month-old children who were followed-up from birth were compared with the performance of 45 toddlers exposed to marijuana or alcohol but not cocaine and 77 non–drug-exposed children ([Chasnoff et al., 1992](#)). Mothers of infants in the two comparison groups were similar to the cocaine-using mothers in socioeconomic status, age, marital status, and tobacco use during pregnancy. On repeated developmental assessments using the [Bayley Scales \(1969\)](#) at 3, 6, 12, 18, and 24 months, albeit with a high rate of attrition from the original cohort, there were no mean differences in either the mental or motor domains. However, the investigators cautioned that a higher percentage of cocaine-exposed infants scored two standard deviations below the mean ([Chasnoff et al., 1992](#)). At least five other investigative groups have reported similar failures to find differences among cocaine-exposed groups on general measures of developmental competence in the first, second, and third years of life ([Anisfeld et al., 1991](#); [Arendt et al., 1993](#); [Billman et al., 1991](#); [Hurt et al., 1997](#)) and extending to no differences in IQ scores by early school age ([Wasserman et al., 1998](#)). Despite these apparent lack of differences in general measures of developmental competence, persistent motor delays at least through 2 years of age have been reported ([Arendt et al., 1999](#)).



Findings such as these have required a reevaluation of earlier concerns about global developmental delay in cocaine-exposed children. Conversely, as more children who were prenatally exposed have been evaluated in a variety of research and clinical contexts, more evidence is beginning to accumulate about impairments in specific functions such as neonatal habituation, attentional or arousal regulation, reactivity to novelty, and conditioned learning ( [Alessandri et al., 1993](#); [Coles et al., 1999](#); [Eisen et al., 1990](#); [Mayes et al., 1993](#); [Mayes et al., 1994](#); [Potter et al., 2000](#); [Struthers and Hansen, 1992](#)). Impairments in these domains would potentially make the normal parenting activities of contingent responsiveness and structuring attention more important for those prenatally cocaine-exposed infants who are more reactive and are easily overaroused ( [Mayes et al., 1994](#)).

In studies of newborns and 3- to 6-month-old cocaine-exposed infants, impairments have been reported in startle responsivity, auditory information processing, habituation, recognition memory, and reactivity to novelty. Persistent electroencephalographic abnormalities including lower spectral power have been reported through 1 year of age in cocaine-exposed infants ( [Scher et al., 2000](#)). [Anday and colleagues \(1989\)](#) reported that cocaine-exposed newborns are more reactive to reflex-eliciting stimuli as well as to specific auditory stimuli. Similarly, using measures of auditory information processing, deficits in habituation to auditory information were described for cocaine-exposed neonates; these same infants showed similar deficits in auditory habituation *in utero* ( [Potter et al., 2000](#)). In the neonatal period, findings of neurobehavioral impairments as measured by the Brazelton NBAS have been inconsistent. [Chasnoff and colleagues \(1989\)](#), among others ( [Datta-Bhutata et al., 1998](#)), reported impairments of orientation, motor, and state regulatory behaviors on the NBAS. In contrast, [Coles et al. \(1992\)](#) reported that NBAS scores for all infants fell within a clinically normal range regardless of cocaine or alcohol exposure, but they did not examine the habituation cluster for all infants. [Eisen and colleagues \(1990\)](#), studying neonates who were urine screen-positive only for cocaine at birth and whose mothers denied opiate use, and [Mayes et al. \(1993\)](#) found significant deficits in cocaine-exposed infants in habituation performance as assessed by the NBAS. No other aspects of neonatal performance were affected.

Links between the dopaminergic system and attentional mechanisms that are likely to involve information processing ( [Coles and Robbins, 1989](#)) make it plausible to hypothesize that prenatal cocaine exposure could affect the infant's early information processing performance as indexed by recognition memory or habituation procedures. [Struthers and Hansen \(1992\)](#) reported impaired recognition memory among cocaine or amphetamine-exposed infants compared with a non-drug-exposed group between 7 and 8 months of age. Similarly, [Alessandri and colleagues \(1993\)](#) reported delays in novelty responsiveness and conditioned learning well into the second half of the first year of life; cocaine-exposed infants also showed less positive affect during play with their mothers and a persistent negative response after the stressful still face procedure ( [Bendersky and Lewis, 1998](#)).

Recognition memory tasks rely in part on habituation processes, and although they measure infant responsiveness to novel versus familiar stimuli rather than decrement of attention over time, they nevertheless require an integrated capacity to attend selectively to novel information. [Mayes and colleagues \(1993\)](#) found that, compared with a non-drug-exposed group, infants exposed prenatally to cocaine were significantly more likely to fail to begin a habituation procedure and significantly more likely to react with irritability early in the procedure. However, most infants reached the habituation criterion, and among those who did not, significant differences emerged between cocaine-exposed and non-cocaine-exposed infants in habituation or in recovery to a novel stimulus. Thus, for at least a subgroup of cocaine-exposed infants, initial reactivity and selectivity toward novel stimuli appear impaired. As a similar finding, in older children, it has been reported that, despite no apparent differences on either motor or mental indices on the [Bayley Scales \(1969\)](#), cocaine-exposed 24-month-old children appear to have more difficulty attending to several objects at the same time and in structuring an approach to a nonfamiliar task on their own in the context of the developmental assessment ( [Hawley and Disney, 1992](#)). Using physiologic measures of arousal regulation (e.g., heart rate in response to novel or stressful stimuli), findings consistent with earlier behavioral observations describe dysregulated arousal and stress response systems (e.g., accelerated heart rate indicating stress instead of deceleration indicating attention) in cocaine-exposed infants ( [Coles et al., 1999](#); [Bard et al., 2000](#)).

Longer-term follow-up of cocaine-exposed children into school age is necessary to explore the implications of impairments in reactivity and attention and state regulation for later learning. A few studies are now available suggesting persisting arousal regulatory difficulties. For example, using the auditory startle response as a measure of stress response, [Mayes and colleagues \(2000\)](#) demonstrated an attenuated startle response in cocaine-exposed 4.5- and 5.5-year-old children. By early school age, cocaine-exposed children also appear to exhibit more disruptive behavior problems ( [Delaney-Black et al., 1998](#)).

However, as a caveat, establishing cause-and-effect relations between prenatal cocaine exposure and emotional regulation or attentional deficits is problematic, given the number of additional variables that may affect vulnerability for such neurodevelopmental outcomes. In addition to a direct effect on developing monoaminergic neurotransmitter systems in fetal brain, prenatal cocaine exposure may potentially affect attentional and state regulation indirectly because of effects on fetal growth, and, in humans, cocaine use is usually accompanied by at least alcohol use. Moreover, continued maternal postnatal use of cocaine affects the child's caregiving environment at two levels, and this may contribute to attention regulation. Adults who are under the influence of cocaine are less able to respond adequately to their children at any given time ( [Bauman and Dougherty, 1983](#); [Mayes, 1995](#)). The effects of cocaine on memory and attention impair the adult's ability to care for a child. More generally, because of the lifestyle adjustments necessary with cocaine use, including, for example, prostitution, crime, exposure to violence, and the overwhelming power of the addiction, the overall environment for these children is often chaotic, violent, and neglectful ( [Black and Mayer, 1980](#); [Regan et al., 1982](#)). Specific outcomes in children such as attentional regulation are also influenced by maternal interactive style ( [Bornstein, 1985](#); [Bornstein and Tamis-LeMonda, 1990](#); [Tamis-LeMonda and Bornstein, 1989](#)). Similarly, the psychological and personality factors that lead an adult to substance abuse may have genetic as well as experiential implications for the fetus. For example, attention deficits or chronic affective disorders in the adult, both of which may be partially medicated by cocaine ( [Khantzian, 1983](#); [Khantzian and Khantzian, 1984](#); [Rounsaville et al., 1982](#)), are associated with genetic risks for similar disorders in the child, and these disorders, particularly depression, also impinge on the adult's capacity to care adequately for the child ( [Fendrick et al., 1990](#); [Field, 1995](#)).

Thus, prenatal exposure to alcohol, heroin, marijuana, or cocaine may contribute to specific short-term and long-term impairments or vulnerabilities in arousal modulation, activity level, or attentional regulation, which may make it more difficult for an adult to parent the child. Moreover, when that adult is involved in substance abuse, his or her addiction and the associated environmental, psychiatric, and neuropsychological effects may further impair the interactions between the child and parent, as assessed through both indirect measures of the incidence of abuse and neglect and direct observational measures of parenting attitudes and behaviors.

## POSTNATAL SUBSTANCE-ABUSING ENVIRONMENT

Each of these presumed effects relating prenatal drug exposure to neurobehavioral and developmental dysfunctions must be viewed in the context of the postnatal substance-abusing environment in which many prenatally drug-exposed children remain. As already mentioned, the postnatal drug-using world carries certain risks to children's development. These include exposure to extreme, often chronic violence, virtual homelessness, poverty, parental neglect and abuse, and parental depression and associated psychopathology. Each of these factors, in turn, influences the parenting behaviors of adults who are also substance abusers.

Addiction to any substance (or condition) points to personality characteristics, disabilities, or impairments, each of which may have significant implications for an adult's ability to parent a child. Moreover, all substances of abuse alter, in varying degree, one's state of consciousness, memory, affect regulation, and impulse control and may become so addictive that the adult's primary goal is to be able to supply his or her addiction to the exclusion of all else and all others in his or her life. These alterations likely influence markedly at any given moment the adult's capacity to sustain contingent, responsive interactions with an infant and young child. For example, neuropsychological impairments in concentration and memory associated with chronic cocaine abuse ( [O'Malley et al., 1992](#)) may be expected to influence certain parenting behaviors such as the capacity to sustain an interaction.

There are differences in the behavioral and personality characteristics of substance-abusing adults according to the specific substance of abuse. Systematic studies of psychopathology among substance abusers find, for example, that abuse of cocaine versus opiates is associated with a different spectrum of psychological disorders ( [Khantzian, 1985](#)). Heroin addicts are generally considered a more psychiatrically deviant group than cocaine abusers ( [Rounsaville et al., 1991](#)), but there are higher incidences of drug abuse and alcoholism among the relatives of cocaine abusers than among the relatives of heroin addicts ( [Rounsaville and Luthar, 1992](#)). These factors influence treatment issues according to the specified drug of abuse and probably also affect the adult's parenting capacities. Moreover, abused drugs differ markedly in their psychological and physiologic effects on the user, and these effects, in turn, differentially influence the adult's capacity to respond to a child. Agents such as alcohol, marijuana, heroin, or antianxiety drugs such as diazepam (Valium) tend to depress mood, whereas stimulants such as cocaine or amphetamines increase activity and contribute to a sense of euphoria and elation. In either case, the adult's moment-to-moment responsiveness to children's needs is impaired, but in one case, the impairment is toward depression and withdrawal and in the other toward unpredictable activity and impulsivity. Although the distinctions are not absolute, because, for example, chronic cocaine abusers often experience depression and persons with alcoholism may be quite agitated, the child's experience will differ depending on whether the parent is predominantly withdrawn or unpredictably agitated. Moreover, as cited earlier, for some substance-abusing adults, the drug of choice may also in part indirectly reflect different preexisting conditions that the drug use may be intended to self-medicate ( [Khantzian, 1985](#); [Khantzian and Khantzian, 1984](#)). These conditions, such as depression or anxiety disorders, not only carry potential genetic risks for the child but also surely influence parenting in the domains of affective availability, capacity to foster the child's independence, and the parent's tolerance for the child's aggression.

The social context of the particular abused substance varies markedly, and these factors also indirectly influence parenting. Alcohol, when abused, poses major



health and psychological problems, but it is legally available, and its use is more socially acceptable for women and men than cocaine, heroin, or even marijuana abuse, all of which are illegal. Abuse of cocaine far more often involves the user directly or indirectly in criminal activities such as prostitution, theft, or drug dealing (Boyd and Mieczkowski, 1990), and it exposes the user, as well as his or her children, to personal and property violence. Because of these activities, cocaine-abusing adults are more likely to be arrested and incarcerated repeatedly, thereby exposing their children to multiple episodes of parental separation and placements usually with different foster families or with other (often substance-abusing) neighbors or relatives (Lawson and Wilson, 1980). Additionally, substance-abusing parents often report feeling more isolated and lonely, with few friends or relatives in their neighborhoods or immediate communities whom they identify as supportive and helpful (Tucker, 1979). Feelings of isolation and self-denigration may reflect both premorbid and postmorbid states related to the adult's substance abuse, but in any case, parents who experience isolation and separateness may be at greater risk of having problems in caring for their children, especially when their isolation is compounded by the psychological effects of their addiction. Multiple studies from substance abuse treatment programs also document the high incidence of unemployment and less than a high school education among participating substance-abusing women (Hawley and Disney, 1992). Among this population, the rate of unemployment has been shown as high as 96% (Suffet and Brotman, 1976). The level of violence in substance-abusing families, particularly between women and their spouses or male friends, is markedly high and exposes children to a considerable amount of witnessed violence (Regan et al., 1982). Studies have repeatedly documented the markedly increased occurrence of severe, often multigenerational, impairments in parenting among substance-abusing families as measured by the incidences of physical and sexual abuse, neglect, abandonment, and foster placement (Black and Mayer, 1980; Lawson and Wilson, 1980; Wasserman and Leventhal, 1993). Neglect and out-of-home placement are extremely common among the children of opiate-using adults. In a sample of heroin-exposed children followed through school age (Wilson, 1989), only 12% were living with their biological mother, 60% lived with extended family or friends, and 25% had been adopted. By their first birthday, nearly half (48%) of these children as infants were living away from their biological mothers.

There are surprisingly few direct observational studies of parent-child interactions among substance-abusing mothers and their children, and most of these have involved adults addicted to alcohol or opiates (Mayes, 1995). In 1985, Lief presented a series of clinical descriptions of interactions between mothers in treatment and their infants and toddlers. Described as points for intervention were the impoverished use of language between substance-abusing mothers and their infants, restriction of exploration that was seen as the infant's "getting into things" (Lief, 1985), and a diminished responsiveness to the infant's bids for social interaction. Few studies have systematically investigated the interactive behaviors between substance-abusing mothers and their infants. The measures employed have been quite variable both in the amount of interactive detail studied and in the aspects of interaction considered potentially impaired by substance abuse.

Most studies have reported impairments in certain interactive domains, although these differences have not always been attributed to substance abuse only. Householder (1980, cited in Hans, 1992), reporting on the interactions between opioid-using mothers and their 3-month-old infants, describes more physical activity, less emotional involvement with the infant, and less direct gaze toward the infant than non-opioid-using mothers. Opiate-addicted mothers tended either to withdraw completely from the interaction or to be persistently physically intrusive. In a study of 15 mothers in a methadone maintenance clinic compared with 15 non-opioid-addicted women interacting with 2- to 6-year-old children (Bauman and Dougherty, 1983), addicted women were more likely to use a threatening, commanding, or provoking approach to discipline and "to reinforce a disruptive method of attention seeking" (Bauman and Dougherty, 1983) in comparison with nonaddicted mothers, who relied more on positive reinforcement. The 2- to 6-year-old children of the substance-abusing mothers in that study also were significantly more provocative and complaining with their mothers.

Studies of attachment profiles among prenatally and postnatally substance-exposed children are, to date, few. Goodman (1990, cited in Hans, 1992) studied attachment patterns in 35 methadone-exposed and 46 nonexposed 1-year-old infants. Methadone-exposed infants more often showed disorganized (group D; Main and Solomon, 1986) or mixed insecure attachment patterns. Similarly, Rodning and colleagues (1989, 1991), studying 18 13-month-old children who were prenatally exposed to cocaine, phencyclidine, heroin, or methadone compared with 41 socioeconomic status-matched preterm children, showed that drug-exposed toddlers were more likely to be insecurely attached to their mothers, whereas most of the non-drug-exposed premature infants were securely attached. In addition, the drug-exposed children showed higher rates of disorganized attachment behaviors. In a similar study of maternal alcohol use (O'Connor et al., 1990, reported in Griffith and Freier, 1992), maternal interactions and maternal prenatal alcohol use significantly predicted infant attachment behaviors at 1 year of age.

Several studies have emphasized that, although the substance-abusing mothers had apparently more impaired interactions than comparison groups, some associated (e.g., comorbid) factors in addition to, or instead of, their substance abuse seemed to predict poor parenting. Jeremy and Bernstein (1984), reporting on the dyadic interactions of a cohort of 17 methadone-maintained women and their 4-month-old infants compared with 23 non-opioid-using mothers (Bernstein et al., 1984, 1986), found that drug use status alone did not significantly predict maternal interactive behavior. Instead, maternal psychological and psychosocial resources, as measured by assessments of maternal IQ and semistructured, diagnostic psychiatric interviews, were more predictive of the quality of the maternal-infant interaction than was drug use status. Indeed, maternal drug use, when analyzed together with other maternal variables, was not a significant predictor of mothers' interactive performance. However, coexisting maternal psychopathology contributes to greater impairments in parenting interactions among substance-abusing adults compared with non-substance abusers and with those substance abusers with no coexisting psychiatric disturbance. Hans and colleagues (Hans et al., 1990, reported in Griffith and Freier, 1992) reported that mothers using methadone who were also diagnosed as having antisocial personality disorders were significantly more dysfunctional in their interactions with their 24-month-old children than were methadone-maintained mothers who had either no significant psychopathology or affective disorders alone. Moreover, the latter group did not differ in their interactions from drug-free mothers. Findings such as these, albeit from small cohorts, point to the importance of not considering drug use alone as the single determining variable for observed differences in maternal interactive behaviors, but rather as a marker for several predictor variables that are more often associated with substance abuse (Mayes, 1995).

## CONCLUSIONS

The field of behavioral and developmental teratology focuses on identifiable effects of prenatal exposure to substances such as alcohol, heroin, marijuana, and cocaine. Children developing amid the violence, substance abuse, poverty, and discord increasingly common in inner city neighborhoods are at risk of dysfunctional development on several accounts. Although specific syndromes such as that associated with maternal alcoholism during pregnancy have been clearly identified, controversy and conflicting findings still pertain regarding long-term effects of any one of these agents on cognitive and intellectual development. Suggestive findings, particularly with prenatal cocaine exposure, point to impairments in more basic neurodevelopmental domains of arousal and attentional regulation, functions that underlie learning and information processing. However, the postnatal substance-abusing environment, both in its more general factors of poverty, homelessness, and violence and in the specific dysfunctions of parenting that accompany substance abuse, almost certainly exacerbates the effects of prenatal substance exposure.

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## 35 SEPARATION AND DEPRIVATION<sup>1</sup>

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The impact of separations from loved ones and of associated or unique deprivation experiences on children's development has been an issue for child psychiatrists and other children's specialists for many years. John Bowlby's groundbreaking investigations into the potentially psychopathologic effects of separation and his ultimate construction of attachment theory in the 1950s and 1960s directed attention to the developmental significance of separation experiences in the lives of young children. Although many of his colleagues at the time believed that childhood experiences could have lasting consequences on personality development, it was not believed that separation from or loss of the primary caregiver could be of particular long-standing importance, if other adults stepped in to care for the child ( [Colin, 1996](#)). Bowlby's work changed conventional views and stimulated an enduring interest in understanding and ameliorating adverse consequences of separation and of depriving experiences on infants and young children.

The clinical questions most basic to understanding separation experiences for children often address how these experiences are handled by adults responsible for the child, including both family and professionals. For example, how can separation experiences in the life of the young child be managed by adults, so that distress is lessened, and adaptive and defensive mechanisms that protect and facilitate development are evoked? How can parents and day care providers best help an infant or young child cope with the stresses inherent in being separated from parents for long hours day after day? How can the impact of separation on disabled or sick infants be lessened, or, similarly, how can hospitalization of a child be made less stressful for the child and family? When the separation includes placement of the child outside the family, what can be done to improve outcomes for children in the foster care system? How can knowledge of child mental health and development be more effectively communicated to influence decision making in child placement and custody cases? Is it possible to design residential group care programs for infants and young children that foster development and address individual needs, including therapeutic needs?

To be sure, issues of separation and deprivation are not the only considerations in the foregoing questions, but they are of central importance and exert a strong influence on recommendations for the care of children and on choice of intervention for a variety of service settings. The role of child psychiatrists and other child mental health professionals includes a wide range of possibilities, from public policy advisor to the more traditional roles of consultant, diagnostician, counselor, and therapist for the individual child and family. Crucial for each of these roles is an understanding of the meaning of separation both for an individual child and for the broader study of children's development.

Separation experiences are myriad in the lives of children and are a part of expected development. Allowing a parent to leave for work or saying good-bye at the door to school, sleeping overnight at a friend's house, and accepting care from another, less familiar, family member are examples of the many expected, indeed, under optimal circumstances, developmentally promoting, separations for the preschool and school-aged child. More potentially disruptive experiences include the loss of a parent through parental divorce or a severe debilitating illness or death. Similarly, separations that occur in the context of child abuse or neglect and subsequent foster placements impose multiple burdens on development in addition to the loss of a parent who may or may not have been the abuser.

Even this altogether too cursory typology illustrates that separation is not a single-dimension experience. The field has moved past the time when separation was regarded as an event that had a predictable effect whenever it occurred in the earliest years. Similarly, deprivation cannot be discussed as though insufficient nutrients, the condition of "not enough," had sufficient explanatory value. Such oversimplification disregards the complexity of brain and psychological development and the influence of developmental change on syndrome expression. It is known that a particular clinical disorder can be the result of multiple factors and that similar insults (e.g., deprivation and traumatic separation) may result in different outcomes. Any one separation experience is mediated by the developmental age and competence of the child, by the previous history of loss and separation for the child and family, and by how the family or responsible adults respond to the particular experience in terms of themselves and the child.

For example, separations occurring in the early preschool years have a potentially greater developmental impact than those that occur later. For children with biologically based vulnerabilities such as a sensitivity to novelty and transitions, even expected separations may impose a greater degree of stress than for another, less sensitive child. Similarly, for children with specific or general developmental delays, age-expectable separations (e.g., entry into preschool) may be experienced with a degree of anxiety and developmental stress more like that of a younger child. From the environmental side, how parents or caring adults help the child cope with the separation experience plays a crucial role in the child's short-term and long-term responses. In many circumstances, the adult most responsible for the child is unable, because of psychological and functional limitations of, for example, substance abuse, to mediate the experience for the child. Or if the adult caring for the child is also acutely affected, as by the death or illness of a spouse, the child may experience a double loss as his or her remaining parent is less available and is more acutely depressed. Situations of extreme family disruption, such as occur when families are displaced from their homes, impose not only the potential loss of cared-for persons but also the loss of familiar surroundings and routines. There is also the mediating factor of how well cared for the child has been up to the separation. Children from chronically depriving or abusing environments are at much greater risk of having severe developmental sequelae of a separation experience. Thus, separation involves more than a single type of experience, either for an individual child or family or for any group of children at a given developmental age or life circumstances. On this and related notes of caution, selected currently recognized constructs about effects of separation and deprivation follow.

### CHANGING DEFINITIONS AND CONCEPTS

In respect to separation, current concepts include consideration of separateness, that is, becoming a separate person, and separation events, which occur across the life cycle ([Bloom-Feshbach and Bloom-Feshbach, 1987](#)). Coping with experiences of separation is a lifelong challenge, not only inevitable but necessary for healthy



development ([Provence, 1987](#)). In childhood, some separation experiences facilitate psychological growth and personality organization by mobilizing new opportunities for learning and adaptation. Others, especially those involving loss of important persons, precipitate states of confusion, anger, anxiety, and grief and are painful and traumatic. Between these two extremes are many separation experiences involving varying degrees of psychological and psychobiological stress; adaptive and defensive mechanisms are called into play with varying degrees of effectiveness, and feelings of mastery or helplessness are aspects of these responses.

Traumatic separation and deprivation often, but not always, go hand in hand. The condition of deprivation, for many years called "maternal deprivation," has most often been seen as the lack of adequate physical care and of social and emotional stimulation and interchange. As investigators and clinicians looked more closely at specific components found in the environments of infants who were developing well and, by contrast, what was missing in the experience of those who were not doing well, and as we have become more aware of the impact of sensory deficits such as deafness and blindness, *deprivation* has been recognized as encompassing other conditions as well. *Experiential deprivation* is a more useful and accurate term than maternal deprivation to express the variety of "not enough" situations that characterize the lives of many children. In addition, multiple risk factors often operate and are cumulative in their impact. [Yarrow \(1961\)](#), in a landmark article, outlines more rigorous and productive methodologic approaches to the study of maternal deprivation and to a conceptual reevaluation of clinical and research studies on the subject. The literature on maternal deprivation has included the following: children residing in institutions for prolonged periods; those only temporarily separated because of illness of child or parent; those cared for by several different persons and, thus, enduring multiple separations; and children grossly neglected or mistreated by their families who experience not only the lack of adequate nurturance in the positive sense but also the trauma of insensitive, intrusive, or hostile treatment by parental figures.

Complex mixtures of experiential deprivation may be encountered. Brain insults and congenital or acquired disabilities, prematurity, and neonatal illness increase the probability that an impoverished environment will have an adverse effect on development ([Shonkoff and Marshall, 1990](#)). Sensory deficits such as deafness and blindness create a situation of needs requiring special environmental response in aspects of child care and education ([Fraiberg, 1977](#); [Freedman, 1981](#)). When sensory deficits are multiple or combined with motor disabilities, vulnerability to the influence of deprivation is heightened. When parents are markedly dysfunctional, however, even the most biologically resilient infant will be at risk for later problems.

## THEORIES OF PROCESS AND MECHANISMS

How does one understand what happens to the young child who experiences traumatic separation or deprivation? There is no unitary theory of psychological development that can be called on to guide diagnostic, therapeutic, and preventive efforts. Nonetheless, empirical data derived from research, practice, and theory from developmental psychologies are organized into sets of constructs. Such systematic efforts permit the type of interaction of clinical experience, data of observation, and theoretical propositions that leads to the refinement of each.

In the following discussion, aspects of attachment theory, psychoanalytic theory, and cognitive psychology are presented because of their contributions to the most current thinking about separation and deprivation. The distress of the young child at separation and the adverse effects of deprivation are understood in part by each. Clearly, one cannot here do full justice to any one of them, but a guide to the salient outlines of each theory is provided.

### Contributions from Attachment Theory

Bowlby's *attachment theory* employs an evolutionary perspective that draws on ethologic principles and animal behavior and combines these with aspects of psychoanalytic theory to understand the formation of human social bonds. The infant's survival mechanisms and the adult's nurturing tendencies "bias" them to behave reciprocally and to form an attachment. *Attachment*, as defined by [Bowlby \(1969\)](#) and as used by Ainsworth and colleagues ([Ainsworth, 1964](#); [Ainsworth et al., 1978](#)), is an affectional tie that forms with another specific person, a tie that endures over time and ultimately becomes a part of an individual's psychic structure.

The biological function of the attachment relationship is protection. Early in life, an infant's biologically rooted attachment behaviors become organized into a goal-directed system with proximity to the attachment figure as the goal ([Colin, 1996](#)). Activators of the system often are external conditions (e.g., frightening events, strangeness, and particularly distance or separation from the attachment figure), but they also may include internal conditions (e.g., fatigue, illness, or pain). Attachment behaviors include a repertoire of actions (e.g., distress, reaching, following) that serve ultimately to bring the infant or child into proximity and contact with the attachment figure. The resulting contact and proximity are system terminators. Although the fundamental nature of this system remains the same throughout childhood, its external character is quite variable. The particular conditions that activate the attachment system, the intensity of activation, the types of attachment behavior used by the infant or child, and the degree of proximity or contact necessary to terminate activation differ with the developmental level of the child and also with the quality of the attachment relationship ([Colin, 1996](#)).

An important result of the attachment interactions between caregiver and child is the internalized social-cognitive model that the child ultimately constructs of close relationships. This "internal working model" ([Bowlby, 1969](#)) is defined as the child's psychological representation of self and of key caregivers, and their relations. It is an important form of mental structure or representation that guides and regulates human functioning and permits, for example, the mental representations of attachment figures to be accessible and emotionally available in time of need and of the self as worthy and lovable.

Distress at separation from an attachment figure is intensified when the young child is placed on a long-term basis in a strange environment and is cared for by a succession of strange persons. A typical sequence of protest, despair, and detachment ensues. Robertson and [Bowlby \(1952\)](#) first defined these three major phases. *Protest* is a phase of angry, loud, tearful behavior during which the child looks for the mother and expects her return. The second phase, *despair*, is one of acute pain and diminishing hope, when there is usually a subdued manner and little interest in the surroundings. In the third or *detachment* phase, the child shows renewed interest in the environment and may be quite friendly and outgoing to others, but specific reactions to the mother, when she reappears, such as ignoring and walking away, suggest that detachment serves a defensive function. The three types of responses are seen as phases of a single process. Acute anxiety recurs when there is fear or anticipation of losing the mother again. The onset of psychiatric conditions, in Bowlby's view, is linked to premature detachment that masks "strong residual yearning for and anger at the lost object . . . ready for expression at an unconscious level" (1961). This leads [Bowlby \(1980\)](#) to argue that, because there are cumulative effects, strong preventive efforts are called for, and "the safest dose [of separation] is a zero dose." Such a prescription, although certainly applicable to acute and unanticipated loss or abandonment, does not as easily apply to the vicissitudes and developmentally promoting aspects of expectable, anticipated, and usual separations in early childhood.

[Bowlby \(1973\)](#) agrees with Freud's view that anxiety is a response to the danger of loss of a love object; pain or mourning is the response to actual loss, and defenses are mobilized to cope with pain. [Bowlby \(1960\)](#) uses the terms *mourning* to characterize the psychological process set in motion by loss and *grief* to denote the sequences of subjective states that follow loss and accompany mourning and suggests that mourning in the infant differs in no material way from mourning at later ages. Anna Freud (1960), in responding to Bowlby's hypotheses, agrees with the descriptions of behavior and the painfulness of the experience but takes issue with his view of mourning. She proposes that the term *bereavement* be used and that the term *mourning* be reserved for later reactions when development in object relations and ego maturity make it possible for the mourning process, as defined in psychoanalysis, to occur.

Since Bowlby, extensive empirical research (see [Bretherton and Waters, 1985](#); [Goldberg, 1991](#); [Waters et al., 1995](#), for reviews) using the *Strange Situation* has documented at least four patterns of 12- to 18-month-old children's response to brief separation: secure, avoidant, ambivalent or resistant, and disorganized. The crux of the separation situation designed to elicit these behavior patterns is a standard sequence of separations and reunions during which the infant experiences increasing distress and a greater need for proximity ([Ainsworth and Wittig, 1969](#); [Ainsworth et al., 1978](#)). The strategies children use to cope with their increasing needs for proximity with the adult are considered indicators of the quality of the attachment. The individual behavioral components that go into an attachment category include seeking and maintaining contact with the adult, avoidance, and resistance to physical contact. Behaviors on reunion appear to be the most salient in distinguishing among these different attachment categories. In the secure category, infants seek their caregiver only when their security is threatened, as when they are left in a novel or frightening place. When the adult returns, these children are able to explore comfortably and securely. Avoidant or dismissing children appear not to notice or be distressed by the caregiver's absence or return, whereas ambivalent children are often overly distressed even when they are not in danger. Children with ambivalent attachment patterns are often unable to resume their usual play even on the adult's return. In [Ainsworth and Wittig's original study \(1969\)](#), 65% of toddlers exhibited secure patterns of attachment, 21% an avoidant pattern, and the remaining 14% an ambivalent pattern. These patterns and their distribution are consistent for infants and toddlers from several different cultures ([van IJzendoorn and Kroonenberg, 1988](#)). Moreover, under stable environmental conditions, individual attachment patterns are stable over short periods and into childhood ([Howes and Hamilton, 1992](#); [Howes et al., 1998](#); [Main et al., 1985](#); [Waters, 1978](#)). Some studies have shown that securely attached infants are more competent than insecure infants in several later cognitive and social domains ([Kerns, 1994](#); [Matas et al., 1978](#); [Sroufe et al., 1983](#); [Waters et al., 1979](#)). A longitudinal study of children followed from the first year through puberty reports the positive relation between

early attachment behaviors and later social competence ([Sroufe et al., 1990](#)).

Studies of attachment under at-risk conditions such as prematurity, maternal depression, and maltreatment have shown mixed patterns of attachment emerging from these early stressful situations and have led to the creation of a fourth attachment category, the disorganized pattern ([Main and Solomon, 1990](#)). Infants classified as disorganized during the Strange Situation exhibit confused, often fearful reactions to the adult's return and have few to no strategies for coping with the stress of separation. Identification of the disorganized pattern has led to a surge of interest in uncovering its determinants and effects ([Solomon and George, 1999](#); [Vondra and Barnett, 1999](#)). Research suggests that disorganized attachment patterns occur more frequently among groups of abused or neglected children ([Barnett et al., 1999](#); [Carlson et al., 1989](#)), and these patterns may result from frightened (e.g., helpless, fearful) or frightening (e.g., hostile, punitive) maternal behaviors ([Main and Hesse, 1990](#); [Lyons-Ruth et al., 1999](#)). In terms of developmental consequences, "attachment disorganization in infancy forecasts controlling behavior with caregivers, aggressive and fearful peer relationships, and internalizing and externalizing problems in preschool, kindergarten, and elementary school, as well as dissociative symptoms and elevated psychopathology during adolescence" ([Lyons-Ruth and Jacobvitz, 1999](#)).

Studies of attachment behaviors beyond the second year of life have also been extended to preschool-aged children, to 5- to 7-year-olds, and to adults. For children, the most common procedures again rely on separations and observations of reunions; however, another method involves asking children to complete stories related to attachment issues ([Bretherton et al., 1990](#); [Goldberg, 1991](#)). For adults, the most validated and widely used instrument for assessing attachment is the Adult Attachment Interview, a semistructured interview about the person's experiences and current thoughts regarding his or her early relationships ([George et al., 1985](#)). Categories similar to those described in toddlers apply to older children and to adults. The secure-autonomous pattern describes a child who acknowledges the adult's return, but who usually continues to play and finds a way to invite the adult to join the activity. For adults, secure attachments are indexed by both a valuing of close relationships and a realistic acceptance of the strengths and weaknesses of those persons. Avoidant preschoolers and older children appear to be more interested in other activities than the adult's return, and although they may greet the adult, they show minimal response to overtures for engagement and little effort to include the adult in their play. Avoidant or dismissing adults idealize their childhood experiences and downplay the importance of close, intimate relationships in their lives. Dependent-ambivalent children, like their infant counterparts, are preoccupied with the adult's whereabouts at the expense of all else including their own play and other activities. Similarly, the adult analog describes a person caught in old struggles, lacking a sense of personal identity of his or her own role within relationships. The fourth category, disorganized, describes a child who is not so much behaviorally disorganized but rather is controlling the interaction. The child works hard to keep the parent happy and engaged or is controllingly hostile and demanding. For the adult, this attachment pattern may be inferred from unresolved mourning over losses of important others in which there is continuing cognitive disorganization around discussions of the lost figure.

These techniques for studying attachment patterns in older children and adults have permitted closer study of both the stability of attachment across the life span and the transmission of attachment patterns across generations. There appears to be a high degree of concordance between adult attachment patterns and their relations to their infants ([Main and Hesse, 1990](#)). Parents who are secure or autonomous in their view of their own parenting tend to have securely attached infants, whereas those who are dismissing tend to have avoidant infants, and those who are preoccupied have ambivalent infants. Studies of adult attachment do not purport to provide a "historically accurate" picture of an adult's childhood—adults with extremely traumatic early childhood experiences may nevertheless be securely attached. Rather, the studies of adult attachment tap into how the adult views important relationships in his or her life (the internal object world in the language of psychoanalysis), and these views, in turn, influence how the adult responds to his or her children.

### Contributions from Psychoanalysis

[Lieberman's \(1987\)](#) discussion of separation in infancy and early childhood presents suggestions for a synthesis of aspects of attachment theory and psychoanalysis. The effort to select relevant propositions from psychoanalysis regarding separation and deprivation is immediately challenged by the complexity of the theory, which emphasizes the uniqueness of the inner life of each child in determining the influence of a reality event, such as separation. The nature of the child's relationships with loved ones is seen as centrally important in determining how he or she will experience, cope with, and defend against separation from them. Antecedent experiences and phases of psychosexual, cognitive, and psychosocial development, with their expectable sources of conflict, anxiety, and satisfaction, as well as the effectiveness of adaptive and defensive mechanisms, play a part in how events acquire psychological meaning. Given this complexity, one can only illustrate this point by defining a few developmental lines and psychological processes, selected because of their demonstrated usefulness in dealing with separation issues in the very young.

In psychoanalytic developmental theory, the concept of separation involves two related but distinct perspectives: separation as a *process*, through which the child develops an awareness of the physical and mental self as distinct from all others, and separation as an *event*, in which someone experiences being parted from those to whom he or she is most strongly attached. Separation events occur throughout the life cycle, as [Bloom-Feshbach and colleagues \(1987\)](#) summarize, and the influence of these events is determined by multiple, interacting factors. The landmark studies of Margaret Mahler and her colleagues ([Mahler, 1963](#); [Mahler et al., 1975](#)) address the separation-individuation process and trace "the psychological birth" of the human infant. Mahler's theory of separation-individuation emphasizes that, although the process is never finished and reverberates through the life cycle, the principal psychological achievements are placed within the period from the fourth or fifth month of life to the 30th to 36th months. "The normal separation-individuation process . . . involves the child's achievement of separate functioning in the presence of, and with the emotional availability of, the mother. . . . This normal separation-individuation process takes place in the setting of developmental readiness for, and pleasure in, independent functioning" ([Mahler, 1963](#)).

The development of a sense of awareness of the body and mental self as distinct from all others and the gradual formation of a personal identity are aspects of the individual's view of his or her totality as a person. The self as a cohesive psychological organization is, as [Loewald \(1980\)](#) emphasizes, contingent on the differentiation between self and other. In the life of the infant, these distinctions develop in myriad day-to-day experiences and transactions with persons, sensations, and actions, in concert and interwoven with other lines of development. The awareness or *sense* of a personal identity is influenced by factors affecting perception of the external world; that is, there are important cognitive elements. Comparisons and contrasts become clearer through intellectual growth. Identity is maintained by continual redefinition ([Greenacre, 1971](#)). In this process, the nature of the child's relationships with others is a vital factor. "There is wide agreement . . . that it is those to whom the child is most strongly attached with whom the most important patterns of separation and individuation are played out as part of the process through which awareness of the separate self identity and self-esteem come into being" ([Provence, 1987](#)).

In psychoanalytic theory, *anxiety* is a response to the danger, real or imagined, of loss of a love object, but it is viewed in much more complex terms than it is in attachment theory. Inherent in the fear of object loss is the mixture of feelings that characterize love relationships: Loving, tender feelings are mingled with hostile feelings. Libidinal and aggressive cathexes of the mental representations of loved persons coexist. Longing for that person and sadness at separation or anticipation of loss, as well as shame or guilt about one's hostile wishes and feelings, stimulate eagerness for reunion.

The studies of Benjamin ([1961, 1963](#)) and Spitz ([1957, 1965](#)) contribute much to psychoanalytic approaches to understanding developmental aspects of anxiety. Freud's ([1926/1959](#)) introduction of the concept of anxiety, psychological pain, and mourning as responses to object loss forms the background of these and other studies. Two overlapping but different anxiety responses occur during the first year, defined operationally as *infantile stranger anxiety* and *infantile separation anxiety*. Fear of loss of the loved person in reality, fear of loss of that person's love and approval, fear of loss of the loved self, and fear of one's own disapproval in sequence overlap and span the years of infancy and early childhood. Each of these phase-associated anxieties influences the way in which separation or the withdrawal of parental interest is experienced by the child.

The developmental progression in the child's psychological life in regard to separation anxiety begins with reaction to loss of what Benjamin calls a *preobject*, a person or persons who can reduce tension and provide security and pleasure, and it progresses to fear of loss of a *differentiated* object, a particular person one knows and loves. [Benjamin's \(1963\)](#) hypothesis that fear of loss of the person one loves and depends on is the immediate dynamic determinant of separation anxiety appears valid. With its later derivative, fear of loss of love, it is universal or nearly universal in our culture in psychologically healthy children, although varying in intensity.

Psychoanalytic developmental theory also brings to bear on separation propositions regarding the stability and permanence of psychic representations. In this connection, the construct of *object constancy* has relevance. It is believed that the child's ability to cope with the stress of separation events leans heavily on the capacity to evoke reassuring mental images of those to whom the child attributes his or her sense of security and well-being. Solnit's definition is useful: "Object constancy is that state of object relations in which the child has the capability to retain the memory of the emotional ties to parents, . . . and to feel their nurturing, guiding presence even if they are a source of frustration or disappointment or when they are absent" ([Solnit, 1982](#)).

[Fraiberg \(1969\)](#) does much to clarify issues regarding the development of object constancy, a term that has been used differently by various psychoanalytic theorists and investigators. She supports a view put forward by [Nagera \(1966\)](#) that one may distinguish between the very beginnings of object constancy in the first year and its attainment at the end of the second year. Similarly, [Mahler \(1966\)](#) indicates that the achievement of object constancy is a characteristic of the fourth subphase of



separation-individuation, when, as [Hartmann \(1962/1965\)](#) suggests, mental representations of the mother become intrapsychically available. Fraiberg reviews the concept of object constancy in its libidinal and cognitive aspects and distinguishes between the formation of the libidinal tie during the first year and the attainment of object constancy, which requires the development of crucial cognitive structures. Evocative memory, demonstrable in the middle of the second year, is probably a necessary, although not sufficient, condition for the child's awareness that the loved figure from whom he or she is separated has not been lost. Object permanence and mental representation in Piaget's sense, occurring at around 18 months, appear to be cognitive predecessors for the development of object constancy in the psychoanalytic sense. Anxiety at separation, however, is not necessarily traumatic. The concept of anxiety as a signal affect through which adaptive and defensive mechanisms are prepared and mobilized is relevant. If not overwhelming at a given point, anxiety sharpens perception of the self and others and of the ways in which challenges can be met and, thus, contributes to personality organization and health.

### Contributions from Other Developmental Psychologies

Other psychological theorists and investigators have contributed constructs that have heuristic value in views of separation and deprivation. [Escalona \(1963\)](#), in bringing together aspects of ego psychology and Lewin and Piaget's studies of the development of intelligence, says

My data suggest the possibility that what Piaget proposes for cognition is true of all adaptive aspects of mental functioning: namely, that the emergence of such functions as communication, modulation of affect, control over excitation, delay and aspects of object relations, and hence identification; all are the result of a developmental sequence in sensory motor terms before they merge as ego functions in the narrower sense.

Deprivation experiences, in particular, would begin to exert a noxious effect on these adaptive functions from early in life. [De Carie \(1965\)](#) demonstrates a significant correlation between stages of affectivity and Piaget's theory of cognitive constructions of the inanimate object. [Bell \(1970\)](#) finds that infants develop a concept of a person's independent existence (person permanence) even more quickly than a homologous concept of inanimate objects, findings that reflect Piaget's realization of the emotional importance of the mother. Piaget sees affectivity as the energy source on which the full functioning of intelligence depends. Neither can exist functionally without the other. Emotional processes can accelerate, delay, or disturb mental acquisitions ([De Carie, 1978](#)). Concepts of recognitory versus evocative memory, means-end relationships, time and space, progression from prerepresentational sensorimotor schemes to representational and symbolic thinking, verbal communication, fantasy, and perception of reality are among other cognitive constructs found useful in examining issues of the effect of separation and deprivation experiences.

### PARENTING REQUIREMENTS FOR SECURE ATTACHMENT AND RELATED DEVELOPMENTAL CAPACITIES

[Winnicott \(1961\)](#) emphasizes that the young baby is essentially part of a relationship; that is, if you set about to describe a young infant, you find you are always describing a baby *and someone else*. Synchronous communication begins in the newborn period as the nurturing adult responds and learns the baby's rhythms, attentional behaviors, comfort-discomfort "messages," and affective states ([Condon and Sander, 1975](#); [Sander, 1980](#)). The very terms *dialog* ([Brazelton and Cramer, 1990](#)) and *the dance* ([Thoman and Browder, 1987](#)) are efforts to capture the reciprocity, mutuality, and transactional nature of the relationship from the beginning. However, it is an uneven partnership because the baby's dependence needs are great, and so we define a facilitating environment as one that parent figures have the responsibility, gratification, and fulfillment to provide. That parenthood demands adaptation and growth and that the tasks and responsibilities are, at times, strenuous and worrisome are reality.

A conviction about the central importance of the child's human relationships for his or her growth and development physically, cognitively, and emotionally is shared by several developmental theories. Whether one prefers psychoanalytic formulations regarding object relations, ethologic attachment theory, social learning theory, self-psychology, or other perspectives, there is strong consensus about the essential role of relationships with parents and others close to the child for his or her psychological well-being and the damage, short-term or long-term, that may occur when relationships are seriously deficient or jeopardized. When infants are deprived of the experiences that comprise good nurturance from their caregivers, and when there is significant discontinuity of care through separation events, there can be interferences and distortion in many aspects of development.

Among the important features of the type of parental care that facilitates development are the following: continuity of care and affection from primary persons who are predictable and trustworthy; the development of a close relationship between adult and child, initiated by the parents' desire to love and nurture and the infant's responsiveness; specific types of stimulation addressed to physical, social, emotional, language, and intellectual growth; models for the development of controls, self-regulation, and socialization through education and guidance in an atmosphere of loving attention; and transmission of societal and cultural values through influencing the manner in which the child solves the tasks imposed by each phase of his or her epigenetic psychosocial development.

Continuity of affectionate care by one or a small number of caregivers who can give of themselves emotionally, as well as in other ways, originates the development of the child's love relationships. The formation of social bonds and of social and emotional communication, the development of a sense of confidence or trust in others, the perception of the parent as the source of security and protection, and feelings of self-esteem depend on such care and its reliability. Affectionate and stable patterns of experience facilitate the baby's ability to organize physiologic and psychological processes ([Escalona, 1968](#); [Greenspan, 1981](#); [Sander, 1980](#)) from early on and influence perceptions of the outer world and of the child's inner sensations, feelings, and thoughts. For example, being loved and esteemed by others is believed to be at the heart of the ability to love oneself, and self-love is a part of the capacity to love another in turn. For another example, having repeated experiences of being comforted when distressed is a part of developing one's own capacity for self-comfort and self-regulation, and later, the capacity to provide the same for others.

Specific types of experiences likely facilitate the formation of secure attachments and, moreover, enhance particular aspects of a child's development. For example, the infant's being talked to by his or her caregivers is very important in speech development. Progress in the ability to communicate verbally occurs when intrinsic maturational events interact with environmental influences that organize and give meaning to the child's utterances. The presence of a speaking, interested social partner is vital. Similarly, motor skills, as opposed to mere motor discharge, are stimulated by an array of tactile-kinesthetic experiences that occur in the process of good care and later by opportunities to practice emerging skills in a safe environment. The growth of intelligence in the early years is enhanced by variety, contrast, and novel stimuli when these occur in the context of essentially stable and developmentally appropriate patterns of experience with the environment. The provision of toys and other inanimate objects to manipulate and explore is important for negotiating the stages of sensorimotor intelligence ([Piaget, 1952](#); [Piaget and Inhelder, 1969](#)), which takes place in concert with structuring of the affective life. The parent also is important as educator and guide, helping the child to identify aspects of reality, that is, both as a source of information and as a reinforcer or modifier of the child's cognitive expectations.

The parental role in the child's coming to interact effectively with the environment is significant in both its learning and its motivational aspects. [Hartmann \(1958\)](#) emphasizes the importance of the child's social relationships in the learning that is essential for many of the child's adaptation processes. Robert [White \(1959\)](#) and Lois [Murphy \(1974\)](#) emphasize the beneficial influences of experiences of mastery in adaptation. Coping involves original, imaginative, and innovative behavior and helps the child to manage both his or her inner experience and the demands of the outer environment. Studies of the origins of competence in the child, motivation for mastery, and relationships between motivation and cognition ([Harter and Zigler, 1974](#); [White, 1975](#); [Yarrow, 1981](#); [Yarrow and Pederson, 1976](#)) further support the view of the importance of an environment in which adults sensitively respond to the child's needs and provide responsive materials.

Parents, as models for the child in the establishment of controls over impulse and behavior, make a significant contribution to the development of a healthy personality. Whether one regards the process as establishing ego control over the drives or as the socialization process, the influence of good parenting is acknowledged. Through the relationship and with the necessary guidance in an atmosphere of loving attention, the child comes to wish to please those persons he or she loves and is motivated to behave in ways of which they approve. The child imitates, identifies with, and gradually incorporates aspects of their behavior, attitudes, and values, making them his or her own. This condensed statement greatly oversimplifies the process through which the child moves toward ego maturity, control over sexual and aggressive impulses, affect regulation, and superego formation. Here, perhaps, it can only be repeated that parents as models and guides play a central role, and, in their absence or severe dysfunction, this aspect of personality development suffers.

### SEPARATION AND DEPRIVATION AS THREATS TO SECURE ATTACHMENT AND RELATED DEVELOPMENTAL CAPACITIES

Traumatic separation and deprivation endanger the development of healthy attachment relationships with others and the formation of social bonds. If one accepts the idea that the child's sense of security, basic trust, and knowledge of others and of self are anchored in and flow from the care of one or a small number of affectionate, available caregivers, then frequent and long disruptions in the interactions with the person one has come to trust and love or deprivation of essential nutrients in caregiver-child interactions can be identified as threats. Infancy research and clinical experience confirm that a growth-inhibiting environment for an infant in the first year exists when caregivers are unavailable, are understimulating or overstimulating, are emotionally distant, are ambivalent, or fail to respond contingently to the

infant's communication across multiple sensory and affective systems ([Greenspan, 1981](#)). In contrast, a growth-promoting environment is characterized by invested, comforting, guiding caregivers who “woo” the infant, encourage the “dialog,” respond appropriately to the infant's communications, and, in the process, transmit feelings as well as bits of “information” about the human and inanimate world that become vital factors in general learning, as well as in relationships with others. Progression from sensorimotor modes to the organization of internal representations of self and other persons and other symbolic elaborations of mental imagery is also sensitive to severe deprivation in the environment and to multiple changes in the continuity of adequate child care. Thus, there is reason for concern about interferences and distortions, especially in the young child's coming to love himself or herself and others, and in his or her relationships with caregivers, peers, and others in his or her social environment.

Language, both speech and nonverbal communication, is particularly sensitive to deprivation and separation. Even though speech and the elaboration of social signals have an inner, biological, and maturational basis, their blossoming as agents of communication is closely related to the releasing and stimulating influence of the environment. Infants and toddlers, for example, who are not talked to by their caregivers in families and elsewhere are delayed in speech development ([Provence and Lipton, 1962](#); [Spitz, 1945](#); [Spitz and Wolf, 1946](#)). Many years ago, Lewis wrote: “When does a child begin to speak? . . . If not at the moment of birth, then certainly during the first day. For as soon as a child cries and someone pays attention to his cry, the first steps are taken; the essentials of language are there” ([Lewis, 1959](#)). The presence and attention of the primary parent, usually the mother, influence the development of speech in several ways, through reinforcing the baby's vocalization, the development of mutually imitative vocalizations that encourage the use of an expanding repertory of sounds, and the “labeling” of people, toys, actions, feelings, and the like ([Bornstein, 1989](#)). The mother's speech is also a carrier of emotions and an organizing influence on the infant's psychological life. The sounds that form the building blocks for speech are believed to gain specific meaning because of the parents' response to them. For example, the “Mama” and “Dada” sounds that emerge as a result of maturation at somewhere around 9 months become names for the parents because of their strong affective response and reinforcement. Speech development is a very complex function, and there is considerable variability in its development within what we consider normal. That being acknowledged, the sensitivity of speech development to disruptions in relationships with the partners in communication and to a verbally impoverished environment is a signal to the clinician to look closely at the child's experiences with his or her caregivers.

The adverse and often long-lasting consequences of traumatic separation or deprivation are rooted in changes to the developing brain. Because of a high degree of neural plasticity, it is generally believed that the infant's and young child's brain is particularly sensitive to experiences that promote or hinder development in not only sensory and linguistic but also emotional domains. The underlying mechanisms, although not completely understood, involve synaptic formation and pruning. “Neuronal groups of cells that are frequently and/or intensely stimulated through environmental stimulation are thought to be selectively amplified, forming cortical maps with defined functions. Conversely, cells that do not form functional synapses are retracted” ([Nelson and Bosquet, 2000](#)). For example, evidence suggests that infants between 6 and 18 months who are repeatedly exposed to negative or flat affect and to little positive affect and synchronization during social interactions with their clinically depressed mothers develop atypical electroencephalographic patterns reflecting a hyperarousal of the right frontal lobe (which mediates negative affect and withdrawal behavior) and a hypoarousal of the left frontal lobe (which underlies positive affect and approach behavior) ([Nelson and Bosquet, 2000](#)). Once established, these and other patterns of brain organization and activity become somewhat resistant to change.

## CONTRIBUTING AND MEDIATING FACTORS

### Poorness of Fit

*Goodness of fit* and *poorness of fit* are terms used to describe the way in which infants and their parents enter into and achieve good adjustment to one another or have great difficulty doing so. Difficult temperamental characteristics ([Thomas and Chess, 1977](#)), unusual sensitivities ([Bergman and Escalona, 1949](#)), and congenital characteristics of activity and reactivity ([Korner, 1974](#)) can make some infants difficult to care for by even the most expert of parents. Some infants' characteristics, even if not difficult, may not fit well with their caregivers' preferences ([Colin, 1996](#)). Other infants may be disappointing to their parents because of their unconscious meaning. These problems in mutual adjustment interfere with a beneficial parent–child relationship and become a pathogenic influence. Various forms of deprivation and traumatic separation events may result from such situations ([Kris, 1962](#); [Provence, 1965](#); [Provence, 1983](#)). Among the more significant opportunities for preventive intervention is the early recognition of parent–child interaction that reflects problems in reciprocity and mutual adjustment.

### Maternal Factors

Many conditions in the mothering person contribute to or complicate deprivation. Maternal depression, for example, often results in a mother's being psychologically unavailable to the child at times, appropriately and lovingly attentive at times, and intrusive and hostile at others. Her failure to respond contingently results in deprivation experiences for the baby, even when she is physically present ([Cohn, 1993](#)). Maternal ambivalence in behavior and attitude toward the child is both confusing and painful, interfering, among other things, with a secure attachment and the orderly progression of separation-individuation, self-esteem formation, and affect regulation. Children who are uncertain of the parent's love, guidance, and protection are especially vulnerable to separation. Mothers with severe psychiatric disorders become dysfunctional in a variety of ways. A mother whose own reality awareness is impaired is unable to represent reality for her infant. If she is unpredictable in her behavior toward her infant, those developmental steps that flow from a sense of trust and security will be impeded; if she is severely disorganized, the baby is likely to suffer from pervasive problems of regulation, both of physiologic and psychological functions. If the baby is a part of her delusional system, the baby may be in gross danger ([Anthony, 1974](#)).

A mother impaired by drugs or alcohol is unable to provide the type of care that promotes the child's development, both because of her unavailability in the positive sense and because poor control of aggressive behavior may result in abuse ([Mayes, 1995](#)). If she has been a substance abuser during pregnancy, the infant is potentially at both biological risk and environmental risk ([Chasnoff et al., 1984](#); [Howard et al., 1989](#); [Jones and Lopez, 1988](#)). Parental substance abuse presents a continuum of vulnerability and risks for the children involved. Chronic substance abuse (e.g., alcohol, heroin, or cocaine) may impair an adult's psychological functioning in domains basic to parenting (e.g., persistence, concentration, memory). Moreover, for the most part, substance abuse involves the adult in related environmental conditions including poverty and homelessness, frequent violence and family discord, poor physical health, and unstable, erratic relationships with others. Mothers who are chronic substance abusers most often cite their social isolation and deep loneliness as the states of being that most interfere with their ability to reduce or stop their addiction ([Mayes, 1995](#); [Tucker, 1979](#)).

Physical abuse and sexual abuse are actions by parental figures against children that stem from the psychological and social pathology of adults. They may be concomitants of deprivation; traumatic separation experiences may be part of the picture. Abusive acts may also occur in child-rearing environments that contain positive features as well. For such children, careful clinical assessment is the basis for recommendations about whether the dangers to physical and emotional survival outweigh the advantages of remaining with the parent. The presence or absence of family members who can adequately protect the young child will be an important determinant of decision making. Separation from abusing parents and placement in a nurturing environment may permit emotional healing and may restore the young child's chances for adequate development ([Goldstein et al., 1979](#)).

### Child Factors

Factors in the child that heighten his or her vulnerability to deprivation and traumatic separation are referred to throughout this chapter. However, young children vary in their vulnerability to noxious biological influences, as well as to environmental situations associated with poor developmental outcome in others.

Genetic disorders associated with mental subnormality (chromosomal or biochemical), malformations of the central nervous system of obscure origin, toxic insults, infections, malnutrition, and perinatal insults such as hemorrhage and hypoxic injury make the young child more vulnerable to the adverse effects of separation and deprivation. Sensory deficits such as deafness and blindness and musculoskeletal disabilities, whatever their cause, delay or disrupt the developmental process. Children deemed vulnerable or in need of particularly skilled parenting, such as those called temperamentally slow to warm up or difficult, those with sensory processing problems, and those with unusual sensitivities, often come to attention because of difficult behavior or delays in aspects of development. Such children often have difficulty with many phase-specific aspects of early development including the usual day-to-day separations between parent and child.

Child characteristics, congenital or acquired, may underlie clinical disorders for two reasons: Intrinsic factors may complicate the nurturing process, and maladaptive child-rearing styles and attitudes may be evoked in the parent by these specific characteristics. Thus, there is often an interaction between basic biology and patterns of parenting that further exacerbates developmental vulnerabilities and responses to situations such as separations.

The child's developmental level and personality organization at the time of separation or deprivation are of great import in determining their influence. A 10-month-old infant, for example, will have both the perception of loss of the differentiated object and distress and despair if a separation is prolonged and there is no satisfactory surrogate. The infant will have very limited resources for self-comfort and will be largely dependent on others to alleviate his or her distress. The 4-year-old child who



has been well cared for and who is developing adequately, in contrast, can identify and verbalize feelings and ask questions. He has enough sense of time (before and after, today and tomorrow) and place to use explanations and reassurance. He has play skills that enable him or her to express as well as to work at mastery of the anger and discomfort through action and fantasy. He will also have available mental representations of those whom he loves and who are sources of security that can be evoked in time of need, and he will have developed coping strategies and defense mechanisms that can be mobilized to deal with the stress of separation.

### Mother-Child Relationship Qualities

The effects of separation or deprivation are mediated not only by the developmental level of the child and by the presence of child and maternal risk factors, but also by the nature of the caregiver-child relationship. "The risk for psychopathology is highest when multiple factors coincide to increase the child's anxiety about his or her parent's availability" (Kobak, 1999). Insecurely attached children are more likely to interpret even temporary disruptions as threats to parental availability. Moreover, for older children, the effects of disruptions such as divorce, parental dysfunction or illness, or even parent-child conflict are likely biased by the nature of current communication in the relationship and not only by the child's internal working model (Kobak, 1999). Although open communication and secure working models often go hand in hand, for insecurely attached children, dysfunctional communication patterns may exacerbate a child's insecurity and symptomatic expressions. Research is only beginning to attend to the role of problematic communication in influencing the perception of separations as threats to availability and, thus, to attachment security.

### Alternative Attachment Relationships

Attachment research generally has focused on the mother-child relationship. However, in the United States, children now are regularly cared for by multiple caregivers (e.g., fathers, grandparents, child care providers), and the role of alternative attachment figures is becoming the subject of increasing empirical research (Howes, 1999; van IJzendoorn et al., 1992). The organization of multiple attachment relationships and internal working models remains unclear (i.e., hierarchical versus integrative or independent), as is their influence on children's long-term development (Main, 1999). Yet there is emerging support for a nonhierarchical model of organization (Howes, 1999; Howes et al., 1998; Suess et al., 1992), and thus developmental consequences may be best predicted by considering the quality of all attachment relationships or even may be linked to particular attachment figures. Further research is needed to clarify how or whether alternative attachment relationships buffer the child who experiences disruption or deprivation in his or her relationship with a mother figure.

### Situational Events

As indicated, whether deprivation and separation are short term or long term and occur in a well-functioning or dysfunctional family will also make a difference in impact on the capacity of the family to respond to a crisis or a planned separation in a way that is protective of the young child. The latter includes, for example, the adequacy of the child-rearing arrangements during the parents' absence. Other factors that precipitate separation, such as illness of child or parent, divorce, natural catastrophe, and social upheaval, are diverse examples of the contexts of separation, each bearing its own set of conditions that affect the child and those closest to him or her.

## CLINICAL OUTCOMES

Clinical experience is replete with examples of consequences of deprivation and traumatic separation, described as delays, syndromes, or disorders.

### Problems in and Disorders of Attachment

Insecure and disorganized attachments, as classified by the Ainsworth Strange Situation, have been increasingly reported as characterizing infants whose mothers, for a variety of reasons, have been unable to respond to them sensitively and contingently (Ainsworth et al., 1978; Bretherton, 1985; Easterbrooks et al., 1993). Although less than ideal in quality, insecure and disorganized attachments reflect typical variation in attachment security observed among infants and children in the Strange Situation. The disorganized pattern is the most problematic of the nonsecure classifications because it is associated with many adverse sequelae (Lyons-Ruth and Jacobvitz, 1999). The Strange Situation is a superb research tool and can be useful to clinicians as a marker suggesting the need for further exploration of the child-rearing environment in that it captures aspects of the mother-infant relationship. However, the results of the Strange Situation are not established as valid for clinical work in individual cases, and the data derived must be integrated with other knowledge about the infant from other sources, to be useful as a clinical instrument (Ainsworth, 1980; Gaensbauer and Harmon, 1982; Greenspan and Lieberman, 1988; Weitzman and Cook, 1986).

Conversely, clinicians, using information from a broad context, are accustomed to diagnosing reactive disorders of attachment in infants and young children that are often the result of multiple separations and depriving environments (Tibbitts-Kleber and Howell, 1985). A current conceptualization of attachment disorders and diagnostic criteria have been proposed by Zeanah and his colleagues (Lieberman and Zeanah, 1995; Zeanah and Boris, 2000). This approach delineates three broad types of attachment disorders: (a) disorders of nonattachment, (b) secure-base distortions, and (c) disrupted attachment disorder. It is recommended that criteria be viewed as dimensional such that if behavioral signs are usually or often present, then an *attachment disorder* is defined, whereas if behavioral signs are only somewhat or sometimes evident, then an *attachment disturbance* is present (Zeanah and Boris, 2000).

First, *disorders of nonattachment* are characterized by the absence of a preferred attachment figure. There are two types: (a) *nonattachment with emotional withdrawal*, in which the child is also emotionally blunted, markedly restricted in comfort seeking and affection, and avoids or fails to respond to social overtures; and (b) *nonattachment with indiscriminate sociability*, in which the child not only fails to use a preferred caregiver for support, protection, and nurturance but seeks proximity, interaction, and comfort indiscriminately, even from strangers, without age-appropriate anxiety toward unfamiliar people (Zeanah and Boris, 2000).

The second general type of attachment disorder is known as a *secure-base distortion* in which a child's relationship with an attachment figure is marked by serious behavioral disturbances *specific to that relationship*. Four different types of secure-base distortions have been described: (a) *attachment disorder with self-endangerment*, in which the child fails to return to his or her secure base and engages in dangerous and provocative behaviors such as running out into traffic, climbing up on ledges, and self- or caregiver-directed aggression; (b) *attachment disorder with clinging or inhibited exploration*, in which the child is unwilling to venture away and explore; (c) *attachment disorder with vigilance or hypercompliance*, in which the child neither clings to the caregiver nor explores, and instead engages in emotionally constricted watchfulness and obedience; and, finally, (d) *attachment disorder with role reversal*, in which the child appears oversolicitous and preoccupied with the caregiver's emotional well-being. The final type of general disorder of attachment is referred to as *disrupted attachment disorder* and applies to a child who is suddenly separated from an attachment figure from days to weeks and reacts to the loss of that attachment relationship with the typical sequence of protest, despair, and detachment (Zeanah and Boris, 2000).

### Object Constancy Delay and Instability

Disturbances in the development of a sense of self and the separation-individuation process occur when the benevolence and reliability of caregivers are grossly inadequate. As already indicated, children who do not feel valued cannot value themselves. Those whose sense of self is not nurtured and reinforced by the affection, protection, and guidance of parent figures have little opportunity to develop the pleasure and confidence in independent functioning that is an integral part of healthy personality development. The attainment of emotional object constancy and a stable sense of self is a main task of the subphase of separation-individuation, occurring, roughly, in the third year of life (Mahler et al., 1975). Disturbances in the separation-individuation process are inferred from the behavior of young children in the third year and beyond, when separation becomes a severe problem, and maladaptive "solutions" to the feelings associated with separation are manifest. Many empirical studies of children entering nursery school support several clinical impressions. Bloom-Feshbach (1987), in studies of 3-year-old children, found clear distinctions between emotionally healthy children and those with problems. After an initial period of adjustment, including protest at separation, emotionally healthy children were better able to seek and use teachers for help and comfort when needed, they investigated the environment more actively and with greater satisfaction, they interacted more confidently with peers, and they regulated their feelings more adaptively. Their behaviors suggest favorable progress in personality organization, healthy coping with stressful events, and the ability to make use of and enjoy a larger world of experience. In contrast, children with problematic separations were marked by the emergence of symptomatic behavior ranging from severe and prolonged distress, to defiance, to apathy. Symptomatic behavior may reflect internal difficulties in differentiation, self-esteem, and identity formation. According to Mahler, Pine, and Bergman (Settlage, 1974), danger signals during the separation-individuation process during the second and third years include the following: the absence of the normal mood of elation related to a sense of power, expected in early toddlerhood; a proclivity toward a depressive mood; and, in some instances, a marked increase in aggressive behavior. Among the behavioral signs of a tendency toward marked ambivalence toward the parent is a greater than average separation anxiety. Clinging, demanding behavior, and protest, as well as superficial indifference and detachment, may occur.

## Affect Regulation Dysfunction

Disturbances in modulation and regulation of affect are frequently found in infants and young children who encounter deprivation and traumatic separations. Range, differentiation, intensity, and expression of feelings as reflected in behavior are interfered with in these children. Regulatory mechanisms that favor the infant's capacity (a) to recognize pleasure and joy, and their opposites, in himself or herself and in others; (b) to tolerate these perceptions; and (c) to develop self-regulation strategies to modulate feelings depend, at least in part, on the infant's interactions with the environment. Insufficient "supplies" and severe disruptions tend to starve or overwhelm the system. Responses and "solutions" ensue, which may have short-term utility but prove to be maladaptive in the long run. At the mercy of poorly regulated affects, young children manifest various problems, including difficulties in peer relationships, severe anxiety, constriction or wide swings in expressive behavior and mood, and conflicts with parent figures. Outbursts of anger, "giddy" excitement, impulsive actions, and panicky behavior may coexist with excessive compliance, blunted affect, depression, or apathy.

## Developmental Delays

Developmental delays in motor, language, intellectual, and social or emotional behavior occur singly or, more often, in combination in instances of repeated separations with deprivation. The classic example of multiple developmental delays found in infants living in foundling homes and other residential institutions has produced a very extensive literature, of which only a small sample is cited here ( [Ainsworth, 1972](#); [Bowlby, 1951](#); [Bowlby, 1958](#); [Provence and Lipton, 1962](#); [Yarrow, 1961](#)). Disturbances in the quality of social relationships and emotional expressiveness occur simultaneously with difficulties in versatility in coping with challenge or stress and in flexibility in thought and problem-solving. Major improvements in the environment, especially the quality of nurturing, are accompanied by encouraging gains, and yet concerns about long-term effects on capacities for mature interpersonal relationships and adequate functioning in the society remain. Less pervasive developmental delays are found in situations that may not immediately be identified as depriving. Infants of mothers with masked depression, for example, may present with delays in discrete areas, such as speech or range and variety of emotional expression, in which developmental findings in the infant are clues to maternal dysfunction ( [Coleman and Provence, 1953](#); [Fendrick et al., 1990](#); [Field, 1995](#); [Provence, 1983](#)).

## Failure to Thrive and Eating Disorders

Environmentally based failure to thrive, as a syndrome defined as a severe deficit in rate of weight gain in the absence of physical disease, was first described, according to [Drotar \(1985\)](#), in association with compromised nurturing in institutional environments ( [Chapin, 1908](#); [Chapin, 1915](#); [Spitz, 1945](#)) and family settings ( [Patton and Gardner, 1963](#); [Talbot et al., 1947](#)). Reduced human potential, as well as the need for a spectrum of services from pediatric hospital care, to foster homes, to psychiatric treatment of severe emotional disturbance, are associated with this condition. Studies have underscored significant individual differences in origin, age of onset, severity of growth deficits, developmental and behavioral problems, and family stress ( [Chatoor and Eagan, 1983](#); [Drotar, 1985](#); [Ferholt and Provence, 1976](#); [Mayes and Volkmar, 1993](#); [Woolston, 1983](#)).

Eating disorders are not uncommon in infants and young children experiencing depriving environments and traumatic separations. They range from anorexia to insatiable appetite, from food refusal to overeating, and from malnutrition to obesity. Deprivation in regard to feeding has several meanings: fewer calories than are required for normal growth; being fed, not according to one's own appetite and rhythms but by the schedules or whims of the caregivers; and missing the physical and social stimulation and connectedness that occur when one is fed by an attentive, attuned feeder. The "stomach love" of the young infant for the mother is a metaphor for a mosaic of sensations, perceptions, emotions, and mutual adjustments. Multiple factors determine the extent to which unsatisfactory feeding experiences will result in clinical disorders, including, as is true in other respects, antecedent experiences, the phase of development during which they occur, and the child's unique endowment and state of physical health. However, when the positive conditions of a satisfactory feeding experience are in short supply, when the relationship with the feeder is disrupted, when mealtime is a time of turbulence or battle, when the child's cognitive and affective expectations are repeatedly violated, eating problems of one type or another may ensue. The finding that these disorders are usually not the sole clinical findings or necessarily the most significant ones for emotional health requires the coordinated efforts of pediatric and mental health experts. A developmental approach to feeding disturbances that looks at numerous factors in the child and the environment is likely to lead to improved understanding, treatment, and preventive intervention ( [Chatoor et al., 1985](#); [Mayes and Volkmar, 1993](#)).

## Psychiatric Disorders

Psychiatric diagnosis in the early years of life is fraught with difficulty. Aside from agreement about certain syndromes associated with troubled parent-child relationships, including those in which deprivation and separation play a part, clinicians encounter the problem that currently available systems of classification are far from adequate. Examples of efforts to clarify the diagnostic nosology for younger children include the *Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood (Zero to Three, 1994)* and Zeanah's typology of disturbances in attachment cited earlier ( [Lieberman and Zeanah, 1995](#); [Zeanah and Boris, 2000](#)). Limited data on the outcome of early-onset disorders, how developmental change affects syndrome expression, and the degree of dysfunction that should be labeled as clinical disorder complicate classification. That infants and young children become depressed and develop psychophysiologic and psychosomatic symptoms and behavior problems is well known, but there is need for a more systematic developmental approach to the classification of early childhood disorders. Disorders of affect, in the early years, for example, can be defined as those that pose serious risk for distortion of expectable development. For a diagnosis of *anxiety disorder*, the child should exhibit the following: (a) excessive levels of anxiety and fear, as manifested by multiple or specific fears; (b) excessive separation or stranger anxiety; (c) panic without clear precipitants; or (d) inappropriate absence of expectable anxiety or fear. *Mood disorders* include the following: (a) prolonged bereavement or grief reactions that interfere with development; that is, normal grief reactions qualify for designation as a disorder if they are excessive; (b) depression of infancy and early childhood, reserved for those who exhibit a pattern of depressed mood with diminished interest or pleasure in developmentally appropriate activities; or (c) a diminished repertory of social interactions and initiative. The term *deprivation syndrome* may be reserved for those in whom gross parental neglect of basic physical or psychological needs or the absence of a stable primary caregiver is evident ( [Volkmar and Provence, 1990](#)).

## INTERVENTION

The rapidly growing literature on early intervention programs addresses effectiveness of treatment for the impact of separation and deprivation. These programs vary widely in sponsorship, specific types of problems addressed, and theoretical orientation of those who conduct them.

### Psychotherapy

In general, *clinically oriented programs* can be classified as (a) those directed primarily at strengthening or supporting families in their child-rearing functions (i.e., caregiver-focused services); (b) those that provide direct services to the child with little or much parent involvement; (c) those whose focus is on simultaneous work with parent and child, aimed at facilitating the relationship between them; and (d) those that take place in extrafamilial settings. One model of caregiver-focused intervention is the parent as recipient of developmental guidance, a generally educative model, but also conceived as a process in which facts and information are provided in the context of a working relationship and in accordance with what parents are ready to hear and able to use. The assumption is that such guidance will enhance the parents' ability to function more competently and comfortably as parents for the child in question ( [Seitz and Provence, 1990](#)). Three models of work with parent and infant simultaneously or conjointly illustrate the use of the child's presence as a catalyst for change primarily in the parent. The child's development, actions, and reactions during the sessions facilitate and shape the direction of the work. For example, the method of parent-infant psychotherapy created by Fraiberg and her colleagues ( [Fraiberg et al., 1980](#); [Fraiberg et al., 1981](#); [Pawl and Pekarsky, 1983](#)) is directed at alleviation of severe conflict between parent and infant or toddler, in which the infant's development has been adversely affected. Experience with this method shows that it can be used in a broad range of cases. Emotional support, interpretation, nondidactic guidance, and counseling are integral to the process.

The Tripartite Therapeutic Design developed by [Mahler \(1968\)](#), in which parent and therapist join in the treatment of the psychotic child, engages the parent in an emotional-intellectual learning experience that permits her or him to sustain the corrective experiences for the child. This method has more recently been effectively used in other types of cases, for example, in cases of young children with severe hearing impairments in which there are problems of social and emotional communication between child and parent ( [Fields et al., 1993](#)). Still another is the Parent-Infant Interaction Model ( [Bromwich, 1981](#)), which focuses on the process of attachment. It is the role of the intervention to assess the nature of the interaction and to enable infant and parent to behave more reciprocally and with mutual enjoyment. The consequences of a positive outcome in the short term can have long-range benefits, primarily because of an increase in the sensitivity and self-confidence of the parent ( [Provence and Naylor, 1983](#)).

Preschool children and their parents are also treated in the conventional child psychiatric modes, in which simultaneous work with child and parent is carried out with careful attention to its coordination. Treating toddlers and young children singly or in small groups and treating mother-toddler groups are other useful and effective



methods of treatment. For the very young, psychotherapy has a considerable educational component directed at enhancing autonomous ego functions but with careful attention to its appropriateness in the child's emotional life ( [Dahl, 1983](#); [Pruett, 1983](#)).

### Home Care for Troubled Families in Crisis

Since the mid-1970s, efforts have intensified to prevent the breakup of families and placement of children in foster care and to promote family reunification through around-the-clock response to troubled families. The Tacoma Homebuilders Model ( [Kinney, 1978](#); [Kinney et al., 1977](#)), using teams of professionals, was a successful effort that has been replicated or adapted for use elsewhere, often under public auspices ( [Schorr and Schorr, 1988](#)). The advantages of improving family functioning to avoid the many separations experienced by infants and young children in the foster care system is obvious. Paraprofessional home visitors backed up by professionals have also demonstrated success ( [Adnopo, 1993](#)). Services for the children that specifically address their psychosocial needs, as well as their educational and health needs, are essential to the success of such efforts, to minimize the impact of the deprivation and traumatic experiences that are common in troubled families. This approach requires sustained efforts to coordinate community services.

### Foster Home Care

Foster care, as part of the child welfare system, is so overburdened that many professionals perceive it as a sentence to certain emotional damage and disability for many children. That families willing to care for a child not their own would be sought as a next-best choice to the child's own family makes eminently good sense, and there are children who have been well served by marvelous foster parents. What has caused great alarm, however, is the frequency of multiple placements, long periods in limbo awaiting decisions about custody or for rehabilitation of dysfunctional parents, the inability of some foster parents to encourage a close relationship with the foster child, and, in some instances, neglect and abuse of foster children. Separation and deprivation are dangers found in situations leading to foster home placement in children of all ages. One of the most urgent needs is for timely and targeted assistance to families in trouble, to prevent their disintegration as child-rearing environments through a spectrum of family support services. Another is to recognize that the passage of time while an infant or young child is in a "temporary" situation can become a great enemy. Clinics, child welfare, mental health services (unless they define themselves as crisis oriented), and, surely, the judicial system have their own rhythms and calendars, not geared to prompt and constructive response to the compelling needs of the very young, in spite of laudable intentions. Whatever can be done to persuade the various systems involved that the dangers of physical and sexual abuse are not the only situations deserving prompt mobilization of services for children or a particular child will make for improvement in the current scene ( [Goldstein et al., 1979](#)).

### Group Residential Care

There is reason to believe that, in the spectrum of services required to care for vulnerable infants and young children, the group residential setting has a place. As a temporary place for healthy infants without families, a residential setting of good quality can be used to avoid "bouncing" from one foster home to another while a permanent placement or restoration to the infant's own family is arranged. In addition, as a therapeutic environment for infants impaired by drugs, repeated traumatic experiences, and other conditions that render them extremely difficult to be effectively cared for in families, a group residential setting may be the treatment of choice.

Such care must be organized as an active therapeutic program that includes carefully designed, individualized treatment, as well as general developmentally appropriate care. The duration of residential care for each child should be determined by the time at which he is judged to be healthy enough and well-organized enough (at whatever level) that home care by parents or foster parents has a good chance of success ( [Provence, 1989](#)).

That such a position is advocated by one whose studies of infants in institutions speak strongly against such care ( [Provence and Lipton, 1962](#)) is a measure of the growing need for alternative solutions for caring for children who cannot be maintained in their own families. It is also a realization that group residential care, in and of itself, does not have to be damaging. It appears to be a much-needed service at this time, and the knowledge of how to create a facilitating environment for infants and young children is advanced enough to make a strong argument for residential care in the spectrum of clinically sound services.

## TREATMENT OUTCOMES

Treatment outcomes in the short term are relatively easy to evaluate in infants and young children in terms of improvement across certain developmental and behavioral domains. However, judgment about the extent to which the type of treatment chosen and its specific elements have brought about change is not a simple matter. Long-term outcomes of treatment are even more difficult to evaluate. Nevertheless, the aggregation of individual carefully studied cases, as well as epidemiologic studies and evaluation of the later status of participants in early intervention programs will continue to inform the mental health field. The effectiveness of early childhood programs for socioeconomically disadvantaged infants and young children is now well established, including their long-term benefits. However, long-term outcomes, important in understanding the natural history of syndromes, should not determine the development of treatment programs. It is enough that we can recognize the need of a young child and family for psychotherapeutic and supportive services and have at hand clinically sound rationales and methods of implementing them.

## CONCLUSION

Studies of children's response to separation or deprivation inherently involve notions of risk and vulnerability and are fundamentally about the interaction between children's endowment and their parenting environment. [Werner's \(1990\)](#) report on protective factors and individual resilience—the positive counterparts of vulnerability and risk—summarizes major studies in this area. In the longitudinal study of the children of Kauai, protective factors, as well as risk factors, were identified. "The majority of resilient boys and girls were characterized by their caregivers as very active, affectionate, cuddly, good-natured, and easy to deal with, when they were infants" ( [Werner, 1990](#)). All elicited a great deal of warmth from their caregivers. Similar reports regarding resilient children ( [Murphy, 1987](#)) in middle-class families describe notable responsiveness to people and objects. Secure attachments to another family member were found in alert, responsive infants of abusive mothers ( [Farber and Egeland, 1987](#)). Resilient infants appear successfully to elicit positive attention, tend to be active, alert, and sociable, and have experienced nurturance from nonparental sources and have learned to trust its availability ( [Werner, 1990](#)).

In the contexts of secure, stable, loving relations, infants and children are more able to respond to separations, brief and long, in developmentally adaptive and facilitating ways that lead to adaptive coping, structure formation, and healthy defenses. In these contexts, separations prime the child's psychological immune system, allowing him or her to accommodate to the experiential viruses of the real world. Persistent distress, as in repeated moves from one foster home to another, predispose a child to failure in developing the ability to feel safe and secure with others or when alone, to enjoy reciprocity, or to be able to tolerate normal frustrations. Such distress is heightened in the presence of biological impairments, chronic illness, parental discord, psychiatric impairment, and inconsistent care. What constitutes a normal, immunizing dose and what overwhelms the mental adaptive immune system remain crucial questions for understanding the growth-promoting and growth-inhibiting factors involved in separation and loss.

Although separation and deprivation are not the principal topics of most contemporary empirical studies, continuing developmental research, in laboratory and clinical settings, in almost any special field one can imagine, will contribute to further understanding of early development and the impact of separation and deprivation. A trend of particular importance is in the acknowledgment of complexity in developmental processes and the trend away from isolation of variables and pursuit of linear concepts of causality toward the study of concurrent and interactive effects of multiple variables and the formulation of nonlinear concepts of causality ( [Sander, 1980](#)). A recognition of such complexity is reflected in the range of factors investigated by research since the 1980s as potential influences on infant and child attachment patterns (e.g., maternal clinical disorders, maltreatment, drug use, social support and marital quality, employment, and infant temperament, premature birth and illness) and by a concomitant increase in the number of studies that examine interactions between multiple variables. Nonetheless, a fuller understanding of the relative influence and complex relationships among identified and unidentified risk and resilience factors remains a task for future research.

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<sup>1</sup> Versions of this chapter appearing in earlier editions of this book reflect the work of our colleague and teacher, Sally Provence, M.D.

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## 36 GRIEF

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The death of a loved one is one of the most painful experiences that one can suffer. Unfortunately, bereavement is not uncommon in children. For example, 4% of children will lose a parent by the age of 15 years ( [National Center for Health Statistics, 1997](#); [Ventura et al., 1998](#)). The death of a parent is a traumatic loss that can jeopardize the child's sense of security and may have long-term adverse effects. Although there has been increased recognition of the impact of parental loss on children, relatively few methodologically sound prospective studies have examined the effects of parental loss, grief, its short-term and long-term sequelae, and the value of intervention. The significance of other losses, such as death of a sibling, is even less well understood.

In a discussion of grief, several terms need to be clarified. *Bereavement* is defined as a separation or detachment that leaves one despondent. It is the physical separation from the deceased. *Grief* is the emotional pain or anguish a person feels after the loss of a loved one. *Anticipatory grief* is a similar emotional pain that may occur before an impending death. It may be experienced before the expected loss of a loved one from a terminal illness such as cancer. *Mourning* is somewhat different from grief. According to Bowlby ( [1961a](#), [1961b](#), [1980](#)), it is defined as the psychological processes that are set in motion by the loss of a loved object that lead to the relinquishment of the object.

According to the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) ( [American Psychiatric Association, 1994](#)), normal bereavement is not considered a psychiatric diagnosis. Instead, it is classified as a V code condition. V codes are conditions that may be a focus of clinical attention, but they are not attributable to any of the mental disorders. The DSM-IV suggests that, sometimes, with additional information, the presence of a mental disorder may become apparent. In some instances, however, a thorough evaluation fails to uncover any mental disorder. In other cases, the scope of the diagnostic evaluation has not been sufficient to establish the presence of a mental disorder. However, it may be useful to note the reason for contact with the mental health care system in these cases.

In adults, a depressive syndrome is frequently a normal reaction to the death of a close friend or relative. The most common symptoms are insomnia, poor appetite, and weight loss. Morbid preoccupation with worthlessness, marked psychomotor retardation, and prolonged impairment are uncommon symptoms that, if present, may suggest that bereavement is complicated by the development of major depression. The reaction to the loss rarely occurs after 2 or 3 months. The duration of "normal" bereavement varies among different cultures. In a classic bereavement study by [Clayton and Darvish \(1979\)](#), 42% of widows were depressed immediately after the death of a spouse. By the end of 1 year, however, only 16% were clinically depressed.

A prime concern regarding bereaved children is whether parental loss in childhood leads to psychopathology in adulthood. There is a lack of consensus on whether the death of a parent during a person's childhood increases the risk of problems in adulthood. Several studies address this issue from different perspectives. However, there is no clear answer. In studies of psychiatrically disturbed adult patients, there are two schools of thought. The first believes that loss of a parent in childhood has a detrimental effect that lasts into adulthood. Psychopathologic conditions such as depression, schizophrenia, drug abuse, and alcoholism have been attributed to childhood bereavement. Other investigators believe that parental loss in childhood does not contribute to adult psychopathology if the care of the child is assumed by parental substitutes ( [Osterweis et al., 1984](#)). Breier et al. (1988) conclude that child care after the loss of a parent is a crucial factor in relation to adult psychopathology.

[Kissane et al. \(1996\)](#) examined the psychosocial morbidity and coping patterns among 115 family members using a family functioning typology. These investigators found that sullen families displayed the most intense grief and the most severe psychosocial morbidity. Supportive and conflict-resolving families were considered well functioning. They resolved their grief and had a more adaptive adjustment than their less functional counterparts. The authors suggest that families at risk of increased morbidity may be identifiable and can be treated preventively to reduce psychosocial morbidity among surviving parents and children.

[Dilworth and Hildreth \(1998\)](#) believe that prior research efforts to study the long-term impact of childhood bereavement often produced ambiguous results because much of this research was conducted in psychiatric populations. When these investigators studied a normative population, the symptoms of unresolved grief of adult survivors of early parental death were identified by using an extended model of Bowlby's variants of grieving in children ( [Bowlby, 1980](#)). These variants included persistent anxiety, desire to die, persistent blame and guilt, compulsive self-reliance, and aggressive outbursts. The authors maintain that practitioners will be better able to identify and treat unresolved grief if these variants can be identified in affected adults. However, [Rainieri and Lester \(1997\)](#) did not find an association between bereavement and increased depression or suicidality in a sample of undergraduate students who lost parents during their first 18 years of life.

Additional research is needed to describe more clearly the prevalence and nature of psychiatric morbidity among adults who were parentally bereaved as children. It seems prudent, however, that practitioners attempt to recognize and address the signs and symptoms of complicated bereavement in children in an effort to minimize the possibility of morbidity and dysfunction in later life.

### EPIDEMIOLOGY

As stated earlier, 4% of children lose a parent by the age of 15 years. Yet childhood bereavement has not been studied, as well as some conditions that are far less common. The reason may be, in part, that death is a topic that many wish to avoid altogether. It is difficult for many people to be objective about this subject.

Although the death of a parent is known to be a traumatic loss for a child, there is little known about how a child may be affected by the death of a sibling. Some investigators theorize that the loss of a child in a family may have a greater impact on parents than the loss of a spouse, and this could, in turn, contribute to a more troubled environment for surviving children ( [Worden et al., 1996](#)). Some have hypothesized that if adults could come to terms with death themselves, they could better handle the emotional reactions of their children to death.

A child's reaction to death depends on his or her emotional and cognitive maturity because this affects one's understanding of death. After studying 378 children, [Nagy \(1948\)](#) believed that a child's concept of death falls into one of three categories, based primarily on age. Three- to 5-year-old children look at death as sleep or a long journey. Children from 5 to 9 years old are able to accept the fact that someone can die; however, they do not believe it happens to everyone and especially not to themselves. By 9 to 10 years of age, children are able to think of death as inevitable and know it may happen to them ( [Chapter 94](#) and [Chapter 104](#)).

There is little consensus among researchers on the percentage of surviving children who are at risk of disturbed behavior. Studies to date have methodologic limitations that include the following: (a) a lack of demographically matched nonbereaved controls; (b) the use of different measures to assess psychological problems, a feature that makes comparisons across studies difficult; (c) the use of parents and teachers as informants to the exclusion of the children; (d) the use of small and unrepresentative samples of bereaved children; and (e) the use of different age cohorts. Most studies use a cross-sectional design rather than a longitudinal design, which is preferred, given developmental changes in children with time ( [Worden, 1996](#)).

The most widely cited research on the impact of parental death on children consists of retrospective studies of psychiatrically ill adults who lost a parent in childhood and prospective studies of psychiatrically ill children who were in psychiatric treatment before their parent's death ( [Furman, 1974](#); [Wolfenstein, 1966](#)). Often such studies have few subjects, so it is difficult to generalize their findings.

Another group of studies comes from three different research groups that studied the same sample of bereaved adolescents at different points in time ( [Bendickson](#)



and Fulton, 1976; Gregory, 1965; Markusen and Fulton, 1971). The bereaved adolescents were compared with normal control subjects who were recruited at the same time and were also followed-up at the same time intervals. When first studied in the tenth grade, the bereaved group had a higher rate of delinquency. In their early twenties, the bereaved group had more legal offenses. Finally, when studied in their thirties, the bereaved subjects reported more serious medical illnesses and less satisfying lives.

Kaffman and Elizur (1983) conducted a prospective study of children 2 to 10 years of age who lived in a kibbutz and who had lost a parent in a national war. These children often had sleep disturbances, social withdrawal, and restlessness. When these children were compared with age-matched children who had lost a parent but who did not live in a kibbutz, living in a kibbutz did not appear to be a protective factor for the development of psychiatric symptoms.

The Child Bereavement Study (Worden, 1996) investigated a community-based sample of 125 parentally bereaved children from 70 families and compared them with matched controls. This study sought to identify behaviors that were overrepresented in bereaved children when compared with matched controls. The surviving parent and the child were assessed using semistructured interviews. In addition, the Childhood Behavior Checklist (CBCL) and measures of self-concept and self-efficacy were obtained. Subjects were interviewed 4 months after the parent's death and again at the first and second anniversaries of the loss. Worden et al. found bereaved children as a group showed the same number of disturbed behaviors as did controls on the total problem scales up to 1 year after the death of a parent. Interviews of these same children at 2 years after the loss showed significant differences from their controls. When age and gender were considered, preteen bereaved girls showed more disturbances than matched controls at 2 years after the parent's death; however, these differences were not apparent at 1 year. After 2 years, preadolescent girls were experiencing more anxiety, depression, and aggressive behaviors than controls. Adolescent boys were more withdrawn and had more social problems than their matched controls. Adolescent girls and preadolescent boys showed no differences from their matched controls at 1 or 2 years. At 2 years, bereaved children exhibited higher levels of anxiety, depression, and social withdrawal. There were no significant group differences in delinquent behavior, aggressive behaviors, somatic complaints, or attention-seeking behaviors at either 1 or 2 years. The bereaved children had significantly lower scores on measures of self-worth and locus of control than did their age-matched controls. Most bereaved children in this study did not exhibit signs of serious emotional or behavioral disturbances. However, a sizable proportion of bereaved children at 1 year (19%) and at 2 years (21%) manifested serious problems that could require intervention.

Some studies suggest that the cause of parental death may influence the adjustment of bereaved children (Ness and Pfeffer, 1990; Shepard and Barraclough, 1976). For example, each year, 7,000 to 12,000 children lose a parent to suicide (Small and Small, 1984). Children who are bereaved as the result of a parent's suicide have been assumed to be at significant risk for psychopathology. However, only a few controlled studies have studied this situation. Pfeffer and colleagues (1997) examined 22 children aged 5 to 14 years from 16 families in which a parent or child had committed suicide. These children showed internalizing symptoms and problems with school adjustment 1 to 36 months after the death. Approximately 40% of these children experienced symptoms of posttraumatic stress disorder; 25% of families had children with elevated Children's Depression Inventory scores. Unfortunately, use of a clinically referred sample, absence of a control group, and wide variations in the time interval between death and assessment limit the generalizability of this study.

Gutierrez (1999) examined the effects of exposure to suicidal behavior in a parent on suicidal behavior in the child. Subjects were 25 parentally bereaved adolescents aged 13.4 to 19.6 years who were not clinically referred. They were assessed with semistructured interviews. Although these adolescents had experienced the death of a parent, they were not clinically depressed, did not express serious levels of suicidal ideation, and had relatively little suicidal behavior. Limitations of this study included small sample size, absence of a comparison group, and lack of minority participation.

Cerel et al. (1999) compared 26 parentally suicide-bereaved (SB) children with 332 parentally non-suicide bereaved (NSB) children in a 2-year longitudinal study. The purpose of the study was to determine the impact of parental suicide on children's grief reactions, psychiatric symptoms, and psychosocial functioning after the parent's death. SB children were more likely to experience anxiety immediately after the death, followed by anger at 6 months and shame at 1 year after death, relative to NSB children. They were less likely than NSB children to report relief immediately after the death or acceptance 1 and 2 years after the death. SB and NSB children did not differ regarding symptoms of posttraumatic stress disorder, suicidality, depressive symptoms, or severity of depression or psychosocial impairment. SB children had fewer missed days of school because of illness and had fewer physician visits for health-related problems. Limitations of this study included small sample size and underrepresentation of minority populations.

In an extension of the foregoing study, Cerel et al. (2000) examined psychopathology in the deceased and surviving parent, family stability before entry into the study, family functioning before and after the parent's death, and changes in social support and religious practices in the same 26 SB children and in 332 NSB children. Parents who had committed suicide demonstrated more psychopathology than parents who died by other means. Surviving SB parents were not more severely impaired than surviving NSB parents. SB families were less stable than NSB families. The SB children appeared to have a poorer relationship with the deceased parent than the NSB children. However, SB and NSB children had a similar relationship with their surviving parent. SB and NSB families had few differences in social supports or changes in religious beliefs after the parent's death. In general, SB children came from families with a history of psychopathology and a high degree of family disruption. Surviving SB parents, however, did not exhibit higher rates of psychopathology than NSB parents, and most had positive relationships with their children.

## SIBLING LOSS

A child's response to the death of a sibling has been studied less than a child's response to the death of a parent. Studies to date suggest that the loss of a sibling is potentially traumatic (McCown and Davies, 1995; Stahlman, 1996). When a child dies, a parent's grief is more likely to be severe, to be "complicated," and to last longer than grief associated with other losses (Rando, 1986). Thus, a sibling-bereaved child must deal both with his or her own grief and with the family environment, which is likely to be altered as a result of the parents' profound grief. For example, the surviving parents' ability to fulfill parental roles may be compromised

McCown and Davies (1995) examined 90 sibling-bereaved children over a 2-year period. Subjects were aged 4 to 16 years. Parents were interviewed about their children's behavior using the CBCL. Most children demonstrated externalizing behaviors that fell onto the aggression subscale of the CBCL. Behaviors such as "demands a lot of attention," "is disobedient at home," "is stubborn, sullen and irritable" were observed in a majority. Boys showed almost exclusively aggressive behaviors. Girls scored high on the internalizing depression subscale "self-conscious" or "easily embarrassed." The highest occurrence of behavior problems was in preschool and school-aged children. Most of the foregoing behaviors declined with increasing age. The lowest occurrence of behavior problems was in adolescents. Behaviors observed in more than 50% of adolescent girls included secretiveness and a preference for being alone. The three behaviors most frequently observed across age groups were "argues a lot," "is stubborn or irritable," and "is self-conscious easily embarrassed." This study was limited by the exclusion of children as informants.

Mahon and Page (1995) assessed sibling bereavement during childhood in 35 subjects from 11 families using qualitative measures. Surviving siblings ranged in age from 4 to 23 years at the time of assessment. Children's perception of their own bereavement and parents' perception of their children's bereavement were studied. Children reported the following as helpful: mother's support, support from friends who had lost someone through death, activities that were supportive before the sibling's death, and open communication with parents and surviving siblings. Children reported the following as not helpful: large number of funeral attendees who gathered at their homes, friends who avoided them after the sibling's death, and withholding of factual information about the death. In a few cases, the father was identified as unhelpful. All the children were functioning well. This study also suggests that children benefit from being informed of imminent death and that children should have an opportunity to decide whether to be at the deathbed or attend a funeral to say their good-byes.

Although the death of a parent and the death of a sibling are both traumatic events, there is limited information on whether a child's bereavement response varies depending on which type of loss occurs. Worden et al. (1999) combined two samples of bereaved children: (a) parent-bereaved children from the Massachusetts General Hospital study by Worden and Silverman and (b) sibling-bereaved children from the study by McCown and Davies (both studies are cited earlier in this chapter). The CBCL was administered to subjects in both studies at 4 months, 12 months, and 24 months after the death of the child's parent or sibling. This study, looking at data from the assessment conducted at 4 months, found no significant difference in the total number of CBCL-identified problems. Nor were there significant group differences between syndrome subscale scores until gender was considered. Overall, most children were doing well and did not need clinical intervention during the first year of bereavement. However, boys who lost a parent had significantly higher scores on withdrawn behavior than boys who had lost a sibling. The difference was greater for preteen boys than for teenage boys. Girls who lost siblings were more anxious, were more depressed, and had more cognitive difficulties than girls who had lost a parent. Girls who had lost a sibling tended to have more attention problems, somatic problems, and delinquent behaviors than did girls who had lost a parent. The boys who had lost a parent were more likely to have scores falling into the clinically significant range on the inward-directed problems scale, specifically withdrawn behavior, than sibling-bereaved children. More of the sibling-bereaved girls fell into the clinically significant range on the inward-directed problems scale, specifically the anxious, depressed, attentional, and thought problem scales. Of the boys who lost a parent, most had lost fathers. This was identified as an important risk factor during the first year of bereavement. For the girls in the sibling study, most had lost a sister.

The few studies of sibling loss suggest that children who lose a sibling are at no greater risk of emotional or behavioral problems than those children who lose a

parent. Most children who lose a sibling go on to do well and do not need clinical intervention. However, some of these children need help after these losses. These studies suggest that younger children (preschool and school-aged children) and girls may have more difficulties adjusting to the loss of a sibling and may manifest these difficulties with internalizing symptoms such as anxiety, depression, withdrawn behaviors, or externalizing behaviors such as attention problems or aggression. Although these studies are preliminary, they highlight the need for further study of this group of children, to help those who care for grieving children understand their behavior and to give them the support they need.

## CLINICAL DESCRIPTION

A child psychiatrist may be asked to evaluate a bereaved child to determine whether the child manifests normal grief or whether symptoms represent more serious psychiatric disturbance. Unfortunately, at present, only limited information is available from research studies to help to delineate normal grief from psychopathologic conditions in children. To provide systematic data to address this issue, Weller and Weller are conducting a research project sponsored by the National Institute of Mental Health to describe grief in parent-bereaved psychologically normal children. Although this study is not yet completed, preliminary data will be presented in the hope of providing some useful information.

In the bereaved prepubertal children studied to date, the most common presentation includes sadness and yearning for the dead parent. Prepubertal children often state that they wish to die, so they can see and visit the deceased parent. They clearly say that they do not seek death in the same way that a child with major depression does. Suicide attempts are very rare in bereaved children and should be immediately evaluated if they occur. Problems with sleep, appetite changes, difficulty with concentration, and not "having fun" with normally pleasurable activities are common. Frequently, surviving parents are not aware of these difficulties in their children. Schools tend to be sympathetic toward bereaved children and will not send them for treatment unless there is a major problem with academic or social functioning. Sometimes, bereaved children with headaches and stomachaches are taken to pediatricians for evaluation. In adolescents, problems are similar but tend to be less frequent than those observed in bereaved prepubertal children. However, it is not clear whether adolescents actually have fewer symptoms or merely report fewer symptoms. It is possible that adolescents may be either more hesitant to disclose their discomfort or unwilling to report symptoms that make them appear different from peers ([Hogan and Greenfield, 1991](#)).

A child's reaction and adjustment to a parent's death often appear to be related to the adjustment of the surviving parent. An earlier study by [Weller et al. \(1991\)](#) compared symptoms of 38 bereaved children and 38 depressed children. DSM-IV suggests that thoughts of death such as "the survivor feeling that he or she would be better off dead" or "should have died with the deceased person" are normal after bereavement. In this study, 61% of the bereaved children had suicidal ideation either by parent or child report. None, however, actually attempted suicide. Eighty-nine percent of the referred clinically depressed children reported suicidal ideation, and 42% of them had attempted suicide. Depression and other psychiatric problems in bereaved children were predicted by preexisting psychiatric disorders in the child or a family history of a psychiatric disorder (most often depression) in the surviving parent. Most children had their psychiatric symptoms "peak" at 1 month after the parent's death. However, one-third had peak symptoms 6 months after the parent's death, and one-sixth had peak symptoms 1 year after the death.

It is important to be supportive of a family that has had a major loss. This is especially true in the case of a bereaved family that includes dependent children. In this situation, the surviving parent often becomes depressed, especially if he or she has a predisposing factor (e.g., past history of depression or family history of depression). Bereaved families experience hardships, both financial and emotional. The socioeconomic status of a bereaved family usually declines. This may result in a move to a home in a neighborhood with a lower socioeconomic level and a change in schools. Moreover, the surviving parent may choose to move closer to his or her family of origin to receive more support from them. This change causes the child to be uprooted from familiar surroundings and to lose friends in addition to losing a parent.

As a practical matter, a grieving child often loses more than the deceased parent or loved one. Surviving parents are often engulfed in their own grief and are unable to provide normal parenting. In the case of the death of a parent, the surviving parent is most often the mother because 70% of parents who die with dependent children are men ([Gutierrez, 1999](#)). As a result of the death, a mother who had not been previously employed outside the home may now have to join the workforce to support the family. Children tend to resent this change, especially when they are asked to do additional chores in the home or to stay at home alone before or after school while waiting for the working parent to return. Children may also resent the surviving parent if he or she shows interest in dating or having adult companionship. Children may feel this as a betrayal of the dead parent. As children grow and mature, the manner in which they manifest grief may change. For example, a child whose grief appears to be resolving may show a resurgence of symptoms when an upcoming birthday or graduation ceremony approaches. Adolescents especially tend to grieve over the role the dead parent would have had in their lives (e.g., the bereaved parent will not be present for special events such as their marriage or birth of a child).

As discussed earlier, there is limited literature comparing parental and sibling loss. Although the death of a sibling represents a loss, it does not usually have the same implications for interfering with development that death of a parent does ([Nagera, 1970](#)). However, the loss of a sibling does present unique issues. The surviving sibling may have particular difficulty dealing with guilt, denial, a heightened sense of vulnerability, and fear about his or her own well-being ([Stephenson, 1986](#)).

As a result of his work with parent- and sibling-bereaved children, [Worden \(1996\)](#) developed the following list of the differences between parent loss and sibling loss.

1. Loss of a child may have a greater impact on parents than loss of a spouse and, thus, may contribute to a more troubled environment for surviving children.
2. Sibling loss leaves two parents to support the child, whereas parent loss leaves one.
3. Marital tensions that occur in child loss are not present in parent loss.
4. Sibling loss increases the child's personal awareness of death more than parent loss.
5. The sibling-bereaved child may experience the parents' need to replace the deceased child.
6. Parents who lose a child may overprotect the surviving children more than the single surviving parent who remains when a spouse dies.
7. After the death of a child, there may be a need to blame or scapegoat. This can also happen in parent loss but is less frequent.
8. In sibling loss, the surviving child(ren) may have been jealous of the sick sibling. Thus, there may be ambivalence to work through after the death.
9. Survival guilt after sibling loss may be exacerbated by sibling rivalries.
10. In sibling loss, children may be angry with parents for not protecting the dead sibling.
11. In some families, one of the parents may feel that the "wrong" child died.
12. Outsiders may provide more support for the child who loses a parent than for the child who loses a sibling.

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### CASE ILLUSTRATION

J.W. was a 9-year-old boy who had two siblings with a rare genetic disorder that manifested cardiac symptoms. Four months after the death of his 7-year-old brother, J.W. was referred to an outpatient psychiatrist for evaluation of "feeling like he wanted to die by choking himself." J.W. believed that he should be with his brother but was not sure whether he would choke himself. His mother, a single parent, reported that J.W. had been sadder, more withdrawn, and less interested in "everything" since his brother's death. He had previously been a good student with many friends but was now performing poorly at school and had lost interest in his friends. He now worried that his mother and his remaining siblings would die, especially his younger sister, who had a similar cardiac condition. He had difficulty sleeping at night and had some appetite loss. He reported feeling guilty because he should have been taking care of his little brother and had sometimes fought with him. He was quite expressive about missing his brother and was worried that his sister and his mother would die. His mother reported that he used to talk to her about things but stopped after his brother's death and would never discuss his sadness about his deceased brother. The family was referred for counseling.

Seven months after his brother's death, J.W. was doing better. He was able to talk to his mother about his worries and concerns. He shared his worries about his sister's dying during surgery and was better able to talk with his mother about his deceased brother. He began to show improvement in his academic performance and to socialize more with his friends. His mood and affect improved, but he acknowledged his sadness about his brother. He denied any further death wishes or thoughts of joining his brother.

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### CASE ILLUSTRATION

M.S. was 14 years old when her father died of lung cancer. She had been anticipating his death for a year. She was the youngest of seven children and was a good student in advanced English and science classes. She was closer to her father than to her mother. When she knew her father was dying, she refused to leave the hospital for 1 week and stayed by his bedside until he died. One month after the death, she had dysphoria, loss of interest, insomnia, loss of energy, recurring nightmares, and trouble concentrating. She was also having repeated stomachaches because of "worrying." Her menstrual cramps had become worse since her father's death, and her grades dropped in school. She was not sure whether her friends understood her feelings and was trying "to be strong for my mother." She was not pleased with her mother's decision to move out of their home "to get away from the memories." Six months after the death, she was not able to talk about her father without crying. She reported her father's death as "a shock," and she was visiting his grave twice a week. Her mother had begun dating, and this was upsetting to M.S. She had no energy and was becoming a loner. She had thoughts of suicide but reported, "I couldn't do it because it would be painful to my mom." She was staying in bed and not going to school because she was unable to fall asleep and was frequently waking up in the middle of the night. She was referred for counseling and evaluation for adjunctive pharmacotherapy.

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## BEREAVEMENT AS A RISK FACTOR FOR PSYCHOPATHOLOGY

As mentioned earlier, bereavement in childhood has been considered a potential risk factor for subsequent psychopathology (Brier, 1989). The most common



psychiatric syndrome experienced both by children and adolescents in the immediate period after parental death is depression ( [Weller and Weller, 1990](#) ). The data for children and adolescents with sibling loss are less clear. However, depression, anxiety, and externalizing aggressive behaviors have all been described ( [McCown and Davies, 1995](#); [Davies, 1998](#); [Worden and Silverman, 1996](#); [Worden et al., 1999](#) ). Interestingly, a preliminary study found that bereaved children with a positive dexamethasone suppression test have more DSM-IV depressive symptoms than do bereaved children with a negative dexamethasone suppression test ( [Weller et al., 1990](#) ).

In regard to anxiety symptoms, earlier studies by the authors found few anxiety symptoms in the short-term period (1 to 3 months) after parental death in children and adolescents ( [Weller et al., 1991](#) ). Children reported anxiety symptoms more often than adolescents. Worrying about their surviving parent and worrying about “little things” were significantly more common in children than in adolescents. [Worden \(1996\)](#) found no difference in the presence of anxiety symptoms in children after the death of a parent until 2 years after the loss. At 2 years, the bereaved children showed higher levels of anxiety, social withdrawal, and social problems, as well as lowered self-esteem and self-efficacy. Although most bereaved children showed no serious emotional or behavioral disturbances, significant numbers of children at 1 year (19%) and 2 years (21%) had severe emotional and behavioral problems.

Somatic symptoms have been hypothesized to be increased in bereaved children because they may use such symptoms to express their emotions. [Sood et al. \(1992\)](#) found that bereaved children reported significantly more somatic symptoms than did bereaved adolescents, but overall, there did not appear to be a marked increase in somatization. The most common complaints were headaches, stomachaches, and nausea. The absence of higher rates of somatization among bereaved children has been reported in studies of parent- and sibling-bereaved children ( [Cerel et al., 1999](#); [McCown and Davies, 1995](#); [Worden and Silverman, 1996](#) ).

One group of parent-bereaved children who appear to be at particular risk of psychopathology comprises those who lost a parent to suicide. In the sample of SB children studied by [Cerel et al. \(1999\)](#), these children had more overall symptoms of psychopathology than NSB children at every time period in the study except the month preceding the death and 2 years after the death. These children were also reported to be more globally behaviorally dysregulated than the NSB children. They also showed higher overall levels of anxiety and significantly more depressive episodes and symptoms in their lifetimes. However, suicidality was not increased. Furthermore, because parental suicide is thought to be associated with a family history of mental illness, specifically mood disorders or substance abuse, these children may be at increased genetic risk of developing these disorders.

### Differential Diagnosis

The DSM-IV clearly differentiates *bereavement* (a V Code) from *adjustment disorder with depressed mood* ( [American Psychiatric Association, 1994](#) ). The DSM-IV categorizes an *acute adjustment disorder* as having symptoms that last less than 6 months after a disturbance. *Chronic adjustment disorder* occurs when symptoms last for longer than 6 months after a major stressor that has enduring consequences. Adjustment disorder with depressed mood does not fulfill the criteria for major depression, but significant depressive symptoms are present over a period of less than 6 months. The disorder is considered chronic if symptoms are present for more than 6 months.

As mentioned earlier, all the signs and symptoms of depression may occur after bereavement. A preliminary study found that 37% of bereaved prepubertal children met criteria for depression 1 month after parental death ( [Weller et al., 1991](#) ). In major depression, suicidal thoughts and attempts are common. In grief, suicidal thoughts may be present. Such thoughts, however, usually focus on wanting to reunite with the dead parent and do not involve self-deprecatory thoughts of wanting no longer to exist. Suicide attempts are uncommon in uncomplicated grief. Thus, if they occur, these children should be brought to immediate psychiatric attention. Anxiety symptoms may occur, but diagnosable anxiety disorders are uncommon immediately after death ( [Sanchez et al., 1994](#); [Worden and Silverman, 1996](#) ). Thus, the presence of an anxiety disorder should not be automatically attributed to the grieving process and should be evaluated. Somatic symptoms should be handled in a fashion similar to that for anxiety disorders because somatization in bereaved children and adolescents is uncommon ( [McCown and Davies, 1995](#) ). When somatic symptoms occur, headaches and stomachaches are the most frequent complaints, especially in prepubertal children ( [Sood et al., 1992](#) ).

The possibility that conduct disorder may occur was suggested by [Markusen and Fulton \(1971\)](#). However, more recent studies ( [Worden and Silverman, 1996](#) ) found no differences in delinquent behavior between bereaved children and age-matched controls.

### TREATMENT

If grief is a normal and necessary reaction to the death of a loved one ( [Bowlby, 1961a](#) and [1961b](#), [Bowlby, 1980](#); [Bowlby, 1982](#); [Freud, 1966](#) ), then intervention may not be necessary. Most people go through the grief process with a painful sense of growth and responsibility. Being a compassionate listener and lending support to grieving families may be all that is necessary for most bereaved families. One of the first decisions a grieving parent has to make is how to inform the children of the death. Questions such as who should tell the children, how should the death be explained, and should the children be allowed to attend the funeral need to be addressed. Earlier studies on the advisability of children's attending their parents' funerals are somewhat contradictory. To address this issue, [Weller et al. \(1988\)](#) studied psychologically normal prepubertal children shortly after the death of a parent. Results found that the surviving parent most often told their children about the death. Most children wanted to go to the funeral and participated in making arrangements for the funeral. Most also reported that participating in these activities made it easier for them to accept the loss and that they were glad they attended the funeral. However, children should be prepared for the funeral process. The parents should identify a familiar person to accompany the child who will be able to comfort the child or to remove the child from the situation if the child becomes unduly distressed. This is especially important if the parent will be unable to comfort the child ( [DeMaso et al., 1997](#); [Schaefer and Lyons, 1993](#) ).

In explaining death to children (especially very young children), it is important not to compare death to sleep because this may increase a child's anxiety about whether he or she too could die when going to sleep. A good explanation is to describe death as cessation of bodily activities. Often grieving parents and children find comfort in reading about the subject. Some, but not all, may benefit from self-help groups. Sometimes these groups combine bereaved and divorced families. This combination, however, should usually be avoided. The issues for the two groups can be quite different, and resentment can occur in such combined groups. For example, grieving children tend to be less angry than children from a family experiencing divorce. Grieving children yearn for the dead parent and want to remember good times. Bereaved children often know that the dead parent did not choose to part from the child. However, children from a divorced family often blame the parents for wanting to part from them. Children from a divorced family often have friends who are from divorced families, whereas fewer children have friends who have lost a parent through death. Thus, it is more difficult for grieving children than for children from a divorcing family to find peers with whom to identify. A parent should not be pushed to join an adult support group unless the parent wants to participate.

For parents dealing with loss of a child, it is important to explain the death to the surviving siblings in an age-appropriate and developmentally appropriate way ( [Nolen-Hoeksema and Larson, 1999](#) ). This requires parents to use concrete language, to be open and honest about the child's illness and death, and to share their feelings of sadness and grief with their children ( [Koocher, 1974](#) ). Consistent family routines and discipline should be maintained. It is also important to secure the support of family and friends who are familiar to the child, to help provide continuity in the bereaved child's daily family life ( [DeMaso et al., 1997](#) ).

Baker et al. (1992) believe that treatment after the loss of a child's loved one should be psychoeducational. Parents of the child may need education on the needs of a grieving child. This information can help them to explain death and mourning to the child and also help with any questions a child may have. Children's literature, such as the E.B. White classic *Charlotte's Web* (1952), *Daddy's Promise* by Cindy Klein-Cohen (1997), and *Sad Isn't Bad* by Michaelene Mundy (1998), may provide some additional help for parents in these discussions ( [Johnson and Johnson, 1982](#); [Sims, 1988](#) ). These and other selections provide life-affirming advice for coping with loss as a child. They can help children to process their grief and help parents to understand the behaviors they may see in their grieving children.

If a child needs to have a new caregiver selected, as a result of the deaths of both parents or the inability of the surviving parent to care for the child, first-degree biological relatives should be given primary consideration in accordance with the permanency planning guidelines of the [Adoption and Safe Families Act \(1997\)](#). Additionally, the developmental and emotional needs of the child should be taken into account when a new primary caregiver is selected. The more permanent a child's placement, the more likely he or she will be to adapt to the new environment and progress through the bereavement process.

Individual therapy can be helpful for those with complicated grief, that is, grief that has become pathologic. Individual therapy may help the bereaved work through their grief and achieve a healthy outcome. Family therapy can be helpful for a dysfunctional family in which some family members are having difficulty adjusting to the loss.

In adults, sleep disturbances and appetite disturbances are symptoms frequently treated by family physicians. However, these are also symptoms of depression. One should ensure that major depression is not overlooked. Sleeping pills are not the best medications for depression. An antidepressant may be helpful to a grieving parent who develops clinical depression and “can't shake it,” especially if there is a history of depression that preceded the bereavement and that responded to

pharmacologic intervention.

In children and adolescents, few data are available on how to differentiate normal grief from pathologic grief. Guidelines on when to intervene are lacking. In cases of pathologic grief, depression should always be considered. Further, in such situations, extreme caution should be taken to ensure that the child is not suicidal or involved in acts that could put his or her life in danger (e.g., drug or alcohol abuse or reckless driving). Major depression that interferes with a child's ability to function (scholastic, family, peer group) should be carefully evaluated and treated with individual therapy and medications as indicated ( [Weller, Weller, and Fristad, 1984](#) ). Children with a history of depression before the bereavement may be more likely to require antidepressant treatment than those who have no such history. In considering the use of an antidepressant medication in children and adolescents, the clinician should always take into account the lethality of such medications in a potentially suicidal patient. In children and adolescents, having a responsible adult (i.e., a parent) in charge of dispensing medication can minimize the risk of medication overdose.

In the parent-bereaved family with a clinically depressed child, the surviving parent may also be depressed. Thus, extra care should be taken in selecting the adult to be responsible for the child's medication. When using medication, a physical examination, thyroid, and liver function tests should always be done. Available evidence suggests that the selective serotonin reuptake inhibitors are well tolerated by children and adolescents and may be a good choice for treating depression in these groups ( [Ambrosini, 2000](#); [Emslie et al., 1997, 1999](#) ). [Emslie et al. \(1997\)](#) completed a randomized, double-blind, placebo-controlled trial of fluoxetine in children and adolescents with severe, persistent major depressive disorder. These investigators report fluoxetine to be superior to placebo and well tolerated. Current recommendations are that fluoxetine be initiated at a dose of 5 mg per day and increased every 7 days until a dose of 20 mg per day is obtained. The recommended maximum daily dose of fluoxetine is 80 mg. Most children respond to 20 mg per day.

Paroxetine has also been found effective and well tolerated in the treatment of major depression in children ( [Rey-Sanchez and Gutierrez-Casares, 1997](#) ). Paroxetine should be initiated at a dose of 10 mg per day and increased every 7 days up to a maximum dose of 60 mg per day.

Generally, bereaved children do not show serious emotional or behavioral disturbances after the death of a parent. However, some children do exhibit serious emotional and behavioral problems after such a loss ( [Cerel et al., 1999](#); [Pfeffer et al., 1997](#); [Worden, 1996](#) ). Many of the children who exhibit persistent psychiatric symptoms are not identified until 1 to 2 years after the death of a parent ( [Weller et al., 1991](#); [Worden, 1996](#) ), even though they did not express these symptoms early in the process of grieving. Children who may be identified at higher risk of developing psychopathologic conditions after parental loss are SB children ( [Cerel et al., 1999](#) ), children with preexisting psychopathology, and children whose families resolve conflicts poorly or exhibit high rates of psychopathology ( [Cerel et al., 2000](#); [Kissane et al., 1996](#) ). These children and their families may benefit from early supportive interventions, education about the potential for difficulties long after the death, and longer follow-up periods after the death of a parent.

## RESEARCH DIRECTIONS

Previous studies of grief in children have suggested a possible link between bereavement and subsequent illness. However, many early studies suffer from small sample size and methodologic weaknesses that limit their generalizability. Several studies have suggested that a depressive syndrome is common sequela of bereavement in adults. Studies of bereaved children and adolescents suggest that depressive symptoms are also common soon after the death of a parent or sibling ( [Cerel et al., 2000](#); [Weller et al., 1991](#); [Worden and Silverman, 1996](#); [Worden et al., 1999](#) ). However, long-term prospective studies of larger groups of subjects using standardized assessment techniques are needed to describe the natural course of grief and possible pathologic outcomes more completely. Some studies have suggested that several of the emotional and behavioral problems related to grief do not manifest themselves until 2 years after the loss ( [Geis et al., 1998](#); [Worden, 1996](#) ). Longitudinal studies that will follow these children prospectively into adulthood are needed. This will be especially important because most hospice programs for families and most bereavement groups currently follow children for only 1 year.

There continues to be a paucity of literature describing the grief process in preschoolers. One of the difficulties in studying this age group has been the lack of standardized instruments to assess 3- to 6-year-old children effectively ( [Kranzler et al., 1990](#) ). There are also few methodologically sound studies on sibling loss. Many of the studies on sibling loss are based on interviews with parents or other adult caretakers and not on interviews with surviving children.

In addition, studies of children's responses to the death of a loved one in relation to the circumstances of death would be important. For example, SB children are at risk of increased psychopathology ( [Cerel et al., 1999](#) ) compared to NSB children. Israeli adolescents who lost a parent in a national war had fewer psychiatric symptoms than those who lost a parent in an accident ( [Bachar et al., 1997](#) ). Younger siblings of murder victims were reported to exhibit more psychiatric symptoms after the death of the sibling than those who lost a sibling from other causes ( [Freeman et al., 1996](#) ). Further research in sibling loss, as well as on the response of children to the loss of close friends and grandparents, is needed. In all these studies, the risk factors for psychopathology need to be identified to allow for early intervention and possible prevention.

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# 37 EFFECT OF DISORDERED PARENTING ON THE DEVELOPMENT OF CHILDREN

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Evolutionary theory suggests that living things should be concerned with the production and rearing of offspring to ensure genetic representation in the next generation ([Barash, 1977](#)). In fact, human adults all over the world see children as helpless, needing protection and training, but also as a valuable asset to society ([Mead and Wolfenstein, 1955](#)). This explains the common aim of parents everywhere to raise their children to be independent, responsible adults capable of full participation in society ([LeVine, 1977](#)), and it may also be reflected in the great similarity that has been found in early child-rearing practices all over the world ([Ainsworth, 1957](#)).

Conversely, because children and their parents have different genetic makeups and different needs, parental behaviors will not always be optimal for each infant at all times. The degree to which these variations can affect the development of children is examined in this chapter.

## THEORETICAL ISSUES PERTAINING TO DISORDERED PARENTING

Textbooks of child and adolescent psychiatry have not traditionally defined what constitutes adequate, optimal, or disordered parenting. This is undoubtedly the result of the enormous complexity of the caretaking function. In addition to the instrumental tasks involved in keeping a child healthy and providing him or her with sufficient clothing and shelter are the many psychological factors that make up the parenting role. Parenting also has an important developmental component. For example, mothering an infant and a teenager requires some common skills, such as patience, but it also draws on very different ones. Whereas a newborn needs a mother who “understands” his or her body language and helps the infant to organize his or her psychological states into predictable rhythms, a teenager will more directly benefit from a parent who can step back and allow him or her to explore the range of socially acceptable experiences.

Describing its instrumental and developmental variations is only one way to characterize good and inadequate parenting. Another way of understanding parenting practices is to see them in terms of theoretical models of development. For example, there is a rich literature, based on psychoanalytic theory, that deals with such issues as maternal overprotectiveness ([Levy, 1943](#)) or the formation of abnormal defense reactions in children with particular family constellations ([Freud, 1937](#)).

Conversely, we have a large literature that describes how specific psychiatric or medical conditions may influence parenting styles. For example, we talk about the effects of maternal depression ([Cox et al., 1987](#); [Puckering, 1989](#); [Weintraub et al., 1986](#)) or abuse by adults ([Cicchetti and Toth, 1995](#)) on children and how children who live with “disordered” parents fare in their overall development. The advantage of this literature is the empirical nature of the presented data. However, many of these studies tend to ignore that the association between parental behavior and child disorders is not unidirectional, and it is necessary to evaluate possible contributions of genetic or other factors within the child in each case.

Other investigators have focused on the ecologic perspective of parenting. For example, parenting is usually embedded in the context of a family or a community, with all its stresses and potential support structures ([Bronfenbrenner, 1979](#)). Degrees of risk emanating from these contexts have been called “distal” factors ([Luthar, 1993](#)).

Despite the interest of developmentalists and clinicians in the effects of the environment on behavior, few studies have examined continuities of contextual risk, because such studies are complex. Nevertheless, there is strong evidence that the best predictors of competence during early childhood are (a) the number of years a family has spent in poverty ([Duncan et al., 1994](#)) and (b) the number of distinct adversities children and families have experienced ([Sameroff et al., 1998](#)).

As can be seen from this brief review, the literature on parenting has been driven by a wide range of theories and empirical data. This situation has often led to seemingly contradictory popular advice. Yet two things seem to be clear. Theories based on classical psychoanalytic or learning principles have failed to stand up to modern empirical findings because they do not sufficiently explain important aspects of children's development. Conversely, increasing evidence considers parenting to be based on genetic principles and the laws of developmental psychopathology ([Rutter et al., 1997](#); [Sroufe and Rutter, 1984](#)), thereby highlighting the importance of genetic and functional factors (e.g., parental emotional support, the role of an intimate confiding relationship) and structural factors (e.g., low socioeconomic status or mental illness in the family) for the quality of parenting. Thus, the emphasis has shifted from the classical psychoanalytic position in which undue importance was placed on the very early years of a child's life to determine later mental health to conceptualizing life as an ongoing developmental process.

This chapter first briefly reviews the history of work that has linked parenting styles with later problems in children. We then attempt to summarize some major characteristics present-day investigators associate with competent caretaking and comment on how specific parental disorders influence these characteristics. Because troubled parents communicate their difficulties through their child-rearing activities, we then examine some of the clinical manifestations of disordered parenting skills and how they develop. At that point, we also evaluate how children influence the parenting they receive and illustrate this with examples of children with developmental disorders. The chapter ends with some thoughts on the efficacy of parent education and draws attention to some unresolved issues requiring further study.

## VIEWS OF PARENTING, THEN AND NOW

The importance of linking specific caretaking experiences in early life with later psychopathology has long been recognized. John Locke (1632 to 1704) wrote an essay in 1693 entitled “Some Thoughts Concerning Education,” wherein he described the consequences of specific parenting styles. He admonished parents to observe their children carefully to understand their “native propensities” and emphasized their role in “weeding out” their faults and “planting good habits” early in life (Locke, 1947). Locke's influence was such that he has been called the Dr. Spock of the 18th century ([Brant and Cullman, 1980](#)).

One hundred years later, Jean-Jacques Rousseau wrote extensively about the consequences of specific parenting practices such as coddling and neglect on the



future development of young children. In his book *Emile* ([Rousseau, 1762/1979](#)), he spoke at length about the need for and style of punishment that should be used to help children in their socialization.

Later, clinicians concentrated on specific child-rearing practices and evaluated them by their effects on a child's alleged later development. For example, Luther Emmett Holt (1855 to 1924) wrote a book entitled *The Care and Feeding of Children*, which went through 75 printings between 1894 and 1943 ([Holt, 1902](#)). Among other things, he warned of the dangers of early swaddling and of playing with children younger than 6 months old because such practices would lead to various mental and physical problems.

In the beginning of the 20th century, clinicians from the psychoanalytic school began to label specific parental activities as pathologic for the development of children. Examples are Freud's early concerns about the prohibition of masturbation in young children and the impact of specific psychosexual stages on children's relationships with their parents ([Freud, 1909, 1958](#)). Anna Freud and others later extended this work by discussing how children develop defense mechanisms that compromise development in response to overindulgent or rejecting parental attitudes ([Freud, 1937](#); [Levy, 1943](#)).

Others write about more practical aspects of caretaking. For example, [Buxbaum \(1959\)](#) discussed the need of children to breast-feed to obtain sufficient oral pleasure, and [Sears et al. \(1957\)](#) emphasized the need for children to be allowed adequate genital play. These authors also believed that certain activities, such as the type and length of toilet training or breast-feeding a child experiences, carry distinct predictions for the infant's future mental health.

However, in a careful review of the then-available data on child rearing, [Caldwell \(1964\)](#) concluded that neither the timing of various aspects of child care nor the method of toilet training have particular psychological consequences. This finding suggests that parenting cannot be explained by studying single dimensions, but it has to be understood as a multifactorial phenomenon, including child factors such as genetic predispositions and developmental status, as well as parental behaviors.

During the last 25 years, developmental theories have changed dramatically as some learning and many psychodynamic principles have been translated into the language of information processing, that is, a cognitive-behavioral mode. This approach presumes that past experiences create predictable biases and distortions in mental representations that can then function as risk or protective factors for later psychological disturbances. Problematic or secure experiences of the child with early attachment figures are frequently used to demonstrate this principle ([Lyons-Ruth and Jacobovitz, 1999](#)), thus indicating the continuing notion that the unfolding of psychopathology occurs in the context of the family and the child's primary socialization environment (Petit et al., 1997).

In the 1990s, quantitative behavior genetics seriously challenged classical socialization theories with their associated emphasis on parenting. For example, [Rowe \(1994\)](#) writes: "parents in most working to professional class families may have little influence on what traits their children may eventually develop as adults." He also states that an undesirable trait displayed by a child cannot be significantly modified by anything a parent does. This strong statement is partly based on the general recognition that the overall connection between early parenting and later socialization turns out to be quite weak, and many of the demonstrated correlations can be interpreted as driven by the child's genetically predetermined predispositions ([O'Connor et al., 1998](#)). This suggests that many influences, previously considered environmental, are or could be actually genetically mediated ([Kendler et al., 1996](#)) and that only up to 20% of behaviors, based on nonshared environmental effects, are related to child-rearing practices ([Plomin, 1994](#)).

## WHAT IS THE CONTRIBUTION OF GENETICS TO DEVELOPMENTALLY APPROPRIATE PARENTING?

We stated earlier in this chapter that children's behavioral adjustment and parent-child interaction operate through bidirectional family processes, some of which have genetic roots ([Kuczynski et al., 1997](#)). Nevertheless, some investigators, using a developmental model of genotype-environment, do not just look at the effect of genes or the environment on specific behaviors because this would ignore the contribution parenting makes in all these conditions. Genetic factors, in their opinion, are therefore seen more as risk or protective factors, and specific behaviors of children are conceptualized as evocative, that is, as challenging and demanding a response from parents ([Deater-Deckard, 2000](#)). Evocative behaviors are thought to have a genetic basis because of the observation that parents behave more similarly toward siblings who are genetically more similar ([Lollis and Kuczynski, 1997](#)). It also seems that evocative behaviors are more closely associated with externalizing symptoms, as described by [Patterson et al. \(1992\)](#) and [Ge et al. \(1994\)](#), and not with internalizing conditions. In fact, in one study, [O'Connor and colleagues \(1998\)](#) assessed 88 adopted boys and their adoptive families when the boys were 7, 9, 10, 11, and 12 years old. They report that both positive (e.g., warmth) and negative (e.g., negative control or lax responses) parenting factors were highly consistent over time, but much more so toward children with externalizing than with internalizing behaviors. This finding suggests that aggressive or other externalizing behaviors are highly evocative, whereas internalizing symptoms are not. [Loeber and colleagues \(2000\)](#), reporting on the stability of family interaction in 1,517 antisocial boys of the Pittsburgh Youth Study, also show a very high "absolute stability" in family interaction measures from ages 6 to 18 years. Across-age correlations generally are between 0.58 and 0.69 on such measures as bad relationship, poor supervision, low positive parenting, physical punishment, and poor communication. The authors suggest that reciprocal behaviors between children and their parents are influenced by genetic factors.

In contrast, gene-environment correlations, which describe underlying common genetic links between parent and child, also called *passive* correlations, do not seem to contribute to child antisocial behaviors substantially ([Hershberger, 1994](#)). Poor parenting in this situation would much more likely lead to depression in children with a genetic "risk" for this condition. An example of this is provided in a study by [Deater-Deckard \(2000\)](#), in which 125 same-sex identical and fraternal twin pairs aged 42 to 45 months were visited at home and were videotaped during interactions with their mothers on two occasions. Parental control, affect, and discipline, as well as the difficult behavior of the children, were assessed based on parent, interviewer, and observer ratings. Results indicate that children who were observed to have more difficult behaviors and who were rated by their parents to have a conduct problem lived in homes with more negative control and less positive affect, showing a genetic component of 40% to 50%. However, correlations between ratings by parents and observers on parenting affect were low. Thus, parents reported themselves more directly to respond to individual children's behaviors than did the observers, a finding suggesting that parents sometimes rate identical twins more similarly, presumably because they think that they should be similar.

The focus of these gene-environment correlation investigations has been the effect of specific child-rearing practices or "proximal" factors on the child. Discipline, personal warmth, the amount and type of interaction, and parental regulation of affect are other examples of dimensions that have been studied ([Emde, 1989](#); [Maccoby and Martin, 1983](#); [Minde et al., 1988](#); [Patterson, 1982](#)).

## WHAT OTHER FACTORS CONTRIBUTE TO DEVELOPMENTALLY APPROPRIATE PARENTING?

On the most general level, raising children requires an environment that provides them with sufficient material and emotional care to further their physical, cognitive, and social development. At least six ingredients have been identified that determine developmentally sensitive parenting: sensitive responsiveness, the parent-child dyad, parental mental health, parenting experience, the extended family, and direct caretaker-child interactions ([Emde, 1989](#)).

### Sensitive Responsiveness

How parents respond to their children's behaviors, demands, and distress ([Mrazek et al., 1982](#)) and how they resolve interpersonal conflicts ([Shure and Spivak, 1978](#)) is a key aspect of parenting. Adequate parents are generally observed to be sensitive to their children's cues in relation to their developmental needs ([Rutter, 1989](#)).

[Emde \(1989\)](#) suggests that it is the parents' sense of protectiveness with regard to their small children that provides the basis for their sensitivity; that is, it is survival-oriented. Parents show this sensitivity when they engage in both the physiologic regulation of their infants' needs (e.g., by keeping them warm and well fed) and the regulation of affect. The latter is demonstrated in the caregiver's empathic responsiveness to the child's overall adaptive needs. This means that parents tend to protect their child from experiencing extremes of excitement, anger, or distress. It is of interest that these clinically observed characteristics have also been found to be essential for the normal development of the brain. For example, it is now established that during the first 2 years of life, sequential growth, proliferation, and, in fact, overproduction of axons, dendrites, and synapses occur in different regions of the brain. This process is genetically determined. However, by the age of 2 years, many synaptic connections are "pruned," that is, have died off from lack of use ([Singer, 1995](#)). This means that the brain in the early stages can be physically changed by environmental input, such as parenting behavior, resulting in change of the "hard wiring" of the brain. In other words, parenting practices can modify the expression of genetically determined behaviors. Another important aspect is the distinction between what [Greenough and Black \(1992\)](#) call the "experience-expectant" and "experience-dependent" aspects of early brain maturation. Other authors state that for an orderly process of synaptic connections to occur in the presence of multiple environmental influences, the maturing brain is genetically programmed to expect stimuli to be presented in a way that is "safe, nurturing, predictable, repetitive, gradual and attuned to a child's developmental stage" ([Perry and Pollard, 1998](#)). To ensure this is the central task of the child's caretakers and fits well with the concept of parental sensitivity, which, in turn, furthers a secure attachment of the child to his or her parents.

In contrast to the genetically driven experience-expectant brain maturation, the experience-dependent process generates new synapses only in response to environmentally determined experiences. This has, for obvious reasons, been most clearly documented in animals. For example, rats whose mothers licked and groomed them a great deal during their first 10 days of life showed lower adrenocorticotrophic hormone and adrenal corticosterone in response to stress when they were adults. They also exhibited behavioral aspects associated with superior “programming” of the hypothalamic-pituitary-adrenal response to stress ( [Caldji et al., 1998](#); [Liu et al., 1997](#)). A more recent study extended this work by fostering rat pups of low-frequency licking and grooming to high-frequency licking and grooming mothers and vice versa. In both cases, the pups behaved like their foster mothers in mothering their own children later ( [Francis et al., 1999](#)). This finding suggests that intergenerationally transmitted parenting patterns can be modified by interventions, and the new patterns, in turn, are transmitted to the next generation. Very similar results have been shown in genetically impulsive rhesus monkeys that were foster reared from birth by unrelated multiparous females and became calm and nurturing mothers to their own infants ( [Suomi and Levine, 1998](#)).

### Parent-Child Dyad

Parenting is always determined by the individual parent–child relationship ( [Minde, 1986](#)). It therefore has very special qualities that are specific to each particular dyad, although it also forms part of a broader system of relationships involving other family members, neighbors, and friends. [Emde \(1989\)](#), in his previously described treatise, suggests that the initial and enduring tie between parent and child is an important determinant of this relationship. Bowlby and others have convincingly shown that the quality of this tie or attachment plays an important role in the overall social and emotional functioning of a child ( [Ainsworth et al., 1978](#); [Bowlby, 1969, 1980](#); [Bretherton, 1985](#)).

Given the complexity of human motivation and behavior, it is not surprising that the early mother–child relationship has a strong intergenerational component. Main and her group showed that a mother’s understanding of her own early experiences with her caretakers is an important predictor of the internal representations she has of her child and that these representations, in turn, determine the quality of the infant’s attachment to the mother ( [Fonagy et al., 1991](#); [Main and Goldwyn, 1984](#); [Main and Hesse, 1990](#)). This means that both the interpretation we as parents make of the behaviors of our children and the way we respond to them are significantly determined by how we have learned to interpret the world through past experiences with our own caretakers.

More recent work has further substantiated these connections. Most important is the repeated observation that interactive processes can both trigger and prevent a genetic vulnerability. For example, [Mrazek et al. \(1999\)](#) reported that high ratings of specific parenting difficulties made 3 weeks after the birth of a child by trained lay personnel, together with frequent infections and an elevated level of immunoglobulin E, predicted the onset of asthma at age 3 years with a 70% probability in a cohort of 100 children born to asthmatic parents. Equally important, in a subsequent study of 140 newborns with the same asthmatic risk factors, a specified family intervention program instituted when these infants were 3 weeks old was associated with a significant decrease in the incidence of asthma in these children at age 3 years versus an untreated control group ( [Mrazek, 2000](#)).

### Extended Family and the Social System

Parenting, even though primarily practiced within the individual parent-child relationship, is also a reflection of a broader social nexus. Because parenting is not a natural behavior but must be learned, society will obviously have a powerful impact on this process, and in this respect, parenting competence must be seen as a reflection of the balance between external stresses and supports ( [Belsky and Vondra, 1989](#)). It is in this area that dramatic changes have occurred in recent years. Most striking has been the change in the percentage of working mothers of young children, a number that has surpassed 50%, whereas fathers’ contribution to domestic and parenting work has not grown correspondingly ( [McBride and Mills, 1993](#)). This leaves more children in day care than ever before. Yet costs for such care have gone up, and parents spend between 9% and 11% of their incomes on child care ( [Marshall and Marx, 1991](#)). The overall cost of raising a child has also gone up and is now estimated to be more than \$150,000 up to age 18 years ( [Arendell, 1997](#)).

At the same time, single parenting has become more common. We have seen divorce rates triple between 1960 and 1982 and now come close to 50% in North America; there are also between 2 and 5 million lesbian mothers who have between 6 and 14 million children, and 25% of children are born to unmarried women ( [Patterson, 1992](#)). In fact, 72% of all teen births in 1993 were by single teens, up from 18% in 1963 ( [Children’s Defense Fund, 1996](#)). In the case of teen mothers, we know that they are less effective in interactions; for example, they have less eye contact with their infants and show less verbal and less positive expressiveness ( [Culp et al., 1991](#)). Most of these single-parent families also struggle financially. The U.S. census in 1990 showed 51.1% of African Americans to be poor, and here again mostly single-parent families. The demands and stresses associated with poverty obviously make it difficult for a parent to be consistently attuned to a child’s needs. Indeed, financial problems are associated with harsh, inconsistent parenting ( [Ge et al., 1994](#)), the use of frequent derogatory comments, and little interaction characterized by praise ( [Conger et al., 1984](#)).

To provide the necessary social supports from the community requires persons who have a commitment and the ability to give emotionally and to share with others. Garbarino calls these communities “free from drain,” in contrast to the many communities that operate on a “scarcity” economy, that is, have citizens who fear exploitation and reciprocity because they distrust the caregiving practices of their neighbors ( [Garbarino and Kostelny, 1993](#)).

Another issue that needs to be considered here is the role and involvement of fathers in parenting. As mothers increasingly have moved into the workforce, fathers have taken on more responsibility in the care of their children. Thus, in 1993, more than 46 million preschoolers were cared for by their fathers while their mothers were at work ( [Casper and O’Connell, 1998](#)). This is 23% of all preschoolers with working mothers. The number of single fathers with children at home has also increased by 25% in the preceding 3 years to 2.1 million in 1998 ( [Bureau of the Census, 1998](#)).

Three components of father involvement have been proposed ( [Lamb, 1997](#)): engagement (father has direct contact), accessibility (father is available to child regardless of type of interaction), and responsibility (father understands and meets economic or planning need of child). It is not yet clear which of these components affect which outcome in children.

### Direct Caretaker-Child Interaction

The actual behaviors parents and children show toward each other can vary from mutually supportive and helpful to oppositional or hostile. Interactions are affected by characteristics of both child and parent ( [Maccoby and Martin, 1983](#)).

The best studied aspect of the caretaker-infant interaction is the impact of specific attachment patterns on caretaking quality. Thus, in keeping with Ainsworth’s original theorizing, ratings of maternal sensitivity in the first year of life are linked to later social and interactional competence in children of all cultures and social strata ( [van IJzendoorn and Kroonenberg, 1988](#)). Likewise, sensitive mothers also provide their children with moderate, appropriate stimulation as well as with warmth, involvement, and responsiveness. These characteristics, in turn, lay the foundations for children to develop an interpretative capacity ( [Fonagy and Target, 1998](#)), which can function as a significant protective factor against externalizing disorders ( [Greenberg, 1999](#)), even when they grow up in families whose members are at risk for such disorders, and to become sensitive and responsive caretakers as adults.

Other systems and factors are also important determinants of parenting competence, among them parenting experience and parental mental health. Experience counts, because both in monkeys and in people we find mothers to be more patient, sensitive, and competent with later-born than with first-born children ( [Rutter, 1981](#); [Suomi, 1987](#)). Experienced mothers also provide more appropriate stimulation ( [Jones et al., 1980](#); [Ragozin et al., 1982](#)). The parents’ psychosocial functioning is another essential component in the quality of the parenting they can provide. As we see in the next section, parenting functions can be severely compromised by mental disorders and the associated distorted internal representations of the world they can cause.

## PARENTAL PSYCHIATRIC DISORDERS

Empirical studies that examine the effects of specific parental disorders on the development of children go back to the early part of the 20th century. [Janet \(1925\)](#) concluded that mental disorders of parents had a negative effect on their children because of their impact on the social life in the family. He based this conclusion on his observations of three of 18 families in which the precipitating mental illness occurred in stepparents and, hence, affected children who were not genetically related to them. He also described cases in which children of psychiatrically ill parents had fared well because they had been brought up away from their biological families. Canavan and Clark ( [1923a, 1923b](#)) undertook the first controlled study by examining, from 1912 to 1921, the 463 children of 136 married patients diagnosed as having dementia praecox, or schizophrenia. The authors compared these youngsters with 581 children of 145 families attending a medical outpatient clinic. These



investigators found the incidence of conduct disorders among the children in the study group to be significantly higher than in the control children (9.5% versus 1.6%).

Other early investigators ([Gardner, 1949](#); [Preston and Antin, 1933](#); Ramage, 1925) confirmed these findings. They suggested that, overall, the children of psychotic patients showed comparatively few mental peculiarities and virtually no occurrence of the parental illness ([Canavan and Clark, 1936](#)). Although this early work suggested a nongenetic intergenerational transmission of psychiatric illness, it was Rutter's monograph, *Children of Sick Parents* ([Rutter, 1966](#)), that set a new standard for examining the effect of a parent's emotional or physical handicap on his or her children. From 922 children who had attended the Maudsley Hospital Children's Department (London, UK) in 1955 and 1959, Rutter selected specific subsamples, children who had at least one parent with a mental illness (n = 137), children who were bereaved (n = 85), and a third group in which the parents had either a chronic or a recurrent physical disease (n = 190). The following are some of the relevant findings of this study:

1. There was a strong association between parental illness and psychiatric disorder in children.
2. This was true for both psychiatric illness and physical illness in the parent.
3. Because parental illness usually preceded the development of the child's disorder, it is reasonable to suggest a parent-to-child effect.
4. Parental disorder was most likely to be followed by behavioral disturbances in children when the parent exhibited a "long-standing abnormality of personality."

Later studies involving general population samples ([Richman et al., 1982](#); [Rutter et al., 1975](#)) and case-controlled comparisons of the children of parents with a mental disorder ([Beardslee, 1986](#); [Cytryn et al., 1984](#)) further extend our knowledge and permit us to ask some specific questions about the linkage between parental and child psychiatric disorder.

### **How Close Is the Demonstrated Link Between Psychiatric Disorder in Parents and Children?**

The answer is "significant but not very close." This is illustrated by data from [Rutter and Quinton \(1984\)](#), who evaluated 137 newly referred psychiatric patients with 292 children who were less than 15 years of age in London and followed them prospectively for 4 years (1966 to 1967 and 1970 to 1971). All were initially interviewed at home to obtain their diagnosis and family characteristics. At that time, about one-third of the boys and one-fourth of the girls were judged to have handicapping problems. Four years later, only 21% of the boys versus 11% of the girls were still seen as abnormal by their teachers.

### **Do Some Parental Psychiatric Disorders Have a More Detrimental Effect on the Development of Children Than Others?**

The evidence seems to support this notion. As early as 1925, Janet observed that children of psychiatrically ill parents were most affected if there were family conflict and other disturbances, in addition to the mental illness. In the previously mentioned study by [Rutter and Quinton \(1984\)](#), similar findings emerged; the presence of a conduct disorder in one or both parents was the most significant factor in the incidence of childhood psychopathology. In those cases, 45% of the children were identified as disturbed on the teacher questionnaire, and 48% were similarly identified in the psychiatric interview. This finding was in contrast to 21% of children whose parents had "just" a psychosis or an affective disorder.

Children with one psychotic or depressed and one healthy parent did not show any increased risk of a psychiatric disorder. However, many of the disturbed parents also had a disturbed partner, and in these families, children showed a high rate of disorders (20% versus 54%, as determined by the psychiatric interview).

These findings support those from Wynne's project in the Rochester Longitudinal Study ([Harder et al., 1980](#); [Kokes et al., 1980](#)), in which boys aged 7 and 10 years who had nonpsychotically ill parents showed far less overall psychological competence than those with schizophrenic or affectively ill parents. A more recent analysis of the 18-year outcome of the Rochester Longitudinal Study sample examined whether cognitive competence and mental health were related to 10 specific environmental risk factors, such as a history of maternal mental illness, high maternal anxiety, and large family size ([Sameroff et al., 1993](#)). Although there were statistically significant effects for each single risk factor at the population level, most children with only one risk factor were doing well, whereas 14% of those with three or four risk factors and 27% of those with five or more risk factors showed clinical problems. This finding indicates that poor parenting is just one of many other risk factors for later mental health.

The Minnesota Study by Garmezy and his group can also be considered here ([Garmezy, 1984](#); [Garmezy and Devine, 1984](#)). In that investigation, children, aged 9 to 11 years, of schizophrenic and internalizing (primarily depressed) mothers were studied using peer and teacher ratings and school records. Initial findings suggested that the children of internalizing (i.e., depressed) mothers were most like the control group. Seven years later, however, only 48% of the children of depressed mothers received an overall good outcome score, whereas 82% of the controls did ([Garmezy and Devine, 1984](#)). [Rutter and Quinton \(1984\)](#), in their previously cited study, also reported that continuing psychiatric abnormalities in their sample of children were not related to a particular parental diagnosis but to the associated presence of quite specific types of family interactions. Most important here was the child's exposure to hostile parental behavior. Thus, 17% of boys with nonhostile fathers and mothers versus 40% of those with hostile mothers and 71% with hostile fathers showed persistent disturbance.

### **Are There Age Differences in the Susceptibility of Children to the Adversities of Having a Mentally Ill Parent?**

This question has never been examined directly. However, some related findings suggest that one can expect to see developmental differences. For example, it is well known that repeated hospitalization is especially difficult for children between the ages of 6 months and 5 years ([Quinton and Rutter, 1976](#)) because young children cannot "understand" that the separation from the family they experience is not permanent and has nothing to do with anything "bad" they have done. One would therefore expect that certain characteristics of a mentally ill mother or father, such as their need to be hospitalized at unpredictable intervals, would be associated with difficulties in younger children, whereas the presence of concrete or overinclusive thinking in some schizophrenic patients may be more traumatic to older children.

These data suggest that some interactive factors, such as hostility, may be pathogenic for a wide age range of children, whereas others, such as parental sadness, may be especially problematic for younger children. Thus, infants of depressed mothers remain disturbed even after their mothers have been treated for their depression and are functioning well ([Field et al., 1997](#)), whereas this is not true for older children.

### **Are There Sex Differences in the Susceptibility of Children to Parental Mental Illness?**

The data here are not very clear. In general, it has been documented that boys are more susceptible to psychosocial stressors than are girls ([Maccoby and Martin, 1983](#)). However, the reason may partly be that, as we have seen, an ongoing parental personality disorder, especially one associated with hostile interpersonal child-rearing practices, shows the strongest association with persistent emotional or behavioral disturbance in the child. Personality disorders are more common in men, and because parental disturbances seem to affect the children of the same sex more, one would expect boys to be more affected by their fathers' problems.

Rutter and Quinton's findings confirm that children of the same sex as the mentally ill parent are more profoundly affected. In their study, 40% of the boys versus 23% of the girls were judged disturbed by teachers when the father was mentally ill, whereas 11% of daughters of mentally ill mothers versus 0% of daughters of mentally ill fathers were seen as abnormal ([Rutter and Quinton, 1984](#)).

### **To What Extent Are Psychiatric Disorders in Children of Mentally Ill Parents Simply a Reflection of the Common Gene Pool Between the Generations?**

Both schizophrenia and major depressive illness have a well-established genetic component. In fact, there are some adoption studies in which rates of schizophrenia were found to be equally high for adopted and nonadopted children ([Gottesman and Shields, 1982](#)). This finding has been taken to mean that a genetic predisposition accounts for much of the disorder seen in children. However, other data suggest that the more severe the parental disorder, the more likely a child is to be placed for adoption ([Sameroff and Zax, 1978](#)). This would indicate that children given up for adoption cannot be readily compared with those who are kept by their original families.

More recent work by Plomin and Daniels introduces the concept of shared and nonshared environmental influences ([Plomin and Daniels, 1987](#)). Plomin and Daniels developed this concept in response to their work on adopted children and twins. In particular, they documented that children living in the same family may nevertheless perceive their environment quite differently. For example, one child may have been influenced by the musical activities she shared with her father and may report her home to have been peaceful. Her brother, in contrast, may have focused on the verbal battles between their parents, grown up feeling rejected, and become a disturbed youngster. Since then, these authors have advanced the precision of measuring the environmental and genetic contributions to specific behaviors or psychopathologic conditions in children. For example, the shared environmental influences are determined by comparing the correlation of a behavior between

adopted children and their adopted siblings with children in other households, using the formula  $E_{NS} = 100 - h^2 - E_S$  where  $E_{NS}$  is the nonshared environment,  $h$  is heritability, and  $E_S$  is the shared environment. This formula suggests that the bulk of the environmental component is nonshared, whereas the shared environment, which includes parental behaviors, accounts for much less of the variance for most disorders ( [Plomin, 1994](#)).

In summary, there is good evidence that children are adversely affected by parents with a psychiatric illness. However, their vulnerability is primarily related to the overall number of stresses the child is exposed to, the pervasiveness with which each parental illness affects the actual caretaking practices of the parent, and by the children's genetic risk status. Thus, personality disorders in parents showing a pervasive and chronic course are more difficult for children to cope with than, say, an intermittent psychotic illness in the parent. Age and sex differences in susceptibility also exist but vary in developmentally expected ways.

## DEPRESSION IN PARENTS

Of all mental illnesses, depression in mothers has received most attention in the literature ( [Rutter, 1986](#)). Because the incidence of this condition among young women is at least 9% ( [Boyd and Weissman, 1981](#)), it potentially affects a large number of children. In fact, there are reports that the rate of depression in nonworking mothers of preschool children may be as high as 40% ( [Richman et al., 1982](#)). Both Richman and her group and Boyd and Weissman defined depression according to criteria of the ninth edition of the *International Classification of Diseases* ( [World Health Organization, 1977](#)), that is, exclusive of postpartum depression, known as "maternal blues," which occurs in more than 80% of women and is usually self-limiting ( [Stein, 1982](#)). However, it includes persons who, by criteria of the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* ( [American Psychiatric Association, 1994](#)), have a single or recurrent episode of major depression (296.2 or 296.3), bipolar disorder, depressed (296.5), or dysthymia (300.40), with all the ensuing differences in each condition's genetic vulnerability.

The effects of maternal depression on children have often been described ( [Beardslee et al., 1993b](#); [Coghill et al., 1986](#); [Cox et al., 1987](#); [Cytryn et al., 1984, 1986](#); [Field, 1988](#); [Gershon et al., 1982](#); [Puckering, 1989](#)). Findings suggest that these children display a wide range of behavior-management problems. Although some appear depressed, especially when parental depression occurs early in the child's life ( [Hammen et al., 1990](#)), others show conduct disorders or hyperactivity ( [Orvaschel et al., 1988](#)).

More important, data from [Richman et al. \(1982\)](#), [Coghill et al. \(1986\)](#), and [Cox et al. \(1987\)](#) show that many children tend to maintain their abnormal behavior even after remission of the mother's depressive illness. For example, Richman and colleagues report that 58% of the 3-year-old youngsters in their study still showed behavior problems at the age of 8 years, even though maternal depression had remitted.

There are some suggestions from a large twin study ( $n = 315$ ) that nonclinically depressive symptoms yield a heritability estimate of 20%, a shared environmental estimate of 27%, and a nonshared environment estimate of 53% ( [Eley and Stevenson, 1995](#)). However, when adolescents on the high end of the continuum of depressive symptoms were analyzed separately, genetic factors accounted for much more (59%) of the variation. In another study, children aged 8 to 11 years showed a high shared environmental variation (60%) and low heritability (18%), whereas in the older group (aged 11 to 16 years), depressive symptoms were primarily accounted for by heredity (78%), with the shared environment explaining only 4% of the variation. Other findings suggest that children of parents with bipolar disorder are at significantly higher risk of showing disturbed behavior than those whose parents have a unipolar illness (20% versus 43%). Children with two affected parents show a far higher morbidity than those with one affected parent (74% versus 24%) ( [Gershon et al., 1982](#); [Puig-Antich et al., 1985](#); [Weissman et al., 1987](#)).

It has also been found that parents who have experienced a single depressive episode versus multiple depressive episodes exhibited few differences in their behavior toward their children, but mothers who in addition to their depression were also diagnosed with a personality disorder had the most severely compromised children overall ( [Radke-Yarrow and Klimes-Dougan, 1997](#)). The same authors also reported in a later article ( [Klimes-Dougan et al., 1999](#)) that children of depressed mothers are also more likely to report suicidality than the children of well mothers. This finding underlines the growing body of literature that shows that suicidality also runs in families ( [Brent et al., 1996](#)). In fact, in a large twin study by [Statham et al. \(1998\)](#), the heritability of serious suicidal attempts was 55%. Within that context, familial depression and suicidal attempts appear to be separately transmitted in families.

Obviously, the children of depressed parents are subject not only to genetic risk but also to a highly stressful environment. Studies examining the parenting style of depressed mothers have focused on direct mother-child interactions. For example, [Richman et al. \(1982\)](#), in a longitudinal prospective study of nearly 200 children evaluated at ages 3, 4, and 8 years, found a high correlation among maternal depression, the frequency the child was read to at home, and his or her reading age measured at age 8 years. This correlation was particularly prominent in boys. In another study, children who, at 2 years of age, had received little reciprocal cognitive stimulation from their depressed mothers performed poorly on cognitive tasks in school at 5 years ( [Meadows and Mills 1987](#)). Likewise, in the foregoing study of depressed mothers by Radke-Yarrow and Klimes-Dougan, children were most affected when their mothers had an associated diagnosis of a personality disorder that behaviorally translated into more critical, irritable interactions and psychological unavailability that was much more severe than in the purely depressed mothers ( [DeMulder et al., 1995](#)). Nevertheless, the literature suggests that, overall, depressed mothers are more preoccupied with themselves and consequently are less sensitive to the needs of their children ( [Billings and Moos, 1983](#); [Cox et al., 1987](#); [Field, 1984](#); [Zekoski et al., 1986](#)). In particular, they are said to stimulate their babies less, that is, use fewer questions, explanations, and suggestions ( [Cox et al., 1987](#)), and have a less consistent attitude toward disciplining. They are also less able to compromise ( [Kochanska et al., 1987](#)). Moreover, there is increasing evidence that, at least in mothers of infants and toddlers, the pharmacologic treatment of mothers does not have clear beneficial effects for the children (Panikkar, 2000). This may be related to Radke-Yarrow's observation that there remain "residuals" of the depression in many of these mothers that are sufficient to compromise at least some socioemotional aspects of young children's development. Furthermore, data by Sameroff and his colleagues indicate that depressed women more often marry or live with depressed or otherwise psychiatrically disturbed men than do women with, for example, obsessive-compulsive disorders ( [Sameroff et al., 1993](#)); that is, assortative mating takes place in this population. The same group of investigators found that overall family functioning is the most powerful mediator of the mental illness variable and is a better predictor of future child mental health than are the details of postpartum and lifetime histories of parental depression ( [Dickstein et al., 1998](#)). Families are compromised only when mothers are symptomatically depressed and not when they are between depressive episodes. However, it seems to be true that depressed mothers overall show a wide variety of interactional patterns, and some of them appear warm and very involved with their children even when these women are depressed ( [Cox et al., 1987](#)).

Another important area of research has been the relationship between maternal depression and children's attachment patterns. It appears that an increased number of children of depressed mothers (65% versus 35% in control populations) display an insecure attachment pattern when they are tested on the Ainsworth Strange Situation Paradigm at 12 or 18 months ( [Radke-Yarrow et al., 1985](#)). However, a similarly high rate of insecure attachment patterns is found among children who live with a wide variety of psychologically compromised parents, such as parents with anxiety or personality disorders ( [Benoit et al., 1989](#); Benoit et al., 1992; [Minde et al., 1990](#)).

Other studies have focused on the children and, here again, on their self-concept as a mediating variable in the intergenerational continuities of depression. Thus, [Jaenicke et al. \(1987\)](#) found that children at risk of depression had a more negative self-concept, a finding these authors thought to be related to the degree of blame and criticism they received from their mothers. [Hammen et al. \(1987\)](#) singled out negative self-concept as a cognitive mediator for later experiences, which then confirm a basically depressive predisposition.

In summary, it appears that parental depression is a significant risk factor for the mental health of children. The abnormal behavior in these youngsters is not limited to depression but can encompass a wide range of both introverted and acting-out behaviors. Although genetic factors seem to be important, the children's interpersonal experiences also affect their attachment patterns, self-esteem, and cognitive style, which, in turn, contributes to and maintains later depressive symptoms.

## OTHER PSYCHOSOCIAL ABNORMALITIES IN PARENTS

There are two other groups of parents whose maladaptive behavior toward their children has been thoroughly examined by mental health researchers. These are antisocial and abusing parents. Although some of their parenting characteristics resemble those of parents who show a personality disorder or other psychiatric disability, and although some antisocial parents also abuse their children more often than do parents who are not antisocial, these two groups display sufficiently distinct patterns of disordered parenting to warrant separate reviews.

### Antisocial Behavior in Parents

Many investigations document that antisocial behavior in parents is often associated with externalizing disorders in children such as delinquency or conduct disorders ( [Hinde, 1980](#); [Le Blanc and Loeber, 1993](#); [Loeber and Stouthamer-Loeber, 1986](#); [Wadsworth, 1979](#)). This has been true in different ethnic and cultural groups ( [Minde and Nikapota, 1993](#); [Rohner, 1975](#); [Werner, 1979](#)). [Loeber and Stouthamer-Loeber \(1986\)](#), in a metaanalysis of those studies, find that data cluster around four risk



factors: conflict, neglect, deviant values, and marital discord.

#### *PARENT-CHILD CONFLICT*

Investigations of parents of children with externalizing disorders identify two ways in which ineffective parenting strategies can contribute to such difficulties. Some investigators label ineffective parenting to be a precursor to child misbehavior ( [Pettit and Bates, 1989](#); [Shaw et al., 1994](#)), whereas others focus on ineffective parenting as a response to these children's conflictual behaviors ( [Patterson, 1982](#); Patterson et al., 1993). Typically, both types of studies support the view that these parents rely on negative, insensitive, and harsh strategies rather than positive ones such as reasoning, negotiation, and compromise ( [Campbell, 1995](#); [Kochanska and Aksan, 1995](#)). Investigators also agree that parents are usually aware of and are displeased with their child's disobedience but are unable to set adequate limits or to employ constructive and nonaggressive ways of disciplining. As a consequence, the child's behavior fails to improve, and this leads to an escalation of maladaptive parental attempts to take control, to a point at which parents and youngsters see each other as enemies. The basic deficit underlying this pattern seems to be parental insensitivity to the child's emotional needs, especially the need for structure and regulation. The children feel rejected, and this feeling, together with nagging and coercive discipline, is directly related to later delinquent behavior. In contrast, competent parents use preemptive rather than reactive strategies to handle conflict in their preschoolers, and this single difference in parenting best distinguishes children who show conduct problems 2 years later from those who do not ( [Gardner et al., 1999](#)).

#### *NEGLECT AND DEVIANT VALUES*

[Loeber and Stouthamer-Loeber \(1986\)](#), in reviewing available studies, concluded that the lack of parental supervision and the absence of parental involvement with their children are both significantly correlated with the development of later conduct disorders. It appears that lack of parental supervision is also strongly associated with delinquency ( [Wilson, 1987](#)). Delinquent children, in turn, often come from homes with no or few house rules, inadequate disciplining, and little parental monitoring of delinquent behaviors ( [Patterson, 1982](#)).

Moreover, parents who exhibit antisocial behaviors themselves also tend to condone abnormal behavior in their children and therefore may serve as models for deviant activity or problem solving ( [West and Farrington, 1973](#)). Support for this concept comes from the observation that delinquency in children is more strongly associated with a parent's current rather than past criminality or delinquency ( [Robins et al., 1975](#)).

#### *MARITAL DISCORD*

As is common in families in which a parent has a specific psychiatric diagnosis, families with antisocial members show much family discord, a powerful predictor of child adjustment ( [Rutter and Giller, 1983](#); [West, 1982](#)). In fact, there is abundant evidence that children in these families are exposed to both frequent and prolonged quarreling among their parents and temporary separations because of arguments, as well as to abuse of alcohol and drugs (Cummings and Davies, 1996).

In general, investigators and clinicians report that marital discord has both direct and indirect negative effects on children. The "direct" negative effect is that children copy the parents' angry, impulsive, and violent behaviors and thereby fail to learn appropriate social interaction skills, such as compromising, necessary for successful peer relationships. More recent work also suggests that children may respond to marital conflict with disturbances of affect regulation and increases in heart rate and blood pressure ( [Lieberman and Van Horn, 1998](#)).

In addition to these direct effects of marital conflicts are "indirect" consequences, mediated through the father-child or mother-child relationship. For example, mothers in high-conflict marriages are less warm and are more erratic in their discipline, and they frequently use guilt- and anxiety-inducing techniques ( [Fincham et al., 1994](#); [Harrist and Ainslie, 1998](#)). Fathers in such situations also withdraw more from the parenting role and show more negative interactions with their children than do fathers in low-conflict marriages ( [Doherty, 1998](#)). Finally, parents in such conflictual relationships are more often depressed, and this is also linked with impaired family functioning.

#### **Child Abuse**

By definition, child maltreatment is the most telling syndrome associated with deviant parenting. As is shown in [Chapter 100](#) and [Chapter 101](#), child abuse is a complex problem that cuts across all classes of society, although it is more commonly seen in poor families ( [Pelton, 1978](#)). Official sources claim that in 1995 there were about 1 million substantiated reports of child maltreatment and 1,000 child fatalities resulting from abuse and neglect in the United States ( [U.S. Department of Health and Human Services, 1997](#)). Children 3 years old and younger comprised 77% of all child fatalities. Life prevalence rates of sexual abuse are estimated to be as high as 62% for girls and 31% for boys ( [Dubowitz, 1986](#)).

Although these figures appear horrendous, the reader should be aware of significant differences in the definitions of abuse, among other things, because of the preconceptions of individual professional groups and differing community values ( [Barnett et al., 1993](#)). The definition can also include qualitative, as well as quantitative, aspects of abuse. For example, physical or sexual abuse may happen once or repeatedly (i.e., are experienced as one or more events), or there may be an ongoing abusive relationship between a caretaker and a child ( [Glaser and Prior, 1997](#)).

What, then, are the parental behaviors associated with abuse? Direct answers are difficult because the maltreatment of a child is usually not just a function of parental deviance but the result of an interaction between child and parent ( [Cicchetti and Risley, 1981](#)). Furthermore, because of its generally low incidence per individual, unique characteristics of abusers cannot be readily identified. However, in many cases, abusing and neglectful parents have been adversely affected by their own past experiences ( [Harmer et al., 1999](#)), and they often live in suboptimal physical and social conditions ( [Boyce et al., 1998](#)); these factors make them less tolerant toward children with a difficult temperament or a chronic illness. Abusive parental behaviors therefore are seen as a consequence of an interaction between parental stresses and available supports ( [Belsky and Vondra, 1989](#)). One can also argue, as [Rutter \(1989\)](#) does, that child maltreatment reflects primarily a substantially abnormal pattern of parenting that may lead to such manifestations as physical or sexual abuse, failure to thrive, or even to deliberately created iatrogenic illnesses such as are seen in the Munchausen by proxy syndrome. Authors who have studied the parental contribution to abuse describe both personality factors and psychological disturbances:

#### *PERSONALITY FACTORS*

There is reasonable evidence that psychological maturity and parental competence are correlated, and it is more often younger and less mature mothers who abuse their children ( [Ragozin et al., 1982](#); [Unger and Wandersman, 1985](#)). This finding has been documented in a longitudinal study of 267 economically deprived families, in which those who abused their children early on had lower scores on scales assessing locus of control and higher scores on aggression, suspicion, and defensiveness ( [Brunquell et al., 1981](#)). In an investigation of 570 German families with abusing parents who came from diverse social classes, [Engfer and Schneewind \(1982\)](#) found the abusing parents to report very unfavorable socialization experiences in their own past. This experience, in turn, was related to heightened irritability and nervous tension as well as family conflict, leading to frequent angry outbursts and harsh punishments of the children.

#### *PSYCHOLOGICAL DISTURBANCES AND INTERGENERATIONAL TRANSMISSION OF MALTREATMENT*

There has been a long-held belief in the scientific literature and popular press alike that the majority of abused children will become abusing parents ( [Friedrich and Wheeler, 1982](#)). Reports emphasizing such continuities are usually retrospective, that is, based on studies of parents who had abused a child and who then reported about their own backgrounds ( [Steele and Pollock, 1968](#)). Investigations using an epidemiologic and prospective approach found a much lower rate of intergenerational transmission of maltreatment. For example, in one prospective study ( [Hunter and Kilstrom, 1979](#)), in which 282 parents of newborns admitted to an intensive neonatal care unit were interviewed and were followed for 1 year, only 18% of mothers who had reported abuse in their own childhood repeated it with their children. Similar figures have been presented by other investigators ( [Kaufman and Zigler, 1987](#); [Strauss, 1979](#)).

The mechanism of transmitting maltreatment from one generation to the other has not yet been established ( [Egeland et al., 1988](#); [Kaufman and Zigler, 1989](#)). Some authors claim that abusive parents teach their children that aggression is an appropriate way of dealing with conflict ( [Burgess and Youngblade, 1990](#); [Hertzberger, 1983](#)). Proponents of attachment theory suggest that children in abusing families develop representational models of the world that are so traumatic that they cannot be remembered in an integrative fashion and therefore cannot serve to prevent a repetition of these behaviors in the next generation ( [Bowlby, 1980](#); [Sroufe and Fleeson, 1986](#)). A maltreating caregiver therefore is both frightened and frightening, and his or her current attachment model is characterized by a lack of resolution of loss or trauma, resulting in contradictory and unintegrated mental contents. There is some support for this concept from studies that prospectively followed abused

children and found that most fit the D (disorganized) category of attachment ( [Lyons-Ruth and Block, 1996](#)). [Crittenden \(1981\)](#) adds an A/C category (describing a mixture of dismissive and overinvolved behavior) to the traditional attachment ratings and has evidence that this combined category is found in more than 80% of abusing caretakers. It is interesting that [Cummings et al. \(1994\)](#) also find that physically abused boys are more behaviorally reactive to interadult anger than comparison subjects. This finding implies that greater anger and aggression experienced across various family subsystems (e.g., parent to child, parent to parent) have a cumulative effect and sensitize rather than habituate children to hostility in others. This also means that attachment can have an important mediating role both in increasing and decreasing the likelihood of abuse.

Belsky (1984, 1993; [Belsky et al., 1984](#)), who has developed the most comprehensive model of abuse to date, suggests that low self-esteem, poor interpersonal skills, and a low intelligence quotient (IQ) provide a matrix that often leads to marriages to deviant and unsupportive spouses. This situation, in turn, creates marital discord, which leads to poorly behaving children whose behavior irritates their parents and causes them to commit abuse. [Quinton et al. \(1984\)](#), [Quinton and Rutter \(1985\)](#), and [Dowdney et al. \(1985\)](#), in a study that assessed parenting capacities of 89 women raised in institutions, also demonstrate that both assortative mating and marital discord provide an important link that continues poor parenting from generation to generation. They also point out that even among these very high-risk women, 40% had no difficulties in psychosocial functioning, and only 18% had their children placed into care, although almost half of these mothers had later children whom they managed to parent well. This finding suggests that many poorly parented persons will nevertheless be able to move from a deviant life path into a more integrated and positive developmental trajectory.

In summary, much evidence points to the association between antisocial and abusive parents and later psychopathology in children and adolescents. However, here again, we do not find a parenting style that is specific to a particular disorder in the caretaker. Rather, we observe that such parents neglect their children, dismiss their need for sensitive understanding, or behave in ways that generally fail to support their children's overall social and emotional development.

## ORIGINS AND CLINICAL MANIFESTATIONS OF DISORDERED PARENTING SKILLS

The final segment of this chapter is devoted to the discussion of the substantial literature that examines how specific maladaptive parenting patterns come about and to what degree specific parental actions or strategies become associated with behavioral difficulties in the children. A better understanding of these pathways is clearly important to facilitate remedial efforts.

In the search for meaningful general dimensions of problematic parent-child interactions, two categories have been conceptualized most clearly. One is characterized by emotional detachment and lack of involvement (i.e., too little control), and the other is characterized by excessive control through hostile harsh disciplining and overt maltreatment.

## PARENTS WHO SHOW EMOTIONAL DETACHMENT AND LACK OF INVOLVEMENT ARE UNRESPONSIVE AND INSENSITIVE

Parents whose behaviors fit this category make children feel misunderstood, rejected, or emotionally abandoned in the sense of attachment theory ( [Sroufe, 1983](#)). The association between parental detachment early in life and later psychological disturbance has often been made. For example, work by Minde and his colleagues shows that a mother's sensitivity to the social cues of premature twins within the first 3 weeks of life predicts which one of the twins will be preferred by her 4 years later ([Minde et al., 1990](#)). Rutter and colleagues, in their study of parenting abilities of women reared in institutions, also showed that among women placed in care before the age of 2 years, a much higher proportion had a personality disorder than did those whose primary attachments were disrupted after the age of 2 years (32% versus 5%). These authors also assessed the degree to which parental disorder determined when the separation took place and found that children who were placed in institutions in early infancy were more likely to have had parents who abused "hard" drugs or alcohol or who had a criminal record. Yet their own later parenting competence was less predicted by their parents' deviance than by the age of placement, with placement at a younger age predicting poorer parenting later in life. Most important, 80% of the girls who were admitted to an institution before the age of 2 years and who remained there until at least age 16 years were poor mothers, whereas only 30% of those who returned home before age 16 years showed poor parenting skills. This occurred even though many of these girls returned to families that experienced severe social disadvantage and marital discord. Of the girls who were placed after the age of 2 years, 45% also showed significant problems in their parenting skills later on, and those who returned home did not differ from those who remained in the institution during all of their childhood. The reason was thought to be the degree of active hostility, rejection, and punitiveness the children encountered on returning home ( [Rutter et al., 1983](#); [Rutter and Quinton, 1984a](#)).

One other longitudinal study highlights the crucial importance of parental sensitivity and care for normal development. Tizard and her group studied a sample of 65 children who had been placed, shortly after birth, in well-run institutions in Britain. Twenty-four of these children were adopted by the age of 4 years; others remained in institutions longer. Most had returned to some type of family by the age of 16 years. In an earlier article, [Tizard and Hodges \(1978\)](#) emphasized that most of the early-adopted children (before age 4 years) had developed good, stable, affectionate relationships with their adoptive parents, whereas those adopted between the ages of 4 and 8 years showed poorer relationships with peers and others. At age 16, they found that the children who were returned to their biological parents (the restored group) sometime after the age of 2 years showed a far higher rate of serious psychiatric disorders than did those who had been adopted early, partly because the former group had lived in a highly disadvantaged environment after their return home ( [Hodges and Tizard, 1989b](#)). However, 12 years later, even the successfully adopted children were more indiscriminately friendly, had fewer close peer relationships, and confided less in others than did the controls. At the same time, their academic performance was not affected ([Hodges and Tizard, 1989a](#)), a finding suggesting that the lack of sensitive early caretaking had left a mark on the quality of social relationships these youngsters had in late adolescence but not on their academic functioning.

Data by [Rutter et al. \(1998\)](#) and [O'Connor et al. \(2000\)](#), reporting on 111 Romanian children who were adopted by British families after a variable stay in highly neglectful institutions, confirm that children who were adopted into caring homes before 6 months ( $n = 58$ ) despite severe initial delays (mean Denver score of 56.8) showed good cognitive recovery by age 4 years (mean Denver score of 115.7). This was not true for the children who were adopted between 6 and 24 months, because their scores at 4 years old were significantly lower (mean, 96.7). More important, however, both early- and late-adopted children showed symptoms compatible with a severe attachment disorder (e.g., indiscriminate approach to strangers, lack of checking back with a parent) and an inability to conceptualize the feelings and thoughts of others as described by [Fonagy and Target \(1998\)](#). However, here again, the rate of disorder in the early adoptees was far lower (7%) than in the late-adopted children (22%), although the number of children exhibiting mild disturbances was similar in the two groups (37% versus 39%). These patterns remained quite stable during the subsequent 2 years. There was also a significant correlation between attachment disorder behaviors and hyperactivity at age 6 years ( $r = 0.42$ ) ([O'Connor et al., 2000](#)).

It therefore seems that a caretaker's severe lack of involvement and responsiveness toward a child's emotional needs has both cognitive and emotional consequences, at least in a minority of children. Moreover, although it appears that later adequate caretaking can repair cognitive shortcomings, this is not necessarily true for the social and emotional development of such children, many of whom are left with behaviors that may compromise their later ability to parent their own children.

## Parents Who Exert Control Through Hostile, Rejecting, Harsh Discipline and Overt Maltreatment

The manner in which parents control the behavior of their children has long been seen as an important predictor of children's later social and intellectual competence ([Maccoby and Martin, 1983](#)). Research has broadened the concept of *control*, which now includes not only the parents' use of discipline and punishment but their overall monitoring of behavior, handling of conflict, and their emotional style of coping ( [Dowdney et al., 1984](#)). Thus, control now not only relates to the degree of power a parent uses in disciplining children but also includes a democracy versus autocracy dimension.

The relevance of this distinction has been shown in numerous empirical studies. Briefly, Baumrind ( [1967, 1971, 1989](#)), in response to the work of [Baldwin \(1955\)](#), developed a tripartite division of parenting styles in which she contrasted authoritarian, permissive, and authoritative categories. Authoritarian parents, in a prospective study involving 134 children, are observed to be controlling and autocratic. They praise and blame without giving clear criteria for acceptable conduct, display anger readily, and are not very involved in their children. Their children, in turn, are passively hostile, anxious, timid, unhappy, and have little self-esteem ([Loeb et al., 1980](#)). They also score low on measures of conscience; that is, they have not developed a successful way of internalizing societal values ( [Hoffman, 1975](#)). Finally, [Dornbusch et al. \(1987\)](#), using a sample of 8,000 children from different ethnic regions, find that the association between low self-esteem and authoritarian parenting holds true in all social classes and among all ethnic groups and, more important, also influences academic achievement. [Steinberg et al. \(1989\)](#) confirm these results and document that children who live in authoritarian families are not only affected in their emotional development but show academic difficulties at least up to the age of 17 years.

Then there are the permissive parents who employ a low level of control, either because they adopt a democratic philosophy and trust their children or because they are not very interested in taking on child-rearing responsibilities and provide little or no active guidance ( [Lamborn et al., 1991](#)). Their children tend to be disorganized,



lack a sense of competence and purpose, and do not apply themselves academically.

The third style of parenting, called authoritative, combines parental warmth and acceptance or involvement with a certain degree of control or strictness. This seems to give the children a feeling that their parents take them seriously and would help them if they need assistance, but that they also have clear expectations of acceptable behaviors that would be enforced. In follow-up studies, authoritative parents have children with the best behavioral outcome and highest school achievement, at least up to late adolescence ([Steinberg et al., 1989](#)). It is remarkable that these children also internalize parental values most readily. This has puzzled investigators because the strong external controls imposed by authoritative parents would be expected to undermine the internalization of moral values, as happens in children of authoritarian parents. Although the debate is ongoing, some investigators suggest that overall parenting is divided into “parenting practices,” which provide the content such as spanking or insisting that children do their homework, and “parenting style,” which refers to the emotional climate such as tone of voice, body language, or display of temper. The authoritative parent communicates feeling comfortable with being in control but at the same time also recognizes and respects the child's wishes and need for separateness. Control can then be seen here as one way to get the child's attention, an essential prerequisite for any effective parenting or disciplining. This is not what an authoritarian parent communicates.

Nevertheless, there is evidence that these constructs do not work for all children equally well ([Darling and Steinberg, 1993](#)). For example, African-American parents who use some aspects of authoritarian child rearing (e.g., are more controlling, more critical, and more inclined to value conformity than independence in their children) have better-adjusted youngsters than those exercising permissive or authoritative child-rearing patterns. This finding confirms the clinical observation that optimal child-rearing techniques may differ among families living in different cultures and socioeconomic circumstances ([Baldwin et al., 1993](#)).

Although these investigators explore the parents' child-rearing styles, others ([Grusec and Goodnow, 1994](#)) study the conditions that determine the child's willingness to comply with or accept a parent's rules or values. Grusec and Goodnow claim that three relationship values—warmth, protection, and mutual compliance—are the primary sources for the development of moral values. This means that for these authors, concepts associated with secure or insecure attachment patterns are important building blocks for the development of children's prosocial behaviors. This indicates that identification with the parents is helpful for internalizing values but that the equally necessary suppression of antisocial impulses requires authoritative parenting practices.

Finally, there is also evidence for an association between an authoritarian parenting style and open childhood aggressivity ([Patterson, 1982](#)). According to Patterson's data, openly aggressive children receive more punishment for an aggressive act than do normal controls, yet they do not modify their behavior. The particular interactions that determine whether a child of authoritarian parents will be passive or openly aggressive have not been precisely identified, although data point to certain moderating factors within the environment and the child. These are discussed in the next section.

## **FACTORS THAT CAN EXACERBATE OR MODERATE THE EFFECTS OF DISORDERED PARENTING ON CHILDREN'S DEVELOPMENT**

Many life events can have a profound effect on the behavior of parents and, through them, on their children. Such events include parental separation, divorce, or the death of a primary caretaker. These topics are discussed in other chapters ([Chapter 21](#), [Chapter 35](#), [Chapter 36](#), [Chapter 103](#), [Chapter 104](#), [Chapter 108](#), and [Chapter 109](#)).

Another significant factor is the parents' marital relationship. [Emery \(1982\)](#), in an earlier review of this literature, points out that despite methodologic variations among empirical studies, the connection between interparental conflict and behavior problems in children is solidly established. Other investigators find evidence of a compensatory process; that is, parents in less satisfactory marriages may show especially sensitive and involved caretaking ([Brody et al., 1986](#)). Still others find evidence of both facilitating and detrimental effects ([Cowan and Cowan, 1987](#); [Goldberg and Easterbrooks, 1984](#)). [Cox and her colleagues \(1989\)](#) hypothesize that the apparent discrepancy in the data is related to the psychological adjustment of the parents. Thus, disturbed persons tend to marry partners with a complementary psychiatric disorder ([Merikangas, 1982](#)), which may lead poorly adjusted partners to have an outwardly harmonious marriage and family life, whereas in fact their children receive compromised parenting.

Additional support for a connection between marital discord and child behavior comes from behavioral observations obtained from laboratory studies. For example, the number of symptoms displayed by children in the laboratory is correlated with specific marital satisfaction scale scores, as supplied by the mothers ([Emery and O'Leary, 1984](#); [Jouriles et al., 1988](#); [Weiss and Summers, 1983](#)). Correlations between marital status and child behavior in these studies usually range between .20 and .25, which is statistically significant, but nevertheless, it explains only about 5% of the variance and leaves much room for other factors.

Another strategy for looking at the impact of parental relationships on children has been developed by Cummings and his colleagues. In experiments, this group and others exposed pairs of children to an adult couple who, after an initial friendly encounter, engaged in a carefully rehearsed verbal argument, followed by reconciliation. The children were observed both during and after each episode. Results showed that children as young as 2 years old become very upset when they see adults quarrel, and they become more aggressive toward each other after they have witnessed such an argument ([Crockenberg, 1985](#); [Cummings et al., 1985, 1989](#); [El Sheikh et al., 1989](#)). It is of special interest that children from families with reportedly higher marital discord show more preoccupation and feel more responsible for the arguments they see in the laboratory. This finding suggests that children living in troubled families become sensitized to parental arguments and are potentially more strongly affected by each recurrent disagreement.

Work by other investigators provides the important additional information that boys are generally more affected by parental strife than girls, but the behavioral consequences of witnessing behavioral discord may not be apparent for years ([Vaughn et al. 1988](#)). More important is increasing evidence that the negative effect of parental arguments can be significantly modified if the parents can make up after a fight ([Cummings et al. 1993](#)). Conversely, [Vuchinich and colleagues \(1993\)](#) document that parents who invariably unite against their children when problems occur, that is, present a “parental coalition,” prevent their children from learning problem-solving skills, and their children present with more externalizing symptoms, at least in preadolescence. This finding supports the notion that arguing things out can be a useful process, and it may be the lack of closure that compromises the children's later emotional well-being.

Finally, it appears that marital patterns are fairly stable and predictable. For example, [Howes and Markman \(1989\)](#) find a significant correlation between a mother's premarital relationship with her husband and their child's later attachment status. Thus, mothers who, before marriage, confided in their husbands and expressed their feelings freely are more sensitive toward their children, who, in turn, are more securely attached. This finding underlines the remarkable intergenerational continuity of good as well as problematic internalized relationships ([Sroufe and Fleeson, 1988](#)), and it may well be one important mechanism through which we transmit psychological normality or abnormalities to our children. It obviously also supports the attachment theory literature, which suggests that children develop specific types of attachment through actual experiences and create inner working models of the world that are based on these same actual events in their lives ([Main and Goldwyn, 1984](#); [Sroufe and Fleeson, 1988](#)).

As we stated previously, modulations in the quality of parenting can come both through changing support and stress factors within the environment and from the object of parental caretaking, the child. Although many child characteristics have been seen as potential contributors to parenting style, the difficult temperament of a child has been most frequently identified as a potential stress factor for parents. It has been observed that parents who view their children as temperamentally difficult display more negative affect toward them, are more critical, and punish them more severely ([Bates, 1980](#); [Campbell, 1979](#); [Dunn, 1980](#)). These children, in turn, appear to be less able to cope with negative parental behavior. For example, [Dunn and Kendrick \(1981\)](#) report that children with a high intensity of emotional expression and a negative mood show more difficulties coping with a new baby than do children with easy temperaments. Conversely, [Washington et al. \(1986\)](#) show that sensitive and committed mothers of small premature infants report an improvement of temperament in their infants between 3 and 12 months of age, whereas mothers who behave less contingently in the nursery do not report such temperamental changes. This finding underlines the multifactorial nature of even such allegedly biological constructs as temperament.

Other characteristics of children contribute to the caretaking they receive. [Patterson \(1982\)](#), in his study of aggressive youngsters, points out that some children react in an unusual way to punishment. When a psychologically normal child is punished by a parent, the probability that the child will continue to be aggressive is reduced. With certain aggressive children, however, the probability is increased. This finding suggests that these children, at least by school age, are less responsive than nonaggressive children to social reinforcement. In fact, there is evidence that aggressive children respond less to all types of social stimuli and may therefore indeed be more difficult to raise ([Maccoby and Martin, 1983](#)). [Block et al. \(1988\)](#), in a study of 105 adolescents who were followed between the ages of 3 and 14 years, were able to make significant predictions about these youngsters' adolescent use of marijuana and hard drugs by examining their early behavior patterns. Thus, children who used marijuana in adolescence had been seen as rebellious, unconventional in their thinking, unable to delay gratification, and without ambition during their preschool years. Those who later used hard drugs were also described as emotionally bland, keeping people at a distance, and lacking in personal charm. Whether such behavior patterns are the result of genetics or are the result of a parenting style that leads to a general hyporesponsivity or rebelliousness of the child is not

known. However, there is currently no solid evidence that juvenile delinquency and conduct disturbances are genetically determined ( [Rutter et al., 1990](#)).

Finally, data suggest that prenatal and perinatal factors may be important contributors to later parental styles. Work by one group ( [Minde et al., 1983, 1988](#)) shows that the severity of illness a premature infant suffers in the neonatal intensive care unit significantly determines the type of parenting he or she receives. Parents of sicker babies initially behave in a more intrusive way, but they seem to give up after about 6 months; that is, they decrease their overall stimulation and contact significantly ( [Minde et al., 1988](#)). Similar findings are reported by [Wasserman and Allen \(1985\)](#) from an observational study comparing the play of premature, physically handicapped children with no central nervous system damage and control children with their mothers between the ages of 9 and 24 months. These investigators report that by 2 years of age, the mothers of the handicapped children have withdrawn from interaction, and this is associated with an average IQ drop of up to 20 points. This finding suggests that the absence of expected events characterizing normal physical or cognitive development can lead to a disruption of mother-child relations through maternal detachment or other parenting disorders ( [Affleck et al., 1982](#)).

In general, one can say that we think differently today about the families of children with mental handicaps than we did in the past. Whereas before 1980, the birth of a retarded child was viewed from a "pathology-based" model, with an emphasis on parents' "mourning the perfect child" ( [Blacher, 1984](#)), today such a child is seen as a stressor that can have both positive or negative effects on the family ( [Hodapp and Zigler, 1995](#)). In addition, there are special caretaking regimens for children with particular disorders, such as fragile X syndrome. A review article by [Byrne and Cunningham \(1985\)](#) supports this more inclusive notion of mental retardation by stressing that the difficulties parents face in raising mentally handicapped children are multifaceted and complex. Thus, parenting patterns of these children appear to be largely determined by factors such as the age and competencies of the child and the practical needs of each family. There is also evidence that mothers are more flexible when teaching their young mentally handicapped children than their nonhandicapped children, but they may fail to provide them with sufficient positive feedback ( [McConachie and Mitchell, 1985](#)). They may also underestimate their children's actual abilities ( [Banu and Begum, 1991](#)). Finally, in a study comparing how mothers feed 15- to 39-month-old physically normal and severely cerebral palsied children, [Reilly and Skuse \(1992\)](#) document that the handicapped children are fed with minimal verbal interaction and in a rather mechanical way. Furthermore, although the duration of feedings for the two groups is almost identical, the handicapped youngsters, because of severe motor dysfunction and poor overall feeding skills, are offered only 60% and eat only 50% as much food as their nonhandicapped peers. The authors suggest that such feeding practices may be responsible for a significant part of the retarded growth exhibited by many mentally handicapped children. This study also confirms the data by [Wasserman and Allen \(1985\)](#) and [Minde and his group \(1988\)](#), which suggest that parents need to be validated in their parenting activities by their children and that one important aspect of this is the child's "normal" look and behavior.

## ASSESSING PARENTING COMPETENCE

Mental health professionals, especially those working in child welfare agencies, have long wanted to deal with their increasing caseloads more effectively by identifying families or individual caretakers who are likely to neglect, harm, or otherwise seriously compromise a child's development. This has led many regional and state government agencies in 40 U.S. states to implement risk-assessment measures ( [Tatara, 1987](#)) to help agency staff decide on service priorities ( [English and Pecora, 1994](#)). Essentially, these measures consist of a systematic collection of information to help determine the likelihood that a child will be abused or neglected in the future ( [Steinhauer et al., 1993](#)). Once that has been established, other protocols are available to predict the likely severity of the maltreatment ( [Miller et al., 1987](#)).

The three basic types of risk assessment are as follows ( [Pecora, 1991](#)):

1. The matrix approach: Clinicians ask parents questions that are bundled in tables of 16 to 35 factors. Each factor measures behaviors that, in theory, are associated with child abuse (e.g., substance abuse; age of children in the house).
2. Empirical scales: These focus on a few risk factors that are most predictive of child maltreatment (e.g., caregiver abuse history as a child, mother's expectations of the child).
3. Family assessment scales: These assess such factors as family cohesion and adaptability ( [Olson et al., 1979](#)), child well-being ( [Magura and Moses, 1986](#)), and family risk ( [Magura et al., 1987](#)).
4. Child at risk field: This method uses an ecologic approach with 14 open-ended questions dealing with family, child, parent, maltreatment, and intervention issues.

All these measures have some face validity and some content validity, and child welfare workers usually agree on low-risk cases ( [English and Aubin, 1991](#)). However, the number of false-positive results is high, and there is no empirical evidence that any of the many available risk scales reliably predicts serious parenting problem behaviors ( [Wald and Woolverton, 1990](#)). Thus, most parents who drink, have a history of abuse, and have two or more preschool children will never hurt their children. Furthermore, a risk factor may be offset by one that can function as a buffer (e.g., a positive abuse history associated with high educational achievements). Some risks may also be more transitory (e.g., number of preschool children at home), and there are important developmental and other unpredictable components in all behaviors. Finally, cultural factors can significantly modify at least some risk factors for psychopathology in children from other cultures ( [Minde and Nikipota, 1993](#)). For example, family boundaries and kinship relationships even in a more collective subsociety within the United States may convey protection against maternal rejection. For example poverty, a high-risk factor for abuse in white neighborhoods, carries a much lower risk in black neighborhoods because poor African Americans generally have more social cohesiveness than equally poor white Americans ( [Korbin et al., 1998](#)). The previously mentioned work on the developmental appropriateness of an authoritarian parenting style among African Americans ( [Darling and Steinberg, 1993](#)) is also relevant here.

In summary, there is general agreement about the need to evaluate parenting competence and associated risks in many cases. However, at present, no instruments can conceptualize and assess parenting practices and styles with sufficient predictive validity to help clinicians in their day-to-day work. Future research in this area must be sensitive to the parameters of developmental psychopathology; that is, it must recognize developmental changes in potential victims, as well as policy, ethical, and cultural issues ( [Hamby and Finkelhor, 2000](#)).

## PARENT EDUCATION

Recognizing the powerful impact of primary caretakers on the behavior and emotional well-being of their children, also as future adults, has long stimulated educators and clinicians to think about how one can educate parents for the task of raising children and how to make them more sensitive to their needs ( [Sunley, 1955](#)). Although many different parenting programs have been offered to the public during the past 50 years, there have been until recently no specific guidelines for empirically validating parent education programs ( [Fine and Henry, 1989](#)).

1. Information sharing: This consists of providing information about developmental expectations, legal rights of parents and children, and other factual information. Lectures, printed materials, or group discussions are used to reach parents.
2. Improving self-awareness: Parents are encouraged to reflect on their own behaviors, to help them understand how their actions may influence their children. Group discussions, specific individual activities, and the use of diaries or logbooks are the most common methods of instruction.
3. Skills acquisition: Parents learn ways to manage children's behavior appropriately. They are trained through group discussions, practice assignments, and analysis of videotapes.
4. Problem solving: Parents are taught the use of more constructive strategies to deal with situations that may previously have led to conflict and arguments. These skills are taught by using feedback from reported actual events at home, direct observations during group sessions, and videotapes or audiotapes.
5. Psychoeducational preventive intervention: These programs aim to identify etiologic factors that may compromise parenting skills (e.g., a parent's mental illness or an infant's high-risk condition, such as prematurity or hyperactivity) and to enhance the strengths of parents and children. Intervention plans are individually tailored and designed to establish a therapeutic alliance within which information about a psychiatric illness or medical condition and the need for adjusting parenting practices become clinically meaningful ( [Beardslee and Poderefsky, 1988](#); [Beardslee et al., 1993b](#); [Brent et al., 1993](#); [Minde et al., 1980](#)).

Parent education programs are generally based on one of three major conceptual models—rogerian, skinnerian, or adlerian, each focusing on different aspects of development and addressing different areas of caregiver and child behavior.

1. Rogerian programs or reflective counseling: These programs are based on Carl Rogers' teaching and emphasize the understanding and acceptance of the child's feelings. Parent Effectiveness Training or PET ( [Gordon, 1975](#)) is a good example of this approach. Reflective counseling also helps parents to become more authoritative in their interactions with their children.
2. Skinnerian programs or behavioral management courses: Managing the actual behavior of children through behavioral analysis is the dominant feature of these parent education courses, such as the Responsive Parenting Program ( [Clark-Hall, 1978](#)). These programs emphasize information sharing and skills training and devote less attention to improving parental self-awareness.
3. Adlerian counseling: Education programs based on Adler's teaching emphasize that parents must understand the goals and meanings of their children's



behavior and use logical consequences to control it. The best known proponents of this approach are [Dreikurs and Soltz \(1964\)](#).

The effectiveness of these programs has not been well established, mainly because of problems with methods, objective measurements, matched samples of control populations, and adequate follow-up evaluations ([Medway, 1989](#)). Another problem is that different programs demand varying degrees of training and sophistication from their respective group leaders and therapists.

Despite such difficulties, there have been attempts to assess the effectiveness of parent education ([Beardslee et al., 1993b](#); [Minde et al., 1980](#); [Patterson et al., 1983](#)). In addition, in a metaanalysis of the 24 scientifically most acceptable programs, including studies of 12 behavioral training programs, 5 PET programs, and 7 adlerian programs, [Medway \(1989\)](#) reports that all of them are effective in that children and parents show an average of 62% improvement over their respective control populations. Those investigators who followed-up their samples for up to 6 months indicate that gains are enduring. Despite similar final overall outcome, data suggest that the behavior-management approach improves child behavior more rapidly than does reflective or adlerian counseling.

Programs using preventive psychoeducational interventions in clinical settings have typically been carefully evaluated ([Beardslee et al., 1993b](#); [Minde et al., 1980](#)). However, because group leaders usually require lengthy training and detailed knowledge of specific medical or psychiatric conditions, it is difficult for others to replicate the reported treatment effects.

In summary, there is some evidence that parent training can be helpful to interested adults who are committed to improving their parenting skills. Although parent education programs may be based on various educational models, there appear to be few if any differences in their effectiveness. This finding suggests that behavioral changes in children can be brought about by parental insight, by various interactional strategies, and through direct behavioral intervention by the parent. However, the long-term effects of such changes and their relation to more global behavioral variables, such as problem solving or social skills in children, have so far not been established.

## CONCLUSIONS AND UNRESOLVED ISSUES

The research discussed in this chapter shows the complexity of the factors that constitute the task of parenting. We have tried to outline some possible processes of psychological development that underlie the various research findings. We have also attempted to show how disorders of parenting are not static single variables but are best understood within the context of developmental psychopathology; that is, they reflect interactions between internal representations within parents and outside stresses and supports, as well as a range of child-related variables.

Although some of our data are strong enough to provide a basis for teaching those who come to us for advice, many of our hypotheses need further elaboration and confirmation. For example, we are still learning about the degree to which genetic factors determine parenting competence and about the compensating influence of nonparental caretakers on the later behavior of a child.

We also still poorly understand the effects of some child-centered mediating variables. For example, does the possibly genetic contribution children make to the parenting they receive operate through temperamental features or overall vulnerability to stress or through ways in which aspects of the environment are selectively perceived by the child, thus leading to greater or lesser levels of experienced adversity? The difference made by sex or the presence of older and younger siblings in the children's vulnerability to abnormal parenting also needs to be further explored. However, it is well recognized in child psychiatry today that developmental pathways show both continuities and discontinuities, and few experiences cannot be made up later in life; therefore, the study of parenting competence is central to the development of preventive and therapeutic interventions.

Finally, we need to be aware of the potential impact of a volatile political climate on the "status" of children and their caregivers. There are now rapidly growing organizations, such as No Kidding (from two to 47 chapters in 5 years) and online sites such as THINKERS (Two Healthy Incomes, No Kids, Early Retirement) versus SITCOMS (Single Income, Two Children, Oppressive Mortgage), whose protagonists aim to influence government support for parents in the United States ([Belkin, 2000](#)). Adequate parenting, although clearly centered within the personal experiences and philosophy of the individual, needs the continuous nurturing by society to ensure not only the best possible development of our children, but also the future health and welfare of this very society.

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## 38 EFFECTS OF CULTURE AND ETHNICITY ON CHILD AND ADOLESCENT DEVELOPMENT

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*Culture* consists of the variable values, attitudes, beliefs, and behaviors shared by a people and transmitted between generations. They constitute “a way of life” that helps to shape the developing personality from infancy through adulthood. It is generally accepted that cultural forces imprint the human personality, the part of us that deals with the outside world. But how? Culture is not simply a layer on top of a biological core. Rather, cultural influences are woven into personality like a tapestry. The development of this tapestry can actually be seen by watching the direct influence of child-rearing practices on the evolving personality of the child. In every country across the globe, the world of the infant is seen through the lens of a parenting person or persons. In many cultures, the main parenting person is the mother, with the father an important but secondary influence. In Asian societies, however, grandparents also have an important child-rearing function, so the refraction of the lens becomes more complex. Conversely, in African societies, infants and preschool children are managed by older siblings, who offer them a very different view of the world (Earls, 1982). However, whoever the primary caretaker may be, the infant's world soon grows to include others—first relatives, and then neighbors, who add new perspective. Later, the child's world expands to include community influence—other adults such as schoolteachers and peers. As the number of interaction partners expands and changes, the mix of new perspectives forms a more and more complex pattern in the evolving personality tapestry.

The view that an infant needs a primary relationship with one individual has been the traditional dominant view of social development, sometimes called the *continuous care and contact model* (Tronick et al., 1992). It was derived from the studies of Spitz (1965), Bowlby (1969), and Provence and Lipton (1962) on institutionalized children in the Western world. However, more recent work with more representative community population samples in other parts of the world suggests that early social experiences may be powerfully shaped by others than the primary parenting person, including fathers, siblings, and other children (Morelli and Tronick, 1992; Whiting and Edwards, 1988; Zukow, 1989). A newer *strategic mode* has emerged, that infant social engagement occurs in an interactive context heavily influenced by group composition, values, and customs. It is based on studies (Tronick et al., 1992) that show that patterns of simultaneous multiple relationships, rather than a single “mothering” person, may be determined by physical and social ecologic factors. An intriguing notion has been put forth by these developmentalists that such cultural practices may develop in the child a basic sense of self that incorporates other people and, thus, buffers against feelings of insecurity. It can even be hypothesized that the enhancement of self-confidence through multiple secure bases in the surrounding world allows children in some societies to explore their social and physical environment more readily.

### CULTURAL VARIATIONS IN CHILD REARING

How has the influence of culture on personality been studied? What is the evidence that differing child-rearing practices produce differences in personality traits? An anthropologist, William Caudill, was the first to scrutinize the interaction between children and the culture in which they grow. He found consistent cross-cultural differences in behavior that persisted from infancy through early childhood (Caudill and Schooler, 1973; Caudill and Weinstein, 1969; Tulkin and Leiderman, 1973). Caudill and Weinstein concluded (Caudill and Weinstein, 1969) that in Japan, the infant is perceived as a separate biological organism who needs from the beginning to be drawn into increasingly interdependent relationships with others, whereas in the United States, the infant is seen more as a dependent biological organism who needs to be made increasingly independent of others.

More recent, systematic studies of larger populations (Joshi and MacLean, 1997) point out that these early researchers focused too narrowly on the single trait, that is, autonomy, one that is valued and encouraged in the Western world, but that no culture, Western nor non-Western, really expects a child to become completely behaviorally autonomous. Indeed, it has been shown (Roland, 1998) that previous anthropologic writers failed to recognize that there can be other kinds of emotional and cognitive variations with their own developmental stages and schedules, which are highly adaptive to different social and cultural contexts.

Later studies further defined some of these differences (Stevenson and Lee, 1990). For example, a systematic investigation of Western and Eastern children's academic performance finds that U.S. mothers view their children as born with or without academic abilities, whereas Asian mothers are more likely to believe any child can succeed at reading or mathematics if he or she works hard enough. In other words, the emphasis given to effort, relative to ability, is much higher among Asian mothers than among U.S. mothers. These cultural beliefs are reflected in the children themselves; Asian children believe in the importance of effort, whereas U.S. children believe in the role of ability. The practical implications, of course, are that Asian children may work harder if they believe achievement depends on effort, or they may believe that effort is important because they have been successful. Similarly, U.S. children may do better because they believe in their ability, or they may perceive themselves as able because of how well they have done. The conclusion is that the belief that increased effort pays off in improved performance may be an important factor and may account for the time and effort Asian children, teachers, and parents spend on children's academic performance.

The effect of this different value system can be seen beyond the individual and small group. It is reflected, for example, at a national (and international) level in the so-called national character that is partly responsible for the economic success of Asian countries. National commitment to the success of the group over that of the individual and the relative absence of adversarial relationships in the workforce have shifted competition from within the group to another level—with other countries. Cultural values, built in from birth, rather than simply population numbers may be important in Asia's economic productivity.

Observations of children's play also reflects the influence of cultural norms about social behavior and self-expression (Farber and Shin, 1997). Children whose culture emphasizes a relational mode characterized by group interdependence, sensitivity to others, and a collectivist orientation reflect these cultural values in play. Conversely, children whose culture emphasizes independence and self-expression show play that is more conflictual. Whereas the former children enact more familial everyday activities and family role themes, the latter children enact more dangerous and fantastic themes.

Reports of child-rearing practices in other cultures featuring group settings with multiple caretakers have also highlighted the factors shaping personality. In the kibbutz (Beit-Hallahmi, 1977), the number of significant others interacting with the child is higher than in the traditional family unit, but the relationships among these figures is nonexclusive and is often discontinuous; that is, the caretaker is likely to be changed several times during childhood, so the youngster is exposed to a uniform, but less personal, treatment than in the traditional family. Theoretically, this weakens the dependence on a single significant figure, such as a parent, and reduces the intensity of feelings toward specific individual, by spreading it among others (Rabin, 1965). Indeed, a “kibbutz personality” has been described, one that shows positive but only moderate attachment to others, with a reduction in both intimacy and rivalry with peers. However, few differences are found between kibbutz and nonkibbutz children within the same culture because the Israeli culture as a whole is more collectivistic than Western societies.

In any case, we can conclude that human development is not bound to a single cultural model. Some cultures are individual centered, placing emphasis on independence and self-reliance, whereas others emphasize the interdependence of individuals within larger groups such as the family (Chiu, 1987). In these cultures, when individuals are successful, that success is attributed to the group rather than to the individual.

The ethnocentricity or culture blindness of Western theorists is illustrated in our most widely held paradigms of emotional and cognitive development. For example, the concept most central to psychoanalytic theory, the Oedipus complex, was shaped by the middle-class family roles of Little Hans and his parents in 19th-century Vienna. Piaget's own grandchildren in turn-of-the-century Geneva served as his research subjects, and generalizations were made from their cognitive development to that of others throughout the world. Kohlberg's six-stage hierarchy of morality clearly reflects a narrow cultural ideal and one that is male oriented. These observations about the cognitive and emotional development of a few middle-class European children led to narrow conclusions and far-reaching generalizations to other cultures.

Investigators who have studied parenting and child development across cultures (Bornstein et al., 1998; Shweder, 1991) argue that culture constitutes a lens through



which reality not only is seen, but also is interpreted and even constructed. Values deemed to be normative in one culture can be deviant in another; those deemed to have one sort of outcome in one society may well have other outcomes in other societies. Studies of children in cultures whose child-rearing patterns differ from one another teach us not only different models of child development but also what those different models have in common. The Whittings were an anthropologic team who conducted the first large-scale international research project to observe children systematically. They studied adult expectations of child behavior in seven different cultures ([Whiting and Edwards, 1988](#); [Whiting and Whiting, 1975](#)). Their findings led to two major conclusions. First, they found that the traditional stereotype of maternal behavior characterized by nurturance must be revised. Across all the cultures studied, parental efforts to control their children's aggression loomed much larger than nurturance in the profile of mothers' behaviors. Second, the influence of siblings and peers appeared to occur earlier and to be more powerful than previously thought. The influence of children on children, whether siblings, friends, or day care companions, must be better understood to outline a model of personality development.

## EFFECTS OF MINORITY STATUS ON PERSONALITY

Let us move from the usual way in which cultural forces are considered—that is, the effects of child-rearing practices on personality formation—to a consideration of the effects of negative subcultural forces on development. Some of the most important findings about personal identity and self-esteem have emerged from studies of minority children in the United States. Much of this work relates to locus of control. Individuals differ in the extent to which they believe that their own efforts can control the environment. This sense of internal control was traditionally found to be stronger in white children than in black children living side by side but having very different sociocultural experiences ([Karon, 1958](#)). In other words, caste or discrimination may result in lowered self-esteem in the minority group children. As one may expect, however, the context is an important variable. Other studies indicate that ethnic minority status itself is not related to lower self-concept ([Long and Hamlin, 1988](#)), if readily identifiable models for the self can be found among the minority group ([Powell, 1985](#)).

Studies of Native American culture carry these findings further. Cultural beliefs about the nature of the world affect locus of control and shape personality development. American Plains Indians emphasize a unity with nature. Thus, acceptance of natural events such as life and death, geography and climate, is the norm, rather than action to control or alter them. These values appear to produce accepting attitudes and behaviors in the children, in contrast to the competitive achievement orientation of Caucasian children in the same culture ([Long and Hamlin, 1988](#)).

Sharing is an important value taught to Native American children ([Yates, 1987](#)). Allegiance to the family and community is valued more highly than allegiance to the self. Competitive striving is not highly valued. Thus, a particular form of individualism emerges, one that is adaptive in Native American culture, but not in the larger outside world ([Wax and Thomas, 1972](#)). Life is considered to be an unhurried natural progression, with disease, death, and disability accepted as part of life's progress. Children progress at their own pace and are responsible for making their own choices. The result is adaptive within the culture but not across cultures. The basic cognitive development of Native American children approximates that of Caucasian children, but progress on culture-bound standardized tests declines as the verbal content of the scales becomes more prominent with increase in age ([Cazden and John, 1971](#)).

## EFFECTS OF ACCULTURATION ON PERSONALITY

*Acculturation* ([Berry, 1979](#)) is defined as a shift between cultures that forces personality change on the individual. The amount of stress on the developing personality is directly proportional to the distance between the two cultural groups and to the degree of insistence on change. The highest stress is seen in those groups that have been uprooted and geographically transplanted to another culture and have lost traditional social supports. In these cases, youngsters are confronted with a conflict between traditional values from the old country and contemporary values from the new country ([Lee, 1988](#)). Differences between conflicting values of the two cultures then become highlighted: group versus individual, extended versus nuclear family, interdependence versus independence, conformity versus competition, past versus future orientation, emphasis on age versus youth, harmony with nature versus conquest of nature, fatalism versus mastery of one's own fate, patience and modesty versus aggression and assertion, suppression versus expression of emotion. In successful acculturation, adaptation can be seen to occur by the formation of various combinations of these personality traits. However, conflict in values between the two cultures may delay acculturation ([Messer and Rasmussen, 1986](#); [Tobin and Friedman, 1984](#)). For example, many Southeast Asian adolescents who come to the United States have been primary caregivers or breadwinners for their families. Young girls may have been responsible for the care of the younger children; young men may have been heads of households, primary wage earners, or soldiers. To come from a culture in which one becomes an adult at puberty to a culture with the concept of adolescence as a prolonged transition between childhood and adulthood disrupts the developmental process. When a new set of values that reinstitutes dependence is superimposed on an old set in which independence had been achieved, the process of separation and individuation must begin all over again. Other studies of refugee teenagers ([Nguyen and Williams, 1989](#)) clearly demonstrate how contrasting values such as obedience to parental authority from the old culture versus freedom of choice regarding marriage and career in the new society are a source of stress to families and adolescents. The difficulty in the shift in values appears to be greater for girls, who experience more distress and resulting disruption of personality organization than do boys.

## CULTURE AND BIOLOGICAL ASPECTS OF PERSONALITY

Cross-cultural studies have consistently demonstrated that the pace and timing of the biological developmental process are significantly influenced by child-rearing practices ([deVries and Sameroff, 1984](#); [Levine, 1974](#); [Thomas and Chess, 1980](#); [Whiting and Child, 1953](#)). In one study, in which major influences on temperament are measured, cultural affiliation proves to be the strongest predictor of infant temperament in the first year of life. In other words, temperament is not a pure biological measure but is exquisitely sensitive to environmental influences. Perhaps temperament can best be viewed as a constellation of traits with a threshold for expression that varies from culture to culture. Temperament traits tend to fall into two clusters, which can be seen as culture related: rhythmicity, approach, and adaptability form one cluster, whereas activity, intensity, and distractibility form another. Group differences in neonatal behavior organization have been reported by comparing different cultural groups ([Coll et al., 1981](#)). Chinese Americans, Navajo Indians, and Japanese Americans are found to be temperamentally less excitable than other groups, as evidenced by lower levels of arousal, fewer state changes, and ease of consolability. Mexican Indians score higher on motor maturity, have smoother transitions from one state to another, and maintain quiet, alert states for longer periods, as do African infants from Kenya, who score higher on motor maturity and are better able to control increased motor tone.

[Lester and Brazelton \(1981\)](#) state that this motor precocity of African babies is facilitated by culturally determined child-rearing practices built on the infant's responsiveness to being handled in the neonatal period. Motor excitement of infants may elicit intense social handling from caregivers, and this, in turn, promotes accelerated developmental advance in motor skills. The conclusion is that child-rearing practices may facilitate early motor development through such an interaction.

Studies of babies' facial expressions of emotion yield somewhat different findings. Basic emotions of happiness, anger, fear, sadness, disgust, and surprise are the six emotions generally accepted as universal ([Ekman, 1992](#)). There is some evidence, however, that cultures may differ in their relative ability to recognize particular emotions, and the situations perceived as likely to give rise to these emotions may also differ among cultures ([Matsumoto, 1992](#); [Mesquita and Fujida, 1992](#)). Studies of the developmental differences in the recognition of emotion ([Markham and Wang, 1995](#)) find facial expressions of the six basic emotions universally recognized, but cultural differences in recognition of *specific* emotions suggest the extent to which learning may operate in moderating a hereditary trait. For example, the finding that children recognize happiness better than the other emotions across cultures suggests this to be the most commonly recognized facial expression. However, differences among cultures on recognition of sadness and anger suggest differences between collectivist and individualistic cultures.

To understand this interface between culture and child development better, Super and Harkness ([Super and Harkness, 1986](#)) conceptualize a construct called the *developmental niche*. (Niche is a term borrowed from biological ecology, meaning place in a biosystem.) It consists of three complementary components: (a) the physical and social setting, (b) the customs of child care and child rearing, and (c) the psychology of the caregivers. These are coordinated systems that relate to the larger culture and environment. Homeostatic mechanisms keep the three subsystems in harmony with each other and appropriate to the developmental level of the child. Although they are synchronous in developmental thrust, they constitute somewhat independent routes for disequilibrium and change.

Here lies the laboratory for studying the interaction of culture and biology. For example, Jerome Kagan's work shows that shyness, reflected in behavioral inhibition, is one of the fundamental dimensions of human social functioning, a universal biological rooted phenomenon ([Asendorp, 1990](#); [Kagan, 1989](#)). However, it is influenced by human inhibitory behavioral systems within social and cultural contexts. Culture not only imparts *meanings* to the behavior but also determines how other persons perceive and react to the behavior, eventually regulating and directing the developmental process of the behavior. For example, a study of behaviorally inhibited toddlers from different cultures, China and North America, finds that mothers differed in parenting practices based on their socialization values; that is, "inhibited" children are more readily accepted by mothers in the Chinese culture as opposed to the North American. Unlike their Western counterparts, shy-anxious children in China are regarded as socially important and are accepted by peers, and thus they adjust well to their social environment ([Chen et al., 1995](#)). In the West, conversely, children are expected to acquire assertive social skills, rather than be reserved and inhibited, so shyness and social withdrawal are observed to be associated with peer rejection and isolation. There appears to be a fundamental difference in cultural acceptance of biologically determined behavior. In Western cultures, as

opposed to Eastern, social inhibition reflects anxiety, inability to express one's self, and lack of confidence, qualities that are generally regarded as socially immature and psychologically maladaptive (Chen et al., 1998). This view and the finding that shyness and inhibition are correlated with peer and teacher acceptance in Asia, indeed, as markers of psychological adjustments indicate the extent to which behavioral inhibition is not simply a biological trait. It is a culturally bound construct that may have different adaptive "meanings" across cultures (Chen et al., 1995). Perhaps we can view this research on inhibition as an illustration of the Chess and Thomas goodness-of-fit model at a cultural, not just an individual, level.

## CULTURE AND SEXUAL IDENTITY

It is generally accepted that sex is biologically determined. However, *gender* is a broader term that includes cultural forces that shape that biological substrate. Indeed, there is evidence that gender orientation is determined as much by postnatal socialization influences as by the prenatal influence of sex hormones on brain development (Money, 1987). This concept is dramatically illustrated in cultures in which bisexuality is sanctioned. Among the peoples of the eastern highlands of New Guinea (Herdt, 1981), a developmental period of homosexuality preceding heterosexual adjustment is institutionalized. Prepubertal boys must leave the society of their mother and sisters and must enter the society of men to achieve the fierce headhunter's role of manhood. In infancy, the boy is fed women's milk to grow. Now, in the secret society of men, he must be fed men's milk—that is, the semen of mature but unmarried young men—to become pubertal and mature himself. Such an exchange is not allowed between adult men but is reserved for the adult-youth relationship. When the prepubertal boy reaches marrying age, a marriage is arranged for him, thus marking the shift to a heterosexual career. He cannot become a complete man on the basis of heterosexual experience alone. Full manhood requires a prior phase that is exclusively homosexual. Thus, homosexual behavior becomes a defining characteristic of masculinity in that culture.

Studies in which traditionally feminine tasks are assigned to boys or in which male-female roles are not differentiated (Whiting and Edwards, 1973) suggest that the nature of the task assigned, rather than the biological sex of the individual, is the best predictor of masculine or feminine behavior. In societies where boys cook, take care of infants, and perform other domestic functions, there are fewer differences between boys and girls. Boys score proportionately lower in aggression, are less dominant, and score higher in responsibility (Harkness and Super, 1985).

Conversely, other studies find that during certain phases of life such as adolescence, maturational forces weaken the influence of culture on the individual (Hsu et al., 1985; McDermott et al., 1983a; McDermott et al., 1983b). In a study of boys and girls from different ethnic groups in the United States, gender differences prove more powerful than cultural affiliation during adolescence. Although family loyalty and emotional constraint characterize Japanese-American families, compared with affective expression and individual orientation in Caucasian families, the adolescent sons and daughters of both sets of families group themselves by gender rather than by cultural affiliation. Girls, whether Japanese or white, value close family relationships and emotional expression significantly more than do boys. In following these Caucasian and Japanese teenagers from early to late adolescence, the researchers (McDermott et al., 1987) also find changes in male-female role perception that are gender related rather than culture related. Traditional roles (male-power and female-support) are perceived as merging by girls, whereas sons hold to an earlier male-female dichotomy. In other words, young men remain relatively fixed in their male-female role orientation regardless of ethnicity, whereas young women shift their role definitions, which earlier have been determined by family and cultural values.

Thus, the powerful influence of culture on personality formation appears to loosen in adolescence. As we have found, culturally determined parental values indelibly imprint the younger child, but broader social forces lessen the intensity of family forces as adulthood approaches.

## CONCLUSION

Culture provides one of the basic organizing principles of human behavior, a set of hidden rules by which individuals develop. Although early work on the interface between culture and development comes from anthropologic studies, more recent research uses large multiethnic population samples and tests of statistical significance to separate universal tendencies in different settings from the unique influence that cultural variation imposes. In any case, the link between cultural forces and personality is clear. Culture is a powerful shaper of behavior in both parents and their children, and it influences the age at which serious learning and work begin, as well as the very existence or nonexistence of adolescence as a transition from childhood to adulthood. However, beyond the direct effects of culture and ethnicity on personality development, current research suggests an illustration of the interaction of the environment with innate biological forces. The clinician may best approach each child or adolescent as a member of a specific culture with its own developmental pathways (and pitfalls), culturally determined symptomatic expressions of dysfunction, and culture-specific pathways of influence.

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# 39 CLASSIFICATION IN CHILD AND ADOLESCENT PSYCHIATRY: PRINCIPLES AND ISSUES

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## PRINCIPLES OF CLASSIFICATION

The ability and the urge to classify are unique aspects of human experience. They provide us with the capacity to observe, to order our observations, and to formulate general principles and hypotheses. Classification enables us to make use of information for purposes of communication, prediction, and explanation. At the present time in child and adolescent psychiatry, classification systems have their greatest role in facilitating communication for both clinical and research purposes; their role in prediction is somewhat more limited, and their explanatory value is often quite limited. The process of assigning a label may itself be associated with some sense of relief on the part of the patient or the patient's parents ([Werry, 1985](#)). Sometimes this reflects the misconception that having a label implies having an explanation ([Jaspers, 1962](#)). Like all human constructions, classification schemes can be abused or ill used ([Hobbs, 1975](#)). This chapter provides an overview of classification in child and adolescent psychiatry and an overview of the current official systems, that is, the 10th edition of the *International Classification of Diseases (ICD-10)* ([World Health Organization, 1992](#)) and the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* ([American Psychiatric Association, 1994](#)). (Specific criteria for each disorder are discussed in detail in subsequent chapters.)

Various authors ([Blashfield and Draguns, 1976](#); [Hempel, 1961](#); [Jaspers, 1962](#); [Quay, 1986](#); [Rutter, 1965](#); [Rutter et al., 1975](#); [Spitzer and Cantwell, 1980](#)) discuss criteria for psychiatric classification systems. There is no single "right" way to classify disorders in childhood. Classification systems vary, depending on the purpose of classification and what is being classified. As described later, "official" diagnostic systems have tended to adopt, on the whole, a categorical approach, but a dimensional approach would be equally as applicable, if perhaps less useful for clinical purposes.

The goals of classification include facilitating communication among professionals, providing information about given disorders that is relevant to treatment or to prevention, and providing information useful for research aimed at understanding the pathogenesis of disorders. To achieve these goals, classification schemes must be readily and reliably used by clinicians and researchers, hence the need for systems that are readily comprehensible. The disorders should be described, so they can be differentiated from one another. Disorders should differ in important ways, such as associated features and course. The classification system must be applicable over the range of development and must be comprehensive and logically consistent ([Rutter and Gould, 1985](#)). A classification of disorders implies that some clinically significant patterns of symptoms, behaviors, and signs are observed and comprise a source of significant distress or impairment ([American Psychiatric Association, 1994](#)). Deviant behavior itself does not necessarily constitute a disorder unless it is a manifestation of dysfunction within the individual person (e.g., conflicts over political beliefs do not constitute a mental disorder). Although it is often assumed that mental disorders must have a biological basis, this need not be the case; for example, maladaptive, enduring personality patterns can readily be classified as disorders ([Morey, 1988](#)).

Development of a general classification system for psychiatric disorders inevitably involves various tradeoffs. General classification systems must cover the entire range of disorders in a logically consistent fashion; classification systems developed for a highly specific purpose do not share this concern. The need for reasonable parsimony must be balanced with the need for adequate coverage ([Rutter, 1965](#); [Werry, 1985](#)). The needs for a clinically relevant system differ somewhat from those for a research system; for example, highly detailed criteria may be useful for research purposes but are cumbersome in clinical practice. Different diagnostic systems address these issues in different ways. Thus, the DSM-IV ([American Psychiatric Association, 1994](#)) is intended to be useful for *both* clinical work and research, whereas the ICD-10 ([World Health Organization, 1992](#)) system provides *separate* clinical and research descriptions.

## ISSUES IN CLASSIFICATION

### Developmental Aspects

Developmental considerations assume major importance in the provision of a classification scheme for children and adolescents, and, indeed, for adults as well ([Zigler and Glick, 1986](#)). Some disorders such as autism have their origin in a specific developmental period, whereas others are frequently associated with developmental problems (e.g., Tourette syndrome may be associated with attentional difficulties). At other times, the child's overall level of development may have a major impact on the ways in which various disorders can be expressed (e.g., the child with mental retardation who also exhibits conduct problems). Classification systems must be able to encompass such issues without simultaneously making the disorder so developmentally specific that the utility of the category is compromised.

The developmental approach to classification is used whenever disorders are viewed in the context of the unfolding of basic developmental processes. The use of standard, developmentally based assessment instruments such as tests of intelligence or communication skills exemplifies this approach. In contrast, many categorical and dimensional classification systems rely on assessment of deviant behavior. The use of such an approach is often complicated because issues of how deviant behavior is to be evaluated and how instruments are to be "normed" become quite important, and reliability among examiners can be low. Both the ICD-10 and the DSM-IV systems include some categories in which the definition is fundamentally developmental (e.g., mental retardation, articulation disorders), whereas in others the deviant nature of the disorder predominates (e.g., autism, schizophrenia of childhood onset).

### Role of Theory

Theoretical models of psychological disturbance have developed from rather diverse historical traditions; they have considerable value for the individual clinician in understanding and treating children with emotional and behavioral problems. For example, Anna Freud proposes a developmental profile based on and applicable to psychoanalytic assessment of children ([Freud, 1965](#)). More phenomenologically based classification systems can be traced to Kraepelin's delineation of schizophrenia and bipolar disorder ([Mattison and Hooper, 1992](#)). In the early "official" classifications, theoretical concerns were reflected in terms such as "schizophrenic reaction of childhood" or "obsessional neurosis." Classification schemes that are driven by theory are limited because, by their nature, they are based on a set of assumptions and hypotheses not usually generally shared and may give rise to different terms used to describe the same clinical phenomena; for example, a learning theorist may invoke principles of conditioning to explain a child's phobia, whereas a psychoanalytically oriented theorist may be more concerned with the child's level of psychosexual organization. Particularly following the work on development of research diagnostic criteria ([Feighner et al., 1972](#)), the phenomenologic approach to classification has predominated in the various "official" diagnostic systems. The more robust diagnostic concepts have typically emerged from clinical experience



rather than from theory ([Weber and Scharfetter, 1984](#)). In some instances, a theory has been invoked to account for a given set of phenomena, but it is the set of phenomena rather than the theory that has endured. For example, Langdon Down provided a complex theoretical explanation for children with the condition now known as Down's syndrome. His theory, based on obsolete racial stereotypes (mongolism), was incorrect, but his observation of some element of commonality among a large group of children with mental retardation has proved enduring.

For clinicians with pronounced theoretical views, the more phenomenon-based approach can be a source of frustration. It is sometimes incorrectly assumed that in such an approach such matters as history, course, and outcome, and, for that matter, etiology and theory are irrelevant to classification. Information on course and outcome may provide important data relative to external validation of diagnostic categories, and information on the development of the disorder may be highly relevant to differential diagnosis regardless of how similar, at one point, two different disorders appear to be. For example, the syndrome of childhood disintegrative disorder clearly appears to resemble autistic disorder once it is established; however, patterns of early development and outcome differ in these conditions ([Volkmar and Cohen, 1989](#)). Theoretical views of conditions and mechanisms remain highly relevant for both clinical work and research because they are more likely to generate truly testable hypotheses.

## Etiology and Classification

It is often assumed that classification systems are developed to approximate some ideal diagnostic system in which the cause could be directly related to clinical condition. This is not, in fact, the case, in that no single ideal system is waiting to be discovered and that cause need not be included in classification systems ([Rutter and Gould, 1985](#)). Similarly, classification need not reflect a "disease" model ([Rutter and Gould, 1985](#)). Different etiologic factors may result in rather similar conditions, and the same etiologic factor may be associated with a range of clinical conditions. Aspects of intervention may be more directly related to the clinical condition than to the cause. Remedial services for children with mental retardation are, for example, much more likely to be oriented around aspects of developmental level than around the precise origin of the specific mental retardation syndrome. With a few exceptions (e.g., reactive attachment disorder in the DSM-IV), etiologic factors are not generally included in official diagnostic systems.

## Contextual Factors

In certain situations and populations, contextual variables such as family, school, or cultural setting pose major complications to diagnosis. The attentional problems of a child whose difficulties arise only as a result of an inappropriate school placement would not, for example, merit a diagnosis of attention deficit disorder. Contextual variables are particularly problematic in disorders of infancy and early childhood in which the infant exerts effects on the parents, who, in turn, exert effects on the child; attributions of causality may be particularly difficult to make ([Bell and Harper, 1977](#)). A few of the traditional categorical disorders can be readily observed in infants and young children (e.g., autism), but generally clinical complaints in this age group are usually centered around problems that encompass the infant in the context of his or her family or life situation. Although research on disorders of infancy is limited ([Zeanah, 1993](#)), it is clear that infants exhibit a tremendous ability to react, even over relatively short periods, to their environment, and change, rather than stability, is often the rule ([Kagan, 1971](#)). Clinical problems often relate more to issues of *goodness of fit* between parents and the infant than to a disorder in the infant ([Chess and Thomas, 1986](#)). As children become slightly older, traditional diagnostic groupings become more readily applicable ([Earls, 1982](#)). Issues of developmental level also become important in specifying inclusion and exclusion criteria for diagnostic categories; for example, a diagnosis of pica may be appropriate for a 12-year-old child with profound retardation but is less appropriate for a normally developing 10-month-old infant.

Cultural differences may also affect diagnostic concepts and practice ([Mezzich and von Cranach, 1988](#)). Clearly, certain sociocultural factors are associated with certain types of problems (e.g., economic disadvantage is associated with conduct and attentional problems), but the meanings of such relationships often remain unclear ([Farrington, 1986](#)).

## What Is Classified?

It is particularly important that clinicians and researchers alike bear in mind that disorders, rather than children, are classified. This is a source of considerable confusion. Concerns have been raised about the possible effects of labeling children ([Hobbs, 1975](#)), and to some extent these concerns are valid. It is, of course, also the case that having an adequate label for a child's disorder may be helpful, for example, in securing needed services for the child. Thus, a diagnosis of mental retardation or learning disability may be associated with social stigma or other untoward effects, or it may be associated with more realistic expectations on the part of parents and teachers and provision of potentially more appropriate services. These tensions are also exemplified in the debate between those who advocate broad and encompassing definitions (e.g., to maximize service provision) and those who advocate narrow definitions (e.g., by defining more homogenous groups of research subjects).

Similar debates arise about aspects of social stigma related to mental illness and behavioral and developmental problems. In this regard, it is always important to refer to the child's disorder, *not to the child as the disorder*. The term *diagnosis* refers both to the notion of assigning a label to a given problem and to the act of evaluation. In important respects, it is the diagnostic process ([Cohen et al., 1988](#)) that is the most important of the two. Although diagnostic labels have considerable value, they do not provide information specifically about the individual person, who is unique and uniquely related to intervention. Diagnostic categories will, and should, change, and children may exhibit a disorder for variable periods.

## VALIDATION AND STATISTICAL ISSUES

As official classification systems have become more complex and sophisticated, issues of reliability and validity have assumed increasing importance. For example, both the DSM-IV and the ICD-10 use results of large national or international field trials in providing definitions of disorders. Categorical and dimensional approaches to classification share certain statistical concerns ([Blashfield and Draguns, 1976](#)).

### Validity

*Validity* is the extent to which a classification system does what it purports to do in terms of facilitating communication, intervention, and research. Various types of validity have been identified, for example, *face validity* (a judgment about whether the description of a category appears to represent the diagnostic construct reasonably), *predictive validity* (whether some aspect of subsequent course or response to treatment is predicted), and *construct validity* (whether the category has meaning in terms of what it purports to assess). Generally, such concepts are most useful in measuring the validity of psychometric assessment instruments; their applicability to classification systems is somewhat different. In general, childhood psychiatric disorders have face validity but not necessarily predictive or construct validity ([Spitzer and Cantwell, 1980](#)). The validity of a given diagnostic category can be established on the basis of its association with various features other than those incorporated in the definition (e.g., response to treatment, natural history in the absence of treatment, family pattern, biological correlates, and developmental correlates such as age at onset and intelligence quotient). The validity of some categories included in the revised third edition of the DSM (DSM-III-R) appears to be questionable (e.g., identity disorder), whereas for other categories (e.g., autism) ([Rutter, 1978](#)), the disorder appears to have considerable validity, but the statistical data on reliability and validity of the various rating scales used to assess it are more questionable.

The sensitivity and specificity of a given categorical diagnostic instrument can be assessed relative to the true presence or absence of a specific disorder. However, a general problem for both categorical and dimensional classification systems is the nature of the standard against which a given category or criteria set is to be judged. Given the usual absence of an unequivocal diagnostic marker for the various conditions, clinical judgment is often used as the standard against which new instruments or definitions are assessed. The issues of "caseness" and diagnostic thresholds are particularly important in the derivation and validation of diagnostic systems ([Swets, 1988](#); [Valliant and Schnurr, 1988](#)).

### Reliability

In addition to validity, classification systems should exhibit *reliability*; that is, users in different locations seeing rather similar disorders should be able to agree on the applicability of a specific category or criterion ([Grove et al., 1981](#)). Various kinds of reliability have been identified: interrater, test-retest, and internal consistency. If a given category is not used reliably, it has little value for purposes of communication. Some disorders, almost by definition, have limited test-retest reliability over a relatively short period, whereas others are presumed to be highly stable (e.g., adjustment disorders versus profound mental retardation). Sources of unreliability in psychiatric diagnosis include differences in the kinds of information clinicians collect, theoretical biases in the clinician, and differences in diagnostic thresholds, as well as, of course, the true differences that persons with disorders will exhibit at various points.

High reliability does not guarantee validity; it is possible that a disorder could be reliably defined but have little or no validity. Conversely, a disorder may have validity, but criteria and diagnostic instruments designed to detect its presence may have little or no reliability. In providing diagnostic criteria and descriptions, there is often a trade off between the level of detail of a definition and its reliability. More detailed definitions, designed for research studies, may, for example, be used reliably in such contexts but may not prove reliable when they are applied by clinicians less experienced with the disorder. What appear to be relatively minor changes in the wording of a criterion can produce major changes in the way in which a diagnosis or diagnostic criterion is applied.

## Statistical Analyses

Various *statistical techniques* have been applied to data derived from assessment methods ([Achenbach, 1988](#); [Achenbach and Edelbrock, 1978](#); [Quay, 1986](#)). These techniques are theoretically of great interest in that they can provide more rational and empirical approaches in the derivation of diagnostic schemes. The fundamental assumption of such techniques is that the variables of interest lie along some dimension of function and dysfunction that all persons exhibit to some degree. For many types of problems, this assumption is probably justified, such as relative to anxiety or depression. However, the usefulness of such techniques is limited in important ways ([Rutter and Gould, 1985](#)). In the first place, these methods are highly dependent on both the sample and the type of data entered in the analysis. For example, *factor analysis* of an instrument designed to detect conduct problems would not likely produce a factor related to eating disturbance. Similarly, cluster analysis of even a very large normative sample would not likely produce a cluster that corresponded to autism, given the low base rate of this disorder in the population. For rare disorders, other statistical approaches may be useful. Other relevant statistical procedures include *signal detection analysis* ([Kraemer, 1988](#)), which can be used to establish which symptoms and symptom combinations are most strongly related to a particular diagnosis. Similarly, *latent trait analysis* and *latent class analysis* ([Szatmari et al., 1995](#); Zoccolilio et al., 1992) provide other approaches.

## MODELS OF CLASSIFICATION

Werry delineated three general approaches to classification of disorders: categorical, dimensional, and ideographic (1985). The *categorical approach*, sometimes referred to as the medical model of classification, views disorders as either present or absent (e.g., the patient does or does not have appendicitis). This approach assumes that patients exhibiting a given disorder display certain similarities, that these similarities outweigh differences, and that this knowledge has certain implications for understanding pathophysiology, course, treatment, and so on. Unlike the categorical approach, which views disorders as dichotomous, the *dimensional approach* to classification relies on assessment of dimensions of function or dysfunction by reducing phenomena to various dimensions along which a child can be placed. Various sources of data can be used for this approach, such as behavioral ratings, parental reports, yes-no criteria, developmentally based test scores, and the like. Although the dimensional approach is more commonly used in nonmedical settings, many medical phenomena also exhibit continuous (i.e., dimensional) characteristics (e.g., stature, blood pressure). For some purposes, categorical diagnoses (e.g., levels of mental retardation) are derived from what is essentially a continuous variable, whereas some dimensional assessment instruments can similarly be used to generate categorical diagnoses. *Ideographic classifications* reject simple labels and focus on the total context of the individual person; this approach may be theory driven (e.g., by psychoanalytic or behavioral theories) or may be used eclectically. Ideographic approaches are commonly used in clinical work; that is, the child or adolescent is viewed in the totality of his or her life circumstance, and various disorders, problems, and psychosocial situations may be viewed as worthy of notation and treatment.

## Categorical Approaches

The most widely used “official” systems are those developed by the World Health Organization (ICD-10) and the American Psychiatric Association (DSM-IV). Both systems have their historical origins in medicine in the 19th and 20th centuries as advances in diagnosis and public health concerns necessitated more systematic approaches to record keeping ([Table 39.1](#)). During the 19th century, many advances in the taxonomy of adult psychiatric disorders were made, and this led Kraepelin to attempt a comprehensive classification system ([Kraepelin, 1883](#)). By the mid-20th century, certain psychiatric disorders were generally recognized. The second edition of the DSM (DSM-II) ([American Psychiatric Association, 1968](#)) includes only a handful of diagnostic categories specific to children: mental retardation, childhood schizophrenia, adjustment, and other “reactions” (hyperkinetic, withdrawing, overanxious, runaway, unsocialized aggressive, group delinquent, and “other”). By the time the DSM-III appeared ([American Psychiatric Association, 1980](#)), the number of disorders first evidenced in infancy, childhood, or adolescence had increased more than fourfold to include the following major classes of disorder, each of which included some specific diagnostic categories: mental retardation, specific developmental disorders, attention deficit disorder, conduct disturbance, eating disorders, stereotyped movement disorders, pervasive developmental disorders, other disorders with physical manifestations, and other disorders of infancy, childhood, or adolescence (see [Mattison and Hooper, 1992](#), for a review). Similar, although not precisely corresponding, changes occurred in the revision of the ICD ([Rutter et al., 1975](#)). In the DSM-III, for example, disorders generally specific to childhood were grouped together, and there were many more DSM-III subcategories ([American Psychiatric Association, 1980](#)). The DSM-III and its successor, the DSM-III-R ([American Psychiatric Association, 1987](#)), differ from the ICD-9 in terms of their greater diagnostic reliance on explicit (if not always truly operationalized) diagnostic criteria ([Puig-Antich, 1982](#); [Spitzer and Endicott, 1978](#)). Both the DSM-III and the ICD-9 incorporate a multiaxial framework, although the specific systems adopted differ from each other in some respects. Both systems are hierarchically organized, although the DSM-III and the DSM-III-R encourage multiple diagnoses.

Kraepelin (1883): Proposal for a comprehensive classification system
ICD-6 (1948): Psychiatric disorders included
DSM-I (1952): First US official classification system
Group for the Advancement of Psychiatry (1966): Diagnostic system for children
DSM-II (1968): Some child disorders, emphasis on theory and Meyer's concept of reaction types
DSM-III (1980): Fourfold increase in child psychiatric disorders, greater diagnostic precision, multiaxial
DSM-III-R (1987): Refinements in criteria, categories
ICD-10 (1992): Separation of research diagnostic criteria from clinical descriptions
DSM-IV (1994): More emphasis on data based modifications in categories and criteria
DSM-IV-TR (2000): Generally minor revisions in text (not criteria)

DSM, Diagnostic and Statistical Manual of Mental Disorders; ICD, International Classification of Diseases

## Table 39.1. Landmarks in the Development of Psychiatric Taxonomies

Changes in the DSM-III-R and DSM-IV are generally less dramatic than in the DSM-III ([Schwab-Stone et al., 1991](#)). Work on the DSM-IV began in 1988 ([Frances et al., 1989](#)), stimulated, in part, by a treaty obligation that mandated terminology compatible with the ICD, which was also undergoing revision at that time. As part of the process of producing the DSM-IV, steps were undertaken to ensure that changes made were based on solid data and thorough documentation ([Frances et al., 1989](#)). This task was somewhat facilitated because it was clear that the DSM-III and III-R had stimulated considerable research of relevance to the DSM-IV ([Widiger et al., 1991](#)). Other issues for the DSM-IV relate to clinical utility and compatibility with ICD-10. As part of the revision process, extensive literature reviews were conducted, data sets were aggregated and analyzed or reanalyzed, and working groups of experts convened to evaluate the available data and, in some cases, to generate new data. For the child disorders section, field trials were conducted for disruptive behavior disorders ([Lahey, et al., 1994](#)) and for autism and related conditions ([Volkmar et al., 1994](#)).

The *International Classification of Causes of Death* was adopted by the International Statistical Institute in 1893 ([Kramer, 1968](#)). By the 1960s, the ICD had been revised six times, and the deficiencies of the ICD-7 for psychiatric disorders ([Rutter et al., 1975](#)) were particularly clear. Many changes were made in eighth edition of the ICD in 1968. Even when this edition was adopted, however, it was clear that further revision would be needed, and a process was instituted for further revision. An important part of this work was the development of a multiaxial classification system for child disorders ([Rutter et al., 1975](#)).

The ICD-9 was published in 1977, and work on its revision began shortly thereafter ([Brämer, 1988](#); [Sartorius, 1988](#); [World Health Organization, 1977](#)). As part of the extensive revision process, the number of categories increased substantially ([Cooper, 1988](#)), and a decision was made to have various versions of the system—one for primary health care providers, another for specialty-based researchers, and one for psychiatric practitioners. The ICD revision process did not, in contrast to the DSM-IV, employ extensive field trials. The ICD-10 draft generally had good clinical utility and good, although variable, reliability (the latter being less optimal for disorders with more subtle symptoms such as personality disorders) ([Sartorius, 1988](#)).



## COMPARISON OF THE ICD-10 AND THE DSM-IV

Although, on balance, the two categorical systems are more alike than different, there are some differences, some more and others less explicit, between the two systems. The ICD system has rather more constraints than the DSM, given its international nature and the fact that the psychiatric section is but one part of a large body of diagnostic coding. The systems also differ in the degree to which diagnoses are operationalized. The ICD-10 provides a comprehensive description of the clinical construct, followed by a discussion of differential diagnosis and major symptoms that should be present. In contrast, the DSM-IV is much more truly operationalized but is also accordingly somewhat less flexible for clinicians. Although both systems allow for multiple diagnostic codes, the ICD-10 also gives the option of applying some combination categories (e.g., depressive conduct disorder). Rutter discusses the pros and cons of this approach ( [Rutter, 1989](#); [Rutter and Tuma, 1988](#); [Volkmar and Woolston, 1997](#)). Although there are some issues relative to certain disorders (e.g., the anxiety disorders), in general the trend has been toward convergence of the DSM and ICD classification systems. The remaining differences largely stem from the emphasis of clinical-diagnostic guidelines in the ICD and the greater specificity for research of the DSM. The more recent text revision of the DSM-IV (DSM-IV-TR) ( [American Psychiatric Association, 2000](#)) includes generally minor modifications in accompanying text but not in criteria. With regard to children, probably the most extensive revision is that for Asperger's disorder; it is also the case that an error in the description of the "subthreshold" category "Pervasive Developmental Disorder Not Otherwise Specified" was corrected ( [Chapter 46](#)).

Detailed critiques of the various diagnostic approaches have appeared ( [Achenbach, 1980](#); [Garnezy, 1978](#); [Quay, 1986](#); [Rutter and Shaffer, 1980](#)). Criticisms have been made of both the overarching framework and its specifics. In the DSM-III in particular, certain categories were introduced on the basis of rather limited data. The reliability and validity of at least some of the various categories proposed (e.g., childhood-onset pervasive developmental disorder) were questionable. Reliability is generally best for the more common and more broadly defined disorders. Information on the stability of the various childhood diagnoses has been limited.

### Dimensional Approaches

In contrast to the more clinically oriented (categorical) approach, multivariate (dimensional) approaches to diagnosis offer several potential advantages in that various behaviors and dimensions of behavior are assessed, rather than single, presumably pathognomonic, features ( [Achenbach, 1988](#); [Achenbach and Edelbrock, 1978](#); [Quay, 1986](#)). Similarly, the dimensional approach can encompass symptom coding in other than a dichotomous fashion; for example, "never," "sometimes," and "always" could be coded, rather than simply presence or absence, to rate-specific diagnostic features. Various rating scales, checklists, and so forth can be used for multivariate classification schemes based on self, parent, or teacher report or on direct observation; many such instruments are described in subsequent chapters. As noted previously, various statistical techniques such as factor and cluster analysis may be used to derive relevant clinician patterns or profiles. These patterns may, in turn, be used to derive categorical diagnoses. Given the inherent problems in sample selection and instrument development, issues of replication are particularly important ( [Rutter and Gould, 1985](#)).

[Jenkins and Glickman \(1946\)](#) and [Hewitt and Jenkins \(1946\)](#) were among the first to examine patterns of relationships (correlations) between variables to derive syndrome groupings. A large series of case records was studied, and the presence or absence of specific behaviors was noted in each case. Clusters of deviant behavior were noted, and broad patterns of disturbance (socialized delinquent, overinhibited, unsocialized aggressive) were identified. Subsequently, more sophisticated methods have been applied to a range of children using a variety of assessment instruments ( [Achenbach, 1988](#)). Studies done using this approach generally identify several factors with relative consistency; factors identified have included conduct disturbance, overactivity, and emotional disturbance ( [Achenbach et al., 1989](#); [Rutter and Gould, 1985](#)). Not surprisingly, the stability of more narrowly defined factors is less robust. Similarly, as would be expected, such techniques have limited usefulness in detecting children with disorders of very low prevalence.

Reliability of dimensions derived from multivariate studies has been assessed and is generally satisfactory ( [Quay, 1986](#)). Stability of dimensions or profiles is somewhat more complex to assess, in that some change is, of course, expected, but short-term stability appears to be within acceptable limits ( [Quay, 1986](#)). The use of dimensional assessment instruments clearly avoids certain of the pitfalls inherent in the categorical approach, for example, in terms of the loss of information inherent in application of dichotomous categories, in increasing reliability, and in issues of "caseness." These issues are relevant to medicine in general and not just to psychiatry.

For some purposes, dimensional assessment instruments are particularly valuable. For such assessments to be clinically useful, their validity must be demonstrated, for example, in terms of some associated features, such as familial pattern or course. The assumption that characteristics have the same meaning throughout their distribution is often questionable (e.g., severe mental retardation differs in a host of ways from normal intelligence) ( [Rutter and Tuma, 1988](#)). Certain disorders clearly do *not* shade off into normality.

### Relationship of Categorical and Dimensional Approaches

There are major areas of agreement between both the categorical and dimensional approaches; this is particularly true for the more common disorders. Analysis of data from dimensional assessment instruments has proved useful in the development of categorical systems, for example, in supporting division of conduct disorder into various types. Probably the greatest drawback to the use of such assessments in clinical practice arises from the difficulty in using such instruments in a simple way for purposes of communication; for clinical purposes, it is more helpful to know that a child has attention deficit hyperactivity disorder and learning problems than to know his or her factor or profile scores on a dimensional assessment instrument. Dimensional and categorical approaches need not be used in mutually exclusive ways; the multiaxial classification used in the DSM-III-R, for example, employs both approaches in that although disorders are categorically defined, assessments of severity of psychosocial stressors and global assessment of functioning are dimensional.

### Ideographic Approaches

Ideographic approaches to diagnosis are common in clinical practice. In the broader sense of diagnosis (i.e., as diagnostic process) ( [Cohen et al., 1988](#)), most clinicians target certain problems or issues for intervention that relate only in part to categorical or even dimensional diagnosis. In some ways, such approaches are more practical in certain situations (e.g., family therapy), although again they can be used in conjunction with categorical approaches. They are less useful for certain purposes (e.g., in considering pharmacological intervention) ( [Werry, 1985](#)). Past the level of the individual cases, the utility of ideographic approaches is limited. Such approaches make it very difficult to communicate information for clinical and research purposes in a concise and readily understood fashion.

## MULTIAXIAL CLASSIFICATION IN CHILD AND ADOLESCENT PSYCHIATRY

*Multiaxial classification* offers considerable potential advantages for child and adolescent psychiatric disorder ( [Cohen et al., 1988](#); [Rutter and Gould, 1985](#); [Rutter et al., 1975](#)). In important ways, it parallels the diagnostic process in that different kinds of information are collected and coded independently. Given that the diagnostic picture is often complex and that different conditions and kinds of conditions are associated with one another, the use of a multiaxial system, at least theoretically, should help clinicians by directing their attention to the major relevant areas of diagnosis. In actual practice, clinicians vary considerably in their use of multiple axes, although the use of such a system would be expected, generally, to increase reliability. Putting developmental disorders on a separate axis would, for example, emphasize their developmental, as opposed to "psychiatric," nature and would remind clinicians to look for such disorders in the course of their regular clinical work. Conversely, the placement of certain disorders within a multiaxial framework is problematic; enuresis, for example, clearly has developmental correlates but is generally included as a psychiatric, as opposed to developmental, disorder.

In the absence of a multiaxial system, certain conditions are particularly likely to be overlooked, such as the developmental learning disorders of a child with conduct disorder. Similarly, coding of medical syndromes is helpful in alerting the clinician to potential problems that contribute to the mental or developmental disorder, are associated with it, or should be considered in the provision of a remedial plan. Theoretically, many different kinds of information could be incorporated within a multiaxial framework (e.g., intellectual level, adequacy of school placement, associated psychosocial problems). One of the dilemmas, particularly for disorders in adolescence, relates to the problem of comorbidity; that is, often children have more than one diagnosis. This comorbidity may be more apparent than real or may represent a more substantive problem in which, for example, having one disorder predisposes to the second or in which the risk factors for one disorder are also risk factors for the second. Artfactual comorbidity adds little to the classification system; for example, for a child with autism to also receive a diagnosis of stereotypy-habit disorder seems pointless because stereotypies are commonly observed in autism and are included as one of the potential defining features of this condition. In other instances, such as conduct disorder with depression, the particular comorbid pattern does appear to have some important distinguishing features ( [Angold and Rutter, 1992](#)). In ICD-10, the general approach is to provide a special code for such conditions, whereas in DSM-IV, the usual diagnoses would be made with no special notation of the concomitant occurrence of the condition. Despite this difference, the general difference in approach to clinical and research diagnosis, and some

differences around a few categories, these two systems have tended toward convergence.

## SUMMARY

Classification in child and adolescent psychiatry has multiple meanings and functions. Complications for classification of child and adolescent disorders are myriad: The child is often not the person complaining; different kinds of data may be used in making a diagnosis; developmental factors may have a major impact on the expression of disorders; and certain features (e.g., beliefs in fantasy figures) are normative at certain ages but not at others. Additional complications are posed by the unintended, but no less real, uses to which diagnostic concepts are put, such as their inclusion in legislation and their use as mandates for services in educational programs or for purposes of insurance reimbursement for services. Different kinds and levels of classification are needed for different purposes.

The 1980s and 1990s witnessed tremendous advances in the area of diagnosis and classification of child and adolescent psychopathology ( [Rutter and Tuma, 1988](#)). These advances are particularly welcome because work in this area has lagged behind that in the adult psychiatric disorders. Various approaches to classification have been employed; each has its advantages and limitations. Issues of reliability and validity remain to be addressed for many categories and classification systems; the attempt to address these issues through examining empirical data rather than theorizing is perhaps the greatest accomplishment of the 1990s.

Tensions between clinical and research utility will continue to exist. As classification systems become more complex, they are less readily used; conversely, simplistic systems fail to capture important aspects of clinical experience. The likely ability, over the next decade, to identify more clearly the role of genetic factors for at least a few conditions and the growing sophistication of statistical approaches to aspects of classification and diagnosis represent important areas for future work.

A classification scheme is best used by persons with considerable training who take the task of diagnosis (in its broadest sense) seriously. Although categorical systems increasingly use diagnostic criteria, these are often not truly operationalized, although they are often abstracted and reified for specific purposes (e.g., development of interview schedules administered by lay interviewers or the use of simple frequency counts or symptom duration that obscures the more central aspects of the underlying clinical construct). Although the various "official" systems present areas of disagreement, the areas of agreement are even more noteworthy. Certain issues, such as classification of comorbid condition versus use of multiple diagnoses, remain to be resolved. Specific issues arise with respect to inclusionary and exclusionary rules and aspects of comorbidity. Although much has been accomplished, considerable work remains to be done.

## Chapter References

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# 40 CLINICAL ASSESSMENT OF INFANTS and TODDLERS

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The developmental assessment of infants involves more than the simple administration of a set of developmental test protocols. Assessments performed in the first 3 years of life require the clinician to function simultaneously as a generalist and as a specialist, to blend quiet observation with active probing, to synthesize information from caregivers with that gathered through direct observation of the child, and to be involved in a curious blend of searching for specifically defined responses from a child with inferences based on behavior. This set of skills is indeed important for adequate clinical assessment of children of all ages. However, the need is even greatest for those providing clinical services to these youngest of patients, because development during the early years is the most rapid, context dependent, and intersystemic. Clinical assessment of infants and toddlers is indeed a subspecialty area of practice. Not all psychiatrists, psychologists, pediatricians, and other professionals possess the training and degree of closely supervised practice necessary to conduct such evaluations competently and independently. Those professionals who do possess these specialized skills typically have acquired them through formal subspecialization near the end of their professional training.

Most scientists and clinicians define the periods of infancy and toddlerhood as being the first 3 years of postpartum life. More specifically, *infancy* refers to the time before the explosion of expressive verbal communication that occurs at about 18 months. The developmental shift that occurs at this time has a massive transformational impact on the child's ability to reason cognitively, to deepen elaborate social relationships, to develop moral reasoning capacities, and to mediate emotional experiences linguistically. *Toddlerhood* is a period of increasing autonomy in which the child uses his or her new skills to explore their world, physically, cognitively, and socially. Regardless of the exact chronologic time frame, infancy and toddlerhood encompass the most rapid and contextually transactional period of neurodevelopmental change throughout the postpartum life span. Therefore, all clinical assessments of infants and toddlers are framed by the context of rapidly changing, growing systems that may be in or out of synchrony with one another.

In this chapter on infant assessment (for ease of communication, the term *infant* is used to identify both infants and toddlers, except when a clear distinction is made in the text), four areas of inquiry implicit in these introductory statements are addressed:

1. What is the history of the field of infant assessment, and what concepts underlie the process of infant assessments?
2. Why perform developmental assessments of infants? When are such assessments indicated, what can we learn from the process, and what, if anything, can we predict about the infant's future development?
3. What kinds of information define an assessment of an infant's development? Does such an assessment entail only a simple quantitative charting of emerging skills and capacities, or does it require a more complex qualitative evaluation of the infant's developing social, affective, and cognitive adaptation within the environmental matrix? What are the sources of data for this more complex assessment profile, and does the process itself have a therapeutic effect?
4. What are the available formal assessment tools? What are their strengths and limitations?

## BRIEF HISTORY OF INFANT DEVELOPMENTAL ASSESSMENT

A brief discussion of the history of infant assessment may provide an illustration of the evolving aims and technologic advances in this field. (For a more complete history of infant assessment, see Brooks and [Weinraub, 1983](#), and [Wyly, 1997](#).) In the late 19th century, the European scientific community was consumed with a fervor and creativity best characterized by the studies of evolution, theories about the unconscious mind, and a growing concern for the mentally deficient and insane. The concept of measuring infant capacities grew out of the concern of scientists of the time to find a metric for human intelligence that would identify the mentally deficient. The French had been the first to distinguish mental deficiency from insanity ( [Esquirol, 1938](#)), and in so doing, they had started a controversy about the educability of the former. Schools for the mentally retarded were being opened throughout Europe. The interest in measuring intelligence reflected a practical need to find criteria for mental deficiency, to develop standards for admission to such schools.

### Early 1900s: The Birth of Intelligence Testing

In 1904, the minister of public education in Paris appointed Binet and Simon to be members of a commission studying the question of special education. Binet, an experimental psychologist, had been interested in mental functioning in normal children, and Simon had worked with him on earlier versions of tests to rank intellectual functioning. In response to their charge from the minister, Binet and Simon developed the concept of *mental age*, which describes how an individual child's performance on tasks involving memory and reasoning compares with the chronologic age of a large group of children who succeed with the tasks. This test, published in 1905, revolutionized the testing movement, and the concepts quickly spread to other scientific and educational communities (Brooks and Weinraub, 1976). In the United States, Terman created an adaptation of the Binet-Simon test and revised their mental age concept by creating the intelligence quotient (IQ), a ratio between mental age and chronologic age multiplied by 100 ( [Terman, 1916](#)). It is not an exaggeration to say that the concept of IQ singularly preoccupied many psychologists for several decades. American psychologists of the early 20th century in particular believed that IQ was fixed, and they found it an exciting possibility that one well-administered test would provide stable, predictive information about a child's underlying potential.

In the decades that followed the development of Terman's IQ test, many psychologists turned their attention toward the subject of intelligence and development in infants. Two basic approaches were used in exploring intellectual development in infants. One approach, the *infant IQ model*, sought to extend the IQ assessment model downward to children younger than school age, eventually including infants. A second approach, the *Gesellian model*, sought to create a new model of assessment that began with the newborn and extended upward. Both these models represented a radical departure from the *case study method* of inquiry employed in the previous century. In contrast to these "baby biographers" of the previous century, the scientists of the 1920s and 1930s brought to the study of infant development a level of empiricism, championed by the behaviorist movement of the time, by relying on direct observations of child behavior across several children



under highly structured conditions.

### 1920s and 1930s: The Infant IQ Movement Versus the Child Study Movement

In the 1920s and 1930s, a few “infant IQ tests” were first introduced. The best example of such an infant IQ or infant cognition test is the *Cattell Infant Intelligence Scale* (Cattell, 1940). Cattell's test was conceptualized as a downward extension of the 1937 Stanford-Binet Intelligence Scale, the much-revised American version of the Binet-Simon test. Although some initial studies were encouraging, the ability of the Cattell and other infant IQ tests to predict an infant's later IQ was weak (Thomas, 1970). Overall, infant IQ tests were found to be inadequate for meaningfully extending the IQ assessment model into infancy and are now all but extinct in use.

#### ARNOLD GESELL AND THE CHILD STUDY MOVEMENT

At about the same time that the first infant IQ tests were being developed, Yale University psychologist and physician Dr. Arnold Gesell began his groundbreaking work on systematically documenting development in infants and toddlers. Several aspects of Gesell's work distinguish it from those of his predecessors and contemporaries. First, whereas the infant IQ tradition grew from an interest in identifying deviant patterns of development, Gesell's interest was mostly in documenting normal developmental trends, as espoused by G. Stanley Hall's Child Study Movement. Of course, this implied a model of developmental maturation, rather than the downward extension of the IQ model endorsed by many of Gesell's contemporaries. Second, Gesell's model supported the conceptualization of development occurring simultaneously in many distinct but interrelated domains (Gesell, 1940), whereas the infant IQ model was predicated on the proposed existence of a singular factor of intellectual ability. This conceptualization of multiple interrelated domains of development revolutionized the fields of developmental assessment and early intervention in a way that endures to this day. Third, the infant IQ model was a product of the prevailing conception of intelligence as an innate genetic endowment with infant assessment viewed as a way of quantifying that endowment. In contrast, Gesell's model of developmental maturation supported an understanding of the effects of the child's environment in altering the course of development and opened the way for the more transactional and dynamic understanding of developmental processes that would later prevail.

For Gesell, diagnosis was the primary objective of measuring infant development. He modeled his approach after the work of Wilhelm His, one of the first investigators of human embryology (Yang, 1979). In 1925, when his observational techniques were well established, Gesell wrote: “Just as the embryologist gets his basic conceptions of morphogenesis by building up indefatigably, step by step, detailed sectional views of growing organisms, . . . so may genetic psychology build up a continuing series of sections corresponding to the stages and moments of development” (Gesell, 1925). Gesell began his descriptions of infants at 3 months of age and initially included five other assessment points in the first 2 years (6, 9, 12, 18, and 24 months). For Gesell, the scales were descriptive, and specific items were included for normative purposes rather than for measuring intelligence. He did argue, however, that the scales were a measure of a child's capacity and personality characteristics (Honzik, 1983).

As early as 1916, Gesell and his colleagues began a series of meticulous observations of infants' and preschool children's behavior at home and in the well-child clinic, to define norms for behavior (Yang, 1979). In fact, Gesell was actually the first to develop the procedure of systematically observing the infant's natural responses in a natural setting (Buhler and Hetzer, 1935). The result of his work was a series of age-specific markers of normative development, known as the *Gesell Developmental Schedules*. His work was far reaching, and the developmental tasks and techniques pioneered by Gesell and his colleagues were borrowed extensively by subsequent researchers (Honzik, 1983). Indeed, many of the modern assessment instruments described later in this chapter bear a striking resemblance to Gesell's earliest schedules.

The Gesell Developmental Schedules addressed several areas of infant and toddler development, including motor development (posture, prehension); language development (comprehension, imitation); adaptive behavior (eye-hand coordination, recovery, and manipulation of objects, alertness); and personal-social behaviors (reactions to people, play behavior, independence). The first completed scale, published initially in 1925, consisted of 144 items divided among these four general fields. Some items appeared in more than one subdomain, and motor items predominated at the early ages, whereas language and personal-social items predominated later. Age placements were determined by the percentage of subjects who passed each item. The first version of the Gesell Developmental Schedules was based on a small, normative sample (107 infants from white middle-class families living near New Haven, CT). Subsequent revisions of the 1925 Gesell Developmental Schedules were directed toward determining ever-finer age gradations of infant and toddler responses (e.g., defining by weeks and days, rather than by months, the appearance of the response to the sound of a bell). Table 40.1 gives examples of items during the first 3 months of development.

Area of Development	Coverage (months)		
	1st	2nd	3rd
Gross motor	Does not hold head up	Holds head occasionally erect, mostly steady	Supports self on hands in prone position
Fine motor	No release grip	Has nonreflexive grasping and prehending	Actively holds objects
Problem solving	Reaches toward	Steady reach beyond the midline	Steady reach beyond 180 degrees
Communication and language	Wakes and reacts to sounds	Reacts to single words	Cries
Social	Attends to faces and voices	Has directed regard of faces, clear social smiles	Initiates being held
Motoric	Quiet when held	Clearly attends to voices	Shows anticipatory excitement

Table 40.1. Sample of Items from Gesell's Developmental Schedules During the First 3 Months

#### NANCY BAYLEY'S LONGITUDINAL STUDY

Meanwhile, on the West Coast of the United States, Dr. Nancy Bayley of the then-named Berkley Institute of Child Welfare, who borrowed many of Gesell's assessment techniques along with concepts more familiar to the infant IQ model, worked on the creation of two scales of infant development that would later revolutionize the field. The *California First-Year Mental Scale* (Bayley, 1933a, 1933b) and the *California Infant Scale of Motor Development* (Bayley, 1935, 1936) were developed by Bayley for use in her own longitudinal study of development in 61 typical infants, with follow-up measures extending into their adulthood.

### 1940s and 1950s: Reconceptualizing Infant Development

In the 1940s and 1950s, the science of infant developmental assessment gained greater international attention, through the publishing of the *Griffiths Mental Development Scale* in London (Griffiths, 1954). At that time, infant assessment techniques were used primarily for diagnosis and categorization purposes, such as for preadoptive screening, testing for admission to special schools, or evaluating physical handicaps (Stott and Ball, 1965). Few scientists, however, used the techniques for longitudinal studies or empirical research. The assessments for diagnosis and categorization were predicated on clinicians' continuing adherence to the belief that intelligence is fixed from infancy. This belief in a fixed IQ still was so well rooted that evidence to the contrary, such as findings documenting an increase in IQ in high-risk infants after nursery school attendance, was dismissed and was attributed to poor standardization of the scales (Brooks and Weinraub, 1976). Indeed, it was largely on the basis of Bayley's longitudinal study (Bayley, 1949) that the concept of an immutable IQ that was fixed at birth was discredited.

The inability of infant developmental scores to predict school-age IQ adequately sparked considerable debate that shaped significantly our understanding of human intelligence and models of assessment. Some scientists claimed that the poor predictive ability of infant developmental tests reflected a fundamental discontinuity between infant and later abilities; that is, intelligence in infants is qualitatively different from intelligence in older children and adults. Others claimed that cognitive development is not fundamentally different for infants, but infant development tests tend to assess different aspects of intelligence than do school-age IQ tests. In the context of this debate, American developmentalists first began to consider seriously the stage theory of Jean Piaget, which clearly favored the view of qualitative, rather than simply quantitative, differences in the abilities of children of various ages.

### 1960s and 1970s: Breakthroughs in Infant Test Development

The 1960s and 1970s brought a new wave of infant development tests, more rigorously standardized on larger numbers of infants, with careful testing of interobserver agreement and test-retest reliability. Developments in the work of Bayley best typify this previously unseen level of commitment to psychometric rigor in infant tests. The *Bayley Scales of Infant Development* (BSID) consolidated Bayley's original two scales into one assessment instrument with norms based on a nationally representative sample of infants ([Bayley, 1969](#)). The BSID set a new and enduring standard of sophistication for the development and standardization of infant developmental assessment tools. Thoughtful research led to far more caution about the predictive validity of early assessment and to important conceptual revisions, such as a growing recognition that IQ is not a unitary construct. In addition, investigators added more global appraisals to their scales ([Escalona and Moriarity, 1961](#)) and began to understand that some items, particularly verbal items, had higher predictive validity than the overall scales ([Cameron et al., 1967](#)). These and other conceptual shifts led to yet another group of infant tests designed to measure specific types of behavior, such as social competency or language development. Moreover, infant developmental tests reflecting Piaget's stage theory began to emerge. Concomitant interest in newborn capacities and the rapidly emerging field of newborn sensory perception also led to the development of certain scales to measure competency in newborns.

### 1980s and 1990s: New Directions in Infant Developmental Assessment

The 1980s and 1990s were dominated by increasing psychometric improvements, by a renewed interest in diagnostic functionality, again primarily driven by the needs of public education, and by the application of information-processing theory to the study of infant development. In 1986, special education laws were revised to mandate special education services for children 3 to 5 years old (P.L. 99-457, Part B) and to allow states to offer early intervention services to children from birth to 3 years old (P.L. 99-457, Part H). Infants and toddlers can qualify for these intervention services based on their having documented developmental delays in motor (gross or fine motor), communication (receptive or expressive), cognition, social, or self-help skills, or by having an environmental or biological condition that is likely to lead to a significant developmental delay in one or more of the foregoing areas. Assessments must draw from information from multiple sources (e.g., both direct assessment and caregiver report), and an individualized intervention plan, based on the unique needs and strengths of the child and family, must be developed. Furthermore, states must create a system for identifying eligible children using various methods of recruitment and developmental screening. These mandates created the need for an arsenal of assessment tools that encompass all the qualifying areas of delay and use information from a variety of sources. Additionally, instruments were developed that were designed to facilitate better treatment planning by focusing on skills that can be targeted easily for intervention (criterion-referenced tests). Moreover, a plethora of brief developmental screening instruments was developed for use by professionals with relatively little training in formal assessment or statistics. In addition to tests for entry into early intervention programs, test developers and state agencies have recently begun to turn attention to the creation of developmental tests specifically for the purposes of measuring developmental progress and holding early intervention programs accountable for improvements in child outcomes ([Ripple and Gilliam, 2000](#)).

The advent of information-processing theories of human intelligence led to more elaborate models of infant cognition. Further, the focus of infant developmental tests on gross and fine motor skills and emerging perceptual motor coordination may contribute to their weak predictive validity for later IQ, because these skills may have relatively little to do with information-processing capacities ([Bornstein, 1989](#)). Several studies have shown measures of infant information processing (e.g., attention, recognition, stimulus habituation, and memory) to be significantly related to later IQ scores ([Bornstein and Sigman, 1986](#); [McCall and Carriger, 1993](#)). However, these measures can be difficult to administer under usual clinical assessment conditions, and the model has not been translated to clinical assessment tools. Regardless, measures of information processing may play an important role in the next generations of developmental assessment instruments.

## OBJECTIVES OF INFANT ASSESSMENTS

Why perform infant assessments? It is perhaps easier to state definitively what infant assessments cannot provide. They do not provide a measure of fixed or immutable intelligence, a trajectory for future development, or a window on future adjustment, nor can they typically parcel out the various potential causal factors. Results are descriptive, with only limited application for etiologic understanding or detailed prognosticating. Questions such as "How much of this infant's delay comes from environment, versus how much from prematurity?" or "What will be the eventual extent of this child's developmental disability?" are not definitively answerable by a developmental assessment. Developmental assessments, however complete and skillfully done, cannot provide sure predictions of long-term outcome or parcel out the complex contributions of endowment, experience, and maturational forces.

Despite the foregoing caveats, assessment of infant development can be highly useful and in many cases essential to proper clinical treatment. Skillfully done, these assessments can help to create a picture of the child's current developmental level and environmental context that can be invaluable to sound clinical decision making and treatment planning. Essentially, developmental assessment results help to provide a lens through which we may be better able to perceive the world from the child's perspective. Indeed, Bagnato and Neisworth point out that the word *assidere* (the Greek origin of *assessment*) literally means "to sit beside" and, hence, to get to know someone ([Bagnato and Neisworth, 1999](#)).

From a problem-oriented stance, there are several general problem areas and more specific concomitant disorders and conditions for which infant assessments may be indicated. These are summarized in [Table 40.2](#) and include the following:

Regulatory disturbances
Sleep disturbances (e.g., frequent waking)
Excessive crying or irritability
Eating difficulties (e.g., slowly eating or food refusal)
Low frustration tolerance
Self-stimulatory movements (e.g., rocking, head banging, excessive finger sucking)
Social and environmental disturbances
Failure to discriminate caregivers
Atypical or no expressions of affect or interest in social interaction
Excessive negativism
Atypical, hostile, or multiple attachments or caregivers
Separation or prolonged separations from caregivers
Psychophysiological disturbances
Recurrent vomiting or chronic diarrhea
Recurrent wheezing
Recurrent skin rashes
Developmental delays
Specific delays (e.g., gross motor, language)
Language delays or delayed development
Genetic and metabolic disorders with known neurodevelopmental sequelae
Down's syndrome
Fragile X syndrome
Prader-Willi syndrome
Exposure to toxins
Fetal alcohol syndrome
Lead poisoning
Central nervous system damage
Traumatic brain injuries
Intracranial hemorrhages
Prematurity and serious stresses early in life

**Table 40.2. Some Clinical and Diagnostic Indications for an Infant Developmental Assessment**

1. Regulatory disturbances: disturbances in self-regulatory capacities, such as sleep or eating disturbances, including food refusal, night terrors, repeated waking, or problems in impulse control such as excessively aggressive behavior. Low frustration tolerance is another mark of regulatory difficulties. Self-stimulatory behaviors, such as rocking or head banging, may indicate a variety of social or regulatory difficulties, may be a manifestation of environmental stress, or may signify more profound difficulties in relatedness, as in pervasive developmental disorder.
2. Social and environmental disturbances: disturbances in social development or the caregiving environment, including serious and profound problems in differentiating mother or caregiver, such as may be seen in pervasive developmental disorder or infantile autism, and disturbances in predominant mood. Infants who are predominantly withdrawn and apathetic are at great risk of developmental difficulties. In this category are also included environmental conditions such as repeated or prolonged separations or neglect, abuse, and exposure to violence, all of which place infants at risk of social and affective disturbances ([Kaufman and Henrich, 2000](#)).
3. Psychophysiological disturbances. These include, among others, failure to thrive, recurrent vomiting, wheezing, or chronic skin rashes. The younger the child, the more likely the response to an environmental stress will be global, involving several organ systems (e.g., failure to grow). Clearly, any one of these problems may have physical causes, but clinicians should be alert to the close connection between physiologic and psychological adjustment in young children.
4. Developmental delays: delays in specific areas of development, including motor development and activity, language and communication, awareness of others and degree of relatedness to others (often seen together with language delay), or delays in more than one of these areas. Such delays may be more common among infants with complicated perinatal courses such as those born severely premature or after parental substance abuse and prenatal exposure to alcohol, cocaine, or other drugs. Thus, infants with such histories are more often referred for assessments early, to plan for appropriate interventions.
5. Genetic and metabolic disorders with known neurodevelopmental sequelae. Various genetic and metabolic disorders have known neurodevelopmental sequelae. These include, but are not limited to, Down's syndrome, fragile X syndrome, Prader-Willi syndrome, certain sex chromosome anomalies (e.g., Klinefelter's syndrome), and poorly managed phenylketonuria ([Madrid and Marachi, 1999](#)). Although certain developmental and behavioral sequelae are associated with these conditions, the extent can often vary considerably, and developmental assessment can be useful to document its course and to target psychosocial interventions better.



6. Exposure to toxins. Exposure to environmental toxins, such as the case with fetal alcohol syndrome and lead poisoning, has been associated with both developmental delays and behavioral dysregulation. Although useful in treatment planning, assessments are not able to determine the proportion of the developmental presentation attributable to these potential causal factors.
7. Central nervous system damage. Central nervous system damage (e.g., traumatic brain injuries and intraventricular hemorrhages) can, of course, lead to developmental sequelae, and follow-up with a developmentalist can be invaluable in understanding the level of functional impairment and in tracking recovery.
8. Prematurity and early illnesses. Prematurity and other serious medical conditions that may result in hospitalization or other restriction of appropriate stimulation early in a child's life may lead to altered parent-child interaction and may adversely affect development ( [Minde, 2000](#)).

Extant research suggests that the specific disturbances and conditions included in the foregoing list are highly interrelated and are mediated by the social context of the child ( [Zeanah et al., 1997b](#)). For example, failure to thrive may also indicate social (e.g., the family and caregiver-child dyadic relationships) or environmental disturbances, or general developmental delay may occur in children with repeated separations or in a withdrawn, apathetic child. A particular developmental profile, such as delayed language skills but age-appropriate motor and problem-solving skills, may occur with different presenting difficulties, and thus, it is not possible to specify a characteristic diagnostic developmental pattern for failure to thrive, sleep disturbances, or the other problems listed here.

In addition to the foregoing caveats, three general points are important to remember. First, language and communication skills are particularly vulnerable to biological and environmental stresses. Moreover, problems in communication also affect personal-social development. For most of the problems listed under social and environmental disturbances, the infant will likely show minimally delayed language and personal-social development. In addition, any adaptive or motor items that require interaction with the examiner are affected by disturbances in relatedness, and the child's skills in these areas appear scattered not necessarily because of motor impairment but rather because of the necessity for social interaction for administering the item. Second, it is possible for an infant presenting with some of the difficulties outlined in [Table 40.2](#) to have an age-appropriate developmental profile in terms of what things the child can and cannot do. In this case, the qualitative observations of how the infant approaches the setting are crucial. The qualitative aspects of the child's interactions with the caregiver and the evaluator, motivations, problem-solving processes, and mood state are infinitely more important than a simple inventory of the infant's skills. Third, an infant's level of developmental functioning may vary considerably between domains. Infants with psychophysiological disturbances often show such a scattered developmental profile. Qualitative observations are again important with this kind of profile, as well as repeated assessments over time, to gain a better sense of the child's developmental trajectory.

Generally, the developmental assessment provides a description of the child's functional capacities, the relationships among the various domains such as language and socialization, the child's ability to adapt, and the range of coping strategies. For the very young infant, developmental assessments describe neurodevelopmental functioning and individual regulatory capacities. For caregivers, the evaluation provides information about both the child and the potential therapeutic value of the alliance established with the clinician. For the referring clinician, the assessment may provide a more integrated view of the infant's psychological as well as physical status. Finally, infant assessments often serve the purpose of facilitating referrals to appropriate educational or rehabilitative services. In such cases, the useful question is not whether the infant is delayed or has problems, but rather what are the most appropriate services to ameliorate these problems or to compensate for these conditions. In cases such as these, the evaluating clinicians will need to be collaborators themselves with persons directing intervention and educational services for infants. (For reviews of the development and effectiveness of early intervention services for infants, see [Clarke-Stewart and Fein, 1983](#); [Guralnick, 1997](#); and [Shonkoff and Meisels, 1990](#).)

## DEVELOPMENTAL FORCES

Several important forces drive or moderate developmental processes. Indeed, clinical assessment of infants and their families represents a process of gaining a better understanding of these interacting forces. Four specific areas are discussed: (a) the interaction of innate and experiential factors, (b) maturational processes, (c) the essential role of relationships with others for healthy development, and (d) developmental stages and critical or sensitive periods. Although these areas are inextricably interrelated, some points are unique to each.

### Interaction of Innate and Experiential Factors

The interactive balance between innate and experiential factors is a well-worn, time-honored controversy in developmental science, and even now it is possible to find proponents emphasizing the singular importance of one over the other. Rarely are these issues clearly distinguishable in a clinical evaluation. At the least, infants bring sets of innate capacities that influence how they respond to the environment and how it responds to them. The clinician is always faced with considering how intrinsic and extrinsic factors have interacted to contribute to an infant's developmental difficulties or strengths. Infants are more vulnerable to developmental dysfunction, even with a supportive environment, if they have biological dysfunction, as in genetic disorders or severe prematurity. Conversely, even "well-endowed" infants are at risk of developmental dysfunction if their environment provides inadequate or inconsistent nurturing. A combination of an impoverished or dangerous environment and biological or genetic risks is a significant predictor of developmental dysfunction, and as the number of risk factors increase so increases the likelihood of poor outcomes ( [Peck et al., 1999](#); [Sameroff and Chandler, 1975](#)). Indeed, an extensive review of genetic research conducted by a special task force of the American Psychological Association concludes that genes, the environment, and the interaction of these two forces all play a large role in cognitive development ( [Neisser et al., 1996](#)). This transactional model of child development, which stresses the dynamic interplay between individual-level and contextual-level factors, is the prevailing paradigm ( [Sameroff and Fiese, 2000](#)).

### Maturational Processes

Depending on the clinician's frame of mind, *maturation*, or the progressive unfolding and differentiation of intrinsic capacities, presents either a complication or a challenge in the process of developmental assessment. Infants change rapidly, and the appearance of behaviors and responses can be highly variable despite certain expected sequences. Moreover, although very young infants begin life in a relatively undifferentiated state, within the first months, perceptual and motor systems differentiate rapidly. Implicitly, we accept Gesell's model of infant development by acknowledging that sequences of development *generally* are based on orderly maturational steps that have been well described and defined. This sequence and the knowledge of when children typically achieve certain skills can be used to establish norms, against which an individual infant's developmental skills can be contrasted. As Provence states: "Maturation . . . is a necessary construct, an invisible process represented by observable behaviors" ( [Provence, 1972](#)).

As described earlier, environment, genetic predisposition, and the interaction of both can alter maturational forces significantly. For example, we expect grasping patterns to follow an expectable, regular sequence of neurologic maturation but know that the timetable for infants' use of a particular grasp to hold a toy or to explore a box is individually variable and can be highly related to the infant's exposure. Or, although the infant may have the neurologic capacity for a responsive smile and the perceptual-motor integration to extend his or her arms toward an adult, experience in interaction with the environment is a necessary factor for such observable behaviors to emerge. In addition, it is true some variants of typical maturational processes are not necessarily associated with later problems. For example, it is typical for infants to learn to crawl on hands and knees at 8- to 9-months old and then to walk at 12-months. However, various alternative pathways of infant locomotion are fairly common and are not necessarily related to underlying problems, and researchers have long known that age of walking alone is not a good predictor of developmental outcomes ( [McGraw, 1932](#)).

It is important to draw a distinction between developmental processes that are primarily delayed and those that represent a qualitative deviation from the typical progression of skills. For example, some infants and young children present a pattern of development that approximates the typical orderly progression of developmental skills, but they are nonetheless developing along that track at a pace significantly behind their same-age peers. Other infants, however, may evidence patterns of development that are substantially different from the normal progression or may show signs of qualitative differences in neuromuscular development (e.g., localized or diffuse hypotonia, abnormal reflex patterns). Significantly deviant patterns of development appear to be more common in children whose overall development is very delayed relative to chronologic age expectations.

### Relationships

It is impossible to overstate the role of human relationships in development. The essential role of stable and nurturing human relationships is well established and is universally acknowledged among researchers ( [Shonkoff and Phillips, 2000](#)). However, most formal infant assessment techniques were developed to focus exclusively on the measurement of maturational forces, as if assuming that development proceeds relatively independent of environmental input. Thus, it is important to emphasize that every infant assessment must consider the other persons in that infant's life. Understanding normal, delayed, or deviant development requires some understanding of the infant's experiences with adults. The younger the child, the more central are such persons to the child's safety and total well-being. Such serious events as traumatic separation, physical abuse, witnessing of violence, deprivation, loss, and neglect often have devastating effects on a child's development

(Rogeness et al., 1986).

Adequate care and nurturing for an infant involve a balance between gratification, comfort, and support and the frustration inevitable in all developmental phases. Adequacy in caregiving, difficult as it is to define, generally includes attempts to mediate painful, tension-producing situations and to adjust the balance between comfort and frustration. The appropriate balance varies depending on the child's age. For example, the infant's frustration at not being fed immediately is different from the toddler's frustration at being unable to reach a favorite toy, and each requires a different response from the parents. In one instance, frustration may produce a painful, tense state; in another, it may lead to an adaptive solution that enhances further learning and appropriate individuation and independence.

Often clinicians are not always dealing with gross parenting deficits or failures, such as in serious abuse and neglect ( Kaufman and Henrick, 2000). For many infants and young children, crucial experiences may have adverse effects that are much harder to identify. For example, we are only beginning to understand the critical effect of maternal depression in the first month to 1 year, when the mother is psychologically and sometimes physically unavailable to her infant ( Garrison and Earls, 1986; Murray and Cooper, 1997). A growing body of research on the issue of caregiver mental illness, however, suggests that serious psychopathology in caregivers can significantly alter dyadic and familial interaction patterns, which, in turn, lead to altered developmental courses for infants. Caregivers, however articulate and enlightened, may be unaware of their own difficulties in responding to their infants or of how their mood states, worries, and frustrations affect their parental responsiveness. It is at this level that the importance of establishing a working relationship between parents and evaluator is clearest.

### Developmental Stages and Critical Periods

Historically, theories of development have conceptualized the phenomenon primarily either quantitatively or qualitatively. A quantitative conceptualization portrays development primarily as a continuous orderly accumulation of skills, dependent on the mastering of prerequisite skills. Qualitative conceptualizations, in contrast, stress the importance of various developmental stages that are qualitatively different and represent a marked shift in the manner in which the person perceives, understands, and interacts with the environment. In short, the quantitative conception represent development as a continuous process, whereas qualitative theories propose that development is a process marked by periodic discontinuities or reorganizations. The concept of developmental stages involves such theories as Freud's theory of psychosexual stages, Erikson's theory of psychosocial stages, and Piaget's theories of cognitive stages. Clinically, the concept is valuable in that it provides schemata for understanding development. Extant research provides some support for both conceptualizations, and the developmental continuity versus discontinuity debate continues.

Research suggesting the existence of certain *critical periods* in human development provides some support for conceptualizing development as occurring in qualitative different stages. The concept of critical periods for the optimal development of different functions suggests that certain capabilities are optimally mastered at certain times, and difficulties arise when this optimal period is disrupted. Although the concept of critical periods was first clearly established in animal models, it has been demonstrated in humans, especially in the areas of social competence and language acquisition. Indeed, it appears clinically true that when the critical period passes without optimal organization of a given function, mastery is fully achieved with far more difficulty, if ever. It is also a clinical truism that when a function is newly emerging, it is most vulnerable to environmental stresses. This statement is supported by the common observation that an infant may stop talking if hospitalized just as the first words appear. Similarly, for an infant, chronic environmental stressors may result in a delay of appearance of age-appropriate skills. For example, a parent's anxiety over a toddler's growing motor independence may slow the development of motor skills and the elaboration of exploration. An infant's particular stage of development may influence which issues are most salient and most vulnerable to stress. During an evaluation session, stage-specific developmental characteristics also may influence not only the child's ability to demonstrate mastery of certain developmental skills, but also how the infant approaches challenges, including challenges elicited during a developmental assessment. For example, toddlers struggling with emerging independence may react differently to an evaluator's assessment tasks than would the younger infant who is focused more establishing social reciprocity and engaging his or her surrounding environment.

Reviewing research conducted in the 1980s and 1990s, Zeanah et al. (1997b) concluded that there are four distinct stages of qualitative reorganization during the first 3 years of life (see also Greenspan, 1981). Although their perspective is primarily focused on social-emotional aspects of development and draws from research and theory, the stages correspond rather closely to those of earlier developmentalists, especially Piaget ( Piaget and Inhelder, 1966). Indeed, many of the qualitative changes in cognitive development first proposed by Piaget seem to provide the prerequisites for qualitative changes in social and emotional functioning now being proposed. The following description of different stages during infancy and toddlerhood illustrates these qualitative changes, as well as the dynamic interaction between maturational processes and social relationships and the interactive nature of development across cognitive, sensorial, social, linguistic, and motor domains.

### Four Qualitative Stages of Infancy

#### STAGE 1 INFANT (0 TO 2 MONTHS)

During the first couple of months postpartum, infants primarily work toward achieving homeostasis, or the capacity for maintaining physiologic equilibrium, in the presence of internal and external stimulation. However, they are also surprisingly active and sophisticated learners, capable of cross-modally exploring and perceiving their environment, visually tracking objects as they move through space, habituating to invariant stimuli, discriminating novelty, and even anticipating caregiver actions.

#### STAGE 2 INFANT (2 TO 7 MONTHS)

The second stage (2 to 7 months) is marked primarily by increased social reciprocity between the infant and caregivers. This qualitative change follows increased awareness of the external world (made possibly by greatly enhanced visual abilities) and improved coordination of sensory input and nonreflexive (voluntary) motor output occurring at about 1 month of age. During the second stage, the infant's responsive cooing, repertoire of increasingly differentiated emotional responses, and a proclivity toward direct imitation of others' behaviors starting at about 4 months serve to facilitate reciprocal or contingent social interactions. During the latter half of the second stage (beginning as early as about 4½ months), infants start to show an understanding of *object permanence* (i.e., the understanding that objects and people continue to exist even when they are no longer within sight or sound) and a rudimentary understanding of the principles of *cause and effect*. These two epiphanies transform the infant's perception of the world and provide the requisite abilities for all future social-cognitive development. The concept of object permanence allows the infant to create mental representations of objects and others. It is therefore a prerequisite skill for imagining and for visual differentiation between caregivers and strangers. Cause-and-effect reasoning leads to increased intentionality of actions. Both these newfound cognitive abilities make possible simple interactive games between infants and caregivers, such as peek-a-boo.

#### STAGE 3 INFANT (7 TO 18 MONTHS)

At about 7 to 9 months, another qualitative shift occurs with profound impacts on reciprocal communication and social preference or familial belonging. At this time, infants develop a sense of *intersubjectivity*, the understanding that their thoughts, feelings, gestures, and sounds can be understood by others. Also at about this age, most infants begin to demonstrate means-end reasoning leading to goal-directed behavior. They can string together several behaviors to achieve a final outcome, often the attainment of some desired object. Through intersubjectivity and means-ends reasoning, the infant is able to consider caregivers as objects that can be used to meet their needs and wants (and the stage 3 infant's now solid grasp of object permanence gives him or her a large inventory of these wants and needs!). Together, intersubjectivity and means-end reasoning lead to the beginning of *communicative gesturing* (e.g., the moment when stretching for an object just out of reach becomes pointing to that object while looking at the caregiver, to request assistance). In the context of all these qualitative changes in the way in which the infant interacts with others, social preferences are established and become increasingly salient. At about 6 to 8 months of age, *separation anxiety* is first observable with most infants, peaking at about 14 to 18 months and declining thereafter ( Kagan, 1976). Relatedly, *stranger anxiety* appears to begin at about 8 months, peaks at about 24 months, and steadily declines thereafter.

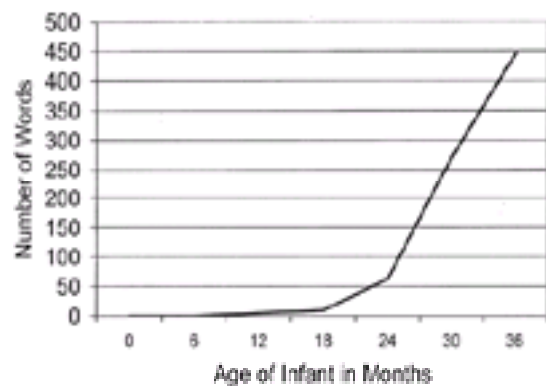
By the second half of stage 3 (starting at about 12 months), several new skills in the cognitive, language, and motor domains create profound changes. At about 12 months, infants typically first learn to walk, and this new form of independent locomotion, more so than crawling, heralds increased independence and a broadening world. Cognitively, the infant's reasoning becomes strikingly less rigid and more open to alternative solutions. For example, before about 12 months, infants who learn that an object is hidden in a particular place will persist in looking for that object at that same location even after watching someone relocate that object to a different location. This is commonly referred to as the *AB error*. However, after about 12 months, the infant's ability to hold increasingly larger amounts of information and to discard outdated information allows for a fluidity of reasoning such that the AB error diminishes or disappears. Given this increased cognitive capacity and fluidity of reasoning, *trial-and-error problem solving* begins to replace conditioned response learning. Moreover, from about 12 to 18 months, infants develop rudimentary communicative speech. By 12 months, most infants understand the meaning of several words and may have an expressive vocabulary of about 5 to 6 words. By the time they reach 18 months of age, infants typically understand the meaning of an amazing number of words, can communicate in one-word sentences, and have



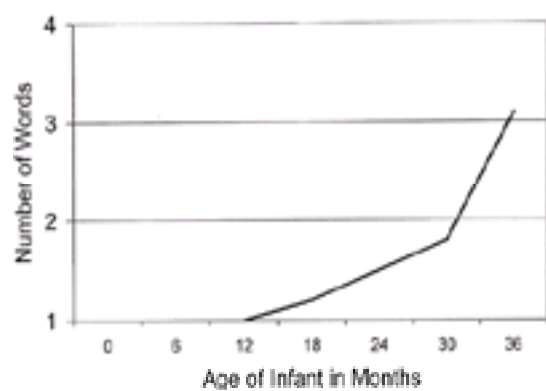
doubled their expressive vocabulary to about 10 words. Their melodic, jargoned speech patterns now closely resemble the inflections and turn-taking pauses observed in conversation.

#### STAGE 4 INFANT (18 TO 36 MONTHS)

At about 18 months, an increased ability to use *symbolic representation* transforms the infant's cognitive and social world. About 12 months earlier, object permanence marked the beginning of the infant's ability to hold in the mind mental representations of objects. The stage 4 infant is now well able to allow symbols to stand for objects, and this advance heralds greatly increased language proficiency. The use of words marks a qualitative change in the way infants think about the world and interact with others, and, likely, the reverse is true as well ( [Hollich et al., 2000](#) ). The beginning of this transformation appears to be marked by a move from direct imitation of others to *deferred imitation*, in which the behaviors of others are remembered and then are practiced later. *Symbolic play* appears as the infant uses a doll to symbolize a baby, and the infant begins to combine words and gestures to label objects in his or her world or make needs and wants known to caregivers. By 18 months, these skills are becoming solidified, and the infant's interactions with others change dramatically. Additionally, at about 18 to 24 months, *internal problem solving* begins to replace trial-and-error problem solving, as the infant's mental ability to hold and manipulate internal representations increases. From 18 to 24 months, infants' expressive vocabularies typically increase from about 10 words to about 50 to 75 words. By 30 months, the infant's expressive vocabulary has increased to nearly 300 words, and by 36 months, many infants have an expressive vocabulary of 500 to 1,000 words and typically speak in three- to four-word sentences ( [Ulrey, 1982](#) ) ( [Fig. 40.1](#) and [Fig. 40.2](#) ).



**Figure 40.1.** Typical rate of increase in expressive vocabulary of infants.



**Figure 40.2.** Typical rate of increase in average number of words per sentence in infants.

### SOURCES OF INFORMATION IN AN INFANT ASSESSMENT

We next discuss general sources of information and three techniques that are central for the clinician doing infant assessments: interviewing skills, observation of children and of caregiver-child interactions (apart from formal structured testing), and synthesis of the information gathered during the evaluation. [Figure 40.3](#) provides a summary of some of the information obtained during an infant assessment. Although interviewing, observation, and synthesis are skills of medical diagnosis in general, there are unique aspects to each in the process of assessing infants.

Caregiver Interview	Observation or Formal Evaluation of Child
<ul style="list-style-type: none"> <li>Family history</li> <li>Genetic influences</li> <li>History of pregnancy, delivery, perinatal period</li> <li>Developmental history</li> <li>Developmental milestones, previous assessments</li> <li>Medical history</li> <li>Children &amp; extended family arrangements</li> <li>Child's psychological role in family</li> <li>Caregiver perceptions &amp; expectations of child</li> <li>Stability of home/family environment</li> <li>Family social support systems</li> <li>Family functioning, stress &amp; coping (abused &amp; drug abuse, domestic violence, etc.)</li> </ul>	<ul style="list-style-type: none"> <li>Physical health, appearance &amp; growth parameters</li> <li>Sensory development (vision, hearing, touch, etc.)</li> <li>Gross motor development</li> <li>Fine motor development</li> <li>Communication development <ul style="list-style-type: none"> <li>Receptive communication</li> <li>Expressive communication</li> <li>Speech clarity &amp; fluency</li> </ul> </li> <li>Cognitive problem-solving</li> <li>Developing sense of self</li> <li>Social relationships &amp; interest in environment</li> <li>Capacity for affect regulation &amp; coping skills</li> <li>Emerging sensory motivation</li> <li>Capacity for symbolic representation &amp; play</li> </ul>
Observation of Caregiver & Infant	
<ul style="list-style-type: none"> <li>Infant's use of caregiver for support &amp; reassurance</li> <li>Caregiver's attentiveness and responsiveness to child's affective state</li> <li>Qualitative &amp; range of positive interactions</li> <li>Quality of transitions between caregiver and infant</li> <li>Caregiver affective response to child's efforts during assessment</li> </ul>	

**Figure 40.3.** Some of the information obtained during an infant assessment.

### Interviewing

It is axiomatic that skillful interviewing is central to a complete developmental assessment because many of the data about infants' daily functioning and their relationships with others come from interviews with the caregivers. Skillful interviewing techniques include the following: letting caregivers begin their story wherever they choose; using directed, information-gathering questions in such a way as to clarify but not disrupt the parents' account; and listening for affect as much as for content. Nearly every step of the assessment process requires an alliance between clinician and caregiver, because infants usually perform better when they are in the company of familiar adults, and the initial interview between clinician and family is crucial in setting the tone for such an alliance. Moreover, establishing an alliance is central to evaluating infants' interactions with the adults in their world. Indeed, infant assessments are quite compromised when there are no familiar adults available to meet with the clinician and to be with the infant. Parenthetically, it is often in cases involving the most severe environmental disturbance that clinicians do not have access to caregivers who are able to describe the infant's history.

### ADDRESSING CAREGIVER FEARS REGARDING THE ASSESSMENT

When parents, foster parents, or other caregivers are available, skillful interviewing is also critical in helping parents to follow through with the assessment process. Coming for a developmental evaluation or participating in one while their infant is hospitalized is enormously stressful and is often frightening for caregivers. Clinicians working with infants and their families need to understand that, regardless of what caregivers have been told about the assessment, caregivers' fears and fantasies about the process are as potent as the facts of the presenting problem. Not uncommonly, caregivers have begun to see the infant as damaged or defective

in some way and are afraid and guilty about the effect of their own behavior on the infant. Their fears of what the infant's problems signify may be expressed in many ways. They may anticipate that their infant has a serious developmental disability, such as autism or mental retardation, or that the infant will have serious emotional difficulties in school, or that they themselves will be, or already are, inadequate caregivers. It is always a vulnerable time for caregivers, and clinicians should keep in mind that what seem inconsequential moments and statements to them may be memorable and powerful for many caregivers. Furthermore, the stress of coming for an assessment affects the caregivers' abilities to report about the infant's development. Often, the "facts" start to change as the alliance between caregivers and clinician develops.

### ACTIVE LISTENING

When first interviewed, caregivers may be reluctant to be candid or may not themselves be fully aware of their own perceptions and beliefs about the infant. Open-ended questions, allowing caregivers to begin their story wherever they feel most comfortable, and conveying a nonjudgmental attitude are crucial beginning points in establishing the working alliance. In addition, at the risk of stating the obvious, such "interviews" involve considerably more listening than active questioning. Indeed, the type of *active listening* involved in this type of interview requires the clinician to do much more than just passively collect and record requested information. Rather, it involves forming numerous connections among "factual" information, observational information (the reactions of the caregivers and their affective responses), and an appreciation the context of the relationship between the caregiver and the clinician.

### CONTENT OF THE INTERVIEW

Practically, the purpose of interviews with parents, foster parents, or other caregivers is to gather information about the infant. Highlighted earlier is the affective atmosphere in which such data gathering best occurs. The important areas to cover in terms of the infant's development are as follows: the infant's medical history and major developmental milestones; the history of the mother's pregnancy, delivery, and immediate perinatal period; the number, ages, and health of family members; and how the infant fits in the family's daily life (Cox, 1999). The meaning of the individual child for all caregivers is an important window on the infant's place in the family. Many infants and toddlers attend child care or early intervention programs, and the perceptions of those teachers, as well as their relationship with the infant's primary caregivers at home, are also important.

More specifically, the interviewer should try to obtain a picture of the caregivers' perceptions of the infant's level of functioning in several areas. These include motor development and activity level, speech and communication, problem solving and play, self-regulation (ease of comforting, need for routines), relationships with others, and level of social responsiveness. Questions about whether the pregnancy was planned or came at a good time for the family and what expectations the parents had for the infant provide important information about perceptions, disappointments, and stresses. Similarly, asking the caregivers of whom the infant reminds them or what traits in their infant they like best and least may be useful avenues for learning about how the parents view both the infant's problem and his or her place within their family.

### TECHNIQUES OF ORGANIZING INFORMATION

Provence suggests that a productive method of gathering developmental and family data is to ask the caregivers to describe a day in the life of their child (Provence, 1977). Provence outlines how this question can be the framework for learning about daily activities, how the infant and caregivers interact throughout the day, and about interactions around mealtime, bedtime, or times of distress. When all major caregivers are present for the interview, this question provides a time for each of them to present descriptions of his or her time with, and perceptions of, the child. Additionally, clinicians may use structured interviews, such as the *Vineland Adaptive Behavior Scales* (Sparrow et al., 1984), currently under revision, to collect both quantitative developmental data and to provide an opportunity to open new areas of clinical discussion. Moreover, the *Infant-Toddler Developmental Assessment* (Provence et al., 1995), appropriate for infants from birth to 36 months, is particularly useful in providing a schema for organizing important information from caregiver interviews, medical and developmental records, and behavioral observations.

Implicit in this overview of interviewing caregivers as a part of the clinical assessment is the assumption that such assessments require several sessions. Minimally, one meeting with caregivers, two or more with the child and caregivers together, and another to present the results to the caregivers are necessary. The sessions with the infant also provide an opportunity to gather more interview information, because other questions will occur in the context of the child's behavior and performance. For example, asking whether the child's response to a particular situation within the evaluation context is usual for him or her may open up another area of information from the caregivers. As is likely clear from these suggestions, in our view, infant assessment is a process of constantly gathering information, revising impressions, and testing hypotheses, and that requires time.

Conversely, clinicians asked to evaluate infants and young children do not always have the ideal situation outlined earlier. At times, consultants, caregivers, or both may insist that the evaluation be done in one session, or the clinician may have limited access to the child, as with many evaluations done in a hospital setting. It is important at these times for the clinician to be clear about the limitations of the evaluation findings. Another situation that occurs increasingly commonly and does not fit the ideal model just outlined is when no parents, family members, or other caregivers are available. Situations of severe abuse, abandonment, multiple placements, or seriously ill parents are examples of times when the clinician will not have available certain critical sources of information. In these instances, certain hypotheses suggested by the child's presentation and status may be left unconfirmed. As in situations when the time for the evaluation is brief, it is most important for clinicians to acknowledge which aspects of their diagnostic formulation are relatively certain, which are not, and what information would likely be clarifying, were it available.

### Observing

*Observation* is the fundamental skill needed for measuring infants' development. After all, most diagnostic evaluations are based on observation of physical signs or behavioral responses. However, what distinguishes the observational skill necessary for developmental assessment is that it occurs on many levels simultaneously and is perhaps the area in which the developmentalist's dual role as both generalist and specialist is most evident. Moreover, the observational skills inherent in assessments of infants require a blend of free-floating attention bounded by a structure. In other words, although the clinician must be comfortable enough in the setting to attend to whatever occurs, he or she must also have a mental framework by which to organize the observations collected during the session. Such a framework entails at least four broad areas: (a) predominant affective tone of the participants; (b) involvement in the situation (curiosity and interest); (c) use of others (child's use of the caregivers or examiner); and (d) reactions to transitions (initial meetings, end of sessions, changes in amount of structure).

### WHAT TO OBSERVE

There are several opportunities for careful observation during the course of a clinical assessment. Clinical observation begins from the very first contact with the caregivers and infant, including the caregiver interview addressed earlier. Many important observations of the infant and infant-caregiver and familial interactions can be obtained during the course of formal developmental assessment. Benham provides an elaborated framework for structuring observations of infants and toddlers that may be useful during clinical assessment (Benham, 2000). In many cases, however, the formal developmental evaluation alone may not provide sufficient opportunity to observe all the important behaviors of the infant and caregiver. Infants may behave differently with different caregivers and in varying contexts. For this reason, both naturalistic and structured analog procedures can often be used to gather additional observational data that can be useful for both clinical and research purposes (Zeanah et al., 1997a). Play-based assessment allows the clinician the opportunity to observe the infant and caregivers in a less structured format than provided by the formal developmental assessment. Moreover, play observations can be very useful in gaining additional information about the infant's cognitive, symbolic and linguistic, social, and motor development, as well as in assessing internal emotional states and conflicts and the infant's internal dynamic representations of the world (Close, 1999).

### LEVELS OF OBSERVATION DURING ASSESSMENT

Within the four broad areas of observation described earlier, the clinician makes observations continuously on at least three levels. Perhaps the most obvious level is the observation of how the child responds to the structured assessment items administered during formal testing. As already stated, observations during formal testing should not be confined solely to whether the child passes or fails a given item, but to how the child approaches the task. The second level of observation during an infant assessment is how the child reacts to the situation apart from the formal testing structure. Does the child approach toys, initiate interactions, refer to the examiner or his caregivers? How does the child react in the beginning of the evaluation versus later when the situation and the examiner are more familiar?

The third observational level is a specific focus on the interactions between caregivers and infant. The clinician makes these observations throughout the evaluation process and revises his or her hypotheses as both caregivers and infant become familiar with the process. How to interpret the behaviors one observes between caregivers and child in terms of their ongoing relationship is learned partially by experience and requires time to gather many observational points. However, several



general areas may provide important descriptive clues. Does the child refer to the caregivers for both help and reassurance? Similarly, does the child show his successes to the caregivers, and do the caregivers respond? Another important observation for toddlers is whether the child leaves his caregivers' immediate company to work with items or explore. For infants, how caregivers hold, feed, and comfort their baby may be windows in the emerging dyadic and familial relationships. A caregiver participates with his or her child during such sessions in varying ways, and the clinician continuously is assessing qualitative aspects of that participation—how intrusively involved, withdrawn, or comfortably facilitative the caregivers are. One of the most important lessons when learning how to observe interactions between infants and caregivers is that clinical observations, even when based in a naturalistic setting, may or may not be an adequate reflection of what is typical for that particular family. Adults may appear very different as individuals in their own right, compared with when they are interacting with their children. Moreover, the assessment context in which one's child (and by implication, one's self as a person and as a caregiver) is observed by another is anxiety provoking in varying degrees for all parents, and it may profoundly alter their parenting style.

Formal quantitative developmental evaluation is only part of the overall clinical assessment of infants. Indeed, in some ways, formal testing is the least critical of the clinical assessment tasks and serves more as a frame for clinicians to guide their observations ( Gilliam, 1999). It is not sufficient in assessing infants simply to say that the infant is either developmentally delayed or age adequate. For very young children, assessing development involves elaborating a more complex view of the child and his or her environment, and at this age, every developmental evaluation must include descriptions of behavior and the qualitative aspects of the child's behavior in the structured setting. For example, *when* the infant first turned to a voice or successfully retrieved a toy in a manner appropriate for age may be less important than *how* he or she responded to these tasks (e.g., with excitement, positive affect, and energy versus slowly, deliberately, and with little affective response). Such qualitative observations are often the best descriptors of those capacities for which we have few standardized assessment techniques but that are absolutely fundamental for fueling the development of motor, language, and problem-solving skills. Through observing how infants do what they do, the clinician gains information about how infants cope with frustration and how they engage the adult world, as well as about their emotional expressiveness, their capacity for persistence and sustained attention, and the level of investment and psychological energy given to their activities.

## Synthesis

The process of synthesizing all the data gathered from the different sources during an assessment is a technique and skill unto itself. Moreover, how this synthesis, with its attendant recommendations, is conveyed to caregivers and other professionals is another essential step in the assessment process, and the assessment is not complete until the therapeutic alliance among all stakeholders is brought to fruition in a collaborative formulation. Infant assessments often involve referring pediatricians and other clinicians, all of whom need to be included individually in the clinician's data-gathering interviews and in the final synthesis.

The synthesis of information from an infant assessment differs from the synthesis involved in other medical diagnostic processes in that very few specific diagnostic categories encapsulate all the findings of an infant assessment. The synthesis involved in an infant assessment requires bringing together all the data gathered from interviews, observations, and testing into a qualitative description of that infant's capacities in different functional areas (motor, problem-solving, language and communication, and social) and of the infant's current strengths and weaknesses. It also involves integrating the assessment information in the context of the infant's individual environment. For example, an infant who has experienced multiple foster placements may be socially delayed, but such a finding may assume a different significance for an infant who has had a stable home environment.

Synthesizing the large amount of data obtained from a comprehensive clinical infant assessment can be quite daunting. As stated in the introduction, in performing the infant assessment the clinician must be both generalist and specialist. The clinician must draw on and synthesize knowledge from child psychiatry, pediatrics, neurology, developmental psychology, speech and language therapy, physical and occupational therapy, and often genetics and endocrinology ( Mayes and Gilliam, 1999). Increasingly, clinicians evaluating young children also need know about early childhood education programs and early intervention, as well as laws regarding child abuse, neglect, and domestic violence. Knowledge from these diverse fields allows a clinician to place the results of a developmental assessment in a meaningful context for the individual child and leads to a better conceptualization of treatment options. For example, understanding the physiologic effects of prolonged malnutrition and episodic starvation in infancy ( Dickerson, 1981; Shonkoff and Marshall, 1990) helps the clinician to evaluate the relatively greater gross motor delays of a child with failure to thrive who has no other neurologic signs. Similarly, understanding the effects of a parent's affective disorder on a child's responsiveness to the external world ( Seifer and Dickstein, 2000) adds another dimension to understanding the infant's muted or absent social interactiveness, babbling, and smiling.

Finally, it is often during the synthesis process that the therapeutic effect for caregivers participating in the assessment is most evident. At the very least, caregivers often change their perceptions of their infant's capacities. They may see strengths in their infant they had not previously recognized, or they become deeply and painfully aware of weaknesses and vulnerabilities that they may or may not have feared before the assessment. Any of these changes in perceptions may affect the caregivers' view of themselves and of their role as caregivers. Moreover, infants often change during the assessment process, as their caregivers become more involved in the alliance with the clinician, and they experience, at least temporarily, another adult's concern and interest in their family. Emphasizing the potentially therapeutic value of an assessment underscores that the synthesis process is not simply wrapping up the assessment and conveying information, but it is also a time to explore with the caregivers the meaning of the process for them and their infant.

## FORMAL DEVELOPMENTAL ASSESSMENT TOOLS

Many different formal developmental assessment tools for infants and toddlers exist. In this section, we discuss the types of quantitative data obtained from these tests and offer basic considerations for choosing an appropriate evaluation tool. Later, we describe some of the most commonly used developmental instruments.

### Types of Quantitative Data

Besides the vast amount of qualitative data obtained by careful observation during testing, most developmental assessment procedures provide several methods of quantifying results. As described later in this chapter, most developmental tests have been standardized and normed on a sample of children selected to represent the performance of typically developing children. It is by comparing an individual infant's performance against this set of norms that most test scores are derived. These scores are then used to convey something about the infant's level of developmental skills acquisition, relative to other presumably typical infants.

Of the several different types of scores available, standard scores and age equivalents are the most commonly used. *Standard scores* are the most robust scores obtainable and represent the infant's performance in relation to other infants of about the same chronologic age. Common forms of standard scores include *Z*-scores, deviation scores, and *T*-scores, the later two being linear transformations of the former. Most tests provide some guidance on how to describe an infant's standard score by placing the scores in descriptive bands (e.g., average, below average, mildly delayed). Often, to increase the interpretability of these scores, they are converted to percentile ranks. Table 40.3 presents one common method for descriptively banding standard scores, although many other methods also are used.

Range	ZScore	Deviation Quotient	TScore	Percentile Rank
Significantly delayed	<-2	<70	<30	≤2
Mildly delayed	-2~-1	70-84	30-39	2-15
Average	-1~+1	85-114	40-59	16-83
Above average	>+1	≥115	≥60	≥84

**Table 40.3. Descriptive Ranges for Standard Scores and Their Corresponding Percentile Ranks**

*Age-equivalent scores* represent an estimate of the chronologic age (typically expressed in months) at which the typically developing infant would demonstrate the skills observed in the infant being assessed. This type of score is often appealing to many caregivers and others with little or no training in psychometric tests

because the interpretation is seemingly straightforward. However, age-equivalent scores are notoriously easy to misinterpret and may lead to erroneous conclusions. First, age-equivalent scores on infant developmental tests tend to be highly unstable, with the infant's performance on only one or two items largely affecting the age equivalent. Second, age-equivalent scores may imply too much about an infant's development, especially when skills are highly scattered. Because of the way in which age-equivalent scores are computed, the score may greatly overestimate or underestimate an infant's developmental level. As a result of the way in which tests are scored, infants who are very consistent in their ability to do tasks typical for their age often receive an age-equivalent score much higher than their chronologic age, even though they are not yet developmentally ready to do the things associated with that age equivalent. Conversely, infants who, for whatever reason, struggle with some less developmentally mature tasks likely will receive an age-equivalent score much below their chronologic age, even if they are able to do many things typical of their chronologic age or older. In cases such as these, age equivalents are best expressed as a range, if expressed at all. Third, developmental delay expressed in age equivalents does not adequately address the frequency or severity of the delays. Clearly, a 6-month delay in development is different for a 12-month-old infant versus a 30-month-old child. Standard scores, with associated percentile ranks, better expresses this delay in terms that are less dependent on the chronologic age of the infant. Finally, the type of data used to compute age equivalents is too weak statistically to support the calculation of confidence intervals that are useful in placing the score within the context of appropriate bands of error. This lack of confidence intervals further exacerbates the preceding limitations. Of course, there are times when age equivalents may be the best or only option available for expressing an infant's performance (e.g., when the extent of developmental delay is so great that standard scores cannot be easily calculated and percentile ranks below the first percentile are obtained).

### Considerations for Selecting a Formal Developmental Assessment Tool

The following discussion includes some basic guidelines for choosing an infant developmental test that best meets the clinician's specific needs. These guidelines also help to provide a framework for evaluating the usefulness of some of the various tests currently in use. Four areas of consideration are presented: purpose, sources of data, standardization, and psychometric properties.

#### Purpose

A developmental test may be useful for one or more of several different purposes: diagnosis, screening, and early intervention planning. Tests designed for these three purposes are quite different, and selecting the correct tests to match the stated purpose is essential. First, *diagnostic tests* are used to provide information necessary for either clinically or eligibility-oriented diagnoses. Clinically oriented diagnoses are used by clinicians to capture succinctly the infant's overall presentation and to provide a common nomenclature for communicating that information to other clinicians. Eligibility-oriented diagnoses, however, are used to identify which infants have levels of developmental delay significant enough to establish eligibility for publicly funded early intervention programs. Often, the infant must demonstrate a particular degree of developmental delay (e.g., performance that is two standard deviations below the mean) to "qualify" for these services. Second, *screening tests* are used when it is desirable to use a relatively brief instrument to identify infants who may be at risk of delayed development and who would benefit from further diagnostic testing. Usually, screening tests are used when many children need to be assessed, and a full diagnostic assessment for all children would be too costly or cumbersome. Third, *intervention-planning tests* are used to plan an individualized early intervention program once children have been diagnosed. These instruments help to identify important program goals and objectives or to track an infant's achievement of these goals over time to document the effects of the intervention.

#### SOURCES OF DATA

Developmental assessment instruments, like all psychometric tests, are methods of collecting and organizing data. Developmental tests for infants use one or more of at least three different types of data: direct assessment, incidental observation, and caregiver report. It is important to acknowledge the strengths and limitations of each data source. Direct assessment has the strength of being potentially very standard in its presentation, so an infant's performance may be directly compared with that of other infants, with the assumption that the material was presented in a similar standard method. The limitation of direct assessment, however, is that it represents only a small sample of the infant's developmental repertoire that will be influenced greatly by current issues regarding the infant's motivation, mood, comfort, and responsiveness to the examiner and the evaluative process. Caregiver report surveys are a useful addition to formal assessment, in particular to document behaviors that occur too infrequently to be observed in a clinical evaluation or to assess the caregivers' individual perspectives of their infant. However, caregiver reports should not be the sole measure of the infant's development, because they are highly subject to rater bias ([Meisels and Wasik, 1990](#)). Given the strengths and limitations of each of these sources of data, a comprehensive assessment should use multiple sources of data across multiple contexts.

#### STANDARDIZATION

Test standardization involves the process of developing a consistent method of administration and collecting normative data regarding children's typical performance on the test. It is by this normative data that evaluators determine a specific infant's standing relative to the normative group. This relative standing can then be expressed in terms of either standard scores or age equivalents, as described earlier. It is the responsibility of the test user, however, to decide whether a given test's standardization sample is representative of the type of infants the test user plans to assess. For instance, it clearly would be inappropriate to compare the developmental performance of an infant raised in a large city in a highly developed nation to a normative sample of infants living in a small village in a less-developed nation, unless one knew beforehand that infants from both these settings scored very similarly on the test. Most decisions regarding the representativeness of a test's standardization are not this apparent, however. Moreover, standard scores and age equivalents are norm sample dependent. In other words, scores derived from a nationally standardized test indicate an infant's standing relative to other infants throughout that nation and not necessarily to other infants of the same specific locality, gender, ethnicity, or economic status. Of course, the inverse is true as well. Some tests are normed on very specific populations of children, such as children from a specific city or state or of a particular economic status. The use of standard scores derived from these tests with infants from other localities or economic backgrounds, without specific empirical evidence to justify their generalization, is generally not recommended. Furthermore, to yield reliable results, normative data should be no more than about a decade old, to keep pace with intergenerational escalation in test performance ([Flynn, 1984](#)). Grossly outdated norms often yield inflated scores that may lead to erroneously disqualifying infants for needed services.

#### PSYCHOMETRIC PROPERTIES

The soundness of a psychometric test is judged based on its *reliability* (the ability to produce similar results under differing conditions) and *validity* (the collection of evidence that suggests that the test measures what it is supposed to measure). Several forms of reliability exist. *Test-retest reliability* is a measure of a test's stability over time. Test-retest coefficients are often lower for infant tests as compared with tests designed for older children, partly because of the rapidity of early development. *Interrater reliability* refers to the degree to which test scores are not dependent on individual differences among test examiners, but rather reflect the infant's abilities regardless of who is administering the test. Finally, *internal consistency* refers to the correlation between various test items and provides evidence of the degree to which a test measures a single unitary construct, as opposed to representing an unrelated collection of test items. As a guideline, reliability coefficients of at least 0.90 for diagnostic tests and 0.80 for screening tests are recommended in each of these three areas of reliability ([Salvia and Ysseldyke, 1991](#)). Besides their use in evaluating the psychometric soundness of a test, reliability coefficients also serve a clinically relevant purpose. Because no test can be completely reliable, scores are often presented within a given *confidence interval*, which bands the infant's obtained score within a certain range of error (e.g.,  $100 \pm 8$ ). This presentation acknowledges the error inherent in all tests, as opposed to presenting a single score, as if it were an exact measurement. *Validity*, in comparison to reliability, is even more multifaceted. There is no single or preferred way to establish a test's validity. Rather, validity represents an accumulation of evidence that together builds a case for the accuracy of that test. Specifically, infant developmental tests are expected to correlate significantly with other similar tests, to reflect developmental changes that result from expected maturation, and to be sensitive to the presence of diagnosable disorders with clear developmental manifestations.

Psychometric reliability and validity are based on a test's properties as demonstrated with groups of test takers. However, psychometric properties that are more individually relevant are also important. For example, developmental tests should have adequate floors and ceilings. In other words, there should be enough lower-level items that significant developmental delays can be detected in even the youngest infants for which the test is to be used (floor). Likewise, there should be enough upper-level items that significant developmental precocity can be detected in the oldest children for whom the test is to be used. Because standard scores are obtained by comparing an infant's performance with that of peers of similar age, the degree to which the normative data approximates the infant's age also is important. The current standard in infant developmental tests is normative bands of 1 month for infants up to 12 or 18 months old and 1 to 2 month bands for infants up to 36 months old. In other words, infant tests typically compare an infant's performance with that of other infants no more than 1 month older or younger, whereas a toddler's performance may be compared with that of other toddlers 1 to 2 months older or younger. Of course, normative bands that are narrow are preferable to those that are wider.

### REVIEW OF SELECTED DEVELOPMENTAL TESTS



A few of the more widely used developmental tests are presented in [Table 40.4](#), and some of the most common examples are further described here, organized under three basic headings: neonatal assessment tests, infant and toddler development tests, and infant and toddler screening tests.

**Table 40.4. Selected Formal Tests of Infant and Toddler Development**

### Neonatal Assessment Tests

Several specific procedures exist for assessing infants during the neonatal period (birth to 4 weeks). Previous studies showed the well-known *Apgar Screening Test* ([Apgar, 1953](#)) to be an inconsistent predictor of subsequent infant development ([Francis et al., 1987](#)), and neonatal tests were created to focus more expressly on describing the range of neurobehavioral differences in normal newborns and highlighting areas of newborn competency. By the early 1960s, newborns and young infants were seen as more active participants in their environment ([Bullowa, 1979](#)). Not only were they seen as developing intrinsic perceptual-cognitive competencies, but they were also viewed as possessing an individualized repertoire of behaviors, known as temperament, that elicited different responses from others. Although clinicians assessing infants are not often asked to evaluate neonates, the conceptual point inherent in these instruments—combining an assessment of innate capacities with attention to individual variability and responsivity—is relevant to all assessments of infants and young children.

#### BRAZELTON NEONATAL BEHAVIORAL ASSESSMENT SCALE

Although the predecessor scale to the *Graham/Rosenblith Behavioral Test for Neonates* ([Rosenblith, 1979](#)) was the first neonatal development test, the *Brazelton Neonatal Behavioral Assessment Scale* (NBAS), currently in its second edition (NBAS-2) ([Brazelton, 1984](#)), dominates the field today. The NBAS-2 is intended to assess the neonate's current level of neurobehavioral organization, capacity to respond to the stress of labor and delivery, and adjustment to the *ex utero* environment. It is designed for use with neonates of 37 to 44 weeks' gestational age who do not currently need mechanical supports or oxygen. Although it is recommended that neonates be at least 3 days old before testing, the test has been used on neonates during the first day of life. It takes about 20 to 30 minutes to administer, followed by about 15 minutes to record and score the neonate's performance. Although the NBAS was originally designed for use with full-term healthy newborns, it has been used extensively with premature and otherwise medically fragile newborns.

The NBAS-2 is used to describe the range of behavioral responses to social and nonsocial stimuli, as the neonate moves from sleeping to alert states. Items assess the neonate's neurologic intactness, behavioral organization (e.g., state regulation and autonomic reactivity), and interactivity and responsiveness with both animate and inanimate stimuli on the basis of 27 behavioral items and 20 reflexes. Relative to preceding neonatal development tests, the NBAS-2 places a greater emphasis on assessing the neonate's social competencies, as opposed to assessing only perceptual capacities or behaviors that were presumed to be related to cognitive functioning (Brooks and Weinraub, 1976). The NBAS-2 is begun optimally when the neonate is sleeping and is completed as the neonate is brought to an alert, interactive state. Each behavioral observation is scored along a seven- to nine-point continuum.

The NBAS-2 does not yield a single score, although Als and colleagues proposes an *a priori* four-factor solution ([Als et al., 1977](#)). The four-factor scores describe interactivity, motor behavior, state control, and physiologic response to stress. Lester and fellow researchers ([Lester et al., 1982](#)) summarize the behavioral items into six factors (habituation, orientation, motor, range of state, regulation of state, and autonomic regulation) and use the neurologic reflex behaviors to define a seventh factor. Each of the seven factors can be used to yield a numeric score describing the infant's performance in that area. Higher scores on the six behavioral factors indicate more mature newborn performance, whereas higher scores on the reflex factor indicate a more deviant neurologic examination result. However, methods to derive factor scores are somewhat complicated, requiring recoding items to a continuous scale to derive meaningful summative scores.

With training, interrater reliability for the 27 items has been shown to be adequate ([Lancione et al., 1980](#); [Sameroff, 1978](#)), although a considerable degree of judgment is required of the examiner for assigning a rating to the neonate's responses. Although interrater reliability for the NBAS is quite high, studies of the test-retest reliability of the NBAS suggest poor temporal stability for most items ([Sameroff, 1978](#)). The validity of the NBAS is supported by research that has *in utero* drug and alcohol exposure, maternal malnutrition, and gestational diabetes. Additionally, the NBAS has been used in studies examining the effect of maternal general anesthesia, withdrawal from methadone, and interventions aimed at teaching mothers and fathers about their newborns' capacities ([Sostek, 1978](#)). In fact, the NBAS-2 has been shown to be an effective intervention tool for increasing the maternal involvement and responsiveness of mothers of low socioeconomic status and adolescent mothers ([Worobey and Brazelton, 1990](#)). Furthermore, the NBAS has been shown to predict infant–parent attachment and subsequent infant development. Unfortunately, research has not consistently shown the NBAS to be a good predictor of infant development much beyond the first year of life ([Horowitz and Linn, 1982](#); [Vaughn et al., 1980](#)). Of the dimensions assessed by the Brazelton, state control appears the most stable and predictive ([Als et al., 1979](#)), possibly reflecting the fundamental importance of early state-regulatory capacities for other more complex functions, such as attention and social interactivity, that emerge in the first year.

### Infant and Toddler Development Tests

Of the various models of infant developmental tests, the norm-referenced multidomain model is arguably the most enduring. Based on the work of Gesell and Bayley, development is assessed in multiple distinct yet interrelated domains. The most notable current examples of these tests (described later) are the second edition of the *BSID (BSID-II)*, *Mullen Scales of Early Learning*, *Battelle Developmental Inventory*, and *Griffiths Mental Development Scales*. Additionally, criterion-referenced instruments are also popular among professionals who wish to compare an infant's development with expectations based either on a particular model of development (e.g., Piaget's model of infant cognitive development) or expected program outcomes. Although not typically useful for diagnostic or screening purposes, criterion-referenced tests may be quite useful when planning intervention programs. To accomplish this goal, criterion-referenced tests are designed to sample extensively the universe of skills that a child is expected to have mastered at various ages. The infant's performance on these tests can then be translated directly into an individualized intervention plan by targeting those skills that the infant was expected to have mastered but as of yet has not.

One of the most popular criterion-referenced tests for use with young children is the *Brigance Diagnostic Inventory of Early Development-Revised* ([Brigance, 1991](#)), which is useful for children birth to 7 years old and surveys skills in 12 different developmental domains (e.g., social and emotional, communicative, motor, preacademic skills). Other similar tests specifically for infants include the *Hawaii Early Learning Profile* ([Furuno et al., 1987](#)) and the *Early Learning Accomplishment Profile for Infants* ([Sanford, 1981](#)). The *Rossetti Infant-Toddler Language Scale* ([Rossetti, 1990](#)) is a popular scale that is designed specifically to assess an infant's verbal and nonverbal communication and level of caregiver-infant interaction. One of the most commonly used piagetian model instruments is the *Infant Psychological Developmental Scale* (described later), which assess an infant's ability to grasp object permanence, to understand means-ends and cause-effect relationships, to imitate vocalizations and gestures, and to manipulate objects in space ([Uzgiris and Hunt, 1975](#)).

#### BAYLEY SCALES OF INFANT DEVELOPMENT-II

The *BSID*, currently in its second edition (BSID-II) ([Bayley, 1993](#)), is the most widely used measure of the development of infants and toddlers in both clinical and research settings. The BSID's extensive history of test development and validation makes it the most psychometrically sophisticated infant test on the market. As previously mentioned, the BSID-II is predicated on the early tests developed by Bayley in the 1930s. During the 1960s, these tests were substantially revised and

subjected to a level of psychometric standardization and validation previously only attempted with IQ tests, resulting in the original BSID ( [Bayley, 1969](#)). The current BSID-II, a substantial revision and renaming of the original scale, is applicable to children from 1 through 42 months of age. Administration time is about 25 to 35 minutes for infants less than 15 months old and up to 60 minutes for children older than 15 months.

The three main components of the BSID-II are the Mental Development Index (MDI), the Psychomotor Development Index (PDI), and the Behavior Rating Scale (BRS). The MDI provides information about the child's language development and problem-solving skills, whereas the PDI assesses the child's gross and fine motor development. The BRS is a form that evaluators may use to rate the child's behaviors during the assessment. Items on the BRS assess attentional capacities, social engagement, affect and emotion, and the quality of the child's movement and motor control. The BSID-II provides a method for obtaining age equivalence scores for four facets of development: cognitive, language, social, and motor. Unfortunately, little empirical evidence exists to support the reliability and validity of these facet scores. Moreover, determining the correct facet age-equivalent score is often difficult because several months of development are often determined on the basis of passing only one item. Therefore, considerably more research is needed before clinicians and researchers can have confidence in these facet scores.

The BSID-II is administered in item sets, determined based on the age of the infant. This represents a substantial revision from the original BSID, which, like most tests, used a continuous series of items. This change has created some confusion among examiners of infants in terms of which item set to use for infants born prematurely ( [Ross and Lawson, 1997](#)). Indeed, the choice of which item set to use as a starting point can significantly affect the infant's final score ( [Gauthier et al., 1999](#)). For testers who use the corrected age procedure, the test developers recommend using the same item set that corresponds to the normative group used for determining that child's standard score ( [Matula et al., 1997](#)).

The BSID-II is normed on 1,700 infants (an exceedingly large normative sample by infant assessment standards) representative of 1988 U.S. Bureau of the Census data as stratified by gender, ethnicity, regionality, and parental education. Test-retest reliability for 1 to 16 days ranges from 0.83 to 0.91 for the MDI and from 0.77 to 0.79 for the PDI. Stability for the BRS varies greatly depending on the age of the child, ranging from 0.55 to 0.90. Because the BRS samples behaviors as observed during a given testing session, it may be more subject to state variation than items contributing to either the mental or the motor scales. Interrater reliability for the BSID-II is reported to be 0.96 for the MDI, 0.75 for the PDI, and 0.70 for the total BRS. Across BRS domains, interrater reliability varies, with the lowest agreement being for ratings of attention and arousal in younger children (0.57). Agreement among observers for the behavioral ratings improves for children older than 12 months. These stability coefficients approach adequacy for the MDI, but they fall somewhat short of optimal for a diagnostic test on the PDI and BRS ( [Salvia and Ysseldyke, 1991](#)). Concurrent validity of the MDI, as correlated with other measures of general cognitive ability, typically falls in the 0.70 range, whereas the PDI correlates best with the motor score of the McCarthy test at .59. In general, the BSID appears to have some ability to predict which infants will score very poorly on intelligence tests in their preschool years, but it shows limited ability to predict specific IQ scores accurately, especially in average developing infants ( [Gibbs, 1990](#); [Whately, 1987](#)).

Bayley did not intend that the MDI or PDI scores be interpreted as IQs. Correlations between BSID performance and subsequent IQ assessments are variable. In her 1933 sample of 61 infants, Bayley found no relation between the mental scale administered before 24 months and the Stanford-Binet test administered from 5 to 13 years of age ( [Bayley, 1949](#)). For the 24-month mental age scale with another sample of infants, she reported a correlation of 0.53 with the Stanford-Binet test. Others have investigated the relation between performance on the 1969 version of the BSID and subsequent IQ tests and have found modest correlations between MDI and PDI measures collected through 24 months with 30- to 36-month Stanford-Binet scores ( [McCall, 1979](#); [Ramey et al., 1973](#); [Siegel, 1979](#)). In the 1993 version, more robust correlations were found between the MDI and subscales of both the *Wechsler Preschool and Primary Scale of Intelligence* and the *McCarthy Scales of Children's Abilities* (1972), a finding suggesting that items added to the 1993 version may tap constructs more similar to measures of information processing ( [Bayley, 1993](#)). However, for the most part, performance on the BSID does not consistently predict later cognitive measures, particularly when socioeconomic status and level of functioning are controlled ( [Rubin and Balow, 1979](#)).

#### MULLEN SCALES OF EARLY LEARNING

A relatively recent addition is the *Mullen Scales of Early Learning* ( [Mullen, 1995](#)). This revision of the original Mullen scales combines earlier versions of the test designed for infants and preschoolers into one test with continuous norms from birth through 68 months. The Mullen takes about 15 to 60 minutes to administer, depending on the age of the child (15 minutes at 1 year old, 30 minutes at 3 years, and 60 minutes at 5 years). The Mullen scales assess child development in five separate domains: gross motor, visual reception (primarily visual discrimination and memory), fine motor, receptive language, and expressive language. The gross motor scale is only applicable to children birth through 33 months old and does not contribute to the overall early learning composite score.

Normative data for the Mullen scales is based on a sample of 1,849 children from across the United States, somewhat overrepresentative of children from the Northeast. Internal reliability ranges from 0.75 to 0.83 for Mullen subtests and is 0.91 for the overall developmental score. Median 1- to 2-week test-retest reliability coefficients range from 0.78 to 0.96 across subtests, and interrater reliability coefficients range from 0.94 to 0.98. The Mullen receptive and expressive language scales show acceptable correlation with similar scales from the Preschool Language Assessment Scale, 0.85 and 0.80, respectively. The gross motor scale correlates with the Bayley MDI at 0.76, and the fine motor scale correlates with the Peabody Fine Motor Scale at 0.70. These correlations for the motor scales also are acceptable. Overall, these validation studies are quite promising. Additional studies of the Mullen scales' concurrent and predictive validity, particularly with specific subpopulations, are warranted, however.

#### BATTELLE DEVELOPMENTAL INVENTORY

The *Battelle Developmental Inventory* (BDI) ( [Newborg et al., 1984](#)) is exceedingly popular, especially among professionals working in publicly funded early intervention and special education programs. It is intended to measure development in children ages birth through 7 years old. Five domains are measured by the BDI: personal-social, adaptive, motor, communication, and cognitive. Each of the five domains of the BDI is divided into subdomains that finely assess the components of each domain, and all domains contribute to a total developmental score. The assessment time required by the BDI is quite long compared with other similar tests, ranging from about 1 to 2 hours, depending on the age of the child.

The standardization sample for the BDI consists of 800 children, stratified to match the 1980 U.S. Bureau of the Census data based on geographic region, race, and gender. In a review of the technical merits of the BDI, McLinden raises several concerns (McLinden, 1981). First, although the test authors report exceptionally strong test-retest and interrater reliability for the BDI, a general lack of procedural details in the manual makes it difficult to evaluate these data adequately. Furthermore, no information regarding the BDI's internal reliability is provided. Second, the concurrent validity of the various domains is based on findings from exceptionally small studies, ranging from only 10 to 37 subjects. Furthermore, resulting correlations for the BDI's cognitive domain are much less than optimal (  $r = 0.44$ ;  $n = 13$  for the full-scale IQ from the *Wechsler Intelligence Scale for Children-Revised*;  $r = 0.50$ ;  $n = 23$  for the *Stanford-Binet Intelligence Scale*; [Newborg et al., 1984](#)).

Third, a potentially more detrimental limitation of the BDI exists in its normative data ( [Boyd, 1989](#)). For the first 2 years, normative data are presented in 6-month bands and in 12-month bands thereafter. Therefore, an infant's performance is compared with that of other infants that can be as many as 6 to 12 months older or younger. Obviously, standard scores for infants who are old for their normative group are inflated, whereas standard scores for infants who are young for their normative group underestimate their true development. Because of these normative discontinuities, children could theoretically score solidly in the average range just before their birthday, and a day or so later, with the exact same performance, score in the range suggestive of serious developmental delay or mental retardation. For this reason, age-equivalent scores may be more stable on the BDI than standard scores ( [Boyd, Welge, Sexton, and Miller, 1989](#)). This limitation greatly reduces the diagnostic utility of the BDI and may even lead to grossly distorted results when BDI standard scores are used for longitudinal research or for tracking the development of individual infants. For these reasons, diagnostic use of the BDI for infants and toddlers in general is not recommended, and BDI results administered by others should be interpreted very cautiously.

#### GRIFFITHS MENTAL DEVELOPMENT SCALES

Although the *Griffiths Mental Development Scales* are seldom used in the United States, they warrant a brief description because of their continued use in Europe and their early influence on extending infant developmental testing internationally. The Griffiths scales consists of two tests: *The Abilities of Babies* ( [Griffiths, 1954](#)), designed for infants birth to 24 months old, and *The Abilities of Young Children* ( [Griffiths, 1979](#)), for children 24 months to 8 years old. The infant scale consists of five domains, modeled closely after Gesell's early work: locomotor, personal and social, hearing and speech, eye and hand coordination, and performance. The test is normed on 571 infants from London, but it is oddly stratified to match the 1940 U.S. Bureau of the Census figures. Reliability studies for the Griffiths scales have yielded mixed results, and validity studies have indicated relatively weak predictive ability for later IQ test scores ( [Thomas, 1970](#)). Despite its outdated and questionable standardization, the Griffiths scales apparently remain popular in Europe and in Quebec.



## UZGIRIS-HUNT INFANT PSYCHOLOGICAL DEVELOPMENT SCALES

Piaget presented a view of the child that was quite different from Gesell's and that reflected, at the least, their different theoretical backgrounds ( [Yang, 1979](#)). Gesell, the essential pragmatist, presented development as the steady march forward of increasingly complex behaviors and capacities that were relatively unaffected by environmental contingencies. Piaget, the essential epistemologist, described development as a hierarchical series of qualitatively different stages that cut across observable behaviors and that are closely linked to environmental influence. For Gesell, children unfolded on a maturational timetable. For Piaget, children grew to understand the world and themselves, and development was the process of "knowing" in ever more complex ways. Maturation of motor skills and other capacities was the vehicle that would lead to such knowing, and progress depended on previous achievements in all functional areas. It was not until the 1950s that American psychologists became seriously interested in Piaget's work ( [Baldwin, 1967](#)), and this was long after the infant assessment field was well established in the tradition of Gesell and Bayley. Thus, the assessment techniques based on piagetian theories are even now far less widely used or considered, but these techniques are available and present a useful contrast to the norm-referenced multidomain tools described earlier. Of the two most widely known examples, the *Einstein Scales of Sensorimotor Intelligence* ( [Corman and Escalona, 1969](#)) and the *Uzgiris-Hunt Infant Psychological Development Scales* ( [Uzgiris and Hunt, 1975](#)), the latter is used more often.

The Uzgiris-Hunt scales, based on Piaget's theory of the sensorimotor period of development, consist of six subscales. The first subscale, visual pursuit and permanence of objects, consists of the infants' increasing awareness of objects outside their immediate perceptual field. Behaviors involving searching for a hidden object fall within this subdomain. The second subscale, development of means for achieving desired ends, covers such activities as using a tool to obtain an object. Development of imitation, the third subscale, is divided into vocal and gestural imitation and includes not only repetition of words but also different sounds for distress and pleasure. The fourth subscale, development of operational causality, includes anticipatory behaviors, such as attempting to start a mechanical toy or, in younger infants, watching one's own hand movements. Object relations in space, the fifth subscale, describes the infant's capacity to discriminate dimensionality, to track and locate objects, or to localize perceptual cues. Finally, the sixth subdomain involves the development of schemas for relating to objects (e.g., how the infant's use of toys changes) and how exploratory behaviors with objects become increasingly differentiated and complex. The position of an item in the Uzgiris-Hunt scales is determined *a priori* by the theory, not by the chronologic age at which most children complete the item, as in the Gesell-based test described earlier. There is also an ordinal assumption in the piagetian-based scales; that is, success on an item at one level presumes success at all previous levels because of the hierarchical assumptions in the theory.

The Uzgiris-Hunt scales are administered using a series of situations and materials that elicit children's responses in the various subdomains. The procedures for administering the situations are quite flexible. The six scales are not presented in a specified sequence, nor does the examiner need to cover all the scales. Specific directions for types of items, number of presentations, and types of expected responses are given, but judging success is more flexible because of the more conceptual nature of the tasks. The scales were tested and revised in three samples of infants drawn exclusively from middle-class families. Interrater agreement percentages and test-retest reliability are adequate in the three original samples, ranging from 92% to 97% and 0.70 and 0.85, for the six subscales, respectively. The subscales are also highly intercorrelated ( $r^3$  0.80), and each subscale is highly correlated with chronologic age ( $r^3$  0.88). Although Uzgiris and Hunt present mean ages for the achievement of each scale stage, they emphasize that their samples are not selected to provide normative data for different ages. Subsequently, Dunst estimated norms for age-equivalent scores based on performance in the various subdomains ( [Dunst, 1980](#)). However, these age-equivalent scores tend to average about 2 months lower than Bayley MDI age equivalents ( [Dunst et al., 1986](#); [Sexton et al., 1988](#)). In terms of concurrent and predictive validity, there appears to be little correlation with current Bayley scores but moderate correlation with later Stanford-Binet scores ( [King and Seegmiller, 1973](#); [Wachs, 1975](#)). Wachs followed a sample of infants between 12 and 24 months and correlated their performance with the Stanford-Binet test at 31 months. By 24 months, all scales except *means for obtaining an end* were significantly correlated with performance on the Stanford-Binet IQ test.

### Infant and Toddler Screening Tests

Screening tests are brief assessments of a child's current level of functioning that are used to determine which children may be developmentally at risk and may require further diagnostic assessment. To fulfill their goal, screening tests should yield scores that are predictive of scores from more comprehensive diagnostic assessments (e.g., the BSID), but they require substantially less time to administer and to score. Screening tests are intended to be used routinely when a comprehensive assessment for all children would be either too costly or unwarranted. These instruments usually are not as reliable or valid as comprehensive assessment tools, largely because of their brevity.

The goal of all screening tests is to identify correctly those children who would likely score poorly on a more comprehensive assessment and to reduce two possible sources of error: false-positive results and false-negative results. The ability of a screener to reduce false-negative rates is referred to as the test's *sensitivity*, its ability to detect children with delays or disabilities accurately. Conversely, the ability of a screener to reduce false-positive results is referred to as its *specificity*, its ability to avoid mislabeling a child as delayed or disabled, when, in fact, that child is not. Although it is desirable to reduce the percentage of both types of error, sensitivity may be more important than specificity with screening tests, because it is assumed that follow-up assessment will correct any false-positive results. [Meisels \(1989\)](#) recommends that developmental screeners possess both sensitivity and specificity levels of at least 80%. Unfortunately, too many developmental screeners do not provide these data. Several of the more common screening tests are presented in [Table 40.4](#). All screening tests presented use direct assessment or observation of the child, unless otherwise stated. Most also allow for caregiver report of information, to gain additional data about behaviors that may be difficult to elicit in the brief assessment period. Some, however, are based solely on caregiver report (e.g., the *Developmental Profile-II* and the *Developmental Observation Checklist System*). In addition, two specific screening tests are further described here.

### DENVER DEVELOPMENTAL SCREENING TEST-II

The *Denver Developmental Screening Test-II* ( [Frankenburg et al., 1990](#)) is one of the most popular developmental screening tests, especially in medical settings. The reason, at least in part, may be its brevity, because it can be administered in as little as 15 to 20 minutes. It is applicable for children birth to 6 years. Items are scored based on a combination of caregiver report, direct assessment of the child, and observation. The Denver-II test produces one overall score, placing children in one of four descriptive categories: pass, questionable, abnormal, or untestable. Because the Denver-II test was normed exclusively on children living in Colorado, caution should be used when employing this screener in other localities. The original edition of the Denver test ( [Frankenburg et al., 1975](#)) has been criticized for not being sensitive enough, missing as many as 80% of children with delays or disabilities ( [Greer et al., 1989](#)). Although the Denver-II test is a clear improvement over the original Denver test, there is evidence that it now significantly overidentifies as many as 72% of children ( [Glascoe and Byrne, 1993](#)). As a result, early intervention administrators in both Kentucky and Tennessee reportedly have requested that evaluators use a different screening instrument before referring children for developmental services, to reduce costly false-positive results ( [Johnson et al., 1992](#)).

### EARLY SCREENING PROFILES

The *Early Screening Profiles* ( [Harrison, 1990](#)) is applicable for children 2 through 6 years old. It screens for cognitive, language, speech, physical, and social disabilities or delays that may interfere with a child's learning and may warrant further diagnostic assessment. Children complete three different subtests: cognitive/language (assessing children's visual discrimination, logical reasoning, verbal concepts, basic school readiness skills), motor (assessing both fine and gross motor skills), and speech articulation. Total testing time per child is only 15 to 30 minutes, depending on the child's age. Additionally, the person who administers the test completes a 2- to 3-minute Behavior Survey documenting the child's behaviors during the assessment (e.g., activity level, attention span, cooperativeness, independence). Caregivers, or sometimes teachers, complete three different rating forms: the Self-Help/Social Profile (which provides a rating of the child's adaptive behaviors); the Home Survey (regarding the caregiver's perception of the child's home environment and caregiver-child interaction); and the Health History Survey (which provides information regarding immunizations, health problems, and prenatal health and delivery). Each of these rating forms can be completed in about 5 minutes. The Early Screening Profiles provide a wide variety of scores for all domains and subdomains, including age equivalents, standard scores, percentile ranks, and easy-to-use six-point screening categories. Screener cut points can be set at several different levels to manipulate the ratio of false-positive to false-negative results. The primary drawback of the Early Screening Profiles, however, is that it has no Spanish version. Psychometrically, the Early Screening Profiles are quite sound and comprise one of the very best screening tests on the market.

### CONCLUSION

Infant assessments are in part clinical explorations involving a fair amount of uncertainty and inference. Whereas the medical diagnostic process always involves some element of uncertainty, the assessments made in infancy require of the clinician a particular comfort with uncertainty and the unknown. Although the second half of the 20th century brought a veritable explosion of knowledge about infancy and the neonatal period, the more we learn, the more we understand how inextricably

woven are the forces of development. As emphasized throughout this chapter, infant assessment involves far more than the infant and is as much a measure of the infant's environment as it is of his or her functional status. Thus, clinicians assessing infants are always dealing more with what they cannot know than with what they can, ever exploring the limits of predictive capabilities and constantly mindful of those distinctions ( [Chapter 22](#), [Chapter 34](#), [Chapter 35](#), [Chapter 37](#), and [Chapter 41](#)).

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# 41 PSYCHIATRIC ASSESSMENT OF INFANTS, CHILDREN, AND ADOLESCENTS

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### Appendix A/Child Psychiatry Emergency Consultation Mental Status Examination Checklist

The psychiatric assessment of infants, children, and adolescents is both complex and subtle, requiring multiple sources of information, including the child, parents, teachers, pediatricians, hospital records, and previous evaluations, as well as observations of family interactions. The range of assessment methods includes the clinical interview, standardized structured and semistructured interviews, questionnaires, rating scales ( [Chapter 42](#)), standardized tests, including developmental, psychological, neurologic, educational, and linguistic tests ( [Chapter 40](#), [Chapter 43](#), [Chapter 44](#) and [Chapter 45](#), and [Chapter 51](#)), and biological studies cited in syndromes (Section VI).

## REFERRAL AND GOALS

Before proceeding with a formal psychiatric assessment of the child, it is important to consider the goals and context of the *referral*. The *goals* of the psychiatric assessment are as follows: to determine whether psychopathology is present, and, if it is, to establish a differential diagnosis; and to determine whether treatment is needed, and, if so, to develop treatment plans and recommendations ( [King et al., 1995](#)).

The first task for the evaluating clinician is to understand clearly the implicit and explicit reasons for the *referral*. Concern over difficulties that may reflect a psychiatric disorder is just one of many possible reasons for parental referral; other reasons, sometimes covert, may include marital problems, child custody conflicts, problems in school, and pending juvenile court decisions. Indeed, the impetus for the referral may frequently lie outside the family, as when the school or court are the source of the referral. When the referral source is external to the family, it is important to know the parents' attitude toward the referral, as well as obtaining the parent's (or legal custodian's) permission for the evaluation.

The parents' concerns often need to be clarified to be understood: Why are the parents coming at this particular time with these apparent referral questions? What help is truly being sought? Parents may be seeking (or fearing) a diagnosis, treatment recommendations, relief and respite, safety for the child or themselves, or answers to questions for which the presenting complaint is but a "ticket of admission" for some other concern that troubles them, such as a marital problem.

A universal set of questions for the clinician defines the basic goals of every evaluation: What are the nature and extent of the child's behavioral difficulties and what degree of functional impairment and distress do these cause? What are the potential individual, family, or environmental factors that may be causing these difficulties or potentially influence them for good or for ill? Does the child indeed have a psychiatric disorder, and, if so, what are the causes of that disorder and what treatment is needed? Sometimes the child's symptoms reflect more a problem of "fit" between parent and child or between the school and child, rather than a psychiatric disorder. At other times, the presenting problem may represent the child's reaction to an environment that is stressful and adverse for that particular child.

Certain evaluation questions, such as custody disputes, require special arrangements and are discussed elsewhere ( [Chapter 115](#)). At all times, a good diagnostic assessment should also be a therapeutic occasion. The child's and family's experience of the diagnostic interviews will, at the very least, influence how the diagnosis and recommendations are heard, understood, and regarded. More than that, the diagnostic interviews may sometimes provide an opportunity to offer a therapeutic intervention.

In short, the basic aims of the psychiatric assessment of a child are as follows:

To elucidate the sources, reasons, and causes that culminate in the referral

To assess the child's overall developmental status, including neurodevelopmental strengths and weaknesses

To characterize any difficulties in thinking, behavior, and emotions

To identify stresses or pathogenic factors in the child, the family, and the environment that may cause or exacerbate these difficulties

To establish a differential diagnosis (according to the *Diagnostic and Statistical Manual of Mental Disorders* or the *International Classification of Diseases*) and arrive at a multiaxial diagnosis

To develop and present a treatment plan

The methods employed in the psychiatric assessment of the child include the following:

Taking a comprehensive history from the parents (or caretaker), including a review of all past relevant evaluations, treatments, and outcomes

Interviewing the child to obtain the history from the child's perspective

Performing a mental status examination of the child

Recommending and reviewing any further indicated diagnostic tests and screening measures

Urgent, highly focused evaluations may be requested for emergencies, especially those involving possible dangerousness to self or others, that require immediate decisions regarding treatment, including the need for hospitalization or medication. Other specialized evaluations concern specific medication consultations, custody evaluations, or other court-ordered evaluations that pose specific questions. Although these evaluations may need to be abbreviated (e.g., in the emergency room situation) or specifically focused, they are informed by the same general principles regarding the need to collect data from multiple informants regarding the child's functioning in multiple adaptive contexts over time.

## Consent

*Parental consent* is required when the referral comes from another source. In an emergency in which the parents are not available, the consultation should go forward while the parents are being sought. Specific statutes that vary by state permit minor adolescents to seek mental health help without parental consent under specific circumstances. However, parental involvement is always preferable. Ethical issues are involved in transmitting information to others. A useful account of the foregoing outline can be found in the American Academy of Child and Adolescent Psychiatry's "Practice Parameters for the Psychiatric Assessment of Children and Adolescents" ([King et al., 1995](#)).

## HISTORY TAKING

The clinician should consider all the foregoing items and questions before proceeding with the first part of the psychiatric assessment of the child, namely, taking the history. At the same time, many of these questions may only emerge or become more sharply focused in the course of taking the history and performing the whole evaluation.

A prerequisite of an informed *history taking* is a knowledge of normal development and its variations and of common psychological reactions to various developmental stresses. A knowledge of psychiatric disorders is, of course, essential. In short, the history and evaluation should help one to understand in detail how this particular child has arrived at this point in his or her life.

The history is the beginning of an exploration, not only of the child's symptoms, behavior, thought processes, feelings, and environment in its largest sense (e.g., family, school, community, culture), but also of the child's total inner life of fantasies, wishes, fears, hopes, and anxieties, as well as the child's strengths and supports.

## PARENT INTERVIEW AND HISTORY TAKING

Parents are most often interviewed first, especially in the case of young children. Adolescents may be given the choice of being seen first or of being present during the initial interview with the parents. Each parent should also be interviewed individually. Seeing the whole family, or at least the patient and parents, together at some point in the assessment is often a useful diagnostic approach, because it permits observing family interactions first hand.

Historical and factual data, such as age, sex, race, legal status, birth history, developmental milestones, and previous illnesses, generally are best gathered by asking specific questions ([Cox et al., 1981](#)), whereas data about feelings and relationships are best elicited by an open, indirect approach. Parents, in any case, frequently recall historical dates incorrectly, although they may offer comparisons; for example, "Johnny was much slower in learning to talk than Jane." To offset these shortcomings, data should be gathered from as many objective sources as possible, including hospital records; school reports; previous tests; and multiple observers (child, mother, father).

A comprehensive and detailed pediatric psychiatric history should include a careful description of the problem as seen by the parents, including why help is being sought now and what previous attempts at obtaining help have been made. The clinician will want to be attentive to what sort of help is being sought by the parent and what explicit or implicit ideas the parents have about the nature of the child's problems.

Having explored the presenting problems, the clinician will also want to obtain the personal and developmental history of the child, including the following: the history of previous illnesses and accidents; the social history; the family history; the school history; the history of such biological functions as appetite, sleep, bladder and bowel control, and menstruation; a description of the child's relationship within the nuclear and extended family and with peers; a description of significant events, such as separations, losses, illnesses, accidents, abuse, and deaths; and an account of the previous and present medical and psychiatric status of the parents, their marriage, and the nuclear and extended family ([King, 1998](#); [King et al., 1995, 2000](#)). Parenting skills, style, and parental expectations of the child are also noted. Inquiry should also be made about the child's temperament, with attention paid to the apparent *goodness of fit* between the child's and parents' temperaments ([Chapter 14](#) and [Chapter 15](#)).

Each question should have a purpose and should be asked with tact. The clinician listens intently to the reply, not only for its content but also for its tone and affect.

The larger context of the child and family should also be considered in terms of school, community, and culture. Finally, various administrative aspects of the evaluation process may be clarified. Thus, fees, confidentiality, releases for information requests, and an outline or overview of what is entailed in an evaluation should be discussed with the parents. There should also be some discussion of what the parents will tell the child to prepare the child for the forthcoming evaluation.

## CHILD INTERVIEW: HISTORY TAKING AND MENTAL STATUS EXAMINATION

History taking and the mental status examination of the child are best performed in the context of the clinical interviews of the child. A major advantage of clinical interviewing techniques with children is that their flexibility offers the clinician the opportunity to explore clinical clues in detail. Disadvantages of clinical interviews are as follows: (a) they may not be sufficiently comprehensive by themselves to establish some diagnoses (e.g., learning disabilities, specific genetically determined forms of mental retardation, certain forms of seizure disorder); (b) the data may not be systematically acquired and hence not valid for comparisons across cases; and (c) because of differences about what realms are assessed and criteria employed, there may be poor interrater diagnostic agreement among clinicians. In addition, the acquisition of the necessary clinical skills requires considerable training.

### General Considerations and Techniques

The clinical interview of the child provides an opportunity to explore the child's own views of the presenting problem and to assess the child's mental status ([King et al., 1995](#)). The child may be a unique source of information only he or she is aware of, such as suicidal or obsessional thoughts, hallucinations, or secrets concerning abuse or antisocial activities. The history taking with the child covers many of the same elements as the developmental history taken from the parents, only with the emphasis on the child's perspective on this history. Although the child is likely to be a less reliable reporter than the parents regarding chronology, family history, and early development, the child is more likely than parents to report anxious or depressive thoughts or suicidal ideation. Although children are more prone than parents to report on their internalizing symptoms, children are less likely to report their disruptive or externalizing behaviors, such as impulsiveness, oppositionality, or aggression.

Although history taking and the mental status examination are two distinct aims of the child interview, in actual practice they are usually not separate processes, but they unfold simultaneously in the interview. Thus, while the clinician is inquiring into the various areas of the child's past and present life and functioning (e.g., how the child gets along with friends), the clinician is concurrently gathering data for the mental status examination (e.g., how warily or trustingly the child relates to the interviewer, how well the child conceptualizes or describes his or her behavior, how much the child tends to blame himself or herself or others for any difficulties). The interview techniques employed in a given child interview depend on the child's developmental, cognitive, and linguistic level.

### DIRECT QUESTIONING

Direct discussion of the presenting problem and other aspects of the child's life requires tact and attention to the child's cognitive and language level ([Lewis, 1974a](#)). It is important to use terms comprehensible to the child and to avoid overly abstract or overly concrete questions that may either lose the child or yield unproductive responses. Tact is required in probing sad or vulnerable feelings with which children may be uncomfortable or perceived transgressions about which the child may be defensive.

### PLAY, PROJECTIVE, AND INDIRECT TECHNIQUES

Young children in particular may be reluctant or unable to reflect and report in detail about their private concerns and feelings. To surmount these limitations, various play and projective techniques can be useful in eliciting these concerns. Although some researchers eschew these methods as insufficiently reliable or validated, many clinicians find them a valuable avenue of exploration as well as a useful means of facilitating the interview process by helping to place the child at ease and introducing an element of fun ([King et al., 1995](#)). Some of these specific techniques are described later. In general, however, it is useful to inquire about what books,



movies, or television shows the child recalls because this provides useful information about his or her interests, preoccupations, and degree of parental supervision. Inquiring about what the child would like to be when grown up provides data about areas of self-esteem and competency, aspirations, and values.

### GENERAL DEVELOPMENTAL CONSIDERATIONS

The child's age and developmental level have important implications for the technique and conduct of the child interview. Young children in particular may be more labile in their behavior and more prone to regress to more immature modes of behavior and relating when they are tired, anxious, or in unfamiliar surroundings. As a result, more than one interview is often desirable because a single interview may not provide an accurate picture of the child's typical or optimal level of function. It is important for the interviewer to be aware of this vulnerability to regression, in order not to be misled by an initial impression and thereby prematurely narrowing the scope of inquiry or the range of the differential diagnosis to be considered.

### Infants and Preschool Children

Clinical interviews with infants and preschoolers require special techniques in a suitably equipped room ( [Chapter 40](#)). A simple screening technique for preschool children is shown in [Table 41.1](#). The parents of infants and preschoolers should be present during the assessment to place the child at ease and hence render the child most likely to function at his or her best (unimpaired by separation or stranger anxiety). Their presence permits them to share in the observations and to learn, and it also allows the examiner to observe parent-child interactions.

Age	Observation Checklist Items	Areas to Assess
2 yr	<ul style="list-style-type: none"> <li>Walks well</li> <li>Stands up (with down stairs—one step) off of knee</li> <li>Stacks blocks</li> <li>Imitates on cardboard</li> <li>Pushes three wheels together</li> <li>Removes grocery card</li> <li>Draws in circles</li> <li>Labels to children with pictures</li> </ul>	<ul style="list-style-type: none"> <li>Stable footing of the child</li> <li>Creative activity</li> <li>Imaginative play with parent</li> <li>Self-expression</li> </ul>
3 yr	<ul style="list-style-type: none"> <li>Went up stairs</li> <li>Walks up stairs</li> <li>Labels to self "penicillin," "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> </ul>	<ul style="list-style-type: none"> <li>Stable footing of child</li> <li>Copies horizontal and vertical shapes (dot in center)</li> </ul>
4 yr	<ul style="list-style-type: none"> <li>Labels to self "penicillin," "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> </ul>	<ul style="list-style-type: none"> <li>Stable footing of child</li> <li>Imagines construction of bridge with three cubes</li> </ul>
5 yr	<ul style="list-style-type: none"> <li>Labels to self "penicillin," "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> <li>Labels and "penicillin" "1"</li> </ul>	<ul style="list-style-type: none"> <li>Stable footing of child</li> <li>Imagines construction of bridge with three cubes</li> <li>Imagines construction of bridge with three cubes</li> <li>Imagines construction of bridge with three cubes</li> <li>Imagines construction of bridge with three cubes</li> <li>Imagines construction of bridge with three cubes</li> <li>Imagines construction of bridge with three cubes</li> </ul>

**Table 41.1. Checklist for Assessment by Observation of Developmental Level of Preschool-Age Child**

An evaluation of the parents' child-rearing skills is particularly important in the psychiatric assessment of the infant. General characteristics to be assessed include the parents' self-esteem, competence, flexibility, and ability to provide a safe, nurturing, and appropriately stimulating environment. Specific characteristics to be assessed include the following: the sensitivity and accuracy of the parents' perceptions of the infant's needs; the goodness of fit between parent and child; the parents' ability to respond rapidly and appropriately on a contingent basis to the infant's expressed needs; the quality of play between parent and infant; and the amount of affection, support, encouragement, and assistance (scaffolding) the parent can provide for the child. The parents should also be able to buffer and titrate the level of stimulation appropriately to foster development on one hand, while preventing the child from being overwhelmed on the other.

Infants less than 18 months old should also subsequently be observed in spontaneous, free play, using such games as peek-a-boo and patty cake. Children between the ages of 18 months and 3 years can participate more fully in regular unstructured play interviews. The play items should look reasonably realistic because children at this age have a limited capacity for abstraction and symbolic play.

A more systematic and detailed assessment requires the use of such scales as the Gesell Infant Scale, the Bayley Infant Scales of Development, the Uzgiris-Hunt Scales, and the Denver Developmental Screening Test ( [Table 41.2](#)) ( [Chapter 40](#)).

**Table 41.2. Selected Developmental and Psychological Tests**

### School—Age Children

Clinical interviews with school-age children similarly require sufficient time (a minimum of 45 to 60 minutes for each interview) and suitable space and the availability of such play items as a ball, crayons and paper, a dollhouse, rubber dolls, puppets, toy guns, a toy doctor's bag, and toy telephones. Elaborate, highly structured games, such as chess or Monopoly, are too likely to interfere with the evaluation by preoccupying the child's attention and cognitive efforts and too readily lend themselves to defensive purposes.

Play and play materials serve several functions. They help to place the child at ease. They provide a context in which to observe many facets of the child mental status, ranging from attitude to the examiner, motor control, thought content, and affective regulation (see the later discussion of mental status). Most important, these unstructured play sessions allow the clinician to make inferences about the child's psychic life, including the child's wishes and preoccupations, fears, impulses, conflicts, defenses, affects, and relationships.

Having previously discussed with the parents how to prepare the child for the interview, the interviewer greets the child from a reasonable physical distance in the waiting room and takes care not to loom too intrusively or intimidatingly. The interviewer should introduce himself or herself to the child and should invite the child to come into the office while reassuring the child that his or her parent or parents will be in the waiting room on return. Once inside the office, the interviewer should ask for the child's preferred name and should make sure the child knows the interviewer's name. The interviewer should not sit behind a desk. He or she should clarify what is the child's understanding of why the child has come, and then the interviewer should give his or her own understanding. Next, the interviewer should tell the child what will take place: "This is a time set aside to see whether I can help you understand what may be bothering you. We will have 45 minutes together, at the end of which you will return to your parents." The interviewer should also clarify the extent of the confidentiality: "I will be meeting with your parents, but I will first discuss with you what I will or will not say to your parents." In some circumstances, such as a court-ordered evaluation, there is no confidentiality, and a report must be rendered to the court. The interviewer should inform the child of this fact.

It is probably best not to take notes during the interview. Note taking may inhibit the child, and it inhibits the interviewer's ability to observe. The interviewer should avoid leading questions or any kind of demanding interrogation because that, too, is unproductive and may inhibit the play and communication. Open-ended

questions (e.g., "What happened then?") are better than leading questions and questions that require only a single-word answer.

In addition to inquiring about the areas of difficulty or worry that have brought the child to clinical attention, it is also helpful to inquire about what the child does for fun and about activities he or she is good at doing. In addition to the clinician's genuine interest in these areas of strength and potential sources of self-esteem, this topic helps to place the child at ease and conveys the sense that the assessment will not be an interrogation focused on what the child may find most embarrassing, anxiety provoking, or humiliating.

In interviewing a child, the clinician can also sometimes offer an interpretation of an obvious preconscious feeling or fantasy. It is often eye-opening, clarifying, and interesting for the child or adolescent, and it sets a model for any subsequent psychotherapy that may be recommended. For example, the child's attention can be directed to the content of his or her actions or verbalizations. Sometimes attention can be drawn to a coincidence that the child has perceived but has not, or professes not to have, registered; more frequently, one can draw attention to certain paradoxes. Thus, in the course of the child's play, the therapist may provide a verbal counterpart to the action being portrayed, an affect that may be present, or the conspicuous absence of certain persons, actions, or affects. (For an example of such an "attention" interpretation, see [Chapter 79](#).)

This kind of interpretation during a diagnostic interview is different from the direct translation of a possible unconscious symbolic representation in the play that may occur in the course of psychodynamic psychotherapy. The play characteristic to which attention is drawn here is in bold relief, capable of being fully recognized and understood by the child.

## Adolescents

Clinical interviews with adolescents require a direct and explicit approach. The clinician can explain to the adolescent that his or her parents came to see the clinician and spoke of their concerns but that the clinician would like to learn directly from the adolescent his or her views on what the parents have said or on what bothers him or her. The clinician should show a genuine interest in the adolescent's view of the matters that have led to the evaluation and should not try to be deceptive, overly ingratiating, or phony. The clinician should not try too hard to appear "with it" or "cool." It is often more productive to employ what Hilde Bruch termed "the constructive use of ignorance" ([King and Schowalter, 1997](#)). If an adolescent enthuses about (or denounces) some activity, group, program, or style with which the clinician is unfamiliar, rather than feigning knowledge of it, one can ask "I don't know much about that; who (or what) is that and what it is that you like so much (or can't stand) about them (or it)." This approach serves several functions. It permits the adolescent to be the expert, educating the grown up, and it also conveys the notion that antipathies or enthusiasms can be thought about and discussed, rather than simply being givens to be taken for granted. In general, it is important for the clinician to be aware of and in control of his or her own predilections to identify with parental authority or adolescent oppositionality.

If the adolescent talks in terms of a third person ("I have a friend who . . ." or "Can a person catch herpes from kissing . . ." or "I read that . . ."), the clinician should answer matter of factly, in the same third-person way. The adolescent is not fooling the clinician, and the clinician is not fooling the adolescent; the adolescent is being allowed room to move and then will not feel so much on the spot.

Rejection, even outright hostility, toward the clinician on the first few visits with an adolescent is not uncommon. The clinician should be patient and should not jump to conclusions. This attitude may turn out to be a test of how much the clinician can be trusted, a defense against anxiety, or a transference phenomenon. The clinician should recognize the adolescent's anger by saying something such as, "I can see you're pretty angry at being here, but you know, I don't know much about how you see things. What are you particularly angry about? Perhaps there is some way I can be of help to you."

Silences should not be allowed to continue for too long, because this risks a useless power game to see who can hold out longer. Similarly, it is important not to be rigid about the length of the interview. The 50-minute session is not a sacred rule; the clinician should feel free to vary the time according to the situation at hand. In some instances, an adolescent may feel more comfortable initially if he or she is invited to go for a walk with the clinician rather than asked to sit face to face in a confined space.

The clinician must be particularly clear with the adolescent about the extent of confidentiality. When appropriate, the adolescent should be informed that a report will be made to a third party, such as a judge. A sense of trust is infinitely preferable to a feeling of betrayal, even at the expense of some tidbit of knowledge.

In general, it is better not to give advice; however, the clinician should not rigidly deny any opinion or advice. An occasional well-judged opinion (if asked) on, for example, the color of a lipstick, or some well-chosen advice may help an adolescent to feel understood and supported.

Eventually, the clinician must inquire about such sensitive areas as suicidal thoughts, hallucinations, drug use, and sexual relationships. This should be done in a matter-of-fact, straightforward manner (see later).

## Countertransference and Other Issues for the Interviewer

During clinical interviews with children and adolescents, the clinician must be aware of important countertransference, as well as transference, phenomena that may occur. For example, children who are aggressive often tend to mobilize strong defenses, such as critical, punitive, or retaliative feelings in the clinician, mentally retarded children are often overlooked or inadequately served, and deformed children may initially repel some clinicians. Other signs of countertransference include the following:

1. The clinician may fail to recognize the developmental level of the child or adolescent. Expectations will then not be commensurate with the child's or adolescent's maturational and developmental capacities.
2. The regressive pull experienced by the clinician interviewing the child or adolescent may give rise to the temptation to identify or act out with the child or adolescent.
3. In too readily regarding the patient's reactions toward the clinician as realistically determined, the clinician may misread transference reactions stemming from the child's or adolescent's feelings toward aspects of the relationship with his or her parents, past or present. Clinicians are usually well aware of a child's or adolescent's aggressive feelings but may be less consciously aware of a youngster's seductiveness toward an adult (parent).
4. Certain specific behaviors in the child or adolescent may stir up old conflicts within the clinician, together with attendant anxieties and defenses. For example, delinquent behavior or disguised masturbation may be upsetting to the clinician resulting in feelings of anxiety, irritation, disapproval, or punitiveness.
5. Sometimes clinicians transfer early feelings from their own childhood onto the parents of the child or adolescent. As a result, clinicians may then overidentify with either the child or the parents in the course of their conflicts. Similarly, residual feelings from the clinician's own childhood relations with brothers and sisters may be an important source of ambivalence toward the child or adolescent.
6. Sometimes the clinician simply cannot understand the meaning of certain behaviors in a child or adolescent. Anyone can occasionally find some item of behavior inexplicable. However, the persistent drawing of a blank in understanding a repeated item of behavior should lead to the suspicion of an interference by one's own conflicts—an emotional blind spot, so to speak.
7. A clinician may feel depressed or uneasy when working with a certain child or adolescent. Assuming that the clinician is not suffering from true depression, the possibility exists that emotions from old conflicts have been aroused and are interfering with the clinician's functioning. A clinician may occasionally become aroused and experience great affection or rescue fantasies toward a child or adolescent, and this also may interfere with the assessment or treatment.
8. A clinician may be unwittingly tempted to encourage acting out in children or adolescents. For example, a clinician may suggest to them that they must stand up for themselves and hit back. This inclination and the suggestion should be carefully examined.
9. A clinician may feel the need for approval from the child or adolescent. Such a desire often represents a need of the clinician and may not be in the best interests of the child or adolescent.
10. Conversely, repeated arguing with a child or adolescent may suggest that the clinician has not only become involved but has become embroiled with the child or adolescent in such a way as to replicate aspects of the child's conflicts at home or at school, with the clinician drawn into a critical or adversarial stance.
11. Recurring countertransference problems commonly arise in relation to specific characteristics of a child or adolescent. For example, a retarded child or adolescent may evoke guilt and defenses against such guilt in the clinician, or the clinician may act out omnipotent rescue fantasies. Passive, hostile children or adolescents may arouse anger or punitiveness in a clinician. Aggressive children and adolescents of either sex may threaten the clinician and may lead either to vicarious and excessive exploitation of sexual issues or to denial and avoidance.

## Mental Status Examination



How and what the child plays, says, and does constitute the raw data for the mental status examination. To bring some order to the understanding of what may seem like random play, it is useful to have an outline of points one particularly wants to observe and why. When completed, such an outline constitutes the report of the child's mental status.

Another useful organizing principle is to keep in mind the major categories of psychopathology one wants to be sure to cover. A list of such categories is shown in [Table 41.3](#).

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Developmental delay
Organic brain dysfunction
Thought disorder
Anxiety
Mood disorder
Temperament and personality (character) problems
Somatiform disorders
Effects of general medical conditions
Mental retardation
Reaction to unfavorable environment

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Adapted from Lewis M: Psychiatric examination of the infant, child, and adolescent. In: Kaplan HI, Sadock BJ (eds). Comprehensive Textbook of Psychiatry. Baltimore, Williams & Wilkins, 1989, with permission.

**Table 41.3. Categories of Psychopathology**

Some of the data emerge spontaneously, some only after questioning. The categories in the mental status examination outline that follows are for convenience only; the behaviors described here usually do not occur in any special sequence and are not isolated items. The child or adolescent acts as a whole and in the context of a given environment, and his or her present behavior is always continuous with past behavior. It is not necessary to elicit the information required in the precise order presented here, and all these categories need not be covered in equal detail or in one sitting. The presenting symptom and history may indicate important areas for close attention. The clinician uses his or her clinical judgment to determine what to look for and how fast and in what detail to proceed. The clinician should also consider the age and developmental level of the infant, child, or adolescent when assessing a given response. An outline of the mental status examination is shown in [Table 41.4](#). A useful questionnaire that can be used as a checklist in the emergency room is shown in Appendix A.

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1. Physical appearance
2. Separation
3. Manner of relating
4. Orientation to time, place, and person
5. Central nervous system functioning
6. Reading and writing
7. Speech and language
8. Intelligence
9. Memory
10. Quality of thinking and perception
11. Fantasies and internal conflicts
12. Affects
13. Object relations
14. Drive behavior
15. Defense organization
16. Judgment and insight
17. Self-esteem
18. Adaptive capacities
19. Positive attributes

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From Lewis M: Psychiatric examination of the infant, child, and adolescent. In: Kaplan HI, Sadock BJ (eds). Comprehensive Textbook of Psychiatry. Baltimore, Williams & Wilkins, 1989, with permission.

**Table 41.4. Mental Status Examination Outline**

#### PHYSICAL APPEARANCE

1. Small stature is often associated with a more immature self-image; the self-esteem of the child who is short may suffer because of his or her size. The cause of shortness is important to determine, and referral for a pediatric endocrine evaluation may be appropriate. Precocious or delayed pubertal development may also have important psychological consequences ([Chapter 24](#)) and should be noted.
2. Head size may indicate microcephaly and mental retardation or hydrocephaly.
3. Physical stigmata, such as unusual facies or other minor physical anomalies, may indicate the presence of a chromosomal disorder (e.g., Down's syndrome, Turner's syndrome, fragile X syndrome, velocardiofacial syndrome) or prenatal toxicity (e.g., fetal alcohol syndrome).
4. Bruising may indicate child abuse.
5. Nutritional state may indicate an eating disorder, ranging from anorexia nervosa to obesity.
6. A level of anxiety may be manifested in the interview by restlessness, hyperalertness, tics, nail biting, or hair pulling. The child's activity may have a driven quality, with an inability to sit still, restless moving from one thing to another, easy distractibility, short attention span, low frustration tolerance, and labile emotions. Differentiating among a primary attention deficit hyperactivity disorder (ADHD), hypomania, and anxiety disorder may prove a challenge to the clinician.
7. Momentary lapses of attention (staring, head nodding, eye blinking) may indicate epilepsy or hallucinatory phenomena. The clinician subsequently inquires about such seizure-associated phenomena as auras (nausea, vomiting, epigastric sensations), micropsia or macropsia ("Do things seem to get smaller or bigger as you look at them?"), and hallucinations.
8. Dress gives some idea of the care the child receives and how much the child cares for himself or herself. In adolescence especially, choice of clothing is an important indicator of ideology and ethnic and group affiliations and identifications (e.g., prep versus grunge or goth). Sexual orientation and conflicts may also be expressed in attitudes, behavior, and dress.
9. Motor mannerisms or stereotypies may provide a clue to a disorder. For example, repetitive flapping or twirling may accompany various forms of pervasive developmental disorder or social relatedness ([Rapin, 2000](#)). Motor or vocal tics may be a transient sign of anxiety or a feature of a chronic tic disorder, such as Tourette's syndrome. Rocking, thumb sucking, or repetitive play may be signs of regressive attempts at self-soothing.

#### EASE OF SEPARATION

Some caution on the part of the child in separating from the parents to go with the clinician is usually appropriate. Too much ease in separating from the parents may indicate superficial relationships associated with frequent separations and maternal deprivation. Difficulty in separating may indicate separation anxiety or an ambivalent parent-child relationship.

#### MANNER OF RELATING

Once in the examining room, the child usually relates to the clinician cautiously at first. However, some children (e.g., deprived or abused children) are indiscriminately friendly and shallow. Children with autism may appear aloof or to look through people.

#### ORIENTATION TO TIME, PLACE, AND PERSON

Orientation may be impaired by organic brain factors, low intelligence, anxiety, or a thought disorder.

#### CENTRAL NERVOUS SYSTEM FUNCTIONING

During the interview of the child, it is often possible to include in a playful fashion many elements of a developmentally oriented neurologic examination to assess a child's neuromaturation status. Such an examination tests functions such as laterality, rapid alternative movements (diadochokinesis), rapid finger tapping, cerebellar functioning (e.g., finger-to-nose test), gait (e.g., heel-to-toe walking), and balance (e.g., Romberg test, hopping on one foot) and looks for the presence of findings such as motor overflow phenomena, motor drift, or choreiform movements. Various standardized formats exist for administering and scoring this examination (Denckla, 1985; Rutter et al., 1970). Abnormalities may include either mild focal or localizing signs (e.g., asymmetry) or signs of developmental immaturity. However, the diagnostic implications of such "soft signs" are unclear (Chapter 44). In some studies they appear to be nonspecific, perhaps correlating with intelligence quotient (IQ) or even with vulnerability to later anxiety disorder, whereas other studies suggest that, when these signs present, they may reflect vulnerabilities in cerebral organization that may predispose the child to learning difficulties.

#### Gross Motor Coordination and Abnormal Movements

The clinician should note the presence of any awkwardness, clumsiness, tremors, motor overflow with extraneous movements, motor tics, and any abnormalities of posture, gait, and balance. These can be observed informally in the course of unstructured activities (e.g., pencil grip in writing, skill in climbing stairs, ball throwing, and catching) or explicitly examined. For example, the presence of any choreiform movements can be elicited by asking the child to stand with his or her arms extended and hands outstretched with the fingers spread.

#### Fine Motor Coordination (Perceptuomotor Capacities)

The child is asked to copy the designs seen in Fig. 41.1 or the more demanding figures of the Bender-Gestalt Test (Bender, 1938) (Fig. 41.2). Formal testing is indicated if the child has difficulty copying the designs at an age appropriate level (Fig. 41.3). The difficulties may include trouble with angulation and juxtaposition, a tendency to verticalize a diagonal, and substitution of loops for dots. Further discussion and illustration of the Bender-Gestalt Test are given in Chapter 43.

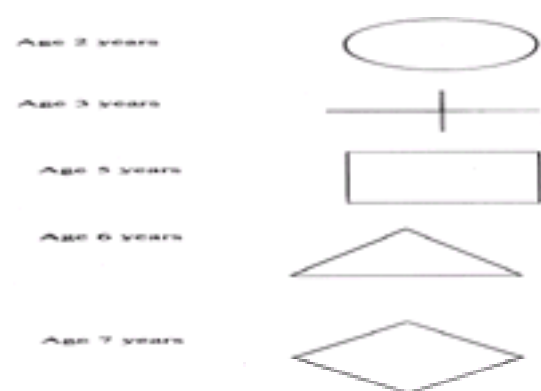


Figure 41.1. Simple designs by age.

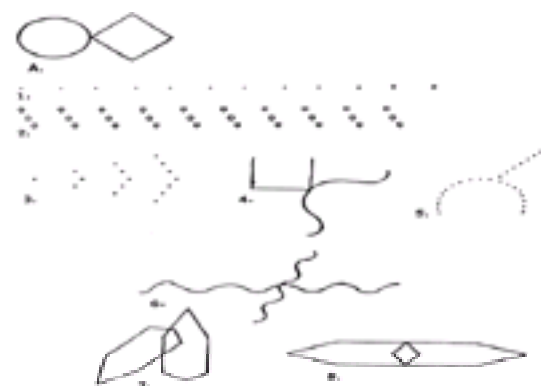


Figure 41.2. The Visual Motor Gestalt Test figures. (From Bender L: *A Visual Motor Gestalt Test and Its Clinical Use*. New York, American Orthopsychiatric Association, 1938.)

Age	Figure 1	Figure 2	Figure 3	Figure 4	Figure 5	Figure 6	Figure 7	Figure 8	Figure 9	Figure 10
Adult										
10 yrs										
9 yrs										
8 yrs										
7 yrs										
6 yrs										
5 yrs										
4 yrs										
3 yrs										
2 yrs										

Figure 41.3. Norms for the Visual Motor Gestalt Test. (From Bender L: *A Visual Motor Gestalt Test and Its Clinical Use*. New York, American Orthopsychiatric Association, 1938.)

#### Laterality

Laterality, preference, and dominance are not identical. *Laterality* is a measurable, specialized, central function of a paired faculty, such as eyes, ears, hands, and feet. *Preference* is the subjective, self-reported experience of an individual, as opposed to laterality, which may be objectively measured. *Dominance* is the term used for the concept of cerebral hemisphere specialization, such as in language and speech. Clinically, one may merely be testing preference, which, in turn, may depend more on the peripheral organ than on any central mechanism.

Handedness is usually consolidated by age 5 years, footedness by about age 7 years, eye lateralization by about age 7 or 8, and ear lateralization by about age 9 (Towne, 1980). Clinically, these may be tested by observing which hand the child writes with, what foot he or she kicks a ball with, and which eye is preferred when the child is asked to look through a rolled-up piece of paper (as through a telescope) or to look at the examiner through a small hole in a sheet of paper.

#### Right-Left Discrimination

The child should be asked to put the right hand to the left ear, the left hand to the right knee, and so forth. At the age of 5 years, children can identify right and left hands (i.e., if they have been taught). At age 6, the child has ipsilateral double orientation (i.e., left hand on left ear), and at age 7, contralateral orientation is achieved (i.e., left hand on right ear) (Silver and Hagan, 1982).



### Short Attention Span

Easy distractability and short attention span during the interview may have a variety of causes in addition to or instead of attention deficit disorder. These include fatigue, anxiety, language problems, or a level of discourse between the parents and clinician that is too abstract for the child to follow. Frequent petit mal or other seizure disorder or sedation from medication may also interfere with a child's attentional focus. In addition, some children with pervasive developmental disorders or disorders of social communication may not be sufficiently entrained by social discourse and, hence, may be inattentive to the clinician's attempts at communication.

### Hyperactivity

Although the possibility of ADHD is a common impetus for referral, hyperactivity may not always be observed in the clinician's office, especially in the one-to-one setting of the child interview. Even children with ADHD may be able to remain focused in a structured one-to-one setting. Hence, reports from multiple settings, including parents and as many different classroom settings as possible, are needed to confirm or rule out this diagnosis. As noted earlier, various other conditions can also cause hyperactivity and disorganized activity in the interview, including anxiety, intense oppositionality, psychosis, and hypomania.

### Sensory Difficulties

**Visual and Oculomotor Difficulties.** These include strabismus, nystagmus, convergence difficulties, and problems in visual acuity.

**Hearing Difficulties.** Hearing should be assessed clinically in the interview, especially if there are difficulties with speech or aural comprehension or a history of recurrent otitis media. In such cases, formal audiometric testing should also be obtained if not already done.

### Reading, Writing, Language, and Speech Difficulties

A prerequisite for assessing the possible causes of a child's academic difficulties is a careful educational history, including the child's previous educational placements and any special educational resources or curricular modifications attempted. For children from bilingual backgrounds, it is also important to know the principal language in which instruction has taken place and the child's and parents' level of fluency in that language.

Formal assessments of reading, writing, and language skills are usually indicated if academic difficulty is one of the chief complaints ( [Chapter 50](#)). Although ADHD or disruptive behavior can interfere with the academic progress and classroom comportment of children with otherwise intact cognitive skills, it is also the case that undiagnosed learning disabilities can be the cause of restless and maladaptive classroom behavior.

Although no substitute for formal psychoeducational assessment, it is often useful to ask the child to read a passage or to write a few sentences during the evaluation. It is also useful to inquire what sort of reading the child does for fun. In addition to providing some insight into the child's reading and concentration abilities, the choice of books also provides information about the child's interests and fantasy life.

Asked to read or produce some sample writing, the child may struggle to read or write and may exhibit poor spelling or handwriting. Typical reading difficulties include reversals and inversions (e.g., d-b, q-p; was-saw, felt-left), confusions (e.g., f-l, m-n; of-off, me-we), omissions (e.g., afraid for afraid, place for palace), and substitutions (e.g., a for the, house for home). At the same time, many normal first-grade (6-year-old) children show reversals. These reversals usually disappear as reading skill matures. The brief screening test shown in [Table 41.5](#) can be used to approximate the child's reading ability ( [Kanner and Eisenberg, 1957](#)).

First grade	40 seconds; 4 errors A little boy had a cat; she ran away; she said, "I want some milk."
Second grade	25 seconds; 2 errors A man took me to see his large barn. There was a horse in the yard; its tail was black.
Third grade	30 seconds; 2 errors One of our favorite birds is the robin. He is a very useful bird. He eats many insects and worms. The robin is less afraid of people than most birds.

Reproduced by permission of Kanner L, Eisenberg L. Childhood problems in relation to the family. *Pediatrics* 20: 155, 1957, with permission.

**Table 41.5. Screening Tests of Reading Ability**

Children with reading difficulties often come from large families and neighborhoods with poor schools and may have symptoms of ADHD or of conduct disorder. Because reading level in general correlates highly with IQ, the most common cause of general reading backwardness, that is, below-average reading level despite adequate educational opportunities, is below-average IQ. Children with *specific* reading retardation (sometimes called dyslexia or developmental reading disorder and often part of a multifaceted, complex learning disability) have reading levels lower than expected from their overall IQ. The causes of dyslexia are still poorly understood, but in at least some children they include a genetic component and are accompanied by more general language processing difficulties, such as difficulties in rapid naming and phonemic segmentation ( [Chapter 32](#)). If the child shows positive findings in any of these clinical screening tests for reading, writing, and spelling, more systematic evaluation is required using standard tests ( [Chapter 50](#)).

### SPEECH AND LANGUAGE

Children who do not use words by age 18 months or phrases by age 2½ to 3 years, but who have a history of normal babbling, who understand commands, and who can use, as well as respond to, nonverbal cues and gestures are probably developing normally. However, delays beyond these ages or disturbances in these and other forms of communication are indicators for further evaluation. The general clinical signs of a language dysfunction include the following:

1. Reduced vocabulary, especially for abstract concepts such as feelings and for question words such as "when" and "where."
2. Overuse of concrete nouns and verbs.
3. Underuse or omission of abstract word classes (e.g., adjectives, adverbs, prepositions, articles, and conjunctions) often giving rise to telegraphic or unintelligible speech. (Some children may then avoid speaking or may have interpreters speak for them.)
4. A tendency to repeat their utterance, or simply nod their head, rather than to clarify what they were trying to say.

Specific language difficulties may result from receptive or expressive problems: Clinically, both commonly occur together.

*Receptive language problems* include sensory impairment (e.g., hearing impairment, also giving rise to delayed and unclear speech) and neurologic damage (e.g., mental retardation, primary disorders of speech) ( [Table 41.6](#)). Children with pervasive developmental disorders may have difficulty in understanding communicative nuances such as metaphor, irony, or humor; these deficits stem in part from relative difficulties in reading paraverbal cues such as facial expression, gesture, and intonation.

	Deafness	Mental Retardation	Infantile Autism	Ectopic W/len
Sound discrimination	↓	Normal	Normal	Normal
Attentiveness	↓ Watches face	↓	↓↓	Normal
Understanding complex orders	↓	↓	↓↓	Normal

From Lewis M. *Clinical Aspects of Child Development*, 2nd ed. Philadelphia, Lea & Febiger, 1982, p. 357, with permission.

**Table 41.6. Receptive Abilities**

*Expressive language problems* involve delays or abnormalities in the development of syntax and semantics and problems of speech production. *Syntax*, the term used to categorize the rules for combining words to form sentences, may be delayed. The normal child can make one-word utterances at age 18 months, two- to three-word phrases at age 30 months, and four-word sentences at age 40 months. *Semantics*, the term used to categorize the meaning of language, may similarly be delayed. Delays in syntax and semantic development may first be encountered in a history of limited or poor babbling. The child may subsequently use gestures instead of verbal language to communicate.

Abnormalities of expressive language include echolalia and delayed echolalia, the persistent use of neologisms, and misuse of pronouns and gender. Such abnormalities are commonly seen in childhood autism and may be associated with a lack of nonverbal communication behavior, including lack of direct eye gaze and communicative use of facial expression. Despite sometimes good vocabularies, children with pervasive developmental disorders have difficulty using language as a means of social interchange, with deficits in taking turns when speaking and an inability to infer the interlocutor's interests or perspective. Some useful language tests are shown in [Table 41.7](#).

Analysis of Spontaneous Speech Samples
Developmental Sentence Scoring (DSS)
Assigning Structure Stages (Miller)
Linguistic Analysis and Remediation Procedure (Crystal)
Systematic Analysis of Language Transcripts (Miller and Chapman)
Language
Pretests Language Scale (Zimmerman)
Language Screening Test (Bishop)
Test for Auditory Comprehension of Language (Fischhoff)
Sequenced Inventory of Communicative Development (Mervis, Proffer, and Inbar)
Revised Developmental Language Scales
Miller Vocabulary Comprehension Test
Clinical Evaluation of Language Fundamentals (Semel, Mick, Wiig, and Merrill)
Word Test of Psycholinguistic Abilities (WPA)
Denver Tests of Learning Aptitude
Vocabulary Tests
Peabody Picture Vocabulary Test, Revised Edition (Dunn)
Expressive One-Word Picture Vocabulary Test (Gooden)
Articulation Tests
Goldman-Fristoe Test of Articulation
The Assessment of Phonological Processes (Hudson)
Auditory Discrimination
Goldman-Fristoe Nonword Test
Wepman Auditory Discrimination Test

From Lewis M. *Psychiatric Evaluation of the Infant, Child, and Adolescent in Hospital*, 3rd ed. Baltimore, Williams & Wilkins, 1989, with permission.

**Table 41.7. Language and Speech Tests**

Common speech difficulties consist of delays, omissions, or distortions in the normal acquisition of particular sounds (e.g., wabbit instead of rabbit). Most uncomplicated, common speech acquisition difficulties of this kind resolve by the age of 7 or 8 years. Stuttering (consisting of repetitions, hesitations, or blocks in the production of a speech sound) often begins about the age of 3 years, follows a fluctuating course, and usually ends at adolescence. In some cases (less than 20%), it may persist into adulthood. There is often a family history of stuttering, a finding suggesting a genetic component.

Other dysfunctional speech patterns may reflect the level of integration at which the central nervous system is affected. Thus, aphonia may occur when the neuromuscular level is involved, including such apparatuses as the lips, tongue, palate, nasopharynx, larynx, and medulla oblongata; dysarthria may occur when the corticobulbar level is involved; scanning, explosive, and monotone speech may occur when the cerebellar level is affected; and agnosia (failure to understand symbols) and aphasia (failure to understand the spoken word or to speak) may occur when the cerebral level is affected.

Speech that is monotonous in tone, with poor, unusual, or unexpressive prosody, is found in many children with pervasive developmental disorders. Complex vocal tics may occur in more severe cases of Tourette's syndrome and may include repetitive syllables or repetitively ejaculated words or phrases, including out-of-context obscenities (coprolalia), pallilalia (repetition of one's own words), and bursts of abnormal prosody; however, such complex tics are rare in the absence of a clear history of prominent simple vocal tics (e.g., sniffing, throat clearing, grunting).

Temporary speech problems may occur during regressive episodes (infantile speech patterns may reappear), during drug intoxication (dysarthria and slurred speech may occur), during anxiety (a high-pitched, tight voice may inhibit speech), and when the child refuses to talk (so-called selective mutism).

When one or more of the foregoing clinical findings are present, a full diagnostic evaluation is indicated. This evaluation may include a complete physical examination, neurologic examination, hearing assessment, reading assessment, comprehensive language and speech tests, and an educational test battery ( [Chapter 45](#) and [Chapter 49](#)).

## INTELLIGENCE

An approximate idea of the child's intelligence may be assessed by an evaluation of the following:

1. General vocabulary, responsiveness, and level of comprehension and curiosity are evaluated.
2. In young children, the ability to identify the parts of the body is evaluated. For example, at age 5 years, the normal child may be able to identify the jaw, temples, forearms, and shins.
3. Drawing ability is assessed. The child is asked to draw a person (see later).
4. The ability to subtract serial sevens or serial threes is assessed.

## MEMORY

At age 8 years, the normal child can count five digits forward and two or three digits backward; at age 10 years, the child can count six digits forward and four digits backward. Very poor performance on the digit span test may indicate difficulties with attention or problems with working memory because of organic factors. Minor difficulties may simply reflect anxiety. The child should also be able to repeat three items 5 minutes after they have been presented.

## QUALITY OF THINKING AND PERCEPTION

Traditionally, the clinical assessment of thought includes two major clinical dimensions: (a) actual thought content and (b) the form, flow, and organization of thought. A variation in any of these dimensions may be of such a degree and duration as to constitute a thought disorder. In actual practice, the distinction between form and content of speech is not always sharp.

*Disordered thought content* may take the form of unusual beliefs (delusions) or perceptions (hallucinations). Other unusual thought contents that also reflect



deficiencies in the formal quality of thought are neologisms and idiosyncratic logic, including developmentally inappropriate transductive reasoning (things that are related in time or space are believed to be related causally), difficulty in discerning differences and similarities, difficulty in distinguishing the relevant from the irrelevant, and excessive concreteness. *Disordered flow of thought* may take the form of slowness of thought (as in primary obsessional slowness) or a press of thought and speech with flight of ideas. Disorder of flow may take the form of blocking, muteness, or excessive repetition of words and sentences. A child may experience any of these manifestations subjectively as being alien, out of his or her control, and sometimes frightening. Loosening of associations entails poorly connected shifts in topic, with the speaker unaware of the incoherence of his discourse. Closely related to loosening of associations is tangential thinking, in which speech veers from one topic to another.

Some of these psychopathologic concepts are difficult to apply to young children without clear developmental criteria. To help distinguish among developmentally normal immaturities in discourse, Caplan and colleagues developed the Kiddie Formal Thought Disorder Rating Scale, which provides operational definitions of illogical thinking, incoherence, loosening of associations, and poverty of speech content applicable to children ( [Caplan et al., 1989, 1990](#)). These authors find that loose associations and illogical associations are rare or absent in normal children after age 7 years, and these features differentiate children with schizophrenia or schizotypal personality disorder from normal children. Children with complex partial seizure disorder sometimes exhibit illogical thinking without loosening of associations.

The causes of a thought disorder may be classified clinically as follows:

1. Genetic (e.g., inborn errors of metabolism, such as Hartnup's disease, Kufs' disease, and schizophrenia)
2. Traumatic (e.g., postconcussion syndrome)
3. Infective (e.g., viral encephalitis and brain abscesses)
4. Neoplastic (e.g., brain tumor)
5. Toxic (e.g., amphetamines, corticosteroids, bromism)
6. Deficiency related (e.g., pellagra)
7. Endocrine (e.g., thyrotoxicosis)
8. Metabolic (e.g., electrolyte disturbance and delirium)
9. Idiopathic (e.g., pervasive developmental disorder, schizophrenia)
10. Posttraumatic (e.g., acute and massive psychological stress reaction)
11. Seizure related (e.g. fugue state, partial complex seizures)

The symptoms that constitute a thought disorder should lead to a systematic review of the foregoing possible causes. Obviously, some causes are easily ruled out, and others immediately appear to be likely possibilities. Once the field has been narrowed in this way, a more detailed study can lead to further narrowing. For example, the associated presence of hallucinations may give a cross-differential diagnosis of specific syndromes.

### *Hallucinations*

The clinician should ask about auditory and visual hallucinations as though he or she were taking a medical history of the eyes and ears. The clinician may ask, "Do you have any trouble with your ears?" "Do your ears ever hurt you?" "Do your ears ever play tricks on you?" "Do you ever think you hear something, but nothing is there?" A similar sequence can be designed for inquiring about visual hallucinations.

One can follow up by asking, "Do you hear it inside your head or outside?" and "Do you think it (or they) are really there, or is it just your imagination?" It is important to try to distinguish between true hallucinations and other phenomena, such as obsessions or compulsions, which children may sometimes describe metaphorically as "a voice telling me to do [some compulsive action]." Dissociative phenomena (including vivid flashback phenomena and intrusive imagery), such as are found in children with posttraumatic stress disorder, may also be difficult to differentiate from hallucinations.

It is also usually relatively easy to distinguish the phenomenon of imaginary companions from true hallucinations by the following features. The child with an imaginary companion is usually normal in other respects and shows no other signs of a thought disorder. The imaginary companion can be imagined or dismissed at will, it is experienced as nonthreatening, comforting, and enjoyable, and it has functions and characteristics assigned at the child's volition. Unlike hallucinations, the imaginary companion is not ego-alien, and the child can freely talk about his or her "friend" in elaborate detail. The natural history of the imaginary companion and its possible relation to dissociative identity disorder ( [Chapter 70](#)) require further study.

Persistent hallucinations in childhood are frequently associated with psychopathology, but they are not necessarily pathognomonic of serious psychotic illness, such as schizophrenia, mania, or melancholic depression with mood congruent or self-reproachful hallucinations (including command hallucinations to hurt oneself) ( [Volkmar et al., 1995](#)). The following additional major clinical categories should also be considered.

**Drug Intoxication.** Many illicit drugs are potentially hallucinogenic; they include marijuana, psychedelics, cocaine, amphetamines, and barbiturates. Furthermore, children and adolescents who abuse drugs may also have an antecedent psychiatric disturbance. However, many legitimately prescribed medications, such as steroids and antihistamines or other atropine-like drugs, can produce hallucinations either when taken in therapeutic quantities or as a result of deliberate or inadvertent overdose or interaction with other medications. The timing and form of the hallucinations (e.g., florid visual hallucinations) sometimes suggest the possibility of drug ingestion. Other symptoms of drug ingestion may be present, including drowsiness, paranoid behavior, confusion, restlessness, excitement, violence, dilated pupils, ataxia, dysmetria, tremor, dysarthria, dyskinesia, akathisia, and hypotensive signs. The clinician must ask about drug ingestion. Urine and blood samples must be tested when drug ingestion is suspected.

**Seizure Disorder.** Hallucinations, particularly hypnagogic hallucinations, may occur in narcolepsy and seizure disorders. Hallucinations may be the first symptom of degeneration after a previous encephalitic illness. A neurologic examination and an electroencephalogram are required.

**Metabolic Disorders.** The metabolic disorders that may give rise to hallucinations include adrenal cortical hypofunction, thyroid and parathyroid disease, hepatolenticular degeneration, porphyria, beriberi, and electrolyte or mineral imbalance (secondary to prolonged parenteral fluid replacement therapy, diuretic therapy, excess vitamin D intake, or diabetic acidosis). Signs of the primary metabolic disorder are usually present.

**Infection.** Encephalitis, meningitis, and acute febrile illnesses (especially in young children) may give rise to hallucinations.

**Immaturity, Stress, and Anxiety.** Hallucinations in children are not always indicators of a serious psychiatric disorder or an organic cause. Hallucinations may also occur transiently in young children overwhelmed by anxiety ( [Volkmar et al., 1995](#)). These hallucinations may begin at night as either hypnopompic or hypnagogic phenomena and may be accompanied by the illusion or hallucination of insects crawling on the skin (formication), a sign of intense somatic anxiety. Young children who are under severe stress and who resort to the defense mechanisms of repression, projection, and displacement may also have hallucinations. Such hallucinations appear to be part of a regressive phenomenon, in which the distinction between fantasy and reality is temporarily lost. In the pediatric intensive care unit, sensory deprivation may combine with intense anxiety, medication, and metabolic factors to produce hallucinations.

In children exposed to too much stimulation or abuse, hallucinations may accompany a broader constellation of dissociative symptoms. Acute grief reactions after the death of a parent may also give rise to hallucinations. Usually, these hallucinations are auditory and consist of admonitions and prohibitions attributed to the dead parent. In all these cases, the content of the hallucination may suggest the underlying psychological conflict.

In older children, external conflicts rarely, if ever, give rise to hallucinations. However, if the stress is massive and overwhelms the child, it can lead to profound regression. The circumstances in which this occurs include severe and sudden illness.

In some instances, severe cultural deprivation, together with a disturbed parent-child relationship, may result in hallucinations. The hallucinations in this situation are often localized, orderly, and related to reality, and they may consist of forbidding voices and overt wish fulfillments. Often, such hallucinations are consistent with the superstitions of the parents. The child may appear to be well organized in other ways. However, often there is evidence of an associated personality disturbance in the child and psychosis in the parent, findings suggesting at least the possibility of a genetic or organic component, as well as powerful sociocultural influences. For example, in some Pentecostal sects, a high value is placed on being possessed by the Spirit. Hysterically inclined children and youths may lend themselves to this

experience and may hear voices.

**Schizophrenia.** When hallucinations are more fragmented, incoherent, and bizarre in content, there is a greater likelihood that schizophrenia is present ( [Bender, 1954](#); [Volkmar et al., 1995](#)). Bodily complaints and paranoid delusions may be associated with the psychosis. The child is often frightened and secretive about the hallucinations, which are often bizarre and out of the child's control. Other signs of a thought disorder, including disordered, illogical thought processes and inappropriate affect, are usually present. A history of psychiatric disturbance in the family and maternal deprivation during infancy may be obtained. Sometimes the child shows delinquent behavior. Psychological tests, particularly projective tests, are indicated.

#### FANTASIES, FEELINGS, AND INFERRED CONFLICTS

The clinician may evaluate the following:

1. The child's spontaneous play is assessed.
2. The child's response is assessed when asked, "Do you have good dreams or bad dreams? Tell me one of your dreams."
3. The child can be asked, "If you could have three wishes—you can have anything you want, or can change the world or yourself in any way you want—what would you wish for?" ([Winkley, 1982](#)).
4. The child's productions in the game of squiggles are evaluated ( [Berger, 1980](#); [Winnicott, 1971](#)). The squiggles game is a way of establishing contact with the child. The clinician briefly explains to the child that he or she will make a squiggle with a pencil or crayon on a sheet of paper and the child will then turn it into something, and then it will be the child's turn to make a squiggle and the clinician's turn to make it into something. As they take turns, a theme may emerge in the sequence of squiggles and constructions. The clinician uses the drawings, the child's approach to the game, and what the child says about the squiggles to help both of them gain some insight into the child's mind.
5. The child's drawings of a person or whatever he or she wants to draw are assessed.

The child's drawings, particularly the child's drawings of a person, are useful in assessing the child's intelligence ( [Harris, 1963](#)) fantasies, and feelings ([Burns, 1982](#); [DiLeo, 1973](#); [Klepsch and Logie, 1982](#)), and various systems have been developed for assessing these aspects of children's drawings ( [Naglieri, 1988](#); [Naglieri et al., 1991](#)).

Because human beings are the objects of most intense interest to the child, the child's developing conceptual abilities may be readily seen in his or her drawings of a person. Thus, a general course or line of progression can be seen in children's drawings at different ages. As long ago as 1921, Burt was able to discern the following sequence ([Burt, 1921](#)):

1. Scribbling (2 to 3 years): The circular motor activity itself is pleasurable to the child as the child increasingly adapts his or her hand to the crayon and also tries to imitate the actions of others.
2. Single lines (4 years): The child soon draws a single line and tends to juxtapose parts.
3. Symbolic representation (5 to 6 years): The child can now draw a person consisting mostly of circles and ellipses representing head and body, "stick" lines representing arms and legs (and an indeterminate number of digits), and some curvilinear lines to represent eyes, mouth, and ears (hair arrangement usually distinguishes the sexes) ([Fig. 41.4](#)).



**Figure 41.4.** Drawing of a person by a 4-year-old child.

4. Descriptive drawing (7 to 10 years): The child now pays more attention to detail, and clothing details appear ( [Fig. 41.5](#)).



**Figure 41.5.** Drawing of a person by an 8-year-old child.

5. Visual realism (11 years onward): The child can draw in profile and attempts a realistic visual representation. The child may subsequently show a tendency to inhibition, with a preference for geometric designs, although later a true artistic talent may emerge ( [Fig. 41.5](#)).

A more precise assessment and measurement of the child's drawings can be obtained through formal psychological testing ( [Chapter 43](#)). The child's drawings may also reveal clinically useful information about his or her feelings and fantasies. However, the scientific reliability and validity of studies of this aspect of children's drawings are modest at best.

Clinically, it is often useful to invite a child to draw a person and then to make up a story about the drawing ("Pretend the boy or girl has just finished doing something, is doing something now, and is going to do something soon").

In observing the child while he or she is drawing, one may notice the following:

1. Motivation and self-esteem: The child may be eager to draw or may be self-effacing ("I'm no good at drawing" or "I can't draw very well").
2. Motor skill and activity: Observe how the child holds the crayon and how careful or slapdash is the child's approach. Does the child erase frequently, perseverate, or confine the drawing to a small corner of the paper? Neurologic impairment may reveal itself in the execution of the drawing.
3. Sex of the figure drawn: In general, children prefer to draw their own sex first and, initially, will identify whether it is a boy or girl "by the hair."
4. Special features of the child's drawing:
  - a. Heavy pressure may suggest tension, assertiveness, aggression, or organicity; light pressure may suggest indecisiveness, timidity, depression, and low self-esteem.
  - b. Large drawings may suggest aggressiveness or perhaps compensatory expansiveness to cover feelings of inadequacy; small drawings may suggest withdrawal, insecurity, or depression.



- c. Drawings placed low or on the edge may suggest insecurity or dependency.
- d. Very enlarged drawings of the head may suggest preoccupation with symptoms related to the head or possibly concern about intelligence.
- e. Omission of facial features may suggest evasiveness; large eyes may suggest anxiety or suspiciousness; small eyes may suggest guilt or a self-absorbed tendency. Sometimes the size of a part (e.g., ear, nose, or mouth) may be related to a possible handicap. Bared teeth may suggest aggression.

Numerous other inferences have been suggested (DiLeo, 1973; Hammer, 1980; Handler, 1985; Koppitz, 1968; Machover, 1949), but again, it must be noted that the scientific validity of these inferences is often low.

## AFFECTS

During the interview with the child, the clinician has the opportunity to observe directly the child's mood, range of affect, and the possible predominance of certain affects, such as anxiety, depression, apathy, guilt, or anger. In addition to observing the child's speech content, facial expression, and other nonverbal cues, the clinician will want to inquire directly about depression, suicidal thoughts or behaviors, and anxiety.

### Depressive Affect, Depression, and Suicidality

Depression is a particularly important affect that must not be overlooked. Depression affect is often accompanied by low self-esteem (e.g., "I can't do that," "I'm no good at drawing"), as well as fatigue, loss of interest and pleasure, guilt, difficulty in concentrating, and disturbances in sleep, appetite, and motor activity. These symptoms constitute a diagnosis of major depression.

The child may be asked: "Do you ever feel really down—sad, upset, or like crying?" "Do you ever feel like no one loves you or cares about you?" "Do you every get really down on yourself, blame yourself a lot, or feel you're no good at things or dumb or ugly?" "Are there ever times when it seems like nothing is any fun any more?" or "Do you ever feel like life sucks?" If the child answers any of these in the affirmative, one should ask "Have you ever felt things are so bad you've wished you were dead?" and "Have you ever done anything to hurt yourself?" In addition to low self-esteem and anhedonia, it is important to inquire about neurovegetative signs, such as changes in sleep or appetite, fatigue or loss of energy, psychomotor retardation, and difficulty concentration.

Suicidal risk may be part of a major depressive disorder and should be specifically investigated. When there is overt suicidal ideation or suicidal behavior, one assesses the events and circumstances leading up to the episode, whether the act was planned or impulsive, lethal in the child's mind, an act of desperation or depression, and whether the child's reaction to his or her suicide attempt was one of continuing desperation and depression or abhorrence and fright. If a child has made a suicide attempt or other nonsuicidal form of self-injury or ingestion, one may, for example, ask the questions in Table 41.8. A more detailed set of questions to ask in the evaluation of suicidal risk in children (Table 41.9) is provided by Pfeffer (1986) (Chapter 65).

1. Present episode:
a. Tell me what happened. And what happened next?
b. Motivation:
1. Were you feeling sad or depressed and frustrated?
2. Did someone approach you and talk to you?
3. Did you have something you'd change afterwards?
4. Did you have something you'd like to do now?
5. Did you have suicidal thoughts or thoughts of suicide?
6. Did you ever do anything to hurt yourself in the past?
7. Have you ever done anything to hurt yourself?
8. What was the reason for it?
9. What did you do?
10. What were the reasons for it?
11. Did anything change for you then, or did things just stay the same?
2. Recurrent episodes:
a. Do you know of anyone else, either in the past or more recently, who has thought about or tried to kill himself or herself?
b. What did you think or feel when you heard about it?
c. Did it make you sad?
3. Concomitants:
a. When you (e.g., had the idea), did you think you would die?
b. Did you hope someone would find you?
4. Symptoms of depression:
a. How is your appetite?
b. Do you have any trouble going to sleep or sleeping through the night?
c. Do you feel tired all of the time?
d. Do you feel sad or down all the time?
e. Do you feel like you're not doing as well as you used to?
f. Do you feel like you're not doing as well as you used to?
g. Have anyone close to you left you or died?
h. How do you feel about your school?
i. How do you feel about your parents?
j. How do you feel about your friends?
k. Do you worry that your parents will be disappointed with you?

**Table 41.8. Questions to Ask About Suicidality**

1. Present episode:
a. Tell me what happened. And what happened next?
b. Motivation:
1. Were you feeling sad or depressed and frustrated?
2. Did someone approach you and talk to you?
3. Did you have something you'd change afterwards?
4. Did you have something you'd like to do now?
5. Did you have suicidal thoughts or thoughts of suicide?
6. Did you ever do anything to hurt yourself in the past?
7. Have you ever done anything to hurt yourself?
8. What was the reason for it?
9. What did you do?
10. What were the reasons for it?
11. Did anything change for you then, or did things just stay the same?
2. Recurrent episodes:
a. Do you know of anyone else, either in the past or more recently, who has thought about or tried to kill himself or herself?
b. What did you think or feel when you heard about it?
c. Did it make you sad?
3. Concomitants:
a. When you (e.g., had the idea), did you think you would die?
b. Did you hope someone would find you?
4. Symptoms of depression:
a. How is your appetite?
b. Do you have any trouble going to sleep or sleeping through the night?
c. Do you feel tired all of the time?
d. Do you feel sad or down all the time?
e. Do you feel like you're not doing as well as you used to?
f. Do you feel like you're not doing as well as you used to?
g. Have anyone close to you left you or died?
h. How do you feel about your school?
i. How do you feel about your parents?
j. How do you feel about your friends?
k. Do you worry that your parents will be disappointed with you?

**Table 41.9. Questions to Ask in the Evaluation of Suicidal Risk in Childrenk values) of the Instruments Considered in This Chapter (Where Available)**

## Anxiety

It is also important to inquire directly about *anxiety*, for example: "Are there any things that get you really nervous or scared, to the point of making you really uncomfortable or keeping you from doing things, such as play dates or sleepovers at friends' houses?"

## RELATIONSHIPS

The clinician may explore the following areas:

1. With the family: The clinician may ask the child who is in the family and which family members the child gets along with best and worst. One can also ask the child to describe the different members of their household, how the child gets along with each of them, and whether there are any "rough spots" in how they get along.
2. With peers: The clinician may ask the child who his or her friends are and whom he or she likes the most and the least. Asking the child what he or she likes about various friends gives some picture of how the child sees others and what aspects of people are most salient to the child. Again, having asked first about whom the child gets along best with, the clinician turns to ask, "Are there any kids you don't get along with?" "Do you ever have any trouble with how you and other kids get along?" and if so, to ask the child's impression of how those difficulties come about. For a child who feels ostracized or unpopular, one can inquire whether there are any things the child is teased about.
3. With teachers: The clinician may ask the child which teachers he or she likes and dislikes and why.

## SEXUAL AND AGGRESSIVE CONCERNS AND BEHAVIORS

1. Sexual: Is the child seductive or inappropriate toward other children or adults? Does the child engage in overt or covert masturbation or other autoerotic behaviors? Does the child have romantic interests, and, if so, what are their nature and object? Does the child have concerns over gender identity or sexual orientation? Is the child sexually active, and, if so, with whom?
2. Aggressive: Does the child have a problem with his or her temper? If so, who are the objects of the child's anger and what provokes it? How does the child express his or her anger—through appropriate verbal means, through verbal abuse, or through physical assaultiveness? Is the child provocative, irritable,

destructive, or violent? For example, does the child have a history of weapon carrying or use or of engaging in serious physical fights? Is there a history of cruelty to animals, fire setting, vandalism, or bullying? What is the child's attitude toward his or her expressions of aggression (e.g., contrition, lack of remorse)? Is the child overly inhibited in expressing aggression?

### DEFENSE ORGANIZATION

What are the child's modes of coping with conflicts or problematic urges (e.g., finding alternative or socially permissible means of satisfying them)? What coping strategies does the child use when anxious, frustrated, or angered? Does the child become phobic, avoidant, or overly inhibited? Does the child resort to counterphobic measures, provoking or deliberately risking what he or she fears? Does the child become overly perfectionistic or resort to denial (saying that there are no problems?)

### POSITIVE ATTRIBUTES

It is also important to make note of the child's positive attributes. For example, does the child have special talents or interests of which he or she is able to make adaptive use? The clinician will want to make note, when present, of the following: physical robustness and attractiveness; intelligence; emotional and cognitive flexibility, good affective coping skills and stability; psychological mindedness, amiability, cooperativeness, and social fluency; stable, supportive, and appropriate relationships with peers, family, and teachers; durable and realistic self-esteem; positive work habits and academic achievement; and reasonable and useful ethical values.

### FORMULATION

The complete psychiatric assessment of the child or adolescent should enable the clinician to formulate a useful description of how and why the particular child or adolescent presents in the way he or she does ([Shapiro, 1989](#)). Thus, the *formulation* is, in essence, an integrated summary of the unique way in which organic factors, environmental stresses, and inner conflicts have interacted over time for a particular child or adolescent in a particular family. Through the formulation, one obtains a full, multidimensional picture of the child or adolescent that goes far beyond the diagnostic label and provides a sound basis for devising an appropriate comprehensive treatment plan. The formulation attempts to spell out the internal and external factors that have brought about and maintained the child's difficulties, as well as protective and ameliorative factors that may have mitigated these difficulties and may serve as potential avenues for remediation. The clinician's focus on the child and family's strengths and adaptive capacities, as well as their areas of difficulty, helps to identify resources in the child, family, and school that may facilitate any therapeutic measures to be undertaken.

### CONCLUDING THE EVALUATION

In the course of the evaluation of the child, the clinician establishes a trusting relationship with the child and the parents. Consequently, the clinician considers the feelings of the child and the parents during the conclusion phase of the evaluation. For example, the clinician informs the child ahead of time as to when the last session will take place. Then, during the final interview, he or she ensures that the child knows what will take place next by asking the child or adolescent: "Are there any things you would particularly like me to tell your parents?" "Are there any things you don't want me to say to your parents?" "This is what I plan to say to your parents. How does that sound to you?" "Do you have any questions?" Sometimes a child will express feelings about the ending. These feelings should be recognized, acknowledged, and dealt with sympathetically and realistically.

At the final review meeting with the parents, the clinician may start by asking how the child reacted to coming for his or her interviews. Sometimes information comes to light that may help the clinician in assessing the child's capacity to form a relationship and engage in psychotherapy. The clinician can then give the parents an account of the child's strengths. Every child has some strengths (e.g., the child may be attractive, intelligent, delightful to be with, well coordinated, able to think clearly), and it is important that the clinician tell the parents about such qualities.

Next, the clinician talks with the parents about his or her assessment of the child's difficulties. The assessment should be discussed in clear language and documented with vignettes from the clinical interviews or from any of the special tests that help to illustrate and clarify the nature of the difficulty. If psychological tests have been performed, the psychologist may wish to participate in the review meeting with the parents.

The parents should be given every opportunity to ask questions. Their reactions should be recognized and understood; the parents need the support of the clinician. They need an explanation of the possible causes of their child's condition. They should also be reassured about all the good things they have done to help the child. Finally, treatment options and recommendations should be discussed with the parents. The parents should not be rushed, and they should be invited to telephone or return if they so wish.

Documentation of the basis for the findings is particularly important when a written report has to be submitted, for example, to a court for evidence in a custody dispute ([Lewis, 1974b](#)). When appropriate, and with the parents' permission, the clinician should also send a report to the referring person. The limits of confidentiality, again, must be clearly understood by the child, the parents, and the clinician, and the clinician must exercise special care to safeguard this confidentiality.

Finally, the psychiatric evaluation of the child and the child's family goes beyond the diagnosis. Each child and each family member has his or her own private experience of life. In a good psychiatric evaluation, the clinician is privileged to enter that private experience momentarily and to empathize with that person's feelings, hopes, and fears. One tries to capture this aspect of a person's life in the descriptive diagnostic formulation that follows the formal diagnosis.

### STRUCTURED INTERVIEWS, QUESTIONNAIRES, AND RATING SCALES

*Structured and semistructured psychiatric interviews*, questionnaires, and rating scales for children and parents have been devised to improve the reliability and validity of information and observations regarding psychiatric diagnosis. Currently, the greatest use for these instruments is in research. However, many of these instruments have been used successfully for screening purposes and as diagnostic aids in the clinical situation. They have also proven useful in providing a quantitative metric for measuring the impact of an intervention on a given set of symptoms. In turn, their use has sharpened clinical inquiry in such areas as the assessment of mood disorders, cognitive functions, attention, and thinking processes.

Structured interviews have the advantage of focusing on important areas and therefore do not leave to chance observations of important items that may not come to light in an unstructured interview. Structured interviews provide comprehensive and systematically obtained and rated data with respect to a specific set of diagnoses; they thus have the advantage that the data elicited by reliably trained interviewers can be compared across subjects, as, for example, in gathering epidemiologic data. One further advantage is the relatively short training period required to be able to administer a structured interview.

Structured interviews are more or less rigid, however, and do not admit much in the way of clinical judgment. Sometimes the conclusions do not correspond to the complexity of the clinical diagnosis. Furthermore, structured interviews pay little attention to the personal, historical, or adaptive context of the child's difficulties and hence are of limited utility in planning treatment interventions. Structured interviews for preschool children are generally unreliable. Questionnaires that have long lists of questions—for example, the Diagnostic Interview for Children and Adolescents (DICA), which has 207 items—may exhaust a child. The clustering of items around symptoms may also be a limiting factor as far as rapport is concerned, because the focus on negative behavior may be too relentless for too long. Often, the rote manner in which the questions are posed conveys little sense to the child that the interviewer has any empathic interest in the child's responses. The lack of normative data, in some instances, is a handicap. Few structured interviews focus specifically on father-child interactions.

On balance, structured psychiatric interviews for children and adolescents supply a useful safety floor and a reliable screening mechanism. In general, children report better about feelings, whereas parents report better about behavior.

The number of schedules and scales now available is formidable and increasing steadily. [Chapter 42](#) and the relevant chapters on syndromes have more detailed descriptions and indications for the use of standardized instruments.

### STANDARDIZED TESTS



Standardized tests, such as developmental, psychological, neurologic, educational, linguistic, and biological studies, are described in Sections V and VI.

## ACKNOWLEDGMENTS

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## Appendix A/Child Psychiatry Emergency Consultation Mental Status Examination Checklist

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Patient name \_\_\_\_\_ Age \_\_\_\_\_ Sex \_\_\_\_\_ Examination date \_\_\_\_\_

Behavior	Yes	No	No info	Comments
Alert	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Cooperative	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Agitated	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Subdued	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Subtle Manner	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
<b>Speech:</b>				
Normal	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Fluently	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Mumbled	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
None	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
<b>Mood (Self Report):</b>				
Sad	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Sober	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Happy	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Angry	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Nervous	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
<b>Other (Observations):</b>				
Complaint	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Reflexes	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Response	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Speech	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Insight	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Intake	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Intake	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Intake	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
<b>Neurovegetative Symptoms:</b>				
Poor Sleep	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Poor Appetite	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Substantive loss of interest	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Early Morning Waking	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Decreased Energy	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Impaired Concentration	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Psychomotor Agitation	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Psychomotor Slowing	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Recurrent Thoughts of Death	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Thoughts of Suicide	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____

**Substance Use:**

Alcohol	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Alcohol	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Marijuana	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Heroin	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Heroin	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Heroin	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Heroin	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Other	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Other	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Specify: _____				
<b>Thought Processes:</b>				
Concentrated	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Loosened Association	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Persecuted	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Obsessed	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Auditory Hallucinations	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Visual Hallucinations	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Idea of Reference	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Grandiosity	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
<b>Insights:</b>				
Preserving Social Behavior	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Preserving Social Behavior	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Preserving Social Behavior	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____
Preserving Social Behavior	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	_____

Note: If "yes" in preceding suitability questions, then complete the following:

1. Type of Event: \_\_\_\_\_

2. Method of Abuse: \_\_\_\_\_

3. Whether to stop or get help to stop: \_\_\_\_\_

4. Abuse Rating: \_\_\_\_\_

5. Abuse Rating: \_\_\_\_\_

6. Abuse Rating: \_\_\_\_\_

7. Abuse Rating: \_\_\_\_\_

8. Abuse Rating: \_\_\_\_\_

9. Abuse Rating: \_\_\_\_\_

10. Abuse Rating: \_\_\_\_\_

11. Abuse Rating: \_\_\_\_\_

12. Abuse Rating: \_\_\_\_\_

13. Abuse Rating: \_\_\_\_\_

14. Abuse Rating: \_\_\_\_\_

15. Abuse Rating: \_\_\_\_\_

16. Abuse Rating: \_\_\_\_\_

17. Abuse Rating: \_\_\_\_\_

18. Abuse Rating: \_\_\_\_\_

19. Abuse Rating: \_\_\_\_\_

20. Abuse Rating: \_\_\_\_\_

21. Abuse Rating: \_\_\_\_\_

22. Abuse Rating: \_\_\_\_\_

23. Abuse Rating: \_\_\_\_\_

24. Abuse Rating: \_\_\_\_\_

25. Abuse Rating: \_\_\_\_\_

26. Abuse Rating: \_\_\_\_\_

27. Abuse Rating: \_\_\_\_\_

28. Abuse Rating: \_\_\_\_\_

29. Abuse Rating: \_\_\_\_\_

30. Abuse Rating: \_\_\_\_\_

31. Abuse Rating: \_\_\_\_\_

32. Abuse Rating: \_\_\_\_\_

33. Abuse Rating: \_\_\_\_\_

34. Abuse Rating: \_\_\_\_\_

35. Abuse Rating: \_\_\_\_\_

36. Abuse Rating: \_\_\_\_\_

37. Abuse Rating: \_\_\_\_\_

38. Abuse Rating: \_\_\_\_\_

39. Abuse Rating: \_\_\_\_\_

40. Abuse Rating: \_\_\_\_\_

41. Abuse Rating: \_\_\_\_\_

42. Abuse Rating: \_\_\_\_\_

43. Abuse Rating: \_\_\_\_\_

44. Abuse Rating: \_\_\_\_\_

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100. Abuse Rating: \_\_\_\_\_

19. Patient's feelings about episode: \_\_\_\_\_

20. Patient's feelings about episode: \_\_\_\_\_

21. Patient's feelings about episode: \_\_\_\_\_

22. Patient's feelings about episode: \_\_\_\_\_

Clinician Signature \_\_\_\_\_ Date \_\_\_\_\_

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## 42 STRUCTURED INTERVIEWING

Adrian Angold, M.R.C.Psych., and E. Jane Costello, Ph.D.

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### BRIEF HISTORICAL INTRODUCTION

Interviews are necessary tools for all forms of clinical medical diagnosis, and they have a singularly prominent position in psychiatry because of the lack of other “tests” for psychiatric disorders. All structured interviews used in psychiatry have their roots in the phenomenologic clinical interview, although different interviews take rather different routes in the standardization of the collection of phenomenologic data relevant to diagnosis.

#### Limitations of Unstructured Diagnostic Interviews

It soon became clear that clinical training was sufficiently varied that colleagues of the same discipline, working in the same establishment, were often unable to agree about an individual patient's diagnosis, even when they were presented with exactly the same information ( [Cantwell, 1988](#); [Gould et al., 1988](#); [Prendergast et al., 1988](#); [Remschmidt, 1988](#)). An apparent difference in rates of schizophrenia between New York and London proved to be almost entirely the result of differences in diagnostic criteria applied to observed phenomena ( [Cooper et al., 1972](#)). Observations such as these motivated the development of the formalized sets of diagnostic criteria familiar to us today from the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) ( [American Psychiatric Association, 1994](#)) and the 10th edition of the *International Classification of Diseases* (ICD-10) ( [World Health Organization, 1992](#)).

The literature on medical decision making had already shown that clinicians suffer from certain detrimental information collection biases: (a) they tend to come to diagnostic determinations before they have collected all the relevant information, (b) they tend then to focus on collecting information to *confirm* that diagnosis (“confirmatory bias”), (c) they tend to ignore disconfirmatory information, (d) they combine information in idiosyncratic ways, and (e) they tend to make judgments based on the most readily available cognitive patterns: the “availability heuristic.” Further problems arise because of a tendency to see correlations where none exist (“illusory correlation”) and, contrariwise, to miss real correlations ( [Achenbach, 1985](#)). Added to all these problems is that, even today, standard diagnostic manuals do not provide very detailed descriptions of how to assess psychopathology at the symptom level. All the criteria for oppositional defiant disorder, for instance, begin with the word “often,” but how often is often? There is a great deal of room for clinicians to adopt very different decision rules about when to regard such symptoms as being present.

In the presence of all these difficulties, it became apparent that means were required to standardize the collection, quantification, and combination of diagnostic information. As a result, all structured interviews aim to (a) structure information coverage so all interviewers will have collected all relevant information (both confirmatory and disconfirmatory) from all subjects, (b) define the ways in which relevant information is to be collected, and (c) structure the process by which relevant confirmatory and disconfirmatory information is combined to produce a final diagnosis.

#### Early Structured Diagnostic Interviews

In the early days of structured interviews, it was supposed that *clinicians* would be using them because it was believed that only they had the necessary training and experience to be able to decide about the presence or absence of symptoms, even when quite detailed definitions were provided. The interview schedule served as a tool to guide the clinician interviewer in determining whether symptoms were present, but the interviewer made the decisions, on the basis of information provided by the child or adult. Interviews of this sort, like the Present State Examination (PSE) ( [Wing, 1974](#)) and the Reynard ( [Guze et al., 1969](#)) for adults and the Isle of Wight interview for children ( [Graham and Rutter, 1968](#), [Rutter and Graham, 1968](#)), were the first to be developed because they sprang naturally from clinical practice. They were called *semistructured* because the interviewer was allowed latitude in the specific form of the questions used.

Although the PSE and Isle of Wight interviews were used extensively in community surveys, it was clear that the use of clinician interviewers created both logistic and budgetary problems. Very large-scale epidemiologic studies, such as the Epidemiologic Catchment Area studies ( [Regier et al., 1984](#)), mandated the use of nonclinician (“lay”) interviewers. Some investigators believed that such interviewers would be incapable of making the judgments about symptoms, so, following methods used by political and marketing surveys, interviews were developed that required only that the interviewer ask a set of fixed questions in a preset order and collect the simple answers to those questions. In such interviews, the *questions* put to the subject are structured, and the interviewer makes no decisions about the presence of symptoms. Hence they came to be called “highly” or “fully” structured interviews. The Diagnostic Interview Schedule was the paradigmatic example of this sort of interview in adult psychiatry ( [Robins et al., 1979](#)), whereas the original Diagnostic Interview Schedule for Children and Adolescents (DICA) was the first child-oriented example ( [Herjanic and Campbell, 1977](#); [Herjanic et al., 1975](#)).

## Emergence of the Diagnostic Interview with the Child and the Need for Multiple Informants

Until the late 1960s, interviews and questionnaires directed to a parent or teacher about a child's behavior and *observation* of the child's behavior were the predominant methods of assessment in child and adolescent psychiatry. Verbal information from the child was typically regarded as being only supplemental or as material for psychodynamic interpretation ([Lapouse, 1966](#)). More attention was paid to playing with the child than to the collection of information through direct questioning. In 1968, a key transitional article reported on the reliability and validity of the Isle of Wight interview with the child ([Rutter and Graham, 1968](#)). The behavior of the child in a face-to-face interview was examined directly, but little was made of the factual content of the child's reports. In 1975, Herjanic and her colleagues asked whether children are "reliable reporters" of factual information and presented evidence that they are ([Herjanic et al., 1975](#)). Since then, a great deal of work has confirmed the importance of children's self-reports as a source of factual information, with the result that fact-finding (as opposed to interpretative) interviews with both parents and children are now regarded as being of equal weight in the diagnostic process, at least from late childhood. The one exception is in the evaluation of symptoms of attention deficit hyperactivity disorder, in which child reports have been found to be of little help ([Lahey, 1990](#); [Loeber et al., 1991](#)). However, the recent growth of interest in attention deficit hyperactivity disorder in adolescence and adulthood has led to the development of new measures in this area ([Conners, 1977](#)).

## Disagreement Among Informants and Its Implications for Combining Information from Multiple Informants

Until the 1980s, agreement between child and parent reports of symptoms was widely regarded as being a test of the *validity* of *child* reports ([Herjanic et al., 1975](#); [Rutter and Graham, 1968](#)). However, it soon became apparent that only low levels of agreement among informants (correlation coefficients around 0.3 for agreement among children, parents, and teachers) could be expected ([Reich et al., 1982](#); [Stanger and Lewis, 1993](#)). It is now considered that low levels of agreement among different informants about the child's clinical state are to be expected and do not invalidate the reports of any of them. Rather, each key informant presents a particular view of the child's problems. Indeed, it is precisely because agreement among informants is low that multiple informants are needed. Were agreement very high, taking the history from more than one informant would be redundant.

The problem is that disagreement among informants means that one has to decide how to weight the information from each informant in arriving at a diagnosis. Because it is uncommon for informants to invent fictitious symptoms, the simple rule of regarding a symptom as being present if any informant reports it usually suffices. When symptoms are combined to make diagnoses, the usual procedure is to "ignore" the source and to add up all positive symptoms from any source. Thus, a diagnosis of a major depressive episode (which requires the presence of at least five symptoms) may be made on the basis of three relevant symptoms reported by the child (say, depressed mood, anhedonia, and excessive guilt), with two other relevant symptoms (perhaps sleep and appetite disturbances) reported by the parent. Although some interview developers recommend *reconciliation*: discussions involving the interviewer, the parent, and the child to clear up discrepancies among their reports, such a discussion is problematic. Reconciliation requires that one informant must modify his or her story, and that means admitting being wrong or at least uninformed. The knowledge that such a discussion will occur could cause informants (e.g., drug-using adolescents) to withhold important information that they did not wish other informants (e.g., their parents) to hear about. Finally, in most research applications, one wishes to assure informants that what they say will not be revealed to anyone else, and this rules out a reconciliation interview.

The remainder of this chapter is concerned with the description of key points relating to "general" psychiatric diagnostic interviews, that is, those that cover a broad range of the common disorders of childhood and adolescence. Some interviews and observational systems exist for more specialized tasks, for instance, the Diagnostic Interview and the Autism Diagnostic Observation Schedule ([Lord et al., 1989, 1994](#)), but such instruments are not considered further here.

## TYPOLOGY OF INTERVIEWS

### Respondent-Based Interviews

As we have already seen, a distinction between semistructured and highly structured interviews has found its way into the description and discussion of diagnostic interviewing techniques. However, these terms are not very helpful for two reasons. First, they imply that the key difference between different types of interview concerns the *amount* of structure they impose. The problem is that the real issue is not one of amount of structure, but rather who makes the final decision on whether a symptom is present. In interviews in which the questions are absolutely prespecified, the respondent makes the final decision (typically by answering "yes" or "no" to each question). The interviewer makes no such decisions, but merely reads the questions. Because the decisions about the presence or absence of psychopathology lie with the respondent in such interviews, we refer to these interviews as *respondent based*. The Diagnostic Interview Schedule for Children (DISC) ([Shaffer et al., 2000](#)), the computer-assisted version of the DICA ([Reich, 2000](#)), and the Dominic-R (Valla et al., 2000) are three representatives of this approach.

### Interviewer-Based Interviews and Glossary-Based Interviews

We call interviews that require the interviewer to make an informed decision based on what the respondent says *interviewer based*. The interviewer is expected to question until he or she can decide whether a symptom meeting the definitions provided by the interview (or known to them from their training) is present. This group of interviews includes the Anxiety Disorders Interview Schedule (ADIS) ([Silverman and Rabian, 1995](#)), the Child and Adolescent Psychiatric Assessment (CAPA) ([Angold and Costello, 2000](#)), the Child Assessment Schedule (CAS) ([Hodges, 1993](#); [Hodges et al., 1982b](#)), the paper and pencil (not the computerized) versions of the DICA ([Reich, 2000](#)) and its close relative the Missouri Assessment of Genetics Interview for Children (MAGIC), the Interview Schedule for Children and Adolescents (ISCA) ([Sherrill and Kovacs, 2000](#)), the various versions of the Schedule For Affective Disorders and Schizophrenia for School-Age Children (Kiddie-SADS or K-SADS) ([Ambrosini, 2000](#)), and the Pictorial Instrument for Children and Adolescents (PICA-III-R) ([Ernst et al., 2000](#)). Three of these interviewer-based interviews (the K-SADS-P IVR, the DICA, and the CAPA) provide extensive sets of definitions of symptoms or detailed guidance on the conduct of the interview, and we call these *glossary based*. Such glossaries are particularly important when an interviewer-based interview is to be used by nonclinician interviewers because they provide detailed guidance about what the interviewer is supposed to be looking for in making symptom ratings. Nonclinician interviewers have been shown to be able to make such "clinical" judgments with high reliability when they have received adequate training with such glossaries ([Angold and Costello, 1995](#)).

The distinction between interviewer- and respondent-based interviews is not hard and fast in actual practice, because there is considerable cross-fertilization between these approaches. For instance, the CAPA, which has its roots in the interviewer-based tradition, includes a subset of questions that are to be asked verbatim of all subjects, as in a respondent-based interview, but then it allows further questioning for clarification. Conversely, the DICA, which had previously been a respondent-based interview, now requires interviewers to question much more flexibly and is now an interviewer-based instrument ([Reich, 2000](#)). Although the distinction between interviewer- and respondent-based interviews provides a useful rough-and-ready typology, it is really better to consider interviews as lying at various locations along three dimensions: (a) degree of specification of questions, (b) degree of definition of symptom concepts, and (c) degree of flexibility in questioning permitted to the interviewers. Interviews that provide extensive definitions and require interviewers to make judgments lie in the interviewer-based region of that three-dimensional space, whereas those that specify every question and allow no interviewer deviation from those questions lie in the respondent-based region.

### Pictorial Interviews

More recently, respondent-based child self-report interviews that add *pictorial* cues have been added as assessment tools. The most highly developed pictorial interview at this time is the Dominic-R ([Valla et al., 2000](#)), which is intended for use with 6- to 11-year-old children. Pictures representing psychopathology relevant to seven diagnoses are shown to the child, and questions about whether each symptom is present are read at the same time. Because no frequency, duration, or onset data are collected, it is not yet clear how such information should be combined with diagnostic information from other sources. This is, however, a general problem for interviews with younger children, because before the age of 8 or 9 years, they simply cannot provide all the frequency, dating, and timing information that full diagnostic interviews require. Although diagnostic test-retest reliabilities cannot be reported for the Dominic-R, its item reliabilities are respectable in comparison with those reported from studies of older children with other interviews.

The PICA-III-R, for children 6 to 16 years of age, adopts a somewhat similar approach, but the questions to be asked with the pictures are more loosely specified, and the test is intended to be used by clinicians. It covers a broader range of diagnoses than the Dominic-R, but no test-retest reliability data are yet available ([Ernst et al., 2000](#)).



## Screened Interviews

The Children's Interview for Psychiatric Symptoms (ChIPS) ([Weller et al., 2000](#)) was designed as a *screening* tool covering 20 DSM-IV Axis 1 disorders. "Cardinal questions" concerning symptoms most often seen in children with a particular disorder are asked at the beginning of each section. If the answers to these screening questions are in the negative, then the rest of that section is skipped. No test-retest reliability data are yet available for the ChIPS. A similarly "screened" version of the CAPA is also available, but in practice it has been found to save only about 10 minutes of interview time, so the loss of information resulting from not asking about all symptoms may not really be worth the time saving.

## BRIEF INTRODUCTION TO THE INTERVIEWS

Here we present a brief introduction to each of the diagnostic interviews, with a focus on their characteristic *response formats*.

### Schedule for Affective Disorders and Schizophrenia for School-Age Children

The K-SADS "family" of interviews consists of a group of very diverse assessments. Indeed, the only features that all the current versions of the K-SADS share in common are the name, the ability to make DSM-IV diagnoses, and the fact that all were designed to be administered *by clinicians*. The original version of the K-SADS (K-SADS-P) ([Puig-Antich and Chambers, 1978](#)) was a downward extension of the adult Schedule for Affective Disorders and Schizophrenia (SADS) and focused on the Research Diagnostic Criteria ([Spitzer et al., 1978](#)). The "P" in its title stands for *present* (not parent). It was designed for use with children aged 6 to 17 years, but it covered only a relatively limited range of symptoms and diagnoses. It was revised to cover DSM-III-R ([Ambrosini et al., 1990](#)) and DSM-IV.

#### K-SADS-P IVR

The version of the K-SADS-P most recently developed by Ambrosini and colleagues is called the K-SADS-P IV-R. This version is closest conceptually to the original K-SADS-P in including quite detailed definitions of severity codings for each symptom. The modal form for these symptom codings is a six-point scale, involving judgments about various combinations of intensity, duration, frequency, environmental responsiveness, psychosocial impairments, and observed behavior.

#### K-SADS-E

The K-SADS-E (E for epidemiologic) collects ratings of the present episode of any disorder *and* the *worst* past episode ([Orvaschel et al., 1982](#)). This interview is not even remotely similar in format to the K-SADS-P, because it rates only the presence or absence of symptoms, rather than employing the carefully defined severity codings of the K-SADS-P. The latest edition is DSM-IV compatible, and it allows the current *episode* (not individual symptoms) to be rated as mild, moderate, or severe.

#### K-SADS-PL

A group in Pittsburgh developed the K-SADS-PL (present and lifetime), as a sort of cross between the K-SADS-P and the K-SADS-E ([Kaufman et al., 1997](#); [Shanee et al., 1997](#)). Symptom ratings are reduced to three-point scales (typically "not at all," "subthreshold," "threshold"), and fairly minimal anchoring definitions of each point are provided. An initial 82-item screen interview, which allows skipping of substantial symptom areas, is also available.

#### WASH-U-K-SADS

Rather brief definitions of symptoms are given, and the level of severity is coded, but the severity codings are idiosyncratic and bear little relation to those in other versions of the K-SADS.

#### COLUMBIA K-SADS

The symptom definitions provided are often simply restatements of the DSM-IV criteria. Sometimes, particularly in relation to depression, a little more guidance is given. Symptom severity is typically rated on what appears to be a six-point scale like the K-SADS-P and K-SADS-P IVR, but closer inspection reveals that two of the points are usually defined only as being "intermediate" between two other points. Thus, only four points (one being symptom absence) are really defined. Although K-SADS interviews were developed for use by clinicians, some are also used with lay interviewers ([Hodges et al., 1987](#); [Lewinsohn et al., 1993](#)).

The K-SADS family differs from most interviews (but not the ISCA) in directing that the parent should be interviewed first and then the child should be seen by the same interviewer, who is then expected to resolve any discrepancies between the child's reports and those of the parent. The interviewer then completes a record representing his or her summation of the two interviews. This procedure is highly dependent on clinical judgment, and it means that the process of combining the information is not structured. It also seems likely to bias the results of the interview in favor of the parental reports ([Angold et al., 1987](#)), and some workers instead score the interviews with the parent and the child separately ([Weissman et al., 1987](#)).

## Child and Adolescent Psychiatric Assessment

The CAPA is one of an integrated group of instruments developed to assess a variety of risk factors for, manifestations of, and outcomes of child and adolescent psychiatric disorders. In addition to the usual symptom and impairment assessments, it also includes extensive ratings of the family environment and relationships, family psychosocial problems, and life events (including traumatic events and physical and sexual abuse). A separate module called the Child and Adolescent Impact Assessment (CAIA) ([Angold et al., 1998](#); [Messer et al., 1996](#)) measures the impact of the child's problems on the family, whereas the Child and Adolescent Services Assessment (CASA) ([Ascher et al., 1996](#); [Farmer et al., 1994](#)) covers service use for mental health problems in multiple sectors and settings. Psychosocial impairment in 17 domains of functioning is measured at both the syndromic level and overall. In the interview with the child, 62 items reflecting the child's observed behavior are also coded.

To facilitate completion of the interview by nonclinicians, the CAPA provides a more molecular approach to symptom codings. Extensive symptom definitions are given in a glossary and on the schedule, and rules are specified to allow nonclinicians to make separate codings of the intensity, frequency, duration, date of onset of symptoms, and psychosocial impairment resulting from them. The CAPA emphasizes obtaining descriptions and examples of possible pathologic features to ensure that codings are not based on the informant's misunderstanding of what was being asked ([Angold and Costello, 1995, 2000](#); [Angold et al., 1995](#); [Costello et al., 1998](#)).

A version of the CAPA has been developed for use with *young adults* (the Young Adult Psychiatric Assessment; YAPA), and a substantially modified version is now available for use with the *parents of preschool children* (the Preschool Age Psychiatric Assessment; PAPA). The latter includes assessment of certain areas not included in the CAPA. In addition, a version with empirically derived screen items is available, and this allows sections to be skipped if screen symptoms are absent. A streamlined version for collecting data for twin studies has also been developed ([Simonoff et al., 1997](#)).

## Diagnostic Interview for Children and Adolescents

The DICA started out as a respondent-based interview in the 1970s ([Herjanic and Campbell, 1977](#); [Herjanic and Reich, 1982a](#); [Herjanic and Reich, 1982b](#); [Herjanic et al., 1975](#); [Reich and Earls, 1987](#); [Reich et al., 1982](#); [Welner et al., 1987](#)). Since then, it has been progressively modified, so its *paper and pencil version* is now an interviewer-based interview ([de la Osa et al., 1997](#); [Ezpeleta et al., 1995](#); [Ezpeleta et al., 1997](#); [Reich, 2000](#)). However, there is also a *computer-based version* of the DICA that remains fully respondent based ([Reich et al., 1995](#)). In addition, the group responsible for the development of the DICA has produced a modification, the MAGIC. The major difference between the DICA and the MAGIC is that the MAGIC has a specifications manual, which includes a great deal of guidance on how to elicit key features of symptoms and a variety of clarifications of coding instructions ([Reich, 2000](#)).

The DICA and the MAGIC provide alternate versions for self-reports from children 6 (or 7) years old to 12 (and 13) to 17 years old, with differently worded questions. The DICA has also been used with even younger children, but special training is required for its administration in children younger than 6 or 7 years old, and the

instructions to interviewers essentially tell them to ignore the usual questioning format laid out in the schedule and to use their own questions.

Symptoms are typically coded on a three-point scale (no, sometimes/somewhat, yes) for items about emotional symptoms, and a two-point scale (no, yes) for disruptive behaviors, with additional information of frequency sometimes added. Impairment is measured at the syndrome level by three items asking about symptoms that make it hard to "get along" with the family, friends, and at school, rated on four-point scales (not at all, not too much, somewhat, quite a bit).

### Child Assessment Schedule

The CAS is organized around thematic topics and provides ratings of many items that are not required if all one wants to do is make ratings of the DSM criteria for disorders ([Hodges and Saunders, 1989](#); [Hodges et al., 1982a](#); [Hodges et al., 1982b](#); [Hodges et al., 1987](#); [Hodges et al., 1989](#); [Hodges et al., 1990](#)). The CAS now exists in child (7 to 12 years), adolescent, and parent report versions. Although originally developed for use by experienced clinicians ([Hodges et al., 1982a](#)), the CAS has now been used by lay interviewers in several studies. Symptoms are scored on a four-point scale (yes, no, ambiguous, not scored). This is followed by questioning about the onset and duration of positive symptoms. The items to be coded are defined in brief sentences that outline each symptom concept. The interviewer is expected to make a judgment about the coding based on the answer to the questions on the schedule (plus any additional questions that may be thought necessary).

The CAS also has a 56-item section for recording observations of the child's behavior during the interview. Diagnoses can be generated manually by interviewers using algorithms provided by the developer of the interview, but computerized scoring is recommended. The algorithms also generate symptom scales pertaining to certain areas of psychopathology.

Psychosocial impairment is measured using a separate instrument, the Child and Adolescent Functional Assessment Scale (CAFAS), which can also be used alone or with another diagnostic interview ([Hodges and Wong, 1996, 1997](#); [Hodges et al., 1998](#)).

### Anxiety Disorders Interview Schedule

Notwithstanding its title, the ADIS also provides brief ratings of symptoms pertinent to other disorders, but its coverage of the anxiety disorders is more thorough ([Silverman, 1991](#); [Silverman and Eisen, 1992](#); [Silverman and Nelles, 1988](#); [Silverman and Rabian, 1995](#)). The interview is designed for *clinician* use, and it is derived from the adult ADIS ([di Nardo et al., 1993](#)). Questions that *should* be asked are provided, and guidance is given about when to use additional questions. Most symptoms are scored on a simple three-point scale (yes, no, other). However, in the anxiety section, a good deal of use is also made of nine-point scales (represented by a thermometer) ranging from "not at all" to "very, very much." Similar scales are used to rate *interference*, which is the ADIS term for psychosocial impairment resulting from disorders. Skip structures are frequently employed, so many persons are not asked about all symptoms.

### Interview Schedule for Children and Adolescents

The ISCA mandates administration by experienced clinicians with extensive structured interviewing training and full understanding of the principles of diagnosis and its application to children with psychopathology. Interviewers must be able to combine the symptom information from both parent and child into diagnoses themselves, because formal algorithms for this process (other than the official diagnostic manual itself) do not appear to be available ([Sherrill and Kovacs, 2000](#)). Questions are clearly specified, but much additional clarification is expected of interviewers, who are not expected slavishly to stick to the written questions. Sixty-nine "major" symptoms and 10 "mental status" items are covered, and they are typically coded on a nine-point severity scale that incorporates judgments about associated distress, functional impairment, and the amount of effort required to counteract symptoms. "Subsidiary information" about many symptoms is also collected, typically in a yes-no format. In addition, 17 intrainterview behavioral observation items are assessed. A version of the interview for use with young adults (the Follow-up Interview Schedule for Adults; FISA) has also been developed.

### Diagnostic Interview Schedule for Children

As already indicated, the questions on a respondent-based instrument are designed in a fixed, ordered sequence and require very simple responses (typically yes or no). Symptom severity is defined by similar yes-no answers on subsidiary questions (asked only if a positive response is obtained to a superordinate question) and forced choices from short sets of frequency ranges. All data combination is performed by computerized algorithms. The DISC covers a wide range of childhood and adolescent disorders and is suitable for use with 9 to 18 year olds. A version for use with the parents of preschoolers is currently under development.

## INTERVIEW TIME FRAMES

The K-SADS interviews, the ADIS, the CAS, the Dominic, the PICA-III-R, and the ISCA all focus on the child's "current" status or the "current episode" of disorder, although the definition of "current" is largely unspecified, except in the case of the ISCA, in which the assessment period is specified (e.g., 2 weeks for irritability). The Columbia K-SADS adds a past 2-week time frame to the "current" frame. The K-SADS-PL, the Columbia K-SADS, and the K-SADS-E explore lifetime histories of "worst" episodes, whereas the ISCA also provides for assessment of lifetime disorder, and an "interim" version provides an assessment of current status and the child's status in the interim between the current assessment and the last assessment for use in follow-up studies. The DICA and MAGIC focus on the whole lifetime, but for some disorders an additional shorter time frame is also included. For instance, in the depression section, the MAGIC asks about the "past month" as well as whether the child has "ever" had symptoms. The CAPA covers a "primary period" of 3 months, but it also notes whether certain uncommon symptoms (such as suicide attempts) have ever occurred, and a version that provides lifetime coverage of major episodes of certain syndromes is also available ([Simonoff et al., 1997](#)). The full DISC-IV can be used to assess either the last month or the last year, and it also offers a module to determine whether certain syndromes that did not occur during the preceding year had occurred at any point since the age of 5 years.

## SPECIFICATION OF QUESTIONS, INTERVIEWER FLEXIBILITY, AND THE DEFINITION OF SYMPTOMS

In a fully respondent-based interview such as the DISC, or the computerized version of the DICA, all questions are completely specified, and no others may be used. In other words, the questions are fully specified, there is no interviewer flexibility, and no need exists to provide definitions of symptoms. All variability in these features concerns the interviewer-based interviews.

The ADIS, CAPA, DICA, and MAGIC all contain questions that, under most circumstances, should be asked as written in the schedule. Additional questions are then asked as necessary to allow the interviewer to determine exactly whether the symptom is present. Many such questions are provided on the schedule of the CAPA, and fewer questions are provided by the CAS schedule (but it is not clear to what extent the "set" questions of the CAS are mandated for use). Most guidance on this process for the DICA and MAGIC is provided in the MAGIC's specifications manual. All these interviews provide at least some additional instructions about when to probe further. The K-SADS group of interviews provides a range of suggested questions but no formal rules about when they are to be used or skipped. Interviewers are also expected to ask any additional follow-up questions that may be necessary to clarify responses.

By *interviewer flexibility*, we mean the degree to which the interviewer is expected or encouraged to use judgment in asking additional clarifying questions. In this respect, the K-SADS and ISCA (and perhaps the ADIS) interviews may be seen as providing most flexibility because they demand that judgments based on clinical experience be made, and in the case of the K-SADS they do not mandate the use of any particular questions. The problem question is "flexible to do what?" The answer is relatively clear in the case of the K-SADS-P IV and the CAPA, in which detailed coding rules are provided for each symptom. The task of the interviewer is to determine whether those coding criteria are met. With the other interviewer-based instruments, it often seems that the interviewer's task's job is to obtain the answer to the *original question*. Variable amounts of guidance on how to do so are provided, in considerable detail in the MAGIC's specifications manual, with brief symptom definitions in the CAS, but with little or no guidance in the K-SADS-E or ADIS.

The K-SADS-P IV and the CAPA provide detailed *definitional* glossaries covering each symptom; this means that the task of the interviewer is quite clearly specified at the level of the *definition* of symptoms. The CAPA glossary also contains many procedural instructions relating both to questioning in general and to questioning about specific symptoms, but these are presented as being secondary to the definitional issues. The MAGIC's specifications manual can also be seen as being a glossary, but it is, first and foremost, a *procedural* manual. It is primarily about *how* to collect the information. In the course of this discussion of procedure, it also includes a good deal of definitional material, but it does not provide formal definitions of items such as found in the K-SADS-P IV or CAPA.



## RELIABILITY OF STRUCTURED DIAGNOSTIC INTERVIEWS

A difficulty with the literature on *reliability* is that the term is applied to several very different sorts of design and statistical approaches. Three of these are commonly seen in the literature on structured interviews, but only one presents a useful test of what one ultimately requires in a diagnostic interview. For instance, one often sees reports of *interrater* reliabilities in the interview literature. However, interrater reliability is not a very useful index of interview performance. With respondent-based interviews, it tests nothing but whether one interviewer can read aloud adequately while another codes a schedule. With such interviews, if interrater reliability is not in the high 0.9's, one should retrain or fire one's interviewers! In an interviewer-based interview, the questions are not fixed, and so different interviewers could use different questions to elicit the same information. Because the interrater reliability paradigm uses multiple raters to score the same interview, this major source of potential unreliability is eliminated, with the result that the interrater reliability is likely to substantially overestimate the reliability of the interview in actual use. However, if interrater reliabilities fall below about .8, that is an indication either that something is wrong with interviewer training or that the interview needs to be revised to provide greater clarity about what is supposed to be rated. The point is that if very high interrater reliability coefficients cannot be obtained, we can be certain that test-retest reliability will be poor.

*Internal reliability* is sometimes reported for scales resulting from diagnostic interviews. Typically, the statistic given is Cronbach's  $\alpha$ . This statistic provides a measure of the degree to which the items in the scale are correlated with one another. There are two problems with the use of such a statistic here. First, it has nothing to do with whether raters are coding items "reliably," but rather, it concerns the degree to which the items in a scale are all measuring the same thing. Second, a perfectly good diagnosis could be associated with low correlations among the symptoms that signal the presence of that diagnosis. Most medical (and psychiatric) diagnoses are more concerned with pattern recognition than with scalar values. For instance, in the general population, it is unlikely that dry eyes, dry mouth, and arthritis are very highly correlated with one another but that does not mean that Sjögren's syndrome is a bad diagnosis. Of course, if the task is to develop a scalar measure of some construct, then the scale's  $\alpha$  is important information. However, from the perspective of a diagnostic interview, low  $\alpha$  values are not necessarily a bad thing, nor are high  $\alpha$  values necessarily a good thing. It is entirely possible to have a perfect measure of a diagnostic entity where Cronbach's  $\alpha$  for the symptoms constituting that entity was close to zero.

Table 42.1 shows the results of studies of the *test-retest* reliabilities ( $k$  values) of diagnoses measured by the instruments considered in this chapter. Test-retest reliability is what is needed in judging an instrument. In the absence of reasonable test-retest reliability, all other reliabilities are moot. It can be seen that all the interviews that have been tested do a reasonably good job, and there is not much to choose among them, as far as test-retest reliability of diagnosis is concerned. These reliability coefficients are similar to those reported for psychiatric interviews with adults. Reliabilities for scale scores derived from these interviews are typically rather higher than they are for diagnosis, but that is nearly always true of comparisons between scale score reliabilities and the reliabilities of categories generated by imposing cut points on the same scales. Moreover, some of the unreliability of diagnostic measures is the product of the diagnostic system itself, thanks to its requirement that numerous unmemorable details about past psychopathology be considered. Particular problems have been identified with the reliability of responses concerning the duration of symptoms and the dates of their onsets (Angold et al., 1996a; Breton et al., 1995).

Int	Symptoms		No		Symptoms		No		No		No	
	ICC	Depression	ICC	Depression	ICC	Depression	ICC	Depression	ICC	Depression	ICC	Depression
KIDDIS	.84	.75	-.01	-.01	-.01	-.01	-.01	-.01	.24	-.01	-.01	-.01
KIDDIS-P	.77	.69	-.01	-.01	-.01	-.01	-.01	-.01	.21	-.01	-.01	-.01
KIDDIS-L	.82	.73	-.01	-.01	-.01	-.01	-.01	-.01	.23	-.01	-.01	-.01
KIDDIS-B	.75	.66	-.01	-.01	-.01	-.01	-.01	-.01	.22	-.01	-.01	-.01
DISC-2.1	.85	.76	-.01	-.01	-.01	-.01	-.01	-.01	.25	-.01	-.01	-.01
DISC-2.1C	.86	.77	-.01	-.01	-.01	-.01	-.01	-.01	.26	-.01	-.01	-.01
DISC-2.1F	.87	.78	-.01	-.01	-.01	-.01	-.01	-.01	.27	-.01	-.01	-.01
DISC-2.1G	.88	.79	-.01	-.01	-.01	-.01	-.01	-.01	.28	-.01	-.01	-.01
DISC-2.1H	.89	.80	-.01	-.01	-.01	-.01	-.01	-.01	.29	-.01	-.01	-.01
DISC-2.1I	.90	.81	-.01	-.01	-.01	-.01	-.01	-.01	.30	-.01	-.01	-.01
DISC-2.1J	.91	.82	-.01	-.01	-.01	-.01	-.01	-.01	.31	-.01	-.01	-.01
DISC-2.1K	.92	.83	-.01	-.01	-.01	-.01	-.01	-.01	.32	-.01	-.01	-.01
DISC-2.1L	.93	.84	-.01	-.01	-.01	-.01	-.01	-.01	.33	-.01	-.01	-.01
DISC-2.1M	.94	.85	-.01	-.01	-.01	-.01	-.01	-.01	.34	-.01	-.01	-.01
DISC-2.1N	.95	.86	-.01	-.01	-.01	-.01	-.01	-.01	.35	-.01	-.01	-.01
DISC-2.1O	.96	.87	-.01	-.01	-.01	-.01	-.01	-.01	.36	-.01	-.01	-.01
DISC-2.1P	.97	.88	-.01	-.01	-.01	-.01	-.01	-.01	.37	-.01	-.01	-.01
DISC-2.1Q	.98	.89	-.01	-.01	-.01	-.01	-.01	-.01	.38	-.01	-.01	-.01
DISC-2.1R	.99	.90	-.01	-.01	-.01	-.01	-.01	-.01	.39	-.01	-.01	-.01
DISC-2.1S	.99	.90	-.01	-.01	-.01	-.01	-.01	-.01	.40	-.01	-.01	-.01

Table 42.1. Diagnostic Test-Retest Reliabilities (k values) of the Instruments Considered in This Chapter (Where Available)

Despite enormous efforts on the part of interview developers, it cannot be said that the reliability of diagnostic interviews has increased much over the years. We now have a fairly mature interview technology that has been repeatedly refined in the hope of increasing the reliability of assessments. The current arsenal of diagnostic interviews probably offers reliability that is as good as can be achieved unless quite different means of arriving at psychiatric diagnoses appear.

A problem with the test-retest assessment of reliability is that it requires that the interview be repeated within a short period of time. With both questionnaires and interviews, one typically finds that fewer symptoms are endorsed at the second interview than at the first (Angold et al., 1996b; Lauritsen, 1998; Lucas et al., 1999; Piacentini et al., 1999). There are many possible explanations for this effect (Jensen et al., 1992), but some evidence suggests that the results of the first interview are probably the most accurate representation of reality. The usual interpretation of test-retest reliability statistics, such as Cohen's  $k$  for categorical data (e.g., diagnoses) and the intraclass correlation coefficient for continuous data (e.g., scale scores), involves the supposition that the relationship between scores at the first interview and those at the second involves two components, agreement and random error. The presence of a *consistent difference* between first interviews and second interviews indicates that such statistics underestimate the "true" (and ultimately unmeasurable) reliability of both interviews and psychopathology scales.

## VALIDITY OF STRUCTURED DIAGNOSTIC INTERVIEWS

The problem with trying to assess the validity of psychiatric interviews is that there is no non interview test for most psychiatric disorders. The structured interview itself is the closest approximation we have to a current standard. So how are we to *validate* the diagnoses obtained from such interviews? This is a version of a problem that psychologists have been grappling with for decades; one that led to the concept of *construct validity*. The central idea here is that the validity of an instrument for the measurement of a psychological construct inheres not in some single agreement coefficient with one external standard, but in the instrument's performance within the *nomologic net* of theory and data concerning the construct or constructs that the instrument purports to measure (Anastasi, 1950, 1986; Cronbach and Meehl, 1955; Gulliksen, 1950; Jenkins, 1946; Novick, 1985; Peak, 1953; Wallace, 1965; Weitz, 1961). As Gulliksen pointedly remarked in 1950, "at some point in the advance of psychology it would seem appropriate for the psychologist to lead the way in establishing good criterion measures, instead of just attempting to construct imperfect tests for attributes that are presumed to be assessed more accurately and more validly by the judgment of experts" (Gulliksen, 1950).

Structured interviews were developed because of the dismal psychometric properties of unaided clinical diagnosis, so comparisons with unstructured clinical judgment (as may be found for instance in chart diagnoses) are hopelessly flawed tests of diagnostic interview validity. That has been obvious to interview developers for a long time, so validity studies have sometimes provided the clinicians with all the information from the interviews to be validated, in addition to allowing them to collect further information should they desire. In such a circumstance, it is often the case that *most* of the information available to the clinician comes from the interview under test. It will hardly be surprising, then, if there is good agreement between clinician and interview, but no real test will have been done.

In considering the validity of any interview, we should take a construct validation approach and describe what we currently know about it in relation to the nomologic net pertaining to child and adolescent psychiatric diagnosis. So far, only the developers of the CAPA have explicitly laid out the evidence for the validity of the CAPA using this approach, but most of the interviews considered here can point to similar chains of evidence. To give a flavor of the sort of evidence relevant to construct validation, the following findings have been adduced as construct validators of the CAPA:

1. Diagnostic rates and age and gender patterns of disorder given by the CAPA are consistent with those found using other interviews.
2. Patterns of diagnostic comorbidity are consistent with those found by other interviews.
3. Symptomatic diagnoses are associated with psychosocial impairment.
4. Parent and child reports of psychopathology on the CAPA are related to parent and teacher reports of problems on well-established scales for detecting psychopathology.
5. Children with CAPA-identified disorders use more mental health services than children without such diagnoses.
6. CAPA diagnosed children tend to come from families with a history of mental illness.

7. There is genetic loading for certain CAPA scales scores and diagnoses.
8. CAPA diagnoses show consistency over time.
9. CAPA diagnoses predict negative life outcomes ([Angold and Costello, 2000](#)).
10. Different CAPA diagnoses are differentially related to the physiologic changes of puberty ([Angold et al., 1999](#)).

There is, of course, a big drawback to this approach. No new interview will be able to point to such chains of evidence until it has been in use for quite a time. How then is any new instrument ever to be developed? The answer lies in the application of common sense and the well-established assessment principles. When a new assessment is needed, it is perfectly proper to use the best available information about the nature of the phenomena to be measured to design what seems, on the face of it, to be an adequate measure. Attention needs to be given to keeping questions short and focused on single constructs and to defining the task of the interviewer clearly and providing good definitions of the constructs to be studied in the case of interviewer-based interviews. If initial test-retest reliability is adequate, then, in general, it is unlikely that the interview will prove to be utterly invalid. Because it is very unusual to try to assess a construct that has been utterly unmeasured before, or about which nothing whatever is known, it is also usually possible to include in the test-retest study other measures that will give some indication of the concurrent, predictive, or divergent validity of the new instrument.

## ADVANTAGES AND DISADVANTAGES OF INTERVIEWER-BASED AND RESPONDENT-BASED INTERVIEWS

Neither interviewer- nor respondent-based interviews are ideal tools, and there is simply no answer to the question of which type is best. In this section, we list the general advantages of each type of interview, and the lack of each advantage can be regarded as a disadvantage of the other type. However, it is worth noting here that it has sometimes been said that respondent-based interviews are more appropriate for general population studies. Because it has now been shown that lay interviewers can provide reliable and valid ratings using interviewer-based interviews, this position is no longer tenable. Indeed, some larger-scale general population studies have used and are still using interviewer-based interviews ([Costello et al., 1996](#); [Lewinsohn et al., 1993](#); [Simonoff et al., 1997](#)).

### Advantages of Interviewer-Based Interviews

Interviewer-based interviews have four theoretical and practical advantages: (a) if the interview has been conducted and coded properly, the meaning of the ratings is precisely known; (b) they provide opportunities to cross-check discrepant or confusing information; (c) they enable the use of efficient open-ended questioning strategies and allow the use of redundant questioning, which has been shown in adults to improve the quality of responses; and (d) they appear to be less prone to overdiagnosis on the basis of symptom reports, so arbitrary impairment scale cutoffs do not have to be employed to produce reasonable diagnostic rates in unselected community samples.

#### WHAT DO THE CODINGS MEAN?

In an interviewer-based interview, a positive coding for a symptom means that it has been determined that the symptom, as defined in the schedule, is present. In a respondent-based interview, one knows only that a child or parent responded positively to a particular question. One does not know exactly what the child or parent understood the question to mean. This problem has been documented with unusual symptoms that most children never experience, such as obsessive-compulsive or psychotic symptoms. Such symptoms are greatly overreported using the DISC ([Breslau, 1987](#)). When clinicians review what the children said, it is obvious that what is being reported is not obsessive-compulsive disorder or psychosis. However, if an unstructured clinician review is added to the diagnostic process, one no longer knows exactly what factors have gone into the final rating, and one great advantage of the respondent-based interview is lost.

#### CROSS-CHECKING DISCREPANT OR CONFUSING INFORMATION

It is common in clinical practice to find that certain answers appear to contradict previously given information or lead to uncertainty about whether a symptom is present. In an interviewer-based interview, one simply attempts to clarify the contradiction or confusion. The respondent-based approach provides no such mechanism for resolving such difficulties, because interviewers are not allowed to exercise their judgment about such matters.

#### USE OF OPEN-ENDED AND REDUNDANT QUESTIONS

The distinction between open and closed questions is not absolute, but open questions are those that offer the chance to provide a wide range of answers or free-recall descriptions of phenomena, whereas closed questions call for one of a limited set of responses. For example, an open question in response to being told by a child that he had received a bad school report may be "How did you feel about your bad grades?" whereas "Did your bad grades make you feel unhappy?" would be a closed question. If a child had just admitted to stealing, responding with "Tell me more about that" involves an open question, whereas "What did you steal?" is a closed question. Basically, closed questions call for a yes or no answer or a date, frequency, duration, or other quite specific piece of information, whereas open questions give the opportunity for the child to provide a description of his or her feelings, which may or may not involve sadness or hostility. The work of Rutter and Cox and their colleagues offers some direct guidance on the best ways to use these different sorts of questions with adults, and in the light of the literature on children's memory, there is little reason not to use a similar approach with children. These investigators conclude that, in general, most factual information is collected when a systematic approach that relies heavily on open questions is used. This approach is also conducive to parental expressions of emotion, because it involves less talking on the part of the interviewer and gives more time for parents to discuss their concerns. Conversely, a noninterventionist approach results in the provision of less relevant information, whereas challenging interpretations and a confrontational style prove less effective in eliciting emotions ([Cox et al., 1981a, 1981b, 1981c](#); [Hopkinson et al., 1981](#); [Rutter and Cox, 1981](#); [Rutter et al., 1981](#)). Thus, open questions can be effective in collecting information efficiently, but, of necessity, respondent-based interviews must rely on closed questions. Conversely, closed questions are necessary to elicit information that is otherwise not forthcoming, and respondent-based interviews have been a substantial help to the developers of interviewer-based interviews in establishing well-thought-out logical structures for series of closed questions.

A redundant question is one that is asked more than once in different ways, that is, questions that contain two presentations of the same item, as in "Have you been more irritable than usual?" followed by "or made angry more easily?" The adult survey literature ([Cannell et al., 1977](#)) suggests that such redundancy is actually helpful in providing both additional time for thought and a second chance to detect symptoms. If the answer to one question is positive and the other is negative, that is no problem for an interviewer-based interview, in which follow-up questions can be used to clarify the situation. However, this cannot be done in a respondent-based interview, in which specific chains of questions in response to initially discrepant information are required. Collapsing the two questions into one is no help because this generates a "multiple question" that requires the respondent to remember and process the two parts of the question simultaneously.

#### REASONABLE RATES OF DISORDER ARE IDENTIFIED IN COMMUNITY SAMPLES WITHOUT RESORTING TO ARBITRARY IMPAIRMENT CUTOFFS

Rates of diagnosis based only on symptom reports have been found to be unreasonably high with the DISC. To correct this tendency, it is now usual to require that a diagnosis be allocated only when the score on the Child Global Assessment Scale (CGAS) is less than 60 (or sometimes 70, i.e., when notable psychosocial impairment is present). However, in most cases, the DSM-IV requires that either impairment or distress be present, not that significant impairment always be present. Furthermore, the typical impairment cutpoint of 60 was established because it generated what appeared to be sensible rates of diagnosis, not because anyone with a score of more than 60 is necessarily unimpaired. Because the K-SADS-P and the CAPA employ exacting definitions at the individual symptom level, community studies using these instruments do not need to apply additional rules about impairment to generate sensible rates of diagnosis.

### Advantages of Respondent-Based Interviews

We can also identify four advantages of respondent-based interviews: (a) less intensive training needs to be provided to interviewers, (b) a lesser level of quality control is required, (c) computer assisted and computer-administered interviews can more readily be developed, (d) computerized interviews offer the prospect of providing "preclinical" screening for use in clinical settings.

#### INTENSITY OF TRAINING

There is no doubt that an interviewer-based interview makes greater demands on an interviewer than does a respondent-based interview. For instance, training on the full CAPA package takes a month, compared with a few days for the DISC.



## QUALITY CONTROL

*Interviewer drift* occurs with respondent-based interviews and necessitates continuing quality control throughout a study, but it is unarguable that there is much more to check with an interviewer-based interview. Care must be taken not only that the interview is being conducted and coded properly, but also that mechanisms must be in place to ensure that interviewers continue to interpret the responses of interviewees correctly. Whether this additional burden is worth the effort depends on the situation in which the interview is being used.

## COMPUTERIZATION

A great advantage of the respondent-based approach is that it lends itself easily to computerization. Questions are arranged in unvarying logical sequences in such an instrument, with stem questions, followed by sequences of further questions contingent on the answers to the stems. Software is available to allow the presentation of such interviews on a personal computer. Computerization can be achieved at two levels. Computer-*assisted* psychiatric interviews (CAPI) employ an interviewer to read questions from the screen and to enter the appropriate codes into the computer as the interview progresses. The machine takes the interviewer to the appropriate stem questions and stores the responses in a database. There is no need for bulky interview schedules to be copied and carried around, and data entry is completed during the interview. Furthermore, the computer will not accidentally skip parts of the interview or vary the order of its presentation. The next level of computerization is referred to as computer-*administered* survey interviewing. Here, no interviewer is used at all. Rather, digitized audio recordings of the questions (sometimes even with digitized video of an interviewer) are played back by the computer as the written form of the question is displayed. The respondent enters a response to the question, which is saved to the database. The DISC has become progressively more complex since the 1980s (largely because of the ever-increasing complexity of the DSMs), and the DISC-IV is now supposed always to be completed in its CAPI format, because it is too difficult to administer it effectively in a paper-and-pencil format. There is also a CAPI version of the DICA, but this differs from paper-and-pencil version of the interview in being fully respondent based (Reich et al., 1995).

## PRECLINICAL SCREENING

We have reached the point at which it is feasible to have parents and children complete a diagnostic interview such as the DISC-IV before they see a clinician. The possible output from the DISC-IV is almost infinitely flexible and requires only programming to allow the production of reports tailored to particular clinical needs that can be generated immediately the interview is finished. Equipped with such a report, a clinician familiar with one of the interviewer-based interviews would then be starting with a very respectable initial diagnostic formulation to guide further elucidation of the clinical status of the child.

## AN APPROACH TO SELECTING A STRUCTURED INTERVIEW FOR RESEARCH OR CLINICAL PURPOSES

As we have already seen, reliability and validity quotients are little help in selecting the “best” interview, because there is little to choose among interviews in this regard. Similarly, all the interviews considered here take quite a long time to complete, and the length of all these interviews is proportional to the symptoms manifested by a child. None has been shown to be notably shorter than any other in practice, and so reported mean interview durations are also no help in selecting an interview for any particular application. Rather, the key to selecting an interview is to be very clear about what that application demands. This means having thought through what the ideal interview for that application would be like, so the characteristics of each can be matched against that criterion. It is also worthwhile to prioritize these demand characteristics in advance. It may not be possible to have everything, so it is a big help to have thought through the relative impact of different tradeoffs. In the following sections, we consider some questions that prospective interview users should ask themselves as part of the process of selecting an interview:

### Question 1: What Content Areas Need to Be Covered?

All interviews do not cover all diagnoses. (Indeed, none covers all possibly relevant diagnoses.) It is essential in the first instance to decide what areas must be covered and what additional areas would be nice to have, but are not essential.

### Question 2: What Assessment Time Frame Is Needed?

The addition of a lifetime frame involves either compromising the assessment of current or recent symptoms (as with the K-SADS-PL) or combining a short time frame interview with a separate lifetime interview, which results in a very long assessment. If one is conducting a 3-month follow-up assessment after treatment, an interview that focuses on diagnostic status over the last year will not be very helpful.

### Question 3: What Sort of Interviewers Will Be Available?

In general, clinicians are not enthusiastic about using a respondent-based interview because they have to follow a fixed schedule of questions, and it provides no means to collect more detailed information that may be relevant to a variety of treatment decisions. So it makes sense for them to use one of the interviewer-based interviews. Such interviews can also be very useful clinical training tools, because they provide excellent models for the clinical interviewing process.

If lay interviewers are to be used, then the K-SADS family of interviews may not be the best choice because these interviews have all been developed for use by clinicians and provide less guidance for the training of lay interviewers. No reliability data on their use with lay interviewers are available. Conversely, the CAS, CAPA, DICA, and ADIS were all developed for use with trained lay interviewers and have been shown to have acceptable reliability in their hands. All these interviews are also acceptable to clinicians.

### Question 4: What Are the Age and Developmental Levels of the Children to Be Assessed?

It is clear that “adult-style” diagnostic interviews (all the interviews considered in this chapter fall into this category) with preschool children are a waste of time, because they simply cannot provide all the information required by the DSM-IV or ICD-10 diagnostic criteria. Several groups are working on this problem at present, but none of the instruments reviewed here is of any use for very young children. The K-SADS and DICA are said to be suitable for face-to-face use with children down to the age of 6 years, but others doubt that a *full* diagnostic interview really works with children younger than 8 or 9 years old, unless the interviewer simply “goes off interview” to obtain a relatively impressionistic view of the child’s symptoms. Even the interview with the parent requires substantial modification for preschoolers. We do not recommend the use of any of these instruments in face-to-face interviews with children less than 8 or 9 years old. Special versions of the DICA and CAPA for use with younger children are currently under development, but their feasibility and reliability remain to be determined. Picture-enhanced interviews such as the Dominic-R may be suitable for children as young as 6 years old, but even here, reliability appears to be somewhat lower with younger children, and such interviews do not generate full diagnoses. The same problem applies to older persons with substantial intellectual deficits. When an individual’s IQ is less than 70, it becomes very difficult to complete a full diagnostic interview. Interviews with parents and teachers may still be conducted, but we know little about their performance characteristics in such circumstances.

At the other end of the “juvenile” age range (late adolescence and early adulthood), the researcher or clinician has the choice of shifting to an “adult” measure or using an age-appropriate modification of a child interview (e.g., the YAPA).

### Question 5: How Much Can I Afford to Spend on Training and Quality Control?

“Spend” here refers to both time and money. If less than a week is available, then none of the interviewer-based interviews is suitable, because good training for them demands a greater time investment. However, DISC training can be provided in as short a time as this because the DISC requires far less of interviewers. Conversely, when amortized over the life of a study or clinical program, initial costs for training usually turn out to be only a small percentage of the total costs, so good training on an interviewer-based interview may constitute a good investment.

Once the interview is over, the DICA and CAPA both expect that it will be reviewed for appropriateness of codings. In addition, regular (both recommend weekly) continuing training sessions for interviewers are required to prevent interviewer drift. Are funds and personnel available to support such continuing quality control and training procedures? The CAS and K-SADS interviews do not appear to have standard recommendations for quality control and continuing training, but there is no

reason to suppose that data quality and consistency are any easier to maintain with these instruments. Because the DISC-IV is computerized and the DISC interviewer's task is much less arduous than that required of interviewers using any of the interviews considered in this chapter, it demands less effort to control interviewer drift.

### **Question 6: What Needs and Resources Do I Have for Data Entry and Manipulation of the Data?**

The K-SADS family of interviews produces diagnoses through the medium of score sheets completed by the interviewer and scored by that person to produce diagnoses. The CAS provides formal algorithms for making diagnoses that can be implemented either by the interviewer or by a computer. The CAPA offers only computerized algorithms because its developers (like those of the DISC) believe that the process of producing a final diagnosis from a large array of symptom data is of such complexity that errors are bound to occur if humans do it unaided. These different approaches to producing a final diagnostic formulation have very different implications for data entry and manipulation. If the computer is to make the diagnoses, then all the symptom information must be entered (this task easily runs to hundreds or thousands of variables per case), and diagnostic algorithms must be available. If the clinician makes the final diagnosis, then one could simply enter only that information (or not computerize any data at all in a clinical setting in which the interview is being used just as a clinical assessment whose results appear only in the medical record). The CAPA is probably the interviewer-based instrument that makes the most demands on data entry (because of its molecular approach to symptom severity), so it has extensive data entry, checking, and diagnostic programs. Even so, one must budget for setting up and training people in the use of these programs. Once these demand characteristics have been determined, one can begin the task of actually selecting an interview:

#### **Action 1: Review the Available Instruments and Make a Shortlist**

It may seem surprising that we place a review of available measures so late in the process. There are two reasons for doing this. First, the choice of measure should depend on the nature of the application, not the other way around, so until the application has been well defined, the issue of instrumentation is moot. Second, once one has answered questions 1 to 6, a brief review of instruments may indicate that only one is even remotely suitable. To put it another way, it is more efficient to decide which interview to use relatively late in the process, because one may be able to save a lot of time that would otherwise have been spent in considering instruments that cannot meet the needs of the study. If there seem to be several possibilities, one has at least reduced the number of instruments that need to be considered to a shortlist for further evaluation.

#### **Action 2: Obtain Copies of the Instruments on the Shortlist and Conduct a Detailed Evaluation**

Unfortunately, there is absolutely no substitute for obtaining copies of the instruments still left on the shortlist and reviewing them in detail. The manifold differences among instruments make it impossible to provide more than a flavor of what an interview is like in a review chapter such as this. It is worth remembering that you will be asking the interview developers to send several hundred pages of schedules, glossaries, instruction manuals, and the like, and these will need to be paid for. At this stage, it should be possible to make a final choice, but if there are still questions that have not been answered in all these materials, a telephone call to someone in the relevant interview development group can be very helpful. If this "homework" is been done, it is also likely to be well received.

#### **Action 3: Plan Training Well Ahead of Time**

It is not unknown for an interview developer to receive a request such as the following: "Hello, I put the \_\_\_\_\_ in a grant proposal, and the funding has just come through. I need you to train my interviewers next month." Interview developers have busy research and clinical lives, and they cannot provide training programs at a moment's notice. The time to be setting a hopeful date for training is *when the grant proposal is submitted*, not after it has been reviewed and funded.

## **FUTURE DIRECTIONS**

A great deal of work has gone into producing the interviews we have today, and there is little sign that recent efforts to "improve" such measures further have had much effect on their reliability. Of course, as we learn more about psychopathology, we will need to modify our measures' content to reflect what we need to measure, but the basic principles used to design new or revised modules will not change in the foreseeable future (and will work just as well or poorly as they do today). What is needed now is to extend the range of structured assessments down to younger ages. There is astonishingly little research on preschool psychopathology, for instance, and it was only in 2000 that the first structured parent-report diagnostic interview specifically designed for use with this age group became available. It remains to be seen how information from the child and caretakers other than parents can usefully be integrated into diagnostic assessments, but work has begun in this area. Newer "self-report" assessments of the mental status of the child that do not tie themselves rigidly to current diagnostic criteria show great promise for the future. For instance, with the MacArthur Story-Stem Battery ([Warren et al., 1996, 2000](#)), the interviewer uses toys to act out the beginnings of stories, which the child is then asked to complete. The videotapes of these interactions are then scored to provide indices of a variety of internal states. The Berkeley Puppet Interview employs two puppets to express two moods or states, and the child indicates the puppet most like himself or herself, thereby providing self-report assessments of perceived academic functioning, social relationships, depression, anxiety, and aggression or hostility. Some simpler questionnaires with pictures have also shown promise with preschoolers in relation to the assessment of depression and anxiety ([Ialongo et al., 1993](#); [Martini et al., 1990](#)).

Now that many diagnostic measures are available, it is likely that they will move progressively into ordinary clinical practice. It seems strange that the unstructured clinical interview has been almost entirely supplanted for research purposes because of its well-documented inadequacies as a diagnostic procedure, but it continues to be the main assessment tool in clinical practice, in which good phenomenologic assessment is surely of the greatest importance. All clinicians dealing with psychopathology can benefit from training on an interviewer-based structured interview (particularly one of the glossary-based interviews), and it is to be hoped that such training will soon become part of all training programs for psychiatric clinicians. However, it must be admitted that the time to conduct a full psychiatric assessment is not always available, and when that is the case, it would be helpful to have shorter interviews available to serve as screening tools. At the time of writing, work is happily beginning on a version of the DISC to fulfill this function. The idea here is not to force slavish dependence on any particular structured interview on clinicians, but to use the strengths of standardized interviews to underpin their explorations of the nature and meaning of psychopathology. Our understanding of psychopathology and of its measurement has come a long way, and it is time to bring the benefits of what are still typically regarded as being research assessment methods to all of our patients and clients.

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## 43 PSYCHOLOGICAL ASSESSMENT OF CHILDREN AND ADOLESCENTS

Nancy E. Moss, Ph.D., and Gary R. Racusin, Ph.D.

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[Referral Questions](#)  
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Psychological testing conducted by a trained clinical psychologist is a method of obtaining developmentally informed, concrete, and standardized data regarding a person's capacities and behavior in a wide variety of domains (Allison et al., 1968; Sparrow et al., 1985, 1995). The domains that are most pertinent to the assessment of children and adolescents include intellectual ability, educational accomplishment, language skills, visual-motor coordination, adaptive behaviors, and personality functioning (Sparrow et al., 1985). (See Chapter 40 for further information on infant assessment.)

In clinical practice, psychological assessment constitutes an important type of inpatient or outpatient consultation. Historically, when done as an inpatient procedure, psychological testing has often been part of a comprehensive, multidisciplinary workup aimed at facilitating long-term treatment. In such circumstances, referral questions may be broad, to contribute to the overall understanding of a patient. Test data are typically integrated with findings from other relevant disciplines. Although such long-range inpatient treatment is still conducted in some sites, recent developments in national health care practices have dramatically reduced the typical length of inpatient stay. To keep pace with these developments, psychologists in these settings have had to respond with flexible, creative approaches to more time-efficient assessment. Careful choices must be made among the test instruments discussed in this chapter to answer highly specific referral questions. More comprehensive psychological assessment often must be deferred until after the patient has been discharged to a less restrictive treatment setting.

Although the foundation of inpatient psychological assessment has been modified, as noted earlier, the basic approach to outpatient assessment has remained fairly constant. Therefore, to articulate most clearly the discrete phases of a comprehensive psychological evaluation, this chapter is organized to reflect the typical flow of an outpatient consultation. Accordingly, the chapter first addresses the identification of appropriate referral questions and the preparation of the child or adolescent patient and of the parents. Choice of assessment procedures and of instruments and test administration are then discussed, with emphasis on recent contributions to the test library. This discussion particularly reflects recent activity in the psychoeducational evaluation of attention deficit hyperactivity disorder (ADHD) and in infant assessment. The chapter then focuses on developments in test interpretation and concludes with a discussion of issues involved in the communication of findings.

### REFERRAL QUESTIONS

To ensure that psychological assessment is most efficacious, the testing should be directed at specific referral questions. Appropriate referral questions facilitate the psychologist's choice of test instruments and, on analysis of the test data, help to determine which are the relevant issues. When not guided by particular questions, the testing often yields general information that lacks the precision necessary to be of clinical utility. Relevant referral questions are often formulated by the referring clinician. When the referring clinician is uncertain about the focus of the testing, consultation with the psychologist may help to articulate referral questions. For children and adolescents, the referral questions are also often designated by parents who make direct requests for psychological assessments.

The many different suitable referral questions for psychological assessment may be grouped into several areas. First, for the very young child, clinical psychological testing offers answers to pressing questions regarding the progress of development. Many times, parents, pediatricians, educators, and mental health professionals form impressions and concerns about the rate and nature of a youngster's development. Testing compares the individual child to age-mates, provides a reliable indicator of developmental level, and permits the substantiation or modification of such assumptions. Such assessment is most useful in the identification of developmental disorders and the sequelae of neurobehavioral insult from sources such as lead poisoning. It also highlights the need for early interventions that may greatly improve a child's and a family's long-term quality of life.

Second, for the school-age child and adolescent, psychological testing provides a more reliable estimate of intellectual capacity than does clinical impression. Often, a clinical estimate of a child's intelligence level based on self-presentation can be misleading. Interpersonal behaviors may be confusing or disturbing if they seem discrepant with the person's apparent level of intelligence. This is particularly true when a youngster's verbal skills are quite different from his or her other intellectual abilities. Intellectual assessment also serves as a foundation on which to base evaluation of other areas of functioning. In this regard, intellectual capacity, as defined by most intelligence measures, is both a global characteristic and the interaction of several specific subdomains. As elaborated on later in regard to choice of test procedures, each intelligence test is constructed based on a particular characterization of global intelligence and the more specific skills of which it is composed. Although some children's intellectual endowment is relatively consistent across these diverse areas, many children display quite striking intellectual strengths and weaknesses. Psychological testing makes a major contribution to clinical care by elucidating the child's strengths and weaknesses and by identifying the ways in which they are manifested in behavior.

In clinical work with children and adolescents, it is often most useful to compare and contrast measured intelligence with results of individualized academic achievement testing. Appropriate academic achievement testing samples accomplishments in reading, arithmetic, spelling and written language, and more specialized skill areas as they may pertain to individual patients. Comparison of intellectual with academic test results allows the psychologist to respond to referral questions regarding the presence of learning disabilities. It is to be expected that academic achievement levels will be commensurate with intelligence levels (Kaufman and Kaufman, 1983; Sattler, 1988). When these levels are inconsistent, with academic achievement statistically significantly higher than intellectual levels, it is likely that the person has expended extreme effort to attain educational goals. It is also possible, although less common, that the person's true intellectual potential is greater than his or her measured intelligence. In contrast, when academic achievement levels are significantly lower than intelligence levels, the child may be considered learning disabled in one or more areas of academic functioning. It is critical to note that diagnosis of a learning disability denotes an inborn information-processing problem requiring particular special educational techniques (Kaufman and Kaufman, 1983; Sparrow and Blackman, 1985). In cases of learning disability, psychological test findings form the basis for educational placement and programming recommendations.

Clinical psychological assessment may also respond to questions regarding psychodiagnosis and psychotherapeutic treatment of children and adolescents. Use of projective test materials and of projective indicators on other instruments makes it possible to elucidate specific areas of psychopathology to aid in diagnosis. Very specific differential diagnoses can be considered, such as documentation in an adolescent of a thought disorder or an affective disorder, or differentiation between psychotic functioning and extreme anxiety in a younger child.

Once a diagnosis has been established, psychological assessment can also assist in treatment. Psychological test results can assist in the selection of the type of therapy to be attempted. For example, information regarding the integrity of a child's thought processes would be helpful in determining the appropriate level of the therapist's supportive activity in promoting the child patient's psychological insight (Pine, 1985). For a case in which testing provides assurance of solid, realistic thinking, intensive therapy may be attempted with confidence. In contrast, therapy would best be approached more cautiously and combined with more comprehensive services after testing that indicated frankly compromised reality orientation.

Similarly, test findings may help to predict the course of treatment. Test data about a youngster's degree of relatedness would help to foresee the child's likely response to a developing therapeutic alliance. For example, documentation of a child's association of intimacy with aggression would prepare a therapist for that youngster's psychological distancing after establishment of a close therapeutic relationship. Test results may also help to address a treatment impasse. Frequently, psychological assessment identifies patient characteristics that were less prominent earlier, attention to which would facilitate progress in otherwise stalled treatment.

Such identification is most helpful in highlighting the severity of disturbances that were previously considered less serious or remained unrecognized.

Psychological testing is also useful to answer very practical questions regarding disposition planning and program eligibility. Many programs designed to meet the special needs of certain children and adolescents, such as classes for the intellectually gifted, services for the retarded, and residential treatment centers, require documentation of need based on psychological assessment results. For children with less unusual needs, psychological test results can also assist with grade placement decisions. With disposition planning in mind, a psychological test battery can be designed efficiently.

## **PREPARATION OF PATIENT AND PARENTS**

For psychological testing to be most useful, the patient should be prepared adequately. With children and adolescents, such preparation typically begins with the parent. When the testing referral comes from another educational or mental health professional, the preparation should be initiated by the referring professional and should be completed by the psychologist.

The referring professional's preparatory function can be divided into three parts. First, the purpose of the testing should be explained to the parents in a clear, forthright manner. Referral questions should be discussed in nontechnical language so parents have a good understanding of the need for consultation. Second, the referring clinician should clarify his or her own ongoing relationship with the patient and parents, apart from the work with the testing psychologist. If the referring professional will continue to work with the child and family after the testing, this should be explained. If the testing referral actually constitutes a transfer to the psychologist for care, this, too, should be explained carefully. The usefulness of psychological testing is compromised severely when parents are confused about the psychologist's role in the provision of care. Finally, pragmatic directions should be provided about how to make contact with the psychologist for the assessment consultation. This sort of direction increases the likelihood that the referral will proceed smoothly.

Preparation of the young patient follows directly from parental preparation. Parents should be encouraged to share the preparatory information with their child and should present information in an honest, open manner appropriate to the child or adolescent's developmental level. In many cases, it is most helpful if the referring clinician assists the parents with this task. Several factors should be emphasized. First, it should be stressed that the testing is being pursued in an effort to provide relevant interpersonal or educational help to the child or adolescent, rather than as a means of being derogatory or punitive. Second, especially with younger children, it is helpful to assure the patient that the psychologist is a "talking doctor" who will not perform any physically invasive procedures. Preparation of the youngster best concludes with assurance that the psychologist will provide complete instructions, guidance, and assistance throughout the assessment.

Even when the patient is well and fully prepared, he or she usually meets the psychologist for the first time with considerable anxiety and denies receiving any information about the assessment to be completed. As part of establishing the necessary testing rapport, the psychologist should reiterate the preparatory information, instruct the child about testing procedures, and move on to carrying out the assessment. In most cases, careful preparation and establishment of initial rapport provide sufficient comfort to enable the youngster to cooperate easily in the collection of reliable and valid test data.

## **PSYCHOLOGICAL TESTING IN RELATION TO CHANGES IN THIRD-PARTY PAYMENT**

In referring patients for psychological testing and in carrying out that testing, clinicians must be aware of changing regulations governing third-party payment. With the more traditional indemnity insurance plans that were prevalent in previous years, decisions about provider, timing, and extent of testing rested with the relevant professionals and the youngster's parents. Likewise, the financial aspect of the psychological testing remained a matter to be agreed on between parents and provider, with the parents later seeking insurance reimbursement as dictated by the terms of their specific insurance policy. Referring clinicians were rarely involved in any financial negotiations regarding psychological testing.

In contrast, with the advent of health maintenance organizations and a wide variety of managed-health care plans, many administrative and financial decisions central to the testing process came under the control of insurance company representatives. Provider choice, preapproval of particular test instruments, specification of number of hours allowed for testing, access to confidential information, and regulation of allowable fees came to be dictated by the insurance companies and their managed-care associates.

In the current insurance climate, it is necessary for clinicians to be cognizant of insurance regulations when they make testing referrals. If use of insurance support is essential to the performance of the testing, clinicians must conform to rules governing choice of psychologist and referral procedures. To be granted access to insurance benefits, referring clinicians must also formulate highly specific referral questions and must specify how the questions will be addressed and to what use the answers will be put.

In some cases, useful psychological testing can be conducted under current insurance conditions. In many cases, however, more current insurance regulations constitute undue interference in the clinical testing process. The most qualified psychologists may not participate in a given plan. Instruments and number of hours approved may not allow for a sufficient response to referral questions. Confidentiality may be sacrificed in potentially damaging ways. If it is at all possible for a family to pursue testing without using insurance coverage, the referring clinician may do well to recommend a private agreement between parents and psychologist to ensure a higher quality of care.

## **TEST ADMINISTRATION**

This section reviews salient test procedures, psychometric issues, and examples of commonly used assessment instruments for each of the functional domains noted earlier. Although it is not an exhaustive list, the instruments included are those that the relevant literature and clinical experience have demonstrated to be of value in constructing test batteries that respond to most referral questions for psychological assessment.

### **Procedures**

#### *BATTERY COMPOSITION*

After clarification of the referral question, the child psychologist is faced with the task of making an initial determination about which instruments will comprise the assessment battery. In rendering this determination, the psychologist must weigh the wish to obtain the most comprehensive possible data with such realistic matters as the need for expeditious completion of the battery, the child's developmental capacity to withstand the rigors of testing, and developmental variables pertaining to the individual child's functioning across domains to be assessed ( [Goldman et al., 1983](#); [Siegel, 1988](#)). The psychologist then must consider adjusting the battery composition as emerging test data refine the known referral question or suggest new questions ( [Sattler, 1988](#)). For instance, if a child's performance on screening measures of academic achievement indicates deficits in acquisition of grade-appropriate reading skills, more intensive examination may be required to specify whether the child's deficits are more attributable to problems with reading decoding or reading comprehension.

#### *ADMINISTRATION*

The length of time required for a child to complete a test battery depends on certain variables, including the complexity of the referral question and the associated number of required test instruments, the child's capacity to sustain work on focused tasks, and the family's cooperativeness in keeping scheduled appointments. As a rule of thumb, experienced psychologists expect that the administration of a comprehensive psychoeducational assessment battery requires four to six sessions of from approximately 60 to 90 minutes each. Scoring test protocols absorbs an additional 1 to 2 hours of the psychologist's time.

Because the goal of testing is generally to obtain a picture of the child's maximum capacity, the psychologist must be certain of sufficient rapport to ensure the child's best effort. To accomplish this aim, the examiner employs the clinical skills, used by most clinicians in evaluating children, that serve to allay the child's anxiety and enlist cooperation in completing the tasks at hand. At the same time, however, the psychologist must bear in mind the distinguishing hallmarks of the psychological assessment enterprise, which are the acquisition of data in a structured setting using appropriate standardized measurements ( [Sparrow et al., 1985](#)). The psychologist must therefore be certain that the establishment of rapport does not violate standardized administration procedures permitting comparison of the child's test performance with those of the normative sample.

Although the principal focus of this chapter is on psychological assessment in outpatient settings, it was noted at the outset that changes in the current health care



climate have compelled psychologists working in inpatient settings to address creatively the need for more time-efficient testing that does not sacrifice the integrity of the testing process. In constructing appropriate test batteries in such settings, it is critical to define the limited number of specific questions to be addressed and to confine inferences accordingly. Typically, appropriate questions for testing under these conditions focus on ruling out major intellectual and psychiatric deficits or disorders (e.g., mental retardation and psychosis). A sample battery to fulfill these needs may include five subtests (Information, Vocabulary, Similarities, Block Design, and Picture Completion) from the Wechsler Intelligence Scale for Children, third edition (WISC-III) (Wechsler, 1987b), the Vineland Screener ( [Sparrow et al., 1994a, b](#)), and the Rorschach test ( [Rorschach, 1921](#)). Each of these instruments is discussed later in this chapter.

## Psychometric Issues

An extensive literature exists pertaining to technical and methodologic principles associated with psychological assessment ( [Anastasi, 1988](#); [Sattler, 1988](#); [Sparrow et al., 1984](#)). To judge the utility of assessment instruments, this literature can be distilled to three concepts: validity, reliability, and standardization.

### VALIDITY

An instrument's *validity* concerns the extent to which it measures what it purports to measure. In summarizing the Standards of Educational and Psychological Testing ( [American Psychological Association, 1985](#)), [Anastasi \(1988\)](#) found that the many methods for determining validity can be organized into three principal groups. An instrument possesses *content-related validity* to the extent that its item content covers a representative sample of the measured domain, a validation issue typically considered in evaluating achievement tests ( [Anastasi, 1988](#)). If an instrument is effective in predicting an person's performance in specified activities, it is said to possess *criterion-related validity*. This form of validity is determined by checking test performance against a direct and independent criterion measure of that which the test is supposed to measure, for instance, by comparing scholastic aptitude test scores with grades obtained in college. Finally, a test's *construct-related validity* refers to the test's capacity to measure a theoretical construct on which the test is based. Derived from documented interrelationships among behavioral measures, this form of validation requires the accumulation of various types of information, including correlations with other tests and statistical techniques such as factor analysis and measurements of the test's internal consistency.

### RELIABILITY

An instrument's *reliability* refers to the degree to which results obtained from the instrument can be reproduced within acceptable levels of agreement. The most commonly required measures of consistency for a psychological test are that its scores can be reproduced over time (*test-retest reliability*) and across examiners (*interrater reliability*). An additional estimate of reliability that is frequently reported is *interitem consistency*, or the consistency of responses to all items in the test ( [Anastasi, 1988](#)). This form of reliability provides information about the degree of homogeneity of the domain which the test is purported to sample and is typically reported in the form of reliability coefficients (either the Kuder-Richardson coefficient for dichotomous responses or Cronbach's coefficient  $\alpha$  for continuous responses) ( [Cronbach, 1951](#); [Kuder, 1937](#); [Novick and Lewis, 1967](#)).

### STANDARDIZATION

To be well *standardized*, an instrument should be pretested on a large, demographically representative sample of subjects. Comparison of a given child's performance on a test with an appropriate standardization group can occur only if such standardization has been conducted ( [Sparrow et al., 1985](#)). Such comparisons provide a context for understanding the child's test performance from a developmentally informed perspective and permit more powerful inferences about strengths or deficits documented in the child's testing.

## Commonly Used Assessment Instruments

The tests presented in this section ( [Table 43.1](#)) are organized according to the functional domain assessed (i.e., intellectual ability, academic achievement, visual-motor functioning, adaptive behavior, attentional capacity, personality organization, neuropsychological functioning, and developmental status). The discussion of each test includes a description of its purpose, format, and appropriate subjects, as well as commentary about the types of scores derived, administration time, psychometric properties, and unique features.

**Table 43.1. Commonly Used Child and Adolescent Psychological Assessment Instruments**

### MEASURES OF INTELLECTUAL ABILITY

#### *Wechsler Intelligence Scale for Children, Third Edition (WISC-III)*

Like its two predecessors, the original WISC ( [Wechsler, 1949](#)) and the WISC-R ( [Wechsler, 1974](#)), the WISC-III ( [Wechsler, 1991](#)) is a downward extension of adult intelligence scales ( [Wechsler, 1955, 1981](#)). In its revision published in 1991, it remains the most widely used individually administered intelligence test for children in the age range for which it was designed, 6 years to 16 years, 11 months. The WISC-III provides several welcome improvements, including updated and more culturally sensitive test items, more user-friendly materials (such as larger color, as opposed to black-and-white, stimulus cards for the Picture Arrangement subtest), and a restandardization providing more contemporary and psychometrically sound norms and standard cores. The WISC-III consists of 13 subtests divided into a six-subtest Verbal scale and a seven-subtest Performance scale (for a description of the 12 subtests retained from the WISC-R and capacities tapped by each, see [Sattler, 1988](#)). The Verbal Scale is composed of the Information, Similarities, Arithmetic, Vocabulary, Comprehension, and Digit Span subtests, and the Performance Scale includes the Picture Completion, Picture Arrangement, Block Design, Object Assembly, Coding, Symbol Search, and Mazes subtests. Symbol Search is a subtest developed for the WISC-III and is designed to measure processing speed. Administration time for the total instrument is approximately 60 to 90 minutes.

The instrument was constructed on the notion that intelligence may be defined as a person's overall capacity to understand and cope with the world ( [Wechsler, 1991](#)). The author emphasizes that this definition requires an assessment instrument that appreciates the global nature of intelligence and that avoids placing undue emphasis on any one ability in assessing overall intellectual capacity. The subtest tasks therefore attempt to challenge a child's abilities in a broad variety of ways, to provide a fuller estimate of capacity. Tasks require children to answer verbal queries and to work several types of visual or visual-motor puzzles.

The test generates Full Scale, Verbal, and Performance intelligence quotient (IQ) scores, individual subtest scaled scores, and test-age equivalents. Factor analytic research has been conducted and summarized in the manual ( [Wechsler, 1991](#)), and it reveals four principal factorial underpinnings of the WISC-III: Verbal Comprehension (Information, Similarities, Vocabulary, and Comprehension), Perceptual Organization (Picture Completion, Picture Arrangement, Block Design, and Object Assembly), Freedom from Distractibility (Arithmetic and Digit Span), and Processing Speed (Coding and Symbol Search). To facilitate scoring and interpretation, the WISC-III provides easy computation of Index Scores for each of these factors.

Interpretation of the test protocol requires several levels of analysis ( [Kaufman, 1979](#); [Sattler, 1988](#)), including placing of the global IQ scores into ranges of intellectual functioning ( [Table 43.2](#)), calculating the significance of any difference between Verbal and Performance IQ scores, comparing the Verbal Comprehension, Perceptual

Organization, Freedom from Distractibility, and Processing Speed index scores, determining the presence of significant individual relative subtest strengths and weaknesses, conducting comparisons of individual subtest scores, and appreciation of the quality of individual item responses. This approach to profile analysis permits generation of hypotheses about cognitive strengths and weaknesses based on statistically significant differences among scores.

Standard Score	Level
130+	Very superior
120-129	Superior
110-119	High average
90-109	Average
80-89	Low average
70-79	Borderline
50-69	Mild retardation
40-49	Moderate retardation
20-39	Severe retardation
Below 20	Profound retardation

**Table 43.2. Commonly Used Intelligence Classification**

The psychometric properties of the WISC-R have been extensively researched, and this research base provides a foundation for the psychometrics of the WISC-III. Reliability is reportedly outstanding (Sattler, 1988; Wechsler, 1974; Wechsler, 1991), with steadily strong internal consistency reliability coefficients (Sattler, 1988; Wechsler, 1974). Sattler also summarizes the plethora of research studies that, collectively, document the instrument's satisfactory concurrent, criterion, and construct validities (Sattler, 1988).

*Wechsler Intelligence Scale for Children, Third Edition as a Process (WISC-III PI)*

A newer test instrument, the WISC-III as a process (WISC-III PI) is designed for use along with or after completion of the WISC-III (Kaplan et al., 1999). Aiming to provide greater insight into the processes by which children or adolescents (ages 6 years, 0 months to 16 years, 11 months) carry out cognitive functions, the WISC-III PI consists of a variety of subtests. Some of these subtests are essentially alternate scoring or administration procedures for standard WISC-III subtests. Others are variations of WISC-III subtests intended to illuminate a range of ways to gain access to cognitive information. Finally, some new subtests are designed specifically for the WISC-III PI to measure cognitive operations related to those on existing WISC-III subtests. Coauthored by well-respected experts in the fields of neuropsychology and psychological assessment, the WISC-III PI makes it possible to investigate low WISC-III scores, to obtain a more detailed summary of a child's strengths and weaknesses, to assist with diagnostic decisions, to help monitor progress, and to support intervention planning. The examiner chooses those parts of the WISC-III PI most relevant to a specific youngster. Standardized scores, percentiles, and age equivalents are derived and are normed to be comparable to most psychoeducational tests in common use. Reliability and validity of the WISC-III PI are good and allow for confident use of the instrument. As educators increasingly request information about students' cognitive processing abilities, in addition to their cognitive levels, the WISC-III PI should gain wider usage.

*Wechsler Adult Intelligence Scale, Third Edition (WAIS-III)*

As noted earlier, the WISC-III represents a downward extension of Wechsler's theories of assessing adult intelligence. The most recent revision of the adult instrument is the Wechsler Adult Intelligence Scale, third edition (WAIS-III), published in 1997 (Wechsler, 1997a). The WAIS-III retains the formulations of the original WAIS but provides more current normative data, updated test content and materials, and improved administration procedures. The format of the WAIS-III is similar to that of the WISC-III in that the test yields Verbal, Performance, and Full-Scale IQ scores along with four Global Index Scores, Verbal Comprehension, Perceptual Organization, Working Memory (a score comparable to the WISC-III Freedom from Distractibility Index), and Processing Speed. These Global Indices are, in turn, based on scores on 14 subtests. Eleven of these subtests are retained from the WAIS-R; three subtests are new. Like the WISC-III, the WAIS-III now also has a Symbol Search subtest to measure efficiency of visual scanning and recognition. The WAIS-III also now includes Matrix Reasoning, a test of nonverbal inferential reasoning ability, and Letter-Number Sequencing, a measure of auditory attention and working memory. Test profile analysis on the WAIS-III is similar to that on the WISC-III. Like the WISC-III, the WAIS-III has excellent statistical properties (Wechsler, 1997a).

The WAIS-III is appropriate for use with persons between the ages of 16 and 89 years. This instrument may therefore be employed in testing adolescents who are more than 15 years old. The age ranges of the WISC-III and the WAIS-III overlap between the ages of 16 years and 16 years, 11 months. Sattler states that a more thorough sampling of ability can be obtained with the children's instruments when it is used with children of below-normal ability in the overlapping age range (Sattler, 1988). For normal and gifted children, however, both instruments provide adequate sampling.

*Wechsler Preschool and Primary Scale of Intelligence—Revised (WPPSI-R)*

Published in 1989, the Wechsler Preschool and Primary Scale of Intelligence—revised (WPPSI-R) (Wechsler, 1987) is a revision of the original WPPSI (Wechsler, 1967), a further downward extension of the Wechsler scales for use with children between the ages of 3 years and 7 years, 3 months. Several significant improvements have been incorporated in the revision to address concerns and limitations raised about its predecessor. For instance, the WPPSI-R age range represents an extension of approximately 1 year both upward and downward. To provide greater comparability to the WISC-III, the WPPSI-R adds a 12th subtest (Object Assembly) to the WPPSI's 11 subtests. Eight of the subtests (Information, Vocabulary, Arithmetic, Similarities, Comprehension, Picture Completion, Mazes, and Block Design) are also found on the WISC-III, whereas three subtests (Sentences, Animal House, and Geometric Design) were developed exclusively for the WPPSI and have been retained in the revision. New items and changes in design intended to enhance interest among young children have made the revision more user friendly. In the tradition of the other Wechsler scales, the WPPSI and WPPSI-R possess well-documented reliability and validity and are standardized adequately (Anastasi, 1988; Sattler, 1988; Sparrow et al., 1985; Wechsler, 1967; Wechsler, 1987). The interpretation of results follows the same course as noted earlier.

*McCarthy Scales of Children's Abilities (MSCA)*

As noted by Kaufman and Kaufman (1977), the McCarthy Scales of Children's Abilities (MSCA) (McCarthy, 1972) comprise a welcome instrument appropriate for the assessment of children between the ages of 2 years, 6 months and 8 years, 6 months. Its 18 subtests are clustered into five domains (Verbal, Perceptual-Performance, Quantitative, Memory, and Motor), and also allow for the calculation of a General Cognitive Index, which serves as the equivalent of the Full-Scale IQ on the Wechsler scales. Administration time is approximately 45 to 60 minutes, depending on the subject's age. Particular advantages of the MSCA are its fine standardization and psychometrics, ease and enjoyment of administration for both examiner and subject, and direct measurement of fine and gross motor skills and acquisition of eye, hand, and leg dominance, which are of particular importance in this age group (Sattler, 1988; Sparrow et al., 1985). The MSCA does not include tasks tapping social judgment. To compensate for this shortcoming, Kaufman and Kaufman encourage administration of the appropriate subtest of the WPPSI-R or WISC-III (Kaufman and Kaufman, 1977; Kaufman and Kaufman, 1983).

As discussed, the MSCA has had a well-earned place among good measures of children's intelligence. At this point, however, the test is quite old and is not widely used. To return to wider use, a thorough revision of the test would be required.

*Kaufman Assessment Battery for Children (K-ABC)*

The Kaufman Assessment Battery for Children (K-ABC) (Kaufman and Kaufman, 1983) measures the cognitive abilities of children between the ages of 2 years, 6 months and 12 years, 5 months. The K-ABC differs from other assessment instruments in its purpose and its theoretical basis. The test attempts to distinguish between a child's acquired knowledge (Achievement) and intellectual capacity as understood from the perspective of cognitive processing abilities. The K-ABC is firmly rooted in theories of cognitive psychology and neuropsychology, which posits that intelligence should be understood in terms of a child's relative facility in processing simultaneously or sequentially presented information. The processing of these different forms of information is understood to require different cognitive styles and to draw from different lateralized hemispheric brain functions, with sequential processing primarily associated with left-brain functioning and simultaneous



processing with activities in the right hemisphere. Reliability levels are highly acceptable and numerous validation studies have appeared in print. The standardization sample closely matches the 1980 U.S. Bureau of the Census data for age, gender, race, geographic region, community size, and socioeconomic status.

The K-ABC requires approximately 45 to 75 minutes of administration time. Its 10 mental processing subtests are divided into Sequential Processing (Hand Movements, Number Recall, and Word Order) and Simultaneous Processing (Magic Window, Face Recognition, Gestalt Closure, Triangles, Matrix Analogies, Spatial Memory, and Photo Series) Scales. A Mental Processing Composite score can be derived from these subtest scores that is equivalent to a Full-Scale IQ score on the Wechsler scales. Six subtests (Expressive Vocabulary, Faces and Places, Arithmetic, Riddles, Reading/Decoding, and Reading/Understanding) contribute to the Achievement Scale. The number of subtests administered varies according to the child's age.

Clinical experience indicates that because of its Mental Processing and Achievement scale construction, the K-ABC provides an efficient means of comparing a child's intellectual capacity and level of academic achievement, a comparison at the core of assessment of learning disability. Conversely, although research has suggested possibilities for richer profile interpretation ( [Kamphaus and Reynolds, 1987](#)), K-ABC profile interpretation does not possess the same heuristic value as the Wechsler scales to generate hypotheses about the interplay between intrapsychic conflict and selective ego deficits. Additionally, critics have labeled the distinction between mental processing ability and acquired knowledge faulty because of the test's artificial distinction between the ways in which children acquire and process information ( [Sattler, 1988](#)).

Since its first publication, the K-ABC has proved to be a well-accepted and useful test instrument for children and adolescents. Over time, several of the items have become outdated, however. In particular, items on the Faces and Places subtest, a test of general factual knowledge, have fallen out of popular awareness and are no longer commonly recognized. At the time of this writing, the K-ABC is being updated to allow it to remain a valuable test instrument.

#### *Kaufman Adolescent and Adult Intelligence Test (KAIT)*

As outlined in an interview with Alan Kaufman presented in Kamphaus' *Clinical Assessment of Children's Intelligence* ( [Kamphaus, 1993](#)), the Kaufman Adolescent and Adult Intelligence Test (KAIT) is an instrument that derives its theoretical foundation from three sources: the Cattell–Horn theory of fluid and crystallized intelligence; Piaget's concept of formal operational thinking; and Luria and Golden's articulation of the planning functions of the prefrontal lobes ( [Kaufman and Kaufman, 1993a](#)). The Cattell–Horn theory is the primary of these three sources and guides the test's organization into Fluid and Crystallized scales. These scales measure novel problem-solving skills and knowledge derived from acculturation and education, respectively. This theoretical construction is developmentally informed and has received strong empirical support, because research has consistently demonstrated that crystallized skills are maintained throughout the life span, whereas fluid skills plummet after peaking in young adulthood. As is true of the Kaufmans' other published tests, the KAIT is well standardized and reports very strong reliability coefficients. Additionally, the authors report construct validity through correlations with other commonly used intelligence tests (WISC-R, WAIS-R, K-ABC), and through studies with a variety of adult clinical populations.

The KAIT is designed to be administered efficiently, requiring only 60 minutes for its Core Battery of six subtests. An Expanded Battery, including four additional subtests, can be used for further clinical or neuropsychological assessment, and customized assessment can be obtained by selecting from among any or all of these additional subtests. The Core Battery's three-subtest Fluid Scale consists of Rebus Learning, Mystery Codes, and Logical Steps, and the Crystallized Scale is composed of Auditory Comprehension, Double Meanings, and Definitions. The Expanded Battery subtests are Memory for Block Design, Famous Faces, Rebus Recall, and Auditory Recall. The 10 subtests provide age-based scaled scores, and global IQ scores are generated for the Fluid Scale, Crystallized Scale, and Composite Intelligence.

Preliminary clinical experience with the KAIT indicates that the instrument is challenging to both administrator and subject and should not be administered without substantial practice on the part of the examiner. Test items often prove difficult for adolescents with less intellectual ability, and examiners may wish to administer another instrument in conjunction with the KAIT, to be most confident of test findings.

#### *Kaufman Brief Intelligence Test (K-BIT)*

Requiring an administration time of 15 to 30 minutes, the Kaufman Brief Intelligence Test (K-BIT) ( [Kaufman and Kaufman, 1990a, b](#)) is designed to provide a quick estimate of intelligence, either as an indicator for, or alternative to, a comprehensive intelligence test. The individually administered test can be used across a very wide age span, ranging from 4 to 90 years, and can be administered by trained nonpsychologists. The K-BIT consists of a Vocabulary subtest, which measures word knowledge. The first part of the subtest is administered to all ages and requires an oral label for pictured objects. The second part of the Vocabulary subtest requires the subject to name a word based on a brief description and partial spelling of the word. This second part of the subtest is administered only to persons age 8 years and older. The K-BIT also contains the Matrices subtest, which measures nonverbal intellectual reasoning ability. A strength of the test, which it shares with the K-ABC, is that the examiner is allowed to teach during administration of the early items, to ensure that the subject's best abilities are tapped. The K-BIT yields standard scores normed to be comparable to the major, commonly used full intelligence and academic achievement tests and is easily interpretable. As is true for other tests authored by the Kaufmans, the K-BIT norms are derived from a representative normative sample stratified to conform to the U.S. Bureau of the Census figures. Furthermore, the test rests on a very sound statistical foundation and boasts excellent reliability and validity. Clinical practice bears out the test authors' assertions regarding the utility of the test. The K-BIT serves a very useful purpose in providing easy access to a trustworthy estimate of a person's intellectual ability level. This easy access is particularly valuable among persons who find it difficult to tolerate full administration of a longer intelligence measure.

#### *Leiter International Performance Scale—Revised (Leiter-R)*

Development of the original Leiter International Performance Scale ( [Roid and Miller, 1997](#)) began in the 1920s. Developed by Dr. Russell Graydon Leiter, the first commercial version of the test was published in 1940. Designed to provide a comprehensive measure of nonverbal intelligence, the original Leiter Scale was used widely for the assessment of diverse populations. Over the years, however, the test materials became outdated, and comparability of Leiter scores with scores on other major test instruments was limited. In addition, it became clear that the Leiter Scale could benefit from application of more modern statistical methods of test construction. Accordingly, Gale H. Roid, Ph.D., and Lucy J. Miller, Ph.D., revised the test. As currently constructed, the revised Leiter Scale (Leiter-R) is designed to measure nonverbal intelligence in persons from 2 years to 21 years of age. On the Leiter-R, nonverbal intelligence is defined as fluid reasoning, visualization, visuospatial memory, attention, and memory. Presented in a tabletop easel format and consisting of attractive, contemporary materials, the Leiter-R is made up of two main groups of subtests, Visualization/Reasoning and Attention/Memory. The test also includes behavior rating scales that capture behavioral observations made during testing. Administered on an individual basis, the Leiter-R can be used in whole or in part, depending on individual needs. The full battery of Leiter-R subtests requires approximately 90 minutes for completion. A full range of standardized scores and percentiles can be derived on the Leiter-R. Standardized on a nationally representative sample, the Leiter-R has good reliability and validity. Given its long history of valuable use and the integrity of its current construction, the Leiter-R should be considered as a valuable tool for the assessment of persons with significant verbal communication difficulties associated with cognitive disorders, developmental disorders, hearing impairments, motor impairments, traumatic brain injury, attentional disorders, learning disabilities, and examiner–subject language incompatibility. The Leiter-R has particular utility for lower-functioning persons on the autistic spectrum. Because the test requires no verbal output from the subject and a minimum of interpersonal interaction, persons on the autistic spectrum are often able to demonstrate more ability on the Leiter-R than they would on other, more traditional instruments.

#### *Stanford-Binet Intelligence Scale, Fourth Edition (SB:FE)*

The Stanford-Binet Intelligence Scale, fourth edition (SB:FE) ( [Thorndike et al., 1986](#)) represents the latest revision of an instrument whose modern history dates back to 1916 and whose roots extend back to at least 1905. This version represents a significant departure from the 1937 Forms L and M and the 1960 Form L-M, for which revised norms were provided in 1972. A three-level theoretical model guided the construction of the instrument and postulated the following: a general intelligence factor (g) at the highest level of inference; a second level of factors, including crystallized, fluid, and short-term memory; and a final level comprising more specific factors such as verbal, quantitative, and abstract visual reasoning. Traditionally, a major advantage of the Stanford-Binet test has been its broad age range, permitting more accurate assessment of persons functioning at either extremely low or high cognitive levels. A significant criticism of the SB:FE is its lack of a comparable battery throughout the scale's age range, meaning that scores obtained by children at different ages are based on different subtest combinations ( [Sattler, 1988](#)).

Administration time for the SB:FE may be quite long, in some cases exceeding 2 hours, because the instrument includes only a single timed subtest. This is a significant drawback for use of the test. The 15 subtests of the SB:FE are divided into four larger areas: Verbal Reasoning (consisting of the Vocabulary, Comprehension, Absurdities, and Verbal Relations subtests); Abstract/Visual Reasoning (Pattern Analysis, Copying, Matrices, and Paper Folding and Cutting); Quantitative Reasoning (Quantitative, Number Series, and Equation Building); and Short-Term Memory (Bead Memory, Memory for Sentences, Memory for Digits,

and Memory for Objects). Standard scores are generated for the subtests, the four areas, and a Composite Score similar to the Wechsler IQ score. Because later factor analytic studies did not support the original factorial structure of the test, [Sattler \(1988\)](#) suggests an alternate, more valid set of factor scores and means of computation. The psychometric properties of the test are very good, with excellent reported reliability. The SB:FE is well standardized on a population similar to that of the 1980 U.S. Bureau of the Census, and weighting procedures are used to ensure maximum conformity to census data. Many validation studies summarized by [Sattler \(1988\)](#) conclude that the SB:FE and the Wechsler scales yield similar scores for subjects in the average range of cognitive functioning, but the SB:FE may produce lower scores for gifted or mentally retarded subjects. Because of the limitations noted, clinical experience suggests that the employment of other instruments (Wechsler scales, MSCA, and K-ABC) be ruled out before using the SB:FE.

#### *Peabody Picture Vocabulary Test, Third Edition (PPVT-III)*

A revision of the earlier PPVT-R ([Dunn and Dunn, 1981](#)), the PPVT-III was designed for persons between the ages of 2½ and 9< years. The Peabody Picture Vocabulary Test, third edition (PPVT-III) ([Dunn and Dunn, 1997](#)) is an individually administered, untimed test intended to measure receptive vocabulary and to serve as a screening instrument of verbal ability. The test is available in two forms: PPVT-III-A and PPVT-III-B. Each form includes four training items and 204 test items. Each of the test items consists of four black-and-white pictures arranged on a page contained in a tabletop easel. For each item, the examiner states a word, and the test subject must designate the picture that best matches the spoken word. Thus, the test is very easy to administer, requires only about 11 to 12 minutes to complete, and is easily scored. The PPVT-III yields standard scores, percentiles, normal curve equivalents, stanines, and age equivalents. Well standardized on a nationally stratified representative sample, the test has excellent reliability and validity. When it is used in conjunction with the Expressive Vocabulary Test (EVT), discussed later, it is possible to derive comprehensive information about a person's single-word vocabulary. Finally, because the PPVT-III is brief, simple to administer, and requires only a pointing response from the subject, it often serves as a good "ice-breaker" when a full test battery is to be administered.

#### *Expressive Vocabulary Test (EVT)*

The EVT ([Williams, 1997](#)) is a measure of word retrieval and expressive vocabulary designed for use with persons between 2½ and 9< years of age. Administered on an individual, untimed basis, the test consists of teaching items and 190 test items. Contained in a tabletop easel, items are divided into an early picture labeling section and a larger, later section that requires the subject to supply a synonym for a stated word accompanied by a picture. Like the PPVT-III, the EVT is simple to administer, requires only 10 to 25 minutes to complete, and is easily scored. The statistical properties of the test are excellent in that it is normed on a nationwide sample stratified to conform to the latest U.S. Bureau of the Census figures, item bias is analyzed and inappropriate items are eliminated from the test, reliability and validity are very good, and the test yields standard scores, percentiles, normal curve equivalents, stanines, and age equivalents that are comparable to those of most other major intellectual measures. The EVT is a very useful tool for screening expressive language problems, determining school readiness, gaining a better understanding of reading difficulties, monitoring development, and assessing progress in mastering English as a second language. In addition, because of its wide age range, the test has great utility for research.

An added attraction of using the EVT is that it is conormed with the PPVT-III. This conorming allows for direct comparison of expressive and receptive vocabulary skills. A significant difference between PPVT-III and EVT scores is to be understood as an index of word retrieval skill.

### *MEASURES OF ACADEMIC ACHIEVEMENT*

#### *Kaufman Survey of Early Academic and Language Skills (K-SEALS)*

The Kaufman Survey of Early Academic and Language Skills (K-SEALS) ([Kaufman and Kaufman, 1993b](#)) fills an important need in the assessment of 3- to 6-year-old children. Although little formal academic knowledge is expected of such young children, it is often useful to assess youngsters' progress in acquiring a foundation of preacademic knowledge. The K-SEALS serves exactly this function. Individually administered in approximately 15 to 25 minutes by both professional and trained nonprofessionals, the K-SEALS consists of three subtests: Vocabulary, a measure of word knowledge; Numbers, Letters, and Words, a test of early recognition of letters and numerals, knowledge of relative quantity and size, and early reading and math skills; and Articulation, a test of skill at pronouncing words correctly. All subtests use pictorial cues on a tabletop easel and require oral responses. Across subtests, the test distinguishes between receptive and expressive skills, as well as between letter and word, as opposed to number, skills. To facilitate easy comparisons between K-SEALS scores and other commonly used intelligence and achievement tests, the test yields standard scores, percentiles, classifications, and age equivalents normed in the same way as most of the other measures. The K-SEALS boasts excellent psychometric properties, that is, good reliability, fine validity, and relative lack of racial, ethnic, or gender bias.

#### *Woodcock-Johnson Psycho-Educational Battery—Revised*

The revised form of the Woodcock-Johnson Battery was published in 1989 ([Woodcock and Johnson, 1989](#)). The revision offers organizational, procedural, psychometric, and scoring improvements as well as new subtests. Although this revised version, like the original Woodcock-Johnson Battery, was published to provide an estimate of both intellectual capacity and academic achievement, it is not currently viewed as a major competitor with the cognitive assessment instruments listed earlier ([Sattler, 1988](#)). Rather, the Tests of Achievement comprising Part II of the Battery are more commonly used to estimate levels of scholastic accomplishment. The Achievement section of the battery measures reading decoding and comprehension skills, math reasoning and calculation skills, written language skills, and knowledge of specific subject areas (science, social studies, and humanities). In clinical practice, most extensive use is made of the Reading, Mathematics, and Written Language Clusters. Although efficient to administer, the instrument is cumbersome to score and, for many psychoeducational purposes, has been supplanted by the Kaufman Test of Educational Achievement (K-TEA) and the Wechsler Individual Achievement Test (WIAT), as discussed later. A third edition of the Woodcock-Johnson Battery has been published too late for review in this chapter.

#### *Wide Range Achievement Test, Third Edition (WRAT 3)*

Earlier versions of the Wide Range Achievement Test (WRAT), the original WRAT and the WRAT-R ([Jastak and Wilkinson, 1984](#)), were used for many years as brief screening instruments for identifying academic skills deficits ([Anastasi, 1988](#); [Sattler, 1988](#)). The current version of the test, the WRAT 3, was published in 1993 ([Wilkinson, 1993](#)). The 1993 restandardization aimed at retaining the ease of administration and reliability of the earlier editions of the test while updating the norms and making it possible to use the WRAT 3 for pretesting and posttesting of academic accomplishments. Accordingly, two equivalent forms of the test were designed for use with persons from 5 years, 0 months through 74 years, 11 months of age. Each form can be administered separately or in conjunction with one another. Both forms consist of three subtests: Reading, a quick measure of decoding skills; Spelling, a test of the ability to write letters and words from dictation; and Arithmetic, a test of basic computational skill. The test requires 15 to 30 minutes to complete and yields standard scores, grade equivalents, percentiles, and normal curve equivalents. The statistical properties of the WRAT 3 are sound, allowing the test to be used with confidence. The strength of the WRAT 3 is that it requires only a brief time to provide a solid assessment of academic skills. Because of its brevity, however, some academic domains receive shorter shrift; that is, there are no measures of reading comprehension or grasp of math word problems. As a result, the WRAT 3 is best used when administration time is a critical factor or when only a basic estimate of academic functioning is required.

#### *Kaufman Test of Educational Achievement (K-TEA), Brief and Comprehensive Forms*

The K-TEA ([Kaufman and Kaufman, 1985](#); [Kaufman and Kaufman, 1998a, b](#)) is a well-standardized screening instrument for assessing academic achievement in children 5 through 18 years of age. It is easy to administer and score, and it takes approximately 30 minutes to complete. Five subtests are included: Reading Decoding, Reading Comprehension, Mathematics Applications, Mathematics Computation, and Spelling. The Comprehensive Form provides a systematic approach for evaluating errors, which is of potential use to clinicians interested in devising curricular interventions to address skills deficits but which is also cumbersome for most examiners. The Brief Form, with a reduced item sample, is appropriate for most psychoeducational applications. Because it provides data about a child's functioning across domains of most general interest, possesses a reasonable item sample and good psychometric properties, and is easy to administer and score, the K-TEA has proven to be the academic achievement screening instrument of choice for children in its age range, especially when the test is used in conjunction with the MSCA or the appropriate Wechsler scale. Although there is some overlap of items between the K-ABC and the Achievement section of the K-TEA, each test does make a unique contribution to the assessment of a child's level of educational accomplishment. For instance, the K-TEA Reading subtest provides a wide range of item difficulty and permits a more refined assessment of reading skills. Conversely, the K-ABC Faces and Places and Riddles subtests assess aspects of acquired knowledge not tapped by the K-TEA.

The K-TEA has become a widely used academic achievement test. In 1997 and 1998, the test norms were updated to improve the statistical properties of the test further and to address intervening changes in curriculum and educational practices, demographics of the population, and overall cultural transitions. In comparing



original and updated norms, it appears that overall student performance improved in the years after the first publication of the K-TEA. As a result, a higher performance level is required to earn a particularly high score using updated K-TEA norms.

#### *Wechsler Individual Achievement Test (WIAT)*

An addition to the available library for the individual assessment of academic achievement, the WIAT ([Wechsler, 1992](#)) is designed to accompany the Wechsler intelligence scales. The instrument was developed for use with children in kindergarten through grade 12, and aged 5 years through 19 years, 11 months. The test requires approximately 30 to 60 minutes to administer the comprehensive battery, depending on age, although a briefer core battery can be administered as a screening instrument in approximately 15 minutes. The WIAT is well standardized and has the advantage of being conormed on the populations used to standardize the Wechsler intelligence scales, thus enhancing the psychologist's confidence in inferring discrepancies between intellectual ability and level of achievement. The Screener comprises three subtests: Basic Reading, Mathematics Reasoning, and Spelling. In conjunction with these three subtests, five additional subtests are included in the Comprehensive Battery: Reading Comprehension, Numerical Operations, Listening Comprehension, Oral Expression, and Written Expression. The last three subtests provide welcome data not typically available in academic achievement tests, and they permit a richer sample of a child's receptive, expressive, and written language functioning that may be contributing to academic performance deficits. Because of these unique aspects and its conorming with the most commonly used intelligence tests (the Wechsler scales), the WIAT has become a significant alternative to the K-TEA.

#### *Oral and Written Language Scales (OWLS)*

The OWLS ([Carrow-Woolfolk, 1996](#)) comprise a measure of receptive and expressive language, both oral and written, in children and adolescents. The test consists of three scales: Listening Comprehension, a measure of receptive understanding; Oral Expression, a test of skill at answering questions, completing sentences, or generating original verbal expression; and Written Expression, a measure of skill at using both the conventions of written language and linguistic forms, as well as skill at communicating meaningful ideas. Whereas tests such as the PPVT-III and EVT assess single-word vocabulary, the OWLS test also examines connected language and linguistic concepts. The oral language scales are for use with persons from the age of 3 through 21 years, whereas the written language scale is for use with subjects 5 through 21 years old. Administered individually, all scales can be used, or the examiner can select the specific scale needed to understand a particular youngster best. Administration time varies according to number of scales completed but generally ranges from half an hour to 1 hour. The OWLS test is standardized on a representative national sample and possesses good reliability and validity. It can be used with confidence to identify learning disabilities, to guide educational interventions, to monitor academic progress, and to contribute to research. The Written Expression Scale is particularly useful. Although there are many easily administered and easily scored measures of oral language, written language measures other than the OWLS tend to be more cumbersome, time consuming, and uninteresting to the subject. In contrast, the relative brevity, ease of administration, and variable item type of the OWLS makes it a much more pleasant instrument to use in assessing written language skill.

### **MEASURES OF ADAPTIVE BEHAVIOR**

#### *Vineland Adaptive Behavior Scales*

A revision of the Vineland Social Maturity Scale ([Doll, 1965](#)), the Vineland Adaptive Behavior Scales ([Sparrow et al., 1984](#)) assess personal and social sufficiency. A semistructured interview is conducted by a trained examiner with a respondent familiar with the subject. Four functional domains are assessed: Communication, Daily Living Skills, Socialization, and Motor Skills (up to age 4 years, 11 months). Domain scores, in turn, generate an Adaptive Behavior Composite. An additional checklist taps behaviors typically construed as maladaptive. The three forms of the instrument are the Survey Form, the Expanded Form, and the Classroom Edition. The Survey Form contains approximately half the items of the Expanded Form, requires approximately 1 hour to administer, and has proved sufficient for the purposes of most clinicians. The Expanded Form divides many of the skills on the Survey Form into discrete steps to be mastered and thus offers valuable programming assistance for work with low-functioning persons. The Classroom Edition is designed to be completed by a child's teacher in approximately 20 minutes and does not require an interview with the teacher. The strongest criticism leveled to date against the Vineland Scales is that there is apparently substantial fluctuation of means and standard deviations across age groups, thus limiting the extent to which the instrument can be used for longitudinal comparisons ([Silverstein, 1986](#)). In clinical practice, however, the scales have proved to provide an excellent estimate of adaptive behavior, which is of significant value in developing a fuller picture of a child's psychosocial functioning. As development of the *Diagnostic and Statistical Manual of Mental Disorders* proceeds toward publication of the fifth edition, it is anticipated that assessment of adaptive functioning will become even more central in psychiatric diagnosis. Thus, it should be expected that the utility of the Vineland Scales in clinical practice will increase.

With this anticipated increase in demand in mind, and to meet current demands for expedited assessment, the Vineland Screener was developed ([Sparrow et al., 1994a, b](#)). Although aimed at research needs with large subject pools, the Vineland Screener's high correlation with the full Vineland test and clinical requirements have led to its increasing use in clinical settings as well. The Screener is administered in the same semistructured interview format as the Survey and Expanded forms of the Vineland Scales. Key items have been selected from the full instrument to allow for speedier administration. Screener raw scores are then equated with raw scores on a fuller version of the Vineland Scales to serve as the basis for the calculation of derived scores. Although in use for a relatively short period, the Vineland Screener has demonstrated its usefulness both in research and in inpatient treatment settings, in which accommodation to shortened hospitalizations has become mandatory.

In the tradition of the original Vineland Social Maturity Scale, the Vineland Adaptive Behavior Scales have become a highly valued instrument in assessing a wide variety of referral questions. The Vineland Adaptive Behavior Scales have been translated into numerous languages and used around the world to document levels of personal and social self-sufficiency. As technology and societal practices change, however, types and standards of adequate adaptive functioning also change. For the Vineland Scales, changes such as the increasing academic, job, and leisure use of personal computers and widespread ownership of cellular phones have necessitated updating and revision of many items. As this chapter is written, a comprehensive revision of the Vineland Adaptive Behavior Scales is under way.

#### *Scales of Independent Behavior (SIB)*

The Scales of Independent Behavior (SIB) ([Bruininks et al., 1984](#)) comprise an individually administered measure of personal and social sufficiency across a broad age range. As opposed to the Vineland Scales, this test is not administered by a semistructured interview and permits estimates of the adaptive behavior functioning of nonretarded adults. In most cases, data are collected from an informant, but the person being assessed may also serve as the respondent. The SIB test was developed to be used in conjunction with the Woodcock-Johnson Broad Cognitive Ability Cluster Score to provide an efficient estimate of both adaptive ability and intellectual capacity. Research questioning the validity of the Cluster Score ([Sattler, 1988](#)), however, requires that clinicians proceed with caution in using these two instruments for the purposes advocated by the authors. The SIB test consists of 14 subtests that produce four adaptive behavior (Motor, Social Interaction and Communication, Personal Living, and Community Living) and three maladaptive behavior (Internalized, Asocial, and Externalized) clusters. The adaptive behavior clusters, in turn, generate a Broad Independence summary Cluster Score. The full SIB test requires approximately 1 hour of administration time, but a 15-minute Short Form has also been developed.

### **MEASURES OF ATTENTION AND CONCENTRATION**

Most recently, increased research and clinical attention have been aimed at gaining a more comprehensive understanding of ADHD. The fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* ([American Psychiatric Association, 1994](#)) summarizes much of the current thinking about this multifaceted disorder.

Advancement in knowledge about this disorder has been accompanied by a need for psychological test data to assist with differential diagnosis. Often, clinical psychologists have relied on indicators embedded in a traditional test battery to help confirm or rule out the diagnosis of ADHD. Such indicators have included behavioral observations in the test setting, Hand Movements performance on the K-ABC, Digit Span scores on the WISC-R, and, more recently, the Freedom from Distractibility Scale on the WISC-III. More currently, however, instruments have been developed that can allow for more direct determination of the accuracy of the ADHD diagnosis. The rationale for administering a variety of instruments is to ascertain the subject's capacity to marshal attention and concentration in response to stimuli presented through different sensory input modes and requiring different forms of response. This cluster should be administered and interpreted as part of a standard, thorough, psychoeducational assessment. Findings of attentional difficulty should be used to place a child accurately in an attentional, behavioral, academic, or emotional diagnostic category or to make the diagnosis of comorbid difficulties.

Given that ADHD is a difficulty in attending to a focused task on demand amid competing stimuli, it should not be surprising if ADHD symptoms do not manifest in the

controlled atmosphere of the psychologist's office. Accordingly, in addition to direct evaluation of the child or adolescent, data must be gathered from multiple observers in settings where the child encounters situational demands that make the emergence of ADHD symptoms more probable. To accomplish this task, relevant instruments can be administered to parents, teachers, and the identified child or adolescent. These instruments are discussed here, organized according to the particular informant or subject.

#### *Child and Adolescent*

**Controlled Word Association Test.** This test ([Benton and Hamsher, 1978](#)) was designed originally as part of the Benton Battery and was used for assessment of aphasia. In a 1-minute period, the subject is required to say as many acceptable words as possible beginning with a specified letter (i.e., F, A, or S). Successful completion requires continued attention. Average response scores are tabulated for each of the three specified letters and for the test as a whole. Normative data are available for children and adolescents ([Gaddes and Crockett, 1975](#)). Percentile scores can be calculated. To make valid interpretations of attentional capacity on this test, accurate information must also be available about the subject's level of knowledge about letter-sound combinations.

**Trail Making Test.** Derived from the Army Individual Test Battery ([U.S. Army, 1944](#)) and revised by [Reitan \(1971\)](#), this is a widely used and popular instrument ([Reitan, 1971](#)) requiring the subject to connect a series of numbered circles in Part A and a series of alternating numbers and letters in Part B under timed conditions. Most pertinent to assessment of ADHD, the test requires sustained concentration for successful completion. Normative information is available in the neuropsychological literature ([Spreen and Gaddes, 1969](#)), including means, standard deviations, medians, and score ranges.

**Wisconsin Card Sorting.** Designed originally in the 1940s and modified in the 1980s ([Heaton, 1981](#)), this test ([Berg, 1948](#); [Grant and Berg, 1948](#)) requires the subject to sort given cards into appropriate piles. The criteria for proper assignment must be determined by the subject using feedback provided either by the examiner directly or by a computer program. Attention to relevant card attributes, sustained concentration, and mental flexibility are all essential for successful task completion. Percentiles, T scores, and standard scores may all be derived from the raw scores.

**Computerized Tests.** Assessment of a child's or adolescent's ability to maintain attention and concentration on a somewhat monotonous, continuous performance task is helpful in the diagnosis of ADHD. Such computerized tests as VIGIL ([Cegalis and Volin, 1991](#)) and PASAT ([Cegalis and Birdsall, 1992](#)) are appropriate choices for this purpose.

#### *Parent*

**Behavior Assessment System for Children (BASC), Parent Rating Scales.** A relatively new addition to the collection of instruments available for assessment of childhood behavior, the BASC ([Reynolds and Kamphaus, 1992](#)) offers a comprehensive, structured assessment of a child's behavioral functioning across a variety of domains. Most salient in this discussion, the BASC contains an Attention Problems and a Hyperactivity Scale, as well as other clinical scales to help provide greater certainty in differential diagnosis. Test authors assert that the BASC is useful in diagnosing attentional deficit disorders with and without hyperactivity (Reynolds and Kamphaus, 1993). Parents are asked to rate 131 behaviors as occurring never, sometimes, often, or almost always. A full range of derived scores, including standard scores, percentiles, and age equivalents, may be derived.

**Home Situations Questionnaire—Revised (HSQ-R).** One of several behavior checklists popularized by Barkley and his colleagues ([Barkley, 1991](#)), the HSQ-R ([DuPaul, 1991a](#)) is a 14-item list of behaviors denoting attentional or concentration problems as they may manifest in home activities, such as riding in a car, playtime, social visiting, and attending religious services. Parents are asked to rate each behavior as either present or absent. If the behavior is rated as present, the parent is asked further to rate its severity. The scale yields total scores as well as factor scores differentiating between difficulty in compliance versus leisure situations. Means and standard deviations are provided. Scores more than 1.5 standard deviations above the mean for the age and sex of the child are considered indicative of diagnosable attentional difficulty.

**Attention Deficit Hyperactivity Disorder (ADHD) Rating Scale.** Another of the scales popularized by Barkley and his colleagues ([Barkley, 1991](#)), the ADHD scale ([DuPaul, 1991b](#)) is composed of 14 items. Each item refers to a problematic behavior suggestive of attentional problems. The parent is asked to rate the degree to which each behavior noted describes the individual of concern. Rating choices are not at all, just a little, pretty much, and very much. Again, the scale yields a total score and factor scores. Factors on this scale are Inattentive-Hyperactive and Impulsive-Hyperactive. Means and standard deviations are provided. These values are interpreted just as they are on the HSQ-R discussed earlier.

#### *Teacher*

**Behavior Assessment System For Children (BASC), Teacher Rating Scales.** The teacher form of the BASC ([Kamphaus and Reynolds, 1993](#)) is quite similar to the parent form in aim, format, scoring, and psychometric properties. The content of the scales is directed to relevant school-based situations, such as adjustment to new teachers, completion of assigned tasks, and disruptiveness of others.

**School Situations Questionnaire—Revised (SSQ-R).** Completed by teachers, this test ([DuPaul, 1991c](#)) is a parallel instrument to the HSQ-R described earlier. One of the instruments used by Barkley and those working with him (1991), the SSQ-R is an 87-item scale. Items refer to school-based settings and ask the rater to note the presence or absence of attentional difficulty in each setting and to rank the severity of the difficulty from mild to severe. Total scores and mean severity ratings are derived, and these scores are then compared with normative means and standard deviations. As on the other Barkley scales, scores more than 1.5 standard deviations above the mean are considered significant for ADHD.

**Attention Deficit Hyperactivity Disorder (ADHD) Scale.** This instrument ([DuPaul, 1991b](#)) is described earlier and is appropriate for use by teachers as well as parents. Whereas the parent completes the form to describe behaviors witnessed at home, the teacher targets school-based behaviors in completing the form.

**Child Attention Profile (CAP).** The CAP ([Edelbrock, 1991](#)) is a 10-item scale also popularized by Barkley and his group of colleagues ([Barkley, 1991](#)). The items refer to behaviors indicative of problems with attention and concentration relevant to completion of structured work. The rater is asked to designate each behavior as not true, somewhat or sometimes true, or very or often true of the child in question. The test yields a total score and two factor scores, Inattention and Overactivity. Means and standard deviations are provided. Scores more than 1.5 standard deviations above the mean are understood as clinically significant.

#### *PROJECTIVE TESTS*

##### *Rorschach Inkblots*

The Rorschach test ([Rorschach, 1921](#)) enjoys great popularity among clinicians ([Erdberg and Exner, 1984](#)). First described in 1921, the Rorschach test consists of 10 inkblots, each printed on a separate rectangle made of heavy cardboard. Five of the cards are monochromatic, two are black and red, and three are polychromatic. Standard administration consists of two phases: free association, in which the subject simply states what each blot may represent; and inquiry, in which the clinician seeks clarification from the subject about what aspects of the blot contributed to the percept identified in the free-association phase. Clinicians typically allow approximately 1 hour for administration time. Although some administration and scoring systems have been developed for use with the Rorschach test ([Beck, 1944](#); [Klopfer, 1954](#)), the work of Exner and colleagues ([Exner, 1978, 1986](#); [Exner and Weiner, 1982](#); [Wiener-Levy and Exner, 1981](#)) represents the most ambitious effort to establish a psychometrically sound basis for scoring this instrument ([Anastasi, 1988](#)). The Rorschach test is primarily used as a clinical device for assessing personality organization and, with children, also provides data regarding the developmental status of such organization ([Zlotogorski, 1986](#)). Particularly significant data are generated regarding a child's developmental capacities for reality testing, integration of affect, and maturational level of object relations. The Rorschach test has been subjected to extensive research that establishes its satisfactory reliability, particularly since the introduction of Exner's Comprehensive System ([Anastasi, 1988](#); [Zlotogorski, 1986](#)).

Computer software has been developed for scoring Rorschach protocols according to the Exner system. The computer scoring yields both quantitative and qualitative interpretive information. The computer scoring facilitates efficiency and objectivity in scoring Rorschach protocols. The comprehensive information provided by the scoring program offers the broadest information possible. Computerized scoring is not, however, a good substitute for the careful sequential analysis of responses carried out by an experienced examiner.



### *Thematic Apperception Test (TAT)*

The Thematic Apperception Test (TAT) ([Murray et al., 1938](#)) consists of 20 picture cards, one of which is blank and 19 of which vary in their degree of ambiguity. Cards are presented one at a time, and the child is asked to tell a story, as opposed to simply stating what the card portrays. The story is to include a beginning, a middle, and an end and is to describe something about the thoughts and feelings of the characters. The story is recorded verbatim by the clinician who may actively request clarification of the story as it is being told. After administration of the test, the clinician analyzes the story to determine the identification of the story's hero, the hero's attributes, and the conflicts, defenses, and adaptations evidenced in the hero's individual and interpersonal functioning. The underlying assumption of this analysis is that the respondent makes particular use of the hero as the object for unconscious projections and identifications.

According to many authors (e.g., [Anastasi, 1988](#); [Siegel, 1987](#); [Siegel, 1988](#)), the TAT is appropriate for use with older children, adolescents, and adults. Other investigators ([Goldman et al., 1983](#)) point out that Murray originally intended his method for use with persons who are more than 4 years of age. Although thematic assessment instruments have been developed for use with younger children, between approximately the ages of 3 and 10 years (e.g., Children's Apperception Test, [Bellak and Bellak, 1961](#); Roberts Apperception Test for Children, [McArthur and Roberts, 1982](#)), in clinical practice the TAT is generally used as the apperception test of choice. This is because the TAT, by virtue of its greater stimulus ambiguity, is considered to elicit more efficiently the subject's projections, and because greater examiner familiarity with the TAT engenders more of an internal frame of reference and face validity. The publication of a version of the Children's Apperception Test in which stimulus cards depict human as opposed to animal figures has generated enthusiasm among clinicians. These pictures appear more developmentally appropriate and better preserve the valuable ambiguity that is a hallmark of the TAT.

Typically, clinicians administer eight to 10 cards selected for the individual child or adolescent being tested. Although this approach reduces the instrument's reliability, it generates a rich lode of clinical data for use in deriving clinical hypotheses that may be confirmed or denied elsewhere in the test battery.

### *Machover Draw-A-Person Test (DAP)*

In this commonly used projective test ([Machover, 1949](#)), the subject is provided with a piece of standard-size, 8½" x 11" paper and a pencil with an eraser and is instructed to draw a person. The only constraints placed on the figure are that it portrays the entire body and that it not be drawn as a "stick figure." On concluding this drawing, the subject is then asked to draw a person of the opposite gender from that of the initial drawing. The examiner notes the subject's behavior, verbalizations, and demeanor while drawing and the sequence in which body parts are drawn. The productions are then subjected to a qualitative analysis, focusing on such variables as figure size, page placement, arm position, clothing, significant omissions and disproportions, shading, and erasures ([Anastasi, 1988](#)).

[Machover \(1949\)](#) provides a guide for interpretations of drawings without providing data in support of those interpretations. Research has been conducted that fails to validate Machover's diagnostic inferences ([Klopfer and Taulbee, 1976](#)). In clinical practice, the Machover Draw-A-Person Test (DAP) is typically used as a source for generating clinical hypotheses. It may be helpful to conceptualize two broad categories of such hypotheses, which may be derived from qualitative analysis of the DAP: (a) hypotheses pertaining to the subject's perception of and feelings about the self, particularly ego development and bodily integrity; and (b) hypotheses pertaining to the subject's perception of and feelings about interactions with the social environment, particularly with primary nurturing caregivers.

### *Kinetic Family Drawing (KFD)*

The Kinetic Family Drawing (KFD) test ([Burns, 1982](#)) is frequently administered by clinicians to generate hypotheses regarding the subject's perceptions of and feelings about family structure and sentient environment, particularly the subject's own sense of connectedness to individual family members and the family as a whole. Administration and interpretation are similar to that of the DAP. The subject is provided with paper and pencil and is asked to draw a picture of the family doing something. It is important that this latter instruction not specify "doing something *together*," because this instruction would contaminate data regarding perceived family cohesion.

One manual for KFD interpretation ([Burns, 1982](#)) cites references for inferences drawn about KFD productions. Machover is one of the most frequent citations, however, and this reduces the reliability of the citations because of the previously noted research failing to support her diagnostic inferences. KFD interpretation is therefore subject to the same caution as other projective data, and it is best used as a source of clinical hypotheses requiring confirmation or denial elsewhere.

### *Rotter Incomplete Sentences Blank*

The Rotter test ([Rotter and Rafferty, 1950](#)) is one of many sentence completion tasks that are widely used by clinicians and researchers ([Goldberg, 1965](#)). The Blank consists of 40 sentence stems, which the subject is instructed to complete to express true feelings. A scoring manual permits ratings of each response on a seven-point scale assessing degree of adjustment or maladjustment. Other more recently developed sentence completion instruments (e.g., Incomplete Sentences Task, [Lanyon and Lanyon, 1980](#)) provide forms for school- and college-age children.

As with many of the instruments noted earlier, many clinicians use a more clinical approach to sentence analysis. Such clinicians often work from a theoretical position that holds that sentence completion protocols can provide a particularly rich source of heuristic data about a child's level of ego development ([Loevinger, 1977](#)).

## PERSONALITY TESTS

### *Behavior Assessment System for Children (BASC), Self-Report*

The BASC ([Reynolds and Kamphaus, 1992](#)), discussed earlier among parent and teacher measures, also includes two editions of a self-report form, one for 8 to 11 year olds and one for 12 to 18 year olds. The instrument contains numerous self-referent statements. The child or adolescent is asked to classify each statement as either a true or false self-descriptor. For youngsters who have difficulty reading, the examiner can provide assistance. A range of standardized scores and percentiles is derived along dimensions pertaining to school, family, and personal functioning. Clinically, the Self-Report Form of the BASC is very useful as a straightforward, time-efficient, easily scored measure of a youngster's self-concept. The drawback of the instrument is that, despite its inclusion of checks on the truthfulness of the youngster's self-report, the test seems to elicit unrealistically positive self-appraisals. Clinical use suggests that only very extreme levels of behavioral and emotional disturbance are likely to be reflected fully on the BASC Self-Report.

### *Minnesota Multiphasic Personality Inventory, Adolescent (MMPI-A)*

The Minnesota Multiphasic Personality Inventory (MMPI) ([Hathaway and McKinley, 1940](#); [Hathaway and McKinley, 1943](#)) has been the most widely used personality inventory ([Anastasi, 1988](#)). A revision, the MMPI-2, was published in 1989 ([Butcher et al., 1989](#)). Like its predecessor, the MMPI-2 is a self-report, criterion-keyed instrument, which means that the instrument's items are selected according to an external criterion, in this case the item's ability to discriminate a group carrying a clinical diagnosis from a normal control group. Although the original MMPI and MMPI-2 were designed for use with an older adolescent and adult population, there has always been caution about the validity of MMPI-based clinical inferences as applied to a broad adolescent population. To address this caution and to provide more valid data regarding adolescents, the MMPI-A ([Butcher et al., 1992](#)) was published in 1992. The MMPI-A is standardized on a school-based national adolescent population. Some possible limitations that exist in the normative population are its relative weighting toward better-educated families of higher socioeconomic status.

The MMPI-A consists of 478 forced-choice, true-false items provided in a test booklet. Answers are recorded on a separate answer sheet and then may be scored by hand or by computer. The MMPI-A includes several validity scales (lie, validity, correction, and variability scores) designed to check on such test-taking variables as carelessness, misunderstanding, malingering, and special response sets, 10 original clinical scales (hypochondriasis, depression, hysteria, psychopathic deviance, masculinity-femininity, paranoia, psychasthenia, schizophrenia, hypomania, social introversion), 15 content scales specifically related to adolescent development (anxiety, obsessiveness, depression, health concerns, alienation, bizarre mentation, anger, cynicism, conduct problems, self-esteem, aspiration level, social discomfort, family problems, school problems, and negative treatment indicators), and supplementary scales to measure anxiety, repression, alcoholism, and immaturity. A profile is generated in which raw scores are converted to standard Z-scores, and interpretation is based on consulting available references providing descriptions of clinical populations consistent with different MMPI-A profiles.

### *Millon Adolescent Personality Inventory (MAPI)*

The Millon Adolescent Personality Inventory (MAPI) ([Millon et al., 1982](#)) consists of 150 true-false items and was designed to be completed in approximately 20 minutes by adolescents between the ages of 13 and 18 years. The instrument's two forms (MAPI-C and MAPI-G) are intended for clinical and guidance applications, respectively. Twenty scales are produced, clustering into measures of personality style (introversive, inhibited, cooperative, sociable, confident, forceful, respectful, sensitive), expressed concerns (self-concept, personal esteem, body comfort, sexual acceptance, peer security, social tolerance, family rapport, academic confidence), and behavioral correlates (impulse control, social conformity, scholastic achievement, attendance consistency). The MAPI is intended specifically for use with adolescents and is standardized on relevant normative populations.

### *Children's Personality Questionnaire (CPQ)*

The Children's Personality Questionnaire (CPQ) ([Porter and Cattell, 1972](#)) represents one attempt to provide a measure of personality functioning in younger children, in this instance ages 8 through 12 years. The test is grounded in the theoretical work of Cattell, which led to the development of the Sixteen Personality Factor Questionnaire (16PF) ([Cattell et al., 1970](#)). Consisting of 140 forced-choice questions that may be broken into short forms of 70 items each, the test's administration time generally does not exceed 45 minutes.

The CPQ generates factor scores tapping 14 hypothesized primary personality traits, including emotional stability, self-concept, excitability, and self-assurance. Consistent with the 16PF, second-order, broad trait factors may be derived, including extraversion, anxiety, tough poise, and independence. Critiques support the instrument's research utility but raise questions regarding its clinical applicability ([Goldman et al., 1983](#)). The relative dearth of theory-based instruments for this age group, however, leads many clinicians to include the CPQ in assessment batteries for children of elementary school age.

## **NEUROPSYCHOLOGICAL SCREENING TESTS AND TEST BATTERIES**

### *Developmental Neuropsychological Assessment (NEPSY)*

The Developmental Neuropsychological Assessment (NEPSY) ([Korkman et al., 1998](#)) is an individually administered, comprehensive assessment instrument that examines a youngster's functioning from a neuropsychological perspective. Ideal for use by clinicians well grounded in developmental theory but not necessarily identified primarily as neuropsychologists, the NEPSY uses a broad range of Core and Expanded subtests to measure abilities in five main Domains: Attention/Executive Functioning, which is the capacity to focus attention, to sustain concentration, and to carry out planning, monitoring, self-regulation, and problem-solving; Language, which comprises verbal labeling, phonologic analysis, verbal efficiency, and verbal comprehension; Sensorimotor Functions, comprising fine motor dexterity, precision, and discrimination; Visuospatial Processing, which is visual-motor integration, judgment of spatial orientation, and nonverbal abstract reasoning; and Memory and Learning, the ability to remember faces, names, isolated bits of verbal data, and fuller narrative information. Subtest materials and procedures are somewhat novel and maintain the interest of young subjects. Because the examiner has some discretion about the number of subtests needed to be administered, administration time varies from about 20 minutes to about an hour. The test is designed for children between the ages of 3 and 12 years. The NEPSY is standardized on a representative national sample. Standardized scores, percentiles, and age equivalents are derived and allow for direct comparison with scores on other mainstream psychoeducational test instruments. Although its statistical properties are thus far superior to many of the poorly standardized but widely used neuropsychological tests, reliability and validity of the test are much better between the ages of 5 to 12 years than in younger children. At the younger ages, normal developmental progress makes it very difficult to measure reliably the functions assessed by the NEPSY. Because of its comprehensive nature and relatively good statistical foundation, the NEPSY is a valuable tool in psychological assessment with children. It should never substitute for a full neuropsychological battery, however.

### *Wide Range Assessment of Memory and Learning (WRAML)*

Intended for use with children and adolescents between the ages of 5 and 17 years, the Wide Range Assessment of Memory and Learning (WRAML) test ([Sheslow and Adams, 1990](#)) is designed to offer a comprehensive measure of the youngster's ability to acquire and remember a variety of types of information. Administered on an individual basis, the WRAML takes approximately 45 minutes to 1 hour to complete. The WRAML has three scales, each of which has three subtests. The first is the Verbal Memory Scale, measuring the ability to complete a rote memory task involving single letters and numbers as well as more complex verbal tasks. The second scale is the Visual Memory Scale, assessing the ability to reproduce a simple visual pattern and to recall more complex visual information. The third scale is the Learning Scale, composed of subtests that measure the impact of repeated trials in remembering verbal, visual, and cross-modal information. Some subtests include a Delayed Recall task to gain preliminary information about intermediate-term memory. The WRAML is standardized using a representative national sample corresponding to the most recent available U.S. Bureau of the Census data. Reliability is good and validity is reasonably good. Accordingly, the WRAML can be used with moderate confidence as part of a more comprehensive test battery.

### *Developmental Test of Visual-Motor Integration (VMI)*

First introduced in 1964, the Developmental Test of Visual-Motor Integration (VMI) has since been renormed twice, in 1981 and again in 1989 ([Beery, 1989](#)). The VMI was developed for use in both academic and clinical settings, and it can be administered either individually or in groups to children between the ages of 2 and 15 years, inclusive. The child is presented with a booklet containing 24 geometric designs and is simply instructed to copy each with no erasures permitted. Designs are scored on a pass-fail basis, according to criteria and examples provided in the manual. Testing is discontinued when the child fails to reproduce three consecutive designs accurately. Administration time is approximately 10 to 15 minutes.

The VMI enjoys satisfactory psychometric properties ([Beery, 1989](#); [Polubinski et al., 1986](#)), and it is commonly employed by clinicians as a measure of children's perceptual-motor ability. Such a measure is an important component of assessment batteries constructed to rule out the presence of learning disabilities ([Jansky, 1988](#)). Because it requires minimal interaction with the examiner, the VMI is often administered early in the assessment battery as a means of introducing children to the task of testing.

### *Benton Visual Retention Test*

The Benton Visual Retention Test ([Benton, 1974](#)) is used as a measure of visual-figural memory in adults and in children beginning at the age of 8 years. The test consists of 10 cards on each of which is reproduced one or more geometric designs. The manual outlines several different administration procedures. In the recommended standard administration, the subject is told that each card will be presented for 10 seconds, after which the card will be removed and the subject will reproduce the figure from memory. Reproductions are scored on both a pass-fail basis ("number correct" score) and in terms of the total number of errors ("error" score). Using tables provided in the manual, both these scores are then compared with the anticipated "normal" score for each age and intellectual level (typically determined by the IQ score obtained on the cognitive instrument used in the assessment battery). Scores deviating from this expected score by a significant number of points are considered to either "raise the question" or "suggest" the presence of a visual-figural memory deficit.

Used together, the VMI and the Benton test provide well-standardized data about a child's visual-motor and visual-memory functioning. Depending on the data obtained, hypotheses about perceptual, motor, or memory deficits can be further explored by administering additional instruments (e.g., Jordan Left-Right Reversal Test, [Jordan, 1973](#); Motor-Free Visual Perception Test, [Colarusso and Hammill, 1972](#)).

### *Bender Visual Motor Gestalt Test (Bender-Gestalt)*

Widely used by clinical psychologists to rule out the presence of brain damage ([Anastasi, 1988](#); [Sattler, 1988](#)), each of the nine Bender-Gestalt cards ([Bender, 1938](#)) presents a geometric design that the subject is asked to copy with the card remaining in sight. This Copy phase is typically followed by the Recall phase, in which the child is next asked to reproduce from memory as many of the figures as possible after they have been removed from sight. Although Bender did not develop objective and standardized scoring criteria and normative data, such systems have subsequently been developed. The best-known scoring system for children is that of Koppitz ([Koppitz, 1964, 1975](#)), which provides norms for subjects in kindergarten through fourth grade.

Similar to the VMI and the Benton test, the Bender-Gestalt provides an efficient screen for detecting brain damage. In the past, clinicians also used the



Bender–Gestalt as a source of projective test data, a practice that has received little validation support ( [Sattler, 1988](#)).

#### *Reitan–Indiana Neuropsychological Test Battery for Children and Halstead–Reitan Neuropsychological Test Battery for Older Children*

These two batteries ([Reitan and Davison, 1974](#); [Reitan and Wolfson, 1985](#); [Selz, 1981](#)) are discussed together because of their conceptual, structural, and clinical similarities. The batteries are designed to assess a broad range of functions, and their content is somewhat flexible, permitting different degrees of emphasis on various aspects of the child's functioning. Most clinicians administer 11 tests including sensorimotor and perceptual tasks as well as an aphasia screening test ([Anastasi, 1988](#)). In addition, the complete battery also includes administration of an intelligence test, typically the WISC-III. Administration time for the full battery can therefore be as long as 6 hours.

The utility of these batteries is compromised by virtue of limited available reliability, validity, and normative data ( [Sattler, 1988](#)). In the hands of sophisticated and well-trained neuropsychologists, the derived data can be employed to differentiate brain-damaged children ( [Reitan and Herring, 1985](#); [Selz and Reitan, 1979](#)) and children with specific associated syndromes and to assist in rehabilitation planning by articulating behavioral deficits related to central nervous system disorders ([Anastasi, 1988](#)).

#### *Luria–Nebraska Neuropsychological Battery, Children's Revision (LNNB-C)*

Similar to the Reitan batteries noted earlier, the Luria–Nebraska Neuropsychological Battery, Children's Revision (LNNB-C) ( [Golden, 1987](#)) was developed to investigate a wide variety of neuropsychological functions, to assess cognitive deficits, and to provide data to guide rehabilitation in children aged 8 to 12 years. This battery differs from the Reitan instruments, however, in its abbreviated administration time (approximately 2½ hours) and greater standardization of content, materials, administration, and scoring ([Anastasi, 1988](#)). The battery's 149 items are grouped into 11 clinical and two optional scales (assessing sensorimotor, perceptual, and cognitive abilities), three summary scales (discriminating brain-damaged from normal children), and 11 factor scales (assessing specific neuropsychological functions and requiring caution in interpretation). All items receive scores of 0, 1, or 2, with higher scores indicating greater evidence of brain dysfunction. A companion 57-category qualitative scoring system permits further evaluation of error items.

The LNNB-C is a downward extension of an adult version of the battery ( [Sattler, 1988](#)), an approach to neuropsychological assessment that has been the focus of substantial controversy and criticism ( [Adams, 1980](#); [Delis and Kaplan, 1983](#); [Sattler, 1988](#); [Sparrow et al., 1985](#); [Stambrook, 1983](#)). The manual provides more substantial standardization, reliability, and validity data than are available for the Reitan batteries, but concern has been raised about the adequacy of the battery for each of these psychometric issues ( [Nolan et al., 1983](#); [Sattler, 1988](#); [Sparrow et al., 1985](#)). Given these concerns, pending completion of further research caution is required in the clinical interpretation of data generated by this battery.

#### *Wechsler Memory Scale, Third Edition (WMS-III)*

More than simply intending to replace its predecessor in measuring simple memory functions, the third edition of the Wechsler Memory (WMS-III) ( [Wechsler, 1997b](#)) was designed to make a major contribution to a comprehensive test battery by measuring the complex interaction between memory and learning. Based on current research in the neurosciences, the test consists of six primary subtests and five optional subtests. The subtests are divided into three main groups: immediate memory, general memory (delayed), and working memory. In all groups, both visual and auditory modes are evaluated. Further scores can be derived to delineate the learning process in acquiring auditory information. The test is administered on an individual basis, requires approximately 30 to 45 minutes for completion, and is appropriate for those between the ages of 16 and 99 years. The age span covered by the WMS-III, thus, makes it a useful instrument for the assessment of older adolescents. A full range of standardized scores can be derived and easily compared with other well-used cognitive test instruments. The statistical properties of the test are sound, so the instrument can be used with confidence.

### **MEASURES OF INFANT AND PRESCHOOL DEVELOPMENT**

#### *Bayley Scales of Infant Development, Second Edition (BSID-II)*

The Bayley Scales of Infant Development, second edition (BSID-II) ( [Bayley, 1993](#)) is a revision of the Bayley Scales of Infant Development ( [Bayley, 1969](#)). Like its predecessor, the BSID-II assesses the developmental functioning of infants and toddlers with the aim of detecting developmental delay and pointing the way toward appropriate intervention strategies. The revision was undertaken to update the normative data, to extend the age range beyond what was covered by the original BSID, to improve the content coverage to make the test more useful clinically, to enhance the test materials to increase their attractiveness for subjects, to improve the psychometric properties of the test, and to improve the clinical utility of the test by including standardization data on such special populations as those with Down's syndrome, prematurity, and prenatal drug exposure, all while preserving the original basic focus of the BSID on deriving sound developmental information from stimulating, standardized interaction with an infant or young child. The test consists of Mental, Motor, and Behavior Rating scales. The Mental scale focuses on the infant's early ability to direct his or her attention to appropriate people and objects in the environment and then on the development of age-appropriate interactive, language, conceptual, and memory skills. The Motor scale focuses on early development of both fine and gross motor skills, as well as sensory integration and visual–motor coordination. Both the Mental and Motor scales are completed using a flexible administration format. Test materials are attractive and resemble captivating toys. Test subjects are offered the materials, along with stimulating guidance about the developmentally adequate way of interacting with the materials. The subject's actual use of the materials is then evaluated. Rather than relying on the type of direct assessment used for administration of the Mental and Motor scales, the Behavior Rating scale is completed based on informal observation of the infant or preschooler during the testing. Styles of interpersonal relating, attunement to others, and accessibility to limit setting are examples of the types of behaviors assessed. Test sessions are typically fast paced, to maintain the very young child's interest, and they require considerable creativity, as well as test familiarity from the examiner. For these reasons, the BSID-II is often considered a challenging instrument for examiners to master. Once mastered, however, the test can be administered in a smooth, systematic, enjoyable manner. The BSID-II is standardized on a representative national sample that conformed to the 1988 update of the 1980 national census reported by the U.S. Bureau of the Census. The sample is designed to conform to racial, ethnic, and socioeconomic proportions in the national population. At each age level, there is equal representation of males and females. Reliability and validity data for the BSID-II are provided in the test manual. The data indicate that the test is a reliable and valid instrument that can be used with confidence. The reliability and validity data pertain to documentation of current developmental status. Early developmental data do not serve to predict long-term intellectual ranking. In clinical practice, the BSID-II provides very valuable information about the status of the youngest of patients. One drawback, however, is that the test does not provide a discrete measure of language development. Such a measure is often needed in the assessment of potential developmental delay.

#### *Mullen Scales of Early Learning (MSEL), American Guidance Service Edition*

The original version of the Mullen Scales of Early Learning (MSEL) was developed by Eileen Mullen, an expert in early childhood development and education (Mullen, 1988). Although the test was useful and well-received, the small, private basis on which the test was constructed compromised its statistical properties. In 1993, the MSEL was purchased by the American Guidance Service (AGS), a well respected publisher of psychoeducational tests. Under the auspices of the AGS, the MSEL was standardized on a nationally representative sample of children between the ages of 2 days and 69 months, 0 days ( [Mullen, 1995](#)). The reliability and validity of the measure are now quite good.

In the original version, there were separate Mullen scales for infants and preschoolers. In the AGS edition, the MSEL has become a single test designed for use with children from birth through 68 months of age. The test is administered individually without time limits. Administration time varies from 15 minutes at the youngest ages to approximately an hour for 5-year-old youngsters. The MSEL is divided into five scales: Gross Motor, a measure of central motor control and mobility; Visual Reception, a test of visual discrimination, memory, organization, sequencing, and spatial awareness; Fine Motor, a measure of visual–motor ability; Receptive Language, a test of auditory comprehension, memory, organization, sequencing, and use of spatial concepts; and Expressive Language, a test of speaking ability, language formation, and the ability to verbalize concepts. Standardized scores, percentiles, age equivalents, descriptive categories, and developmental stages are all derived on the MSEL. The test is particularly useful for determining eligibility for early intervention and special education services, for assessing children with special needs, and for helping to design individualized psychoeducational programs.

### **PSYCHOLOGICAL TEST RESULTS**

As discussed earlier, psychological testing yields a wealth of data regarding a child's or an adolescent's functioning. It is important to address both the nature of the data gathered and the means by which it is most usefully communicated to young patients and their parents.

## Nature of the Data

Consistent with the multifaceted test battery described earlier, psychological assessment provides data on a youngster's cognitive, behavioral, and personality functioning. For each domain assessed, test findings place the youngster at a particular level of functioning overall. To obtain the most sophisticated benefit of psychological testing, however, it is critical to go beyond a summary analysis of each distinct domain to analyze both the interrelationships among the domains assessed and, further, the profile of individual strengths and weaknesses within each global domain ( [Novick and Lewis, 1967](#)). This is because an identical performance in one area of functioning by several youngsters may have vastly different implications depending on how each one of them performs in other areas. For example, projective evidence of highly original, complex, rich, reality-oriented internal mental processes is reassuringly unremarkable for a child with a high IQ. The same projective data from a child with a low-average or borderline IQ, though, raises important questions about the accuracy of measurement of that child's intelligence. Once the individual features of a youngster's test profile have been analyzed, the psychologist provides a formulation that integrates the diverse findings into a comprehensive view of the patient. The data analysis concludes with the offer of recommendations on how best to address the needs noted in the assessment.

## Communication of the Findings

To facilitate appropriate interventions on behalf of the child or adolescent assessed, psychological test results must be communicated in understandable language and with interpersonal sensitivity appropriate to the recipient of the information. If the results are communicated in overly technical language or without regard for their impact on parents, clinicians, and others invested in the child or adolescent patient, the test data are likely to be ignored, and the benefits of testing will be negated.

Assessment findings should also be shared in a particular order to facilitate their incorporation into clinical care. The initial step of this sequence depends on the referral source. If the test referral came from another professional, test data and recommendations should be communicated initially to the referring clinician. By communicating first with this colleague, appropriate primary responsibility for patient care and relationships to the patient are safeguarded. The psychologist and referring clinician can then decide together how best to proceed with further communication of the test findings to those concerned. In some instances, the referring clinician and testing psychologist may elect to present the findings together. The advantage to this presentation mode is that all concerned can develop a shared, multidimensional understanding of the child or adolescent. In other cases, it may be best for the psychologist to present the assessment data alone. The data can then be brought back to the clinical relationship with the referring professional. This method is best when time constraints make a joint meeting difficult. The method also has special advantages for those cases in which the psychological test data may be at odds with clinical expectations. Separate communication of the data by the psychologist allows the referring clinician to maintain better a therapeutic alliance with the patient and family. Together, the clinician and family can then begin to incorporate the data obtained from the testing. Finally, for those cases in which fragile patient functioning highlights the importance of the relationship with the referring clinician, it is often best to have that clinician relay the test findings to those concerned. For these patients and their families, the information may be best accepted and understood in the context of a supportive, therapeutic relationship.

For cases of the type discussed earlier, findings and recommendations should then be communicated to the parents in the most efficacious presentation style. If the parents themselves initiated the referral, sharing these findings and recommendations with the parents constitutes the first step in the interpretive process. Just as good rapport is necessary in the testing situation to guarantee testing accuracy, so is it necessary in consulting with parents to facilitate their willingness to make good use of the data. Care should be taken to demonstrate some empathy for the parents' understanding of the child or adolescent so they can feel confident about the veracity of the test results. Findings should be communicated in a forthright but tactful manner, with particular sensitivity to shared family traits and for parental attitudes toward the youngster involved.

With children of elementary school age and with adolescents, it is always important to then consider providing the test findings directly to the patient. The offer of an interpretive meeting for the child or adolescent should be made pending parental approval. If the child or adolescent requests such an interpretive meeting, a careful clinical decision should be made by parents and professionals regarding the best setting and participants for this meeting. Some children prefer to safeguard their self-esteem by hearing the results privately from their parents. Those who have developed an initial attachment to the testing psychologist often want to hear about their test performance from him or her. Still others, who have therapists to whom they are closely attached, request that their therapists communicate the test results. These latter two formats may or may not include the parents. With the patients themselves, test findings should also be shared in a straightforward, honest manner. The child's or adolescent's strengths should be emphasized, his or her weaknesses discussed in an empathetic manner, and hopeful recommendations offered.

At times, other interested parties also need to share in the assessment findings. For children and adolescents, the school is the likeliest other recipient of the information. It may also be helpful to communicate the test findings to programs for young people with special needs as well as to other caregivers. Parents should make informed decisions with the psychologist and, when relevant, the referring clinician about the format in and extent to which the findings are to be shared with outside parties. These decisions should balance protection of family privacy with sufficiently full communication of findings to allow for appropriate interventions on behalf of the child or adolescent.

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## 44 NEUROLOGIC MEANING OF SOFT SIGNS

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The term *soft signs* has become a subterfuge for clinicians who are avoiding hard scientific thinking. It is almost a slang expression, like “stroke,” which can mean cerebral thrombosis, embolus, hemorrhage, convulsion, or even heart attack. The term *soft signs* has been used to refer both to *historical evidence* of events that can be associated with brain damage and certain kinds of behavior (e.g., hyperactivity, impulsiveness, poor attention span) and to *physical findings* elicited by examination of the patients. Many different symptoms and signs are subsumed under the title soft signs. Some of these are maturational and probably represent variants of normal. Others relate to hereditary conditions and diseases, and still others refer to acquired disorders of the brain. Hyperactivity, emotional lability, disorders of attention, and impulsiveness have been considered soft signs by some. Children certainly may have motor, perceptual, linguistic, adaptive, social, and educational problems of various types as the result of brain abnormality. To identify useful evidence of organic brain dysfunction, there must be a search for other findings that will reliably reflect neurologic abnormality. This has only partly been accomplished, hence the term “soft.”

Asymmetrical physical and neurologic findings are clearly reliable indicators of disease. Still, there are certain “hard” neurologic findings, on which neurologists rely daily in the performance of physical examinations that have not actually been subjected to careful scientific scrutiny. For example, it is not known how often patients who are free of nervous system diseases have Babinski reflexes, especially when these patients are tired or stressed. Not only do we not know how many neurologically normal persons manifest this sign, we also do not know exactly how many people with demonstrable brain damage—even those with corticospinal tract damage—fail to manifest this sign.

Despite this failure, great and appropriate emphasis is placed on the significance of the neurologic examination in general and on the Babinski reflex in particular, even though the neurologist may be influenced in his or her interpretation by the fact that patients have been referred because of their failure to conform to some normal behavioral pattern and are therefore abnormal almost by definition. It is up to the neurologist to discover independent evidence of abnormality. Therefore, the signs the neurologist elicits are taken as confirming the presence of lesions in the brain.

Exactly the same thing can be said of soft signs. There have been relatively few systematic studies of large populations of children in whom meticulous neurologic examinations have been performed. The Isle of Wight Study ([Rutter et al., 1970](#)) suggests that there is not as close an association between the presence of abnormal neurologic findings and behavioral abnormalities as may have been assumed from earlier publications. Neurologic findings do not always predict behavioral distortions, and behavioral abnormalities do not always predict physical abnormalities; yet there is a statistical correlation between the two. Other studies confirm the lack of specificity of some motor and sensory signs in the neurologically impaired and in neurologically normal persons ([Werry and Aman, 1976](#)).

When a soft sign is identified as reliably indicating the presence of brain dysfunction, it is no longer soft. If it reflects brain damage in some patients and can be seen in others who have no evidence of brain dysfunction, then it is soft indeed and is probably not very helpful in determining whether a patient has brain dysfunction. Signs and symptoms that are found in neurologically normal persons more frequently than in people with brain damage are not soft signs of abnormality but, rather, are within the normal range. To determine which signs are truly soft—that is, those that can be associated with organic damage but need not be—requires correlational statistical studies in patients who are known to be abnormal and in people who are known to be normal, with the recognition that the former is easier to establish than the latter. This has not been completed for each of the physical signs that are called soft.

Consistency has been considered important in determining the long-term significance of soft signs in children. Although follow-up studies can provide considerable information, the finding that minor or major neurologic abnormalities may disappear in the course of development does not necessarily mean that the findings originally elicited were not significant or should be considered “merely maturational.” Mild hemiparesis or diapaesis may disappear, hypotonia may become normotonia, seizures may stop, but the importance of these findings as evidence of encephalopathy in the past remains. Such historical encephalopathy may still be relevant to contemporary cognitive and behavioral disturbances.

One of the most common clinical settings in which learning and behavioral disturbances turn up is that of seizures originating in the temporal lobe. The etiologic dilemma of this association has not been fully resolved. Are the learning and behavior problems secondary to the emotional trauma of susceptibility to seizures? Does the uncontrolled seizure disorder, or the medication that is used to control it, lead to a partial functional disruption of the system that is sufficient to cause defects of attention and learning? The weight of evidence indicates that, in most cases, seizures and behavior problems are coincident symptoms of the same basic neurologic abnormality. This explains why the seizures often resolve while the behavior and learning problems remain.

The search for validating correlations among various findings requires a standard against which to compare them. There is a tendency to ascribe certainty to tools that are used by an allied profession. Child psychiatrists may tend to regard the physical examination of a child neurologist with greater respect than do neurologists themselves. Consistency of findings and interrater reliability can be a problem. The electroencephalogram (EEG) may provide borderline or frankly paroxysmal abnormalities in a certain percentage of patients with learning disability who have no clinical seizures. This may be a soft finding to many neurologists, but it may not be to some psychologists. Conversely, psychological tests, especially the Wechsler Intelligence Scale for Children and Bender tests, may provide evidence that to neurologists is less equivocal than it may be to psychiatrists or even to psychologists. All groups are somewhat in awe of computed tomography (CT) and magnetic resonance imaging (MRI) of the brain, although findings such as cerebral atrophy and enlarged ventricles are quite soft because they do not reliably predict behavioral or cognitive dysfunction ([Shaywitz et al., 1983](#)). The truth is that we lack an acceptable standard against which to measure each of the multitudinous symptoms and signs included in the vague expression, soft signs. This being the case, we have been forced to make correlations among tests of unproven value ([Barlow, 1974](#)).

### INVESTIGATION OF PARTICULAR SOFT SIGNS: MOTOR AND SENSORY

[Precht and Stemmer \(1962\)](#) first pointed out the significance of choreiform movements. This phenomenon consists of random, quick, irregular movements, most prominently seen in the fingers of the supinated outstretched forearms. Precht and Stemmer correlate this phenomenon with perinatal distress and neonatal abnormality. Wolff and Hurwitz report an overall incidence of the choreiform syndrome of roughly 10% in elementary school children aged 10 to 12 years, with a significant difference between boys (17%) and girls (5%) ([Wolff and Hurwitz, 1966](#)). The incidence of choreiform signs drops from 50% at age 10 years to 3% among 12 year olds. Additional studies document that boys with choreiform twitches have more reading and spelling difficulties than boys without such twitches, but there is no such correlation in girls ([Wolff and Hurwitz, 1973](#)).

Development of the ability to appreciate simultaneous touch to face and hand (the face–hand test, a test of attention to sensory stimuli, a useful measure of frontal cortical function in adults) is clearly age related. Most neurologically normal children achieve this ability by age 7 years. This is also the age during which concepts of body image enlarge to include a body under the large head of children’s drawings ([Bender et al., 1951](#)). [Kraft \(1968\)](#) basically confirms these findings but finds no



correlation with the scored Goodenough Draw-a-Man test, a test that measures parietal lobe functions. Kraft finds that 30% of 7-year-old nonreaders give an immature response to the face–hand test, whereas 20% of normal readers give an immature response, a difference that is neither impressive nor useful. In adults, none with normal cortical function, as defined by the Halstead–Reitan battery, have an abnormal face–hand test, and 91% of those with abnormal cortical function are abnormal on the face–hand test ( $p < .01$ ), (Jenkyn et al., 1985). No one knows at what age failure on the face–hand test becomes a reliable indicator of cerebral cortical dysfunction.

In a longitudinal study of 66 prematurely born children from advantaged homes, neurologic examinations were performed at 8 years of age. Thirteen of these children had localizing neurologic findings, and 20 were found to have two or more nonlocalizing (soft) signs of central nervous system (CNS) dysfunction. These included tests of speech, balance, coordination, gait, sequential finger–thumb opposition, muscle tone, graphesthesia, stereognosis, and choreiform movements. These 33 neurologically abnormal children were significantly more likely to have sustained perinatal complications than were children whose neurologic examinations were normal. Although children with soft signs were at a consistent disadvantage—in relation to posterior cerebral cortical functioning as shown by intelligence quotient (IQ), reading, and arithmetic achievement test score levels—compared with those who were neurologically normal, these differences did not reach conventional levels of significance. However, the children with soft signs were significantly more likely to have received special education and to have been referred for psychiatric consultation than were the neurologically normal children. Almost half of the children with two or more of the physical soft signs elicited by the investigators required special educational intervention to reach the same overall level of academic school performance achieved by the neurologically normal children. Ninety percent of the neurologically normal children made satisfactory progress without any modification of the school program. This finding, coupled with the high frequency of psychiatric consultations and continuing intervention in the group with soft signs, provided support for the view that children with soft signs at 8 years old are, indeed, at greater risk for the emergence of behavioral or school learning problems (Hertzog, 1981). Soft signs tend to reflect frontal lobe dysfunction, whereas difficulty with reading, writing, and arithmetic represents parietal, temporal and occipital dysfunction. The association of soft signs with behavioral disorders and not with abnormal reading, writing, and arithmetic could be understood as the result of premotor frontal dysfunction.

## RELIABILITY AND STABILITY OF SOFT SIGNS

Any conclusion regarding the validity of soft signs assumes a satisfactory interrater reliability, internal consistency, and retest stability of subtle signs. Although this has not been studied extensively, some information is available (Stokman et al., 1986). The Physical and Neurological Examination for Soft Signs (PANESS) was used with 54 psychiatric patients and 25 neurologically normal children aged 5 to 17 years. Acceptable interrater reliability was found for most of the items tested. Test-retest reliability at 2 weeks was not satisfactory for some of the categorically scored items. However, continuous items, such as the time needed to perform 20 consecutive movements, remained relatively stable at retest (Vitiello et al., 1989).

To assess the persistence of soft signs outside a referred sample, 159 members of a local birth cohort of the United States National Collaborative Perinatal Project were traced, and their performance on six neurologic test scales was measured at age 17 years by examiners who were unaware of the status of these children when these patients were 7 years old. A comparison group was also formed that had been free of signs at age 7 years. On four of the six tests (dysdiadochokinesia, mirror movements, dysgraphesthesia, and motor slowness), index boys did significantly worse than the comparison boys. In contrast, index girls scored significantly worse than comparisons only on motor slowness. The consistency of neurologic test performance through the years contradicts the idea, at least in boys, that soft signs are only maturational lags destined to fade with age. The worse the boy's neurologic score at age 17, the stronger is the relationship with his status at age 7.

This important study makes another observation of great significance. Analysis of the data that categorizes findings as either absent or present, rather than of varying intensity, would have shown almost no statistically significant relationship in the 7- to 17-year data. If neurologic test performance is intrinsically a continuous function, such categorical analysis, which was used in most of the earlier studies, would be likely to underestimate the true strength of relationships (Shafer et al., 1986). Unfortunately, the large population studies that have been interpreted as casting doubt on the usefulness of abnormalities found on neurologic examination have used a categorical approach to describing them.

## SOFT SIGNS AND ATTENTION DEFICIT HYPERACTIVITY DISORDER

The literature on this subject is somewhat confusing. Wikler et al. (1970) and Camp et al. (1978) find no difference in the prevalence of signs between children with and without *attention deficit hyperactivity disorder* (ADHD), but every possible sign is not individually validated. Nichols and Chen (1981) analyze a large sample drawn from all 7-year-old participants in the National Collaborative Perinatal Project and find only a small excess of overactivity and inattention among children with neurologic soft signs. Shaffer et al. (1985) find no relationship between early soft signs and attention deficit or conduct disorders in 63 male and 27 female adolescents known to have had neurologic soft signs at the age of 7 years, but the same group does relate three signs (motor slowness, motor inaccuracy, and dysgraphesthesia) to poor IQ and poor attention span (Schonfeld et al., 1989). Castellanos et al. (2000) report abnormal frontal oculomotor function in girls with ADHD. Lucas et al. (1965), Mikkelsen et al. (1982), and McMahon and Greenberg (1977) all find soft signs to be more common in generally younger groups of children with ADHD than in controls (Wikler et al., 1970).

There is, thus, considerable evidence that, although soft neurologic signs reflect organic brain dysfunction, they are rather inconsistently seen in the syndrome of ADHD, once called minimal brain damage. This does not mean that ADHD is unrelated to brain dysfunction. If ADHD or any cognitive–behavioral syndrome were associated with soft neurologic findings, this would presumably constitute evidence in favor of the organic nature of such a cognitive–behavioral syndrome. If a cognitive–behavioral syndrome were not associated with soft neurologic findings, it would not necessarily mean that the disturbance arose from some source other than brain dysfunction. In fact, dysfunction confined to the premotor portion of the frontal lobes is likely to produce a major distortion in behavior, attention span, and personality without concomitant major motor signs, difficulty with reading, writing, arithmetic, or a drop in IQ.

The frontal lobes have increasingly been targeted as the possible source of abnormality in ADHD (Benson, 1991). Quantitative analysis of MRI scans of 18 boys with ADHD and 18 carefully matched controls supports the theory of abnormal frontal lobe disturbance in ADHD (Giedd et al., 1994). Semrud-Clikeman et al. (2000) report similar findings. MRI studies of the cross-sectional area of the corpus callosum also reveal that the anterior regions are smaller in boys with ADHD (Giedd et al., 1994; Hynd et al., 1991). A similar study using positron emission tomography (PET) identifies hypometabolism in the left anterior frontal lobe as a significant correlate of ADHD severity in adolescents (Zametkin et al., 1993). Functional MRI studies are consistent with the foregoing, showing abnormality in the putamen, a portion of the frontal-basal ganglia-thalamic circuit (Teicher et al., 2000). Event-related potentials also indicate abnormal frontal function in ADHD (Pliszka et al., 2000). Brain mapping reveals abnormal premotor processing in ADHD (Steger et al., 2000).

Reports of psychological tests that generally reflect frontal dysfunction generally support the linkage of ADHD with frontal lobe dysfunction. Deficits in verbal fluency are found in male ADHD inpatients compared with inpatient controls (Koziol and Stout, 1992). Other tests reflecting frontal lobe dysfunction distinguish children with ADHD from controls, although general cognitive impairment as measured by the IQ is not found in the former group (Shue and Douglas, 1992). Some measures of frontal lobe dysfunction do not reliably distinguish attention deficit disorder (ADD) with and without hyperactivity, but the Continuous Performance Test and the Stroop Test do distinguish groups with ADHD from neurologically normal groups (Barkley et al., 1992). Others (Loge et al., 1990) report impaired Wechsler Intelligence Scale for Children performance in ADHD but no difference from controls in the Wisconsin Card Sorting Test or tests of verbal and design fluency (Grodzinsky and Barkley, 1999; Doyle et al., 2000).

Clearly, the preponderance of evidence (MRI, PET, psychological tests) favors the concept that frontal dysfunction explains some of the behavior in ADHD, even though none of these tests is specific enough to provide a reliable diagnosis, absent other data. We must now ask: Are there physical neurologic signs that reflect frontal lobe dysfunction? The answer is yes, but none of them are part of the ordinary neurologic examination of school-age children, and, for the most part, they have not been standardized and validated in these age groups as they have been in adults (Jenkyn et al., 1977, 1985). These are the snout reflex (Paulson and Gottlieb, 1968), suck and grasp reflexes (Shahani et al., 1970), paratonia, persistent glabellar reflex (Damasio, 1985), abnormal visual tracking movements (Rodin, 1964), abnormal response in performing reciprocal coordination of the hands, and three-step motor sequences of the hands (Luria, 1962; Ozeretski, 1930). The presence of three frontal signs discriminated between subjects with normal and abnormal MRIs with 93% accuracy, and normal and abnormal neuropsychological testing with 85% accuracy, when applied to 31 murderers whose average age was 32 years. Half had been diagnosed as having ADHD in their youth: Grasp reflex, suck reflex, and paratonia were the best discriminators (Blake et al., unpublished data). Three or more abnormal signs on the battery of tests performed by Jenkyn and his colleagues reliably distinguish normal subjects from mildly, moderately, and severely impaired adults, using the Halstead–Reitan battery (Jenkyn et al., 1977, 1985). If ADHD were, in fact, a reflection of frontal lobe dysfunction, we would predict that physical signs of frontal lobe dysfunction of the type validated by Jenkyn et al. and Blake et al. would be more prevalent among children with ADHD than controls. The age at which findings on these tests become abnormal must be known. Clearly, normal 2 year olds would not be able to perform tests of complicated motor sequences. At what age does the inability to accomplish these tasks become abnormal? Are abnormalities on these tests characteristic of children with ADHD? It is disappointing that no study has yet answered these questions.

## Clinical Examination of the Brain

The neurologic examination is a way of determining whether various parts of the brain and spinal cord are functionally intact. It is easiest to test the lower, phylogenetically more primitive parts of the nervous system and then progress to more complex functions.

The human brain is organized in layers of interacting regions. At the base, just behind the mouth, is the brain stem, which controls most elemental functions such as breathing, blood pressure, and swallowing. It also controls the cranial nerves subserving eye movement, facial movement, and the muscles of the throat, mouth, and neck. The brain stem mediates sensation in the head, face, and neck and, through the cerebellum, general motor coordination. Above the brain stem, behind the nose and its sinuses, is the diencephalon. This controls body temperature, appetite, sleep, and wakefulness. Circling around that central region of the brain is the limbic system and amygdala–hippocampus complex, the seat of the primitive emotions involved in sexual behavior, fear, anger, and aggressive attack, as well as memory. Virtually, all mammals share these features. Over all this, covering the entire surface of the brain and lying just below the skull, is the cerebral cortex, the thick layer of gray matter that is the source of intelligence. The cortex is anatomically and functionally distinct from the rest of the brain. Complex perceptual and cognitive functions take place in the cortex. Vision registers and is integrated at the back of the brain in the occipital lobes. Somatosensory information like touch, pain, and position in space is interpreted in the parietal lobes; smell and taste are interpreted in the temporal lobes. Speech and the understanding of language are usually identified with the left perisylvian region and in the transcortical areas surrounding that. Voluntary movement derives from the motor strip in the posterior part of the frontal lobes. Voluntary movement is modified by deep subcortical centers called the basal ganglia. Almost all mammals have well-developed occipital, parietal, and temporal lobes and motor control systems that are very similar to the human ones.

To determine whether the brain is intact, the examiner must test reflexes and sensorimotor functions, must compare the patient's right and left sides, and must observe stance and gait. The head circumference should be measured with a tape measure and compared with a table of normal standards. If the circumference is more than two standard deviations below the mean, the patient is microcephalic and will have an 85% chance of being cognitively impaired.

Tests of motor coordination include skipping, hopping, and walking a straight line forward and backward. The patient should be asked to spread the fingers apart and hold the hands apart while the examiner looks for discontinuous, involuntary, jerky movements of the fingers and arms, called *choreiform movements*. The patient should be tested to see whether he or she is right handed and right footed. If dominance is mixed, it may mean that his nervous system has not developed the way that it should.

Abnormality on a single test usually does not mean that the person is brain damaged, but a pattern of abnormalities does indicate that the brain is malfunctioning. Asymmetrical (lateralized) abnormalities are likely to reflect structural abnormalities of the CNS, but severe damage to the brain can be present in patients whose sensorimotor functions, coordination, and reflexes are normal.

### COGNITIVE FUNCTIONS

Because the brain is the organ of cognition, cognitive functions must also be tested in the neurologic examination. The Mini-Mental Status Examination (MMSE) is a standard tool for the investigation of the cognitive status. The patient must be able to identify the day, date, time of day, and location. The patient is asked to name several objects that have just been presented (pen, wristwatch). The patient is given three words and is asked to repeat the three words immediately and to remember them, is asked to subtract seven from 100 and to perform five serial subtractions of seven, and is asked to spell the word "world" forward and backward. The examination requires patients to be able to read, repeat, and write a sentence, copy a standard diagram, follow a three-step command, and recall the three words he or she was instructed to remember earlier. The MMSE is an excellent screening test for cognitive disorders that affect the posterior two-thirds of the brain, but the frontal lobes can be damaged or even surgically removed without causing any abnormality in speech, arithmetic ability, reading, writing, or memory. Although these functions, tested by the MMSE and IQ tests, often remain intact, frontal lobe dysfunction can cause profound and devastating changes in the patient's social life.

### EVALUATION OF THE FRONTAL LOBES

Fibers from the frontal lobes project to other regions of the brain such as the corpus striatum and the dorsomedial nucleus of the thalamus. Lesions in the dorsomedial thalamus can cause the kinds of abnormalities that frontal lesions can produce. For this reason, it is a mistake to conceptualize the functions that are mediated by the frontal lobes as residing exclusively within this area of the brain, but for the sake of brevity, we refer to them as *frontal functions*.

Judgment and the ability to deal with complexity are crucial functions of the frontal lobes. Unfortunately, there is no reliable, objective standard or test for measuring judgment that is sufficiently sensitive and specific. Asking a patient what he or she would do with a stamped addressed envelope found lying in a street is not an adequate test of frontal lobe function. It is insensitive and nonspecific. There are, however, several simple physical tests of frontal lobe function that rely on motor and sensory functions. Neurologists and psychiatrists can easily assess them.

Regressive reflexes (e.g., snout reflex; suck, nuchocephalic) ([Jenkyn et al., 1977](#)) are associated with diffuse cerebral dysfunction (e.g., delirium, dementia). These reflexes are called *regressive* because they are normally present in very young children and infants but not in adults ([Jenkyn et al., 1985](#)). When interpreting these reflexes, it is wise to recall that neuroleptic medications can cause regressive reflexes.

The most useful of these reflexes engages the extensive *frontal eye fields*, the portion of the frontal lobes that enables the eyes to track a moving object. The examiner asks the patient to follow the examiner's smoothly moving finger as it goes slowly from left to right horizontally in front of the patient. An abnormal response would be visual tracking in brief, staccato, discontinuous (saccadic) jerks or inattentiveness with brief deviations of the patient's eyes from the examiner's finger rather than smooth movements. A normal response would be smooth visual tracking of the moving finger. The patient should also be able to stare for 30 seconds at the examiner's stationary finger without deviating his or her gaze. This helps to demonstrate an ability to concentrate.

Tapping gently on the bridge of a patient's nose normally elicits *blinking*. If the blinking persists for more than three taps, it means that the patient cannot accommodate to a benign stimulus. After three taps, it should be clear that there is no potential threat to the patient's eyes. If the patient cannot suppress the urge to blink even though there is no threat, it shows that he or she cannot adapt to a new situation. This inflexibility reflects dysfunction in a frontally mediated pathway ([Jenkyn et al., 1977](#)).

Paying attention to two sensory stimuli simultaneously can be difficult for frontally damaged people. They tend to disregard the more distal of two unilaterally applied stimuli. For example, if the examiner touches the patient's right cheek and right hand simultaneously while the patient's eyes are closed, the patient may report having been touched only on the cheek. This is called the *face–hand test*. If the patient extinguishes (pays no attention to) the more distal stimulus, it shows that his or her attention to somatosensory stimuli by the frontal lobes is attenuated.

To see whether a patient can concentrate and suppress an urge, the examiner creates a visual urge and then asks the patient to ignore it. The examiner faces the patient, holds his or her hands to either side of the patient's eyes, and asks the patient to look at the examiner's nose. He or she instructs the patient to deviate his or her eyes to the side of the examiner's briefly raised index finger and then return his or her gaze to the examiner's nose. Alternately, the examiner moves his or her right and then left index fingers. The patient looks towards the moving finger and then back at the examiner's nose. Once it is clear to the examiner that the patient has mastered this and looks to the moving finger and then back to the examiner's nose, the examiner asks the patient to look toward the opposite side, toward the side that *does not* move. Patients with frontal damage have trouble suppressing the urge to look at the moving finger and first look there before correcting. This test uncovers impulsivity and inattentiveness. It is called the *antisaccade test*. It is performed five times, twice to the left, twice to the right, and then once to the left. Two or more failures count as an error.

Frontally damaged patients often cannot perform two- and three-stepped motor sequences repetitively even though their strength and coordination are normal. This makes complex, repetitive motor tasks sensitive indicators of frontal function. The patient is asked to strike his or her thighs repeatedly, simultaneously, with the right palm and the left fist, then with the right fist and left palm, alternating with each blow. Patients should be able to perform three sets perfectly on their own after having been taught how to perform this by the examiner. This is the *two-stepped Luria test*. Then the examiner shows the patient how to strike the thigh successively with the right palm, fist, and the edge of the hand. The patient attempts it alone after being shown how to do this by the examiner. The patient is then asked to perform the task with the left hand, striking the palm, the fist, and the side of the hand in sequence. This is called the *three-stepped Luria test*. Frontal lesions often interfere with the proper sequencing of this task. Abnormality on either the two- or three-stepped Luria test indicates frontal dysfunction.



Patients with frontal disturbances often cannot relax their limbs when asked to do so and allow them to remain suspended in the air when one is raised and then is dropped by the examiner. This is the *placement test*. Sometimes patients try to help by anticipating the position the examiner wishes them to assume after the examiner instructs them to be "limp as a rag doll." The abnormality is called *paratonia*. When this sign is severely abnormal, the limb will remain in any position in which it was placed. This phenomenon is called *waxy flexibility or catatonia* and can also be encountered in psychotic states of depression and schizophrenia.

Upward gaze is another frontally mediated function. When asked to look upward, the patient should be able to move the outer limbus of the iris upward at least 5 mm and downward 7 mm. The examiner must place a ruler vertically in front of the patient's eyes to measure the extent of movement.

Testing the *nuchoccephalic reflex* reveals the patient's ability to adapt to postural change. It is often helpful in diagnosing frontal disease. With the patient's eyes closed, the examiner places his or her hands on the patient's shoulders and quickly turns the patient to the right or to the left. Normally, the head follows the shoulders. Some frontally impaired patients do not move their heads with their shoulders. They cannot adapt to this postural change.

The *snout reflex* is tested when the examiner uses the middle phalange of his or her flexed index finger and firmly presses the patient's relaxed lips and then draws the finger away. Any contraction of the orbicularis oris to this stimulus results in a puckering of the lips or the chin.

The *suck reflex* is similar. The examiner firmly places the knuckle of his or her flexed index finger between the subject's lips. There should be no response from either stimuli. Any pursing or sucking motion by the subject's lips or movement of the chin is recorded as a disinhibited reflex and an abnormal response.

The *grasp reflex* is tested both with and without distraction. First, the patient is told to relax his or her hand while the examiner uses his or her own finger to stroke the palmar surface of the subject's hand. Next, the patient is distracted by being instructed to spell a simple word such as "fist" both forwards and backwards. Each hand is stroked without distraction and then with distraction. The addition of a second task ("spell 'fist' backward") to the first task ("relax your hand") increases the stress on the person and allows a disinhibited reflex to become manifest in frontal lobe dysfunction. Absence of any flexion of the subject's fingers is normal. Any flexion of the fingers represents disinhibition and is abnormal.

The results of each of these tests are not abnormal in every case of frontal damage. A pattern of abnormality, that is, three or more abnormal tests, sustains the conclusion that the frontal lobes are damaged. Three abnormal test results reliably correlate with abnormalities in both the MRI of the brain and neuropsychological testing with the Halstead-Reitan battery ([Blake et al., 1995](#)). The correlation is significant but not absolute. We have found that the most sensitive and specific of these frontal neurologic tests are the inability to perform smooth visual pursuit and the inability to perform accurately the three-step Luria test. MRI scans of patients who fail both these tests have a great likelihood of being abnormal. ([Bae et al., 1998](#)).

#### COGNITIVE ASPECTS OF THE FRONTAL LOBE EVALUATION

The *word fluency test* examines frontal function by testing the ability to generate words that are not in a context. In this test, the examiner asks the patient to name as many words as possible in 60 seconds that begin either with the letter "F" or "M." A normal score is 14 plus or minus five. It is abnormal to name fewer than nine words. This is not an intelligence test but a test of improvisation. A person with frontal damage may perform normally on an IQ test or the MMSE. Because frontally damaged people have trouble improvising or in using old knowledge in a new way, the word fluency test provides an indication of frontal dysfunction.

The "king story" can be very useful in separating patients with attentional deficits from those with memory deficits. The story has a familiar sound but contains no familiar details:

Once upon a time there lived a king who was very ill. His physicians could not help him, so he sought the advice of his wise men. His wise men advised him to obtain the shirt of a happy man. The king sent out messengers who scoured the kingdom and finally found a happy man, but the happy man did not own a shirt.

Before this story is recited, the patient is asked to pay close attention to the details and to repeat it, including as many details as possible. When patients are unable to provide all the details of this story, it indicates inattention. Sometimes the inattention is the result of frontal damage, and sometimes it reflects nervousness, depression, mania, schizophrenia, obsessiveness, or intoxication. After the examination has been completed and 15 minutes have elapsed, the patient is asked to repeat the story. If the patient recalls the details that *he or she* included in his initial repetition of the story, the examiner can conclude that the patient's memory is intact. The combination of poor attention span and intact memory is frequently seen in patients with frontal dysfunction.

The abnormalities on the physical and cognitive tests described earlier are age dependent. What is abnormal in adults may not be abnormal if found in children. Unfortunately, there are no studies of normal children that indicate at what age a frontal sign is abnormal.

The psychological tests that most reliably reflect frontal dysfunction are those that assess executive functions. These are mediated in large part through the frontal lobes. The most sensitive tests are the Wisconsin Card Sorting test, Trail Making Tests A and B, the Categories subtest of the Halstead-Reitan battery, and tests of continuous performance such as the Stroop Color and Word Test. The pediatric psychologists and school psychologists, who are ordinarily the ones who test children with behavioral, cognitive, and learning disorders, rarely use these tests of frontal, executive functioning, although many of these tests have been standardized to age 6 years. No psychological evaluation is complete that does not evaluate executive functioning, especially in attentional disorders.

The other tests that are available for the evaluation of the brain include static and dynamic imaging and tests of body fluids. MRI is much superior to CT for identifying brain abnormalities, but even MRI can be misleadingly negative in patients suffering from devastating diseases such as epilepsy, mental retardation, cerebral palsy, torsion dystonia, chorea, and a variety of dementing disorders.

The dynamic tests such as single photon emission CT, PET, and functional MRI are misapplied when used just to see whether the brain is abnormal. All these reflect cerebral blood flow, which is greater in those portions of the brain that are being used. The radiologists who perform the tests usually ask the subjects to remain still, eyes closed, and thinking of nothing, in other words with their brain not working. It is not possible to be sure whether a person is thinking or about what he or she may be thinking. The most trivial thoughts represent brain activity and can result in a change in the regional blood flow as measured by these sensitive tests. For this reason, it is difficult to determine what activity on the imaging test is normal and what is abnormal. Patients with severe frontal dysfunction or severe dyslexia may seem normal on scans performed when these patients are not trying to perform a task with which they have trouble. In other words, when they are not using their brains, their defective brains can seem normal. Only during a task that normally engages a particular defective region of the brain is the abnormality identified. Patients with dyslexia would have to be studied while they are reading. Those with attentional difficulties would have to be studied while they are attempting to attend. So variable are the details of testing that the normal control values of one laboratory cannot be used by another.

The mind and the brain can be disordered by conditions whose presence can only be detected by examinations of body fluids, namely, endocrine, infectious, metabolic, and toxic disorders. These conditions generally require investigation by blood tests such as toxicology screens, complete blood count, sedimentation rate, urea, electrolytes, glucose, tests for syphilis or human immunodeficiency virus, and endocrine functions. Vitamin B<sub>12</sub>, folate, calcium, phosphorus, magnesium, porphyrin screens, lead levels, antinuclear antibodies, and other laboratory tests may be required as clinically indicated. Lumbar punctures have become less necessary as imaging has improved, but suspected inflammatory conditions still require cerebrospinal fluid examination for diagnosis.

#### Differential Diagnosis of Attention Deficit Hyperactivity Disorder

The most common form of ADHD is probably the result of a lag in the rate of normal maturation of the brain. The brain does not complete its growth until early in the third decade, and an important factor in brain growth is myelination of the frontal lobes, the last part of the cortex to join the electrical grid that operates the brain. Such maturational ADHD may largely reflect genetic factors (Wilcutt et al., 2000).

The large male preponderance in children with ADHD ([Arnold, 1996](#)) may be explained if the growth and development of the brain and the frontal lobes are generally slower and more variable in boys. This concept has some experimental support (Benes et al., 1994, [Bachevalier and Hagger, 1991](#)) and clinical support as well ([Flannery et al., 2000](#); [Castellanos et al., 2000](#)). ADHD may often be the reflection of the slow but normal development of frontal circuits. Behaviorally, this would

appear as social immaturity, hyperactivity, and short attention span. ADHD as a manifestation of immaturity is a condition that many would outgrow.

There is a marked predilection of cognitive-behavioral disorders such as ADHD, autism, dyscalculia, developmental disorders of motor execution, and some forms of mental deficiency for boys by margins of four to one. These abnormalities may be X-linked traits such as the fragile X syndrome. Sex steroids may affect early brain maturation. Testosterone could slow the development of certain cortical functions, usually associated with the left hemisphere, and it may speed the development of other functions. This could explain the superiority of girls for verbal tasks and boys for visual-spatial tasks ( [Rapin and Allen, 1988](#)).

Increasingly, ADHD is being recognized not as a separate disease entity but as a behavioral syndrome that has many causes with differing prognoses and treatments that depend on the origin. Neurologic diseases of frontal circuits may produce the same behavioral distortions in a school-age child as immaturity, but these would be less likely to resolve with time. Examples of such disorders may be lead poisoning and traumatic brain injury.

Mental illnesses can also produce symptoms of ADHD in school-age children. Of the mental diseases, mania has received a great deal of attention ( [Carlson, 1998](#); [Geller et al., 1998](#); [Giedd, 2000](#)). Obsessive-compulsive disorder (OCD) ( [Clayton et al., 1999](#); [Moll et al., 2000](#)), Tourette's syndrome ( [Freeman et al., 2000](#)), and schizophrenia ( [Erlenmeyer-Kimling et al., 2000](#)) are other possible causes of ADHD in children. Experiential factors can produce ADHD, often with conduct disorder and oppositional defiant disorder. The symptoms of ADHD can be thought of as one of the childhood expressions of these conditions, rather than being considered another, unrelated, comorbid disease.

## NEUROLOGIC SOFT SIGNS AND OTHER PSYCHOPATHOLOGY

Neurologic soft signs have been described as being overrepresented in patients carrying a diagnosis of childhood autism, Tourette's syndrome, schizophrenia, borderline personality disorder, or anxiety and withdrawal.

### Autism

Like ADHD, *autism* is not a disease entity itself but, rather, a behavioral syndrome of childhood that can result from many different disorders of the CNS that cause bilateral dysfunction. Defective speech is a hallmark, but autistic children often have varying deficits in comprehension, symbolic thinking, and the formation of abstract concepts that reflect varying degrees of CNS dysfunction and the diseases that cause it. This is consistent with the variegated pattern of cognitive functioning in autism, which is often uneven in an individual case, with preserved "islets of intelligence." Autism is often seen in children who show strong independent evidence of neurologic abnormality. It may be associated with phenylketonuria, tuberous sclerosis, and infantile spasms during the first year of life. Many autistic children have multiple cognitive deficits, and perhaps half have, or once had, electroencephalographic abnormalities or a history of seizures or both. The symptoms of autism resemble those of congenital aphasia. Permanent aphasia can be produced in children only by bilateral cerebral lesions.

Hardly any information on the neuropathologic correlates of autism has been published. It is a nonprogressive, nonfatal syndrome, and brain biopsies cannot usually be justified. Some defects in cerebral myelination and myelination of the cerebellum have been reported, but it seems highly unlikely that these constitute the root of the disturbance, which symptomatically resembles more a problem of the gray matter and cerebral cortex than of the white matter or the cerebellum.

Children with autism were compared with neurologically normal children matched for both chronologic and mental ages on two tests of motor imitation and on the Herzig battery for nonfocal neurologic signs ( [Jones and Prior, 1985](#)). The latter includes tests of speech, balance, coordination, double simultaneous stimulation, gait, sequential finger-thumb opposition, muscle tone, graphesthesia, astereognosis, and choreiform movements. Performance was rated as normal, mildly impaired, or markedly impaired. Imitation ability was defective in the children with autism who showed an average of four soft signs. All had two or more such signs, the most frequent of which were choreiform movements (100% markedly impaired); imbalance (70% markedly, 30% mildly impaired); extinction to double simultaneous stimulation (60% markedly impaired); incoordination (40% markedly, 50% mildly impaired); finger-thumb opposition (40% markedly, 30% mildly impaired); and speech disarticulation (40% markedly, 40% mildly impaired).

The significant prevalence of soft signs in this group of 6- to 10-year-old children with autism was marked, universal, and excessive, compared with both chronologically and mentally age-matched controls. Yet some of the soft signs were also seen among the controls. The lack of true, normative developmental data makes it difficult to use the soft signs to support the theory of the organic pathogenesis of autism. The lack of complete understanding of autism makes it unsound to use the supposed organic pathogenesis of autism to validate the meaningfulness of soft signs ( [Jones and Prior, 1985](#)).

### Tourette's Syndrome

*Tourette's syndrome* is a familial neuropsychiatric disorder of unknown origin that is characterized by waxing and waning multiform motor and phonic tics and a range of complex and intriguing behavioral symptoms. Typically, it is a lifelong disorder that can be disabling and that usually begins in the first decade. In addition to the motor and phonic symptoms, several behavioral symptoms are often seen in patients with Tourette's syndrome, including diminished ability to concentrate, impulsiveness, impaired regulation of activity, and disabling obsessions and compulsions. As many as half of the children with Tourette's syndrome satisfy the diagnostic criteria for ADHD. Although most do not have primary learning disabilities, their conduct disturbance can substantially impair school performance. Many patients with Tourette's syndrome do have specific learning disabilities as well, however. Obsessive-compulsive symptoms seen in patients with Tourette's syndrome usually appear late in the developmental course of the syndrome. These include obsessive doubting of decisions, concern about touching objects or persons, and elaborate compulsive rituals.

Family studies and twin data support the notion that Tourette's syndrome and multifocal tics are inherited as an autosomal dominant trait on a single major gene locus ( [Shapiro et al., 1978](#)). The finding that Tourette's syndrome is genetic strongly implicates brain dysfunction as the source of the symptoms. The relationship between Tourette's syndrome and ADHD is controversial. Some studies suggest that ADHD is etiologically related to Tourette's syndrome, whereas other reports are more consistent with the hypothesis that ADHD is a comorbid condition and that the high prevalence of ADHD in a clinical population of patients with Tourette's syndrome may result from an ascertainment bias ( [Cohen et al., 1982](#)). If so, ADHD is still the most common "comorbidity" in Tourette's syndrome ( [Freeman et al., 2000](#)). Increased rates of obsessive-compulsive symptoms in patients with Tourette's syndrome and the finding that about 20% of first-degree relatives of Tourette's probands display prominent obsessive-compulsive symptoms have led to the provocative hypothesis that some forms of OCD may be an alternate phenotypic expression of the Tourette's gene.

### Obsessive-Compulsive Disorder

Obsessive-Compulsive disorder (OCD) has been linked to altered neurologic function after head trauma, encephalitis, abnormal birth events, and Tourette's syndrome. Abnormalities in CT scans, EEGs, PET scans, and evoked potentials have been described in this disorder, but they are neither consistent nor pathognomonic of OCD. Forty-one medication-free patients with OCD who met the criteria of the third revised edition of the *Diagnostic and Statistical Manual of Mental Disorders*, as well as 20 normal controls matched for age, sex, and handedness, were studied on 20 individual tasks that involved fine motor coordination, involuntary movements, and sensory and visual-spatial function. There were significantly more signs of CNS dysfunction in the OCD group, as shown by abnormalities in fine motor coordination, involuntary and mirror movements, and visual-spatial function. An excess of findings on the left side of the body and abnormalities in drawing cubes may suggest right hemispheric dysfunction in a subgroup of patients with OCD. Soft signs correlated with the severity of obsessions. There was also a correlation between abnormalities in visual memory and recognition on neuropsychological testing and total soft signs. These findings provided additional evidence for a neurologic deficit in some patients with OCD. We lack understanding both of the neurologic basis of soft signs and of OCD ( [Hollander et al., 1990](#)), but both may derive from subcortical dysfunction, either within the frontal lobe or in the basal ganglia and thalamic gray matter that communicate with the frontal lobe ( [Cummins, 1993](#)).

### Schizophrenia

Minor neurologic abnormalities and physical anomalies are commonly found in *schizophrenia*. This is not what one would expect in a "functional" disease, and, for this reason, such signs have often been overlooked or considered epiphenomena. One has only to walk through the chronic wards of a large state hospital to be impressed by the number of patients who suffer from neurologic dysfunction, in terms of impaired equilibrium, gait, and coordination, and even gross mental retardation. The effects of medication, malnutrition, and multiple electrical shock treatments may have something to do with these abnormalities. Certainly, many of the studies of neurologic changes in state hospital populations, for which both the cause for admission and original symptoms often have been long forgotten, are suspect when a high incidence of neurologic findings in patients with schizophrenia is cited. The few such studies done on patients with acute illness, however, have



documented a significant degree of neurologic dysfunction. Abnormalities include minor motor and sensory (soft) neurologic signs on physical examination, EEG abnormalities, and abnormal CT scans.

[Rochford et al. \(1970\)](#) examined 65 hospitalized, untreated psychiatric patients for the presence of the following minor signs: (a) motor impersistence; (b) stereognosis; (c) graphesthesia; (d) extinction during bilateral simultaneous stimulation; (e) bilateral marked hyperreflexia; (f) coordination defects; (g) disturbance of balance and gait; (h) cortical sensory abnormalities; (i) mild movement disorders; (j) speech defects; (k) abnormal motor activity; (l) defective auditory–visual integration; (m) choreiform movements and adventitious motor overflow (tremor); (n) cranial nerve abnormalities, such as slight anisocoria, esotropia, auditory deficit, and visual field and retinal defects; and (o) unequivocally abnormal EEGs. They found neurologic abnormalities in 36.8% of the psychiatric patients (all diagnostic groups). This was significantly different from an age-matched, normal control population (5%). Neurologic soft signs were found in 65.5% of the patients with schizophrenia. By way of comparison, there were no soft signs in patients with primary affective disorders.

Neurologic deficits have been included in descriptions of schizophrenia since Kraepelin first defined dementia praecox. One group of studies reported a greater-than-normal overall number of neurologic signs in patients with adult and childhood schizophrenia and in the children of patients with schizophrenia ([Rieder and Nichols, 1979](#)). Another group reported more neurologic signs in the schizophrenic twin of identical twins discordant for schizophrenia. A third group of studies found deficits in a broad range of specific neurologic areas, including psychomotor integration, vestibular reactivity, lateralization, and involuntary muscle coordination and tone ([Marcus et al., 1985](#)). In fact, the presence of neurologic signs in schizophrenia is so strong a finding that a congenital neurointegrative defect is now believed to be at the base of at least some subtypes of the illness.

[Fish et al. \(1965\)](#) reported in detail on 10 infants, each of whom had a parent with schizophrenia. During the neonatal period, these children showed poor muscle tone and an unusually torpid biological state, and, later, they showed irregular or uneven rates of physical growth and motor development.

The original neurologic examination for the Israeli study of children of patients with schizophrenia ([Marcus et al., 1985](#)) was constructed in 1966 and used child neurology scales. Results showed a subgroup of children of parents with schizophrenia who had poorer cumulative scores than controls.

Five years after the original data were collected, another neurologic assessment was made of the same children using a similar examination. Several statistically significant group differences in neurologic functioning were found. In the original assessment, neurologic signs were more severe in boys than in girls. This sex difference is consistent with the higher incidence of many kinds of major and minimal brain dysfunction and developmental delay observed in boys. A group of children with multiple areas of neurologic deficits was identified, consisting almost exclusively of the children of parents with schizophrenia. In contrast, children with good neurologic functioning were a mixture of index and control cases. This pattern of results is consistent with the model described previously: Only a subgroup of the children of parents with schizophrenia would show biological deficits, whereas others would be indistinguishable from neurologically normal children. Forty-four percent of the children of parents with schizophrenia in this study had signs of a neurointegrative defect during at least one of the time periods. This result is consistent with the hypothesis that approximately half of the children of parents with schizophrenia have neurologic deficits.

The types of neurologic signs most typical of the children of parents with schizophrenia were perceptual–sensory signs, poor motor coordination, poor right–left orientation, poor balance, and motor overflow (the last especially at the older ages). Such minor deficits in attention and inhibition suggest a strong resemblance between this subgroup of children of parents with schizophrenia and children with ADHD. Although many of the children of parents with schizophrenia had other neurologic signs, such as dyskinesias, abnormal reflexes, and facial asymmetries, it was the foregoing group of motor and sensory signs that best defined the poorly functioning subgroup and probably represents the genetically determined dysfunction.

If there are soft signs that correlate with psychosis, they may derive from the same brain abnormality that causes the psychosis. Premorbid deficits in verbal memory, gross motor skills, and attention during childhood were characteristic of the children of parents with schizophrenia who ultimately became psychotic when they reached adulthood ([Erlenmeyer-Kimling et al., 2000](#)).

Reports of EEG abnormality in patients with schizophrenia who were referred at random range from 5% to 80%, with an average of 25%, but the vagaries of EEG interpretation and the variability in diagnosing schizophrenia obscure the meaning of these data. Patients diagnosed as having catatonic schizophrenia seem to show consistently higher rates of EEG abnormality, usually manifested as nonspecific slowing. Because catatonic states are often acute and have a relatively good prognosis, one wonders whether all cases so labeled are really catatonic schizophrenia. The higher rate of EEG abnormality in catatonic schizophrenia raises some question about the diagnosis.

Yet EEG abnormalities are seen in patients with schizophrenia, especially catatonic schizophrenia, more often than in the general population. This statement seems valid even when allowances are made for an occasional misdiagnosis and the effect of drugs and shock therapy. It is not known whether the schizophrenic process or an underlying or associated brain defect causes these EEG abnormalities. One possibility is that brain damage, which may be reflected in the EEG, could facilitate the development of schizophrenia in persons with a genetic tendency to the disease. The theory that brain damage makes it more likely that the gene for schizophrenia will be expressed clinically would also explain the inconsistent but significant association of soft signs on the physical examination with schizophrenia.

CT and, more recently, PET have opened exciting possibilities for studying the structure and function of the CNS in patients with schizophrenia. Since 1976, there have been persistent reports of ventricular enlargement, hemispheric asymmetry, and cerebellar atrophy in patients with schizophrenia. [Rieder et al. \(1983\)](#) and [Shelton et al. \(1988\)](#) have shown ventricular changes in schizophrenia and have shown that these ventricular changes correlate with other findings, such as increased neuropsychological impairment, poor response to treatment, and poor premorbid adjustment. The ventricular enlargements demonstrated would not be called abnormal by radiologists but, rather, are subtle variations within the normal range, and even these are not present in all the patients with schizophrenia who are studied. Other investigations have not confirmed the findings of ventricular enlargement. Almost all the studies in this area, whatever their results, suffer from serious methodologic problems, such as the following: (a) the study populations have been disparate, ranging from old to young, chronic to acute, rigidly diagnosed to less rigidly diagnosed; (b) the techniques of measuring ventricular size in each study are not standardized and vary from manual measurements to computer measurements, thus leading to a great variation from study to study in the incidence of abnormal findings and in their comparability; and (c) the control populations have varied from none, to reported norms in the literature, normal populations, or neurologic patients with headaches who are referred for evaluation. Very few of these studies have compared other chronic psychiatric patients to the patients with schizophrenia, and none has used a blind technique for reading CT scans.

PET is now being used to study schizophrenia, and this technique is so sophisticated that we may be able to observe dynamic changes of cerebral function in patients with schizophrenia during different psychopathologic states and to follow these changes through the course of illness. The PET studies seem to show some decrease of blood flow in the frontal lobes of patients with schizophrenia. Minor asymmetrical hemispheric differences have been noted as well ([Berman et al., 1988](#); [Buchsbaum et al., 1984](#)). These studies, however, have involved so few patients with such varying diagnoses and drug regimens that we must await larger studies and more standardization of the research methods to lend them more weight.

### **Borderline Personality Disorder**

Extensive investigation of patients with *borderline personality disorder* suggests that underlying organic factors may play a role in this complex disorder that consists of affective, behavioral, and cognitive abnormalities. One area of focus has been neurologic and neurophysiologic dysfunction. Findings in patients with borderline personality disorder of abnormal EEG results, histories including head trauma, seizures, and learning disabilities, and similarities to patients with episodic dyscontrol syndrome and adult ADHD have led several authors to postulate that a neurophysiologic dysfunction of the CNS may be responsible for mediating some of the symptoms experienced by patients with borderline personality disorder ([Gardner et al., 1987](#)).

Patients with borderline personality disorder were found to have a significantly greater number of soft-sign neurologic abnormalities when compared with a group of normal control subjects. The presence of two or more soft signs differentiated the two groups statistically. The authors speculated that the nonfocal, soft-sign neurologic abnormalities may reflect underlying CNS dysfunction, which may, in turn, be associated with the development of borderline personality disorder.

### **Anxiety and Withdrawal**

In their study of soft signs, [Shaffer et al. \(1985\)](#) find an odd correlation of early soft signs with *anxiety and withdrawal*. Sixty-three male and 27 female adolescents at 17 years of age, known to have had neurologic soft signs at the age of 7 years, were compared with controls with no soft signs at age 7 years. Adolescents with early soft signs had significantly lower IQs and were more likely to have a psychiatric disorder characterized by anxiety, withdrawal, and depression. All the girls and 80% (12 of 15) of the boys with a diagnosis of anxiety and withdrawal showed early soft signs. There was no relationship between early soft signs and attention deficit or

conduct disorders. Examination of relative contributions of anxiety at age 7 years, IQ, and social and family disadvantage to later diagnosis showed that most of the variance was accounted for by soft signs, independently of IQ. Soft signs and anxious, dependent behavior at age 7 were strongly predictive of persistent psychiatric disorder characterized by anxiety and withdrawal.

### Origin of Soft Signs

The origins of soft signs remain obscure. It is widely held that they have a developmental origin, by which it is usually meant that they diminish in prevalence or severity with age. This hypothesis is supported to some extent by cross-sectional studies that find a higher prevalence of individual signs among younger than older children. However, there have been relatively few longitudinal studies. [Shaffer et al. \(1985\)](#) find that a large proportion of children who have soft signs at age 7 years continue to show such signs at age 17. [Hertzog \(1981\)](#), studying a neurologically deviant population, finds that although there is diminution in amplitude and range of signs in an individual child, children with a sign at one age are likely to show signs, not necessarily the same ones, 5 years later. Further, the notion of signs being "developmental" does not elucidate the problem. The study by [Nichols and Chen \(1981\)](#) on the cohort of children aged 7 from the entire National Collaborative Perinatal Project finds few prenatal predictors, other than maternal smoking or diabetes and chorionitis, to be related to soft signs. Although the relative risk of having soft signs given one of these maternal factors is significantly increased, it is nevertheless low. Postnatal infections, illnesses, and injuries do not predict soft signs.

Nichols and Chen have also studied concordance for signs among the monozygotic and the dizygotic twins in the study population. The difference between them was statistically significant, with greater concordance found in the monozygotic twins. This finding, coupled with observations on the ratio of concordance between full siblings and first cousins, is compatible with a genetic origin of soft signs.

One is led to the conclusion that soft signs on the neurologic examination do reflect brain dysfunction in most cases and are influenced by age and heredity. They do not reliably predict ADHD or any other mental-behavioral-cognitive syndrome. In all likelihood, many soft signs reflect premotor frontal lobe underdevelopment or abnormal frontal development. The prevalence of soft signs is greatest in disorders that have been linked to frontal lobe disease, such as ADHD and schizophrenia, or to basal ganglia disorders of frontally mediated circuits, such as OCD and Tourette's syndrome.

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## 45 PSYCHOEDUCATIONAL EVALUATION IN THE SCHOOLS

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*Psychoeducational evaluation* refers to the use of standardized psychological and educational tests and other procedures to evaluate the aptitudes, abilities, skills, knowledge, attitudes, and behaviors that affect school performance and adjustment. This chapter overviews test use in the schools. It begins with a synopsis of some of the debate provoked by school-based testing. Next comes an examination of the implications of U.S. federal law for psychoeducational evaluation. With these overviews providing a context for discussion, the chapter continues with a presentation of testing goals, types of tests, and domains of functioning typically assessed through school-based testing. The chapter ends with conclusions and implications for the child psychiatrist. (For a general discussion of psychological testing, see [Chapter 43](#).) [Chapter 43](#) presents information about psychological assessment that is essential to a full understanding of psychoeducational evaluation in the schools. All the chapters in the Section VIII B contribute information for understanding the purposes of school-based assessment and the circumstances under which it occurs. Several of the chapters on syndromes in Section VI detail disorders such as mental retardation, developmental learning disorders, and attention deficit hyperactivity disorder (ADHD) that may be first identified through school-based psychoeducational evaluation ([Chapter 120](#) and [Chapter 122](#) provide further essential information).

### SCHOOL TESTING CONTROVERSIES

A long history of controversy has surrounded the use of standardized tests in schools ([Bolon, 2000](#); [Sattler, 2001](#)). More recently, proponents and opponents of testing have rekindled debate. The catalysts for renewed argument include high levels of illiteracy, unacceptable rates of school dropout, increasing proportions of students identified as disabled, and young adults unprepared for certain sectors of the economy ([Broadfoot, 1996](#); [U.S. National Commission on Excellence in Education, 1983](#)). The disparate rates of illiteracy, dropout, disability, and vocational unpreparedness evident among communities that differ with regard to social, cultural, and economic variables add intensity to the discussion ([Duncan and Brooks-Gunn, 1997](#); [Kozol, 1991](#); [McDonnell et al., 1997](#); [Utley and Mortweet, 1999](#)).

At the level of mandatory large-scale testing, supporters reason that measuring academic progress with uniform, objective procedures is the only way to hold students and educators accountable for achievements. These same proponents adhere to the premise that standardization of educational goals and objectives will lead to equality of educational opportunity and therefore will enhance outcomes for all ([Bolon, 2000](#); [Pullin, 1994](#)). Foes of mandatory testing note the questionable reliability and validity of group-administered measures. Opponents maintain that situational factors can idiosyncratically and capriciously affect a student's test scores, thereby reducing reliability and making interpretation difficult. Further, opponents point out that concretizing achievement criteria into paper-and-pencil formats necessarily narrows the scope of the skills, behaviors, and knowledge that tests can tap. This limits test validity by reducing the correlation between test performance and classroom learning (concurrent validity) and future academic and vocational outcomes (predictive validity). Arguments opposing uniform standards regarding the measurement of educational goals and objectives also emphasize the tremendous diversity of the students and communities in the United States. Many educators worry that testing programs can lead to "teaching to the test" at the expense of comprehensive and integrated curricula that take into account the diverse backgrounds, expectations, needs, and goals of students and their families ([Cizek, 1998](#); [Madaus, 1991](#); [Pullin, 1994](#); [Sacks, 1999](#)).

At the level of individual testing, educators and policy makers who support the frequent use of psychoeducational evaluation highlight the importance of understanding individual learning styles when designing instruction and planning curriculum ([Gardner, 1993](#); [Sattler, 2001](#); [Sternberg and Grigorenko, 1997](#)). Educators who back comprehensive individualized psychoeducational assessment also point out that rates of educational disability requiring special education have increased over the past 2 to 3 decades. Nationally, rates of disability exceed 10% ([Reschly, 1996b](#); [U.S. Department of Education, 1999](#)). Thus, high rates of disability justify putting significant resources into identifying and planning for students with special needs. This approach assumes that any attempt at intervention or remediation requires a thorough analysis of the problems to be solved. In essence, accurate diagnosis must precede effective intervention, especially for remediating deficits that are associated with disability. Critics of the regular use of psychoeducational assessment respond that extensive evaluation takes resources away from instruction. Further, they argue that few data support the idea that matching instruction and curriculum with learning style leads to improved learning and performance ([Englemann and Carnine, 1982](#)). Finally, they note that norm-referenced tests have historically been used to justify the exclusion of poor and minority students from mainstream classrooms ([Artiles and Zamora-Duran, 1997](#); [Lopez, 1997](#); [Oswald et al., 1999](#)).

### FEDERAL GUIDELINES ABOUT PSYCHOEDUCATIONAL EVALUATION

State and federal regulations regarding education and disability directly affect the use of educational and psychological tests in the schools, at both large-scale and individual levels ([American Psychological Association, 1999](#)). The U.S. Individuals with Disabilities Education Act (IDEA), most recently amended and reauthorized in 1997, contains many principles and guidelines that govern assessment and evaluation. The IDEA succeeded Public Law 94-142, the historic act passed by Congress in 1975 that transformed public education in this country for all persons ([Martin et al., 1996](#)) (an overview of this law is presented in [Chapter 121](#)). This section covers only those aspects of the act that bears directly on test use and the psychoeducational evaluation process.

Federal law, as amended in 1997, promotes six principles designed to ensure that all students receive a Free and Appropriate Public Education. The six principles are (a) zero reject, (b) nondiscriminatory evaluation, (c) appropriate education, (d) least restrictive environment, (e) procedural due process, and (f) parent participation ([Turnbull et al., 1995](#)). Each of these principles has implications for the regulation of psychoeducational assessment in the schools. *Zero reject* means that no student will be denied an education. Therefore, all students are entitled to evaluations that will yield useful educational data. No students may be judged "untestable" or unable to benefit from education. *Nondiscriminatory evaluation* refers to adhering to standards and maintaining procedures that guarantee that no student will be incorrectly categorized in the special education identification process. This principle puts into place safeguards such as seeking parent permission, assessing in the student's primary language, employing tests that meet professional standards, interpreting test scores in the context of the student's cultural and social background, and using multiple measures. *Appropriate education* refers to the development of an Individual Education Plan (IEP) that takes into account the student's strengths and weaknesses and provides services that ensure that a student will actually benefit from the instruction delivered. For special education students, the goal of psychoeducational evaluation is always development or modification of an IEP. Appropriate education, by definition, leads to measurable increases in the skills, knowledge, attitudes and behaviors that are the target of a student's IEP. *Least restrictive environment* means that instruction must occur in "normative" or "inclusive" settings whenever possible. The implication of this mandate for psychoeducational evaluation is that findings and recommendations must be applied to the regular classroom setting or must provide clear data about the justification for a more restrictive setting. *Procedural due process* principles put in place steps that assure parents that they may challenge educational decisions. Parents have the right to challenge findings from school-based evaluations, either by having findings from their own experts considered by the student's team or by requesting an independent evaluation. *Parent participation* means that parents take part in all aspects of their child's educational planning, including serving as informants in the evaluation process.

The IDEA defines several categories of disability including (a) autism, (b) mental retardation, (c) specific learning disability, (d) serious emotional disturbance, (e) speech and language impairment, (f) orthopedic impairment, (g) traumatic brain injury, (h) other health impairment, (i) visual impairment including blindness, (j) hearing impairment including deafness, and (k) multiple disabilities. Further, the law requires identification of preschool children as developmentally delayed or as having a diagnosed disorder or disability that interferes with education. Gifted and talented students are to be identified, but service is not mandatory ([Wehman, 1997](#)). Psychoeducational evaluation may be an important component of the evaluation of any special education student. It is an essential component of the identification, classification, and program planning process for students with autism, mental retardation, learning disability, serious emotional disturbance, other health impairment (when identified because of ADHD), and developmental delay. It is also usually a critical component of program planning for students with traumatic brain



injury and other neurologic impairments ([Anastasi and Urbina, 1997](#); [Sattler, 2001](#)).

## PURPOSES AND TYPES OF PSYCHOEDUCATIONAL EVALUATION

School-based psychoeducational evaluation serves three major functions. The first, and the one that applies universally to all students, involves the measurement and documentation of academic progress. Almost all students participate in some kind of large-scale, standardized testing of scholastic competencies. These mass testing programs aim at documenting that students have achieved curricular goals, ensuring that schools are adequately serving their communities, and screening for students who are experiencing academic difficulty. Large-scale programs do not, and cannot, employ the kind of assessment procedures needed for diagnosis and special education identification.

The second major purpose of psychoeducational evaluation is the identification of disabled students in need of special education. Psychological and educational tests provide crucial information for establishing differential diagnoses and appropriate educational classification. The third goal of psychoeducational assessment is the development of IEPs. The use of standardized instruments by psychologists and educational specialists can elucidate neuropsychological profiles, learning styles, and cognitive strengths and weaknesses that may give teachers keys to adapting curricula for individual students. Testing may also be one of the methods used to document progress toward IEP goals.

Tests differ on several different dimensions including group versus individual, norm-referenced versus criterion-referenced, and domains of functioning assessed. Group-administered tests may be helpful screening devices. Only individual tests, administered by certified or licensed professionals, are appropriate for diagnosis and special education classification ([American Psychological Association, 1999](#)). Norm-referenced measures compare a student's performance with a specific group. Typically, the normative sample consists of age or grade peers. Supplemental samples may include special populations. If a student's background diverges from that of the normative sample, interpretation of test scores may be compromised. Inappropriate application of scores derived from normative data is one of the major criticisms leveled against test use in the schools. Criterion-referenced measures evaluate a student's performance against absolute and objective measures of skill ([Cohen and Spruill, 1990](#)). Criterion-referenced measures may delineate prerequisites for entry into a program. They may specify successful exit criteria. They may also serve as instructional benchmarks that document progress through a curriculum or course of study. Criterion-referenced measures may also be used as a part of classifying students for special education in certain circumstances and for certain disabilities.

## DOMAINS OF FUNCTIONING ASSESSED

The set of skills, abilities, and behaviors that may be assessed for educational planning is essentially infinite. For practical purposes, domains may be categorized in terms of aptitude, achievement, development during early childhood, and behavior.

### Aptitude and Achievement

Psychological and educational theories have traditionally distinguished between aptitude and achievement and have treated them as orthogonal constructs, even though considerable research documents their complex entanglement ([Ceci, 1991](#); [Daniel, 1997](#); [Hunt, 1995](#)). *Aptitude* refers to the abilities and endowed capacities that a student brings to instruction. *Achievement* refers to the repertoire of acquired knowledge and skill that a student demonstrates as a function of educational experience. In the United States, school-based testing developed during the 20th century as a reflection of these seemingly dichotomous streams. Those measuring aptitude followed in the tradition of Alfred Binet, Theodore Simon, Lewis Terman, Robert Yerkes, and David Wechsler, in search of pure measures of intelligence and other specific abilities ([Anastasi and Urbina, 1997](#)). Other researchers and pedagogues attempted to objectify and quantify achievement through development of standardized measures that could be used on a broad scale ([Bolon, 2000](#)). The press to measure aptitude and to document academic progress in the service of enhancing achievement intensified in the 1950s with the Cold War and the race for space ([Rickover, 1959](#)). In this climate, [Cronbach \(1957\)](#) advanced the still popular tenet that focus on aptitude-by-treatment interactions would increase the efficacy of all educational interventions. The idea that careful assessment of the unique individual characteristics that a student brings to the classroom is a requisite to appropriate instruction continues to guide psychoeducational assessment today.

Assessment strategies that distinguish between aptitude (or ability) and achievement remain a fixture of school-based testing today. However, researchers have typically failed to find aptitude-by-treatment interactions ([Ayers and Cooley, 1986](#); [Good et al., 1993](#); [Reynolds, 1986](#)). Search for aptitude-by-treatment interactions continues apace, however, bolstered by the knowledge that it is not possible to test the null hypothesis and the faith that it is right to fit interventions to individual circumstances and characteristics ([Naglieri, 1997](#)).

### Assessing Aptitude

Certain group-administered tests of ability or aptitude continue to be used in schools today. Group measures may tap some problem-solving aptitude that is different from the academic skills that are targeted by educational curricula. Group aptitude measures may also screen for students who are not achieving up to potential or for students who are intellectually gifted. However, interpreting scores from these tests as measures of intelligence or intelligence quotient (IQ) is *not* appropriate. Determining a student's intellectual potential is typically an integral part of most comprehensive, individual psychoeducational evaluations. In the school setting, individually administered intelligence tests are typically used for three purposes. First, scores are used to predict academic potential and to make judgments about underachievement. Second, scores from intelligence tests are used to determine criteria for disorders such as mental retardation and specific learning disability and to make special education classification decisions. Finally, patterns of strength and weakness evident on intelligence tests may be used to make inferences about learning style.

Intelligence measures can be derived from a variety of psychometric instruments; however, several tests are most commonly used in educational settings. Perhaps the most widely used test today is the Wechsler Intelligence Scale for Children, third edition (WISC-III). The WISC-III yields an overall measure of intelligence or IQ, referred to as a Full-Scale IQ, as well as quotient scores that describe verbal and nonverbal (performance) intellectual abilities. The WISC-III is most commonly used in educational settings to establish a baseline for the child's intellectual functioning. Other standardized measures, such as achievement tests or tests of language ability, can then be compared with IQ scores. Large discrepancies between intelligence and achievement, or between verbal and nonverbal abilities, may indicate potential learning disabilities or language impairments.

Another widely used measure of intelligence is the Kaufman Assessment Battery for Children (K-ABC). Similar to the WISC-III, this test provides a measure of the child's overall intellectual ability. On the K-ABC, this summary score is referred to as a Mental Processing Composite. Compared with the WISC-III, the K-ABC relies less on the child's verbal abilities and acquired knowledge and focuses more on problem solving. Based on Luria's neuropsychological model of intellect and cognition, the K-ABC provides measures of a child's sequential and simultaneous processing. In addition, it attempts to separate academic achievement and acquired knowledge from processing ability. The K-ABC can be a useful tool for determining intellectual potential when one works with children who are very young, who evidence delayed language acquisition, or who come from culturally different or culturally impoverished backgrounds. Historically, many school systems have used another measure of intellectual ability, the Stanford-Binet test. This test was one of the earliest widely used measures of intelligence and is still used by many school systems nationally. However, the more rigorously designed and widely normed WISC-III and K-ABC have become the instruments of choice for many evaluators.

Children with unique learning problems or severe disabilities often present a challenge to psychologists who wish to assess the intellectual aptitude of these children. Many children with severe motor, visual, hearing, or emotional problems may require alternative forms of assessment. Structured clinical interviews, classroom observation, behavioral and play assessments, and curriculum-based assessment are examples of alternatives often used when assessing challenging students. Alternate tests of cognitive functioning abound. When one is confronted with challenging students, perhaps the most important assessment tool is a well-trained professional who has had experience and specialized training in clinical child assessment as it relates to school performance.

### Assessing Achievement

The assessment of academic achievement remains the cornerstone of comprehensive psychoeducational evaluation and provides important information about student skill levels in various areas related to specific programs of instruction. Achievement test results are also used in conjunction with other data to determine eligibility for special education services, gifted and talented programs, and other academic curricula. As mentioned previously, results of achievement testing are also used to measure the effectiveness of individual teachers, schools, and school districts, although this practice is controversial and is fraught with difficulty. More broadly, the goal of achievement testing is to facilitate the process of learning through the assessment of academic progress.

Academic achievement can be measured in several ways. Most commonly, acquired skill and knowledge are assessed using a survey or general achievement battery. These group tests are administered by school personnel in the classroom setting and are designed to assess a range of academic competencies. At the elementary school level, measures of achievement typically focus on the academic skills of reading, mathematics, and written language. At the secondary level, advanced reading comprehension, mathematics, and writing skills continue to be measured, and achievement in content areas becomes another aspect of academic progress to be evaluated. Achievement batteries typically allow for comparison of student scores within a particular grade or age range and permit tracking of individual achievement as a student progresses from grade to grade. Typically, these tests have good psychometric properties and offer norm-referenced scores. However, these tests do not provide the detailed diagnostic and prescriptive level of assessment that is necessary for the identification of strengths and weaknesses necessitating alternative educational programming.

In contrast to general achievement batteries, diagnostic achievement tests are designed to assess specific areas of knowledge, skill, or cognitive processes thought to underlie essential academic competence. These tests are typically individually administered and allow for the identification of problem-solving deficits or gaps in knowledge or skill that may contribute to learning problems. Diagnostic tests may also tap advanced skill levels or cognitive strengths suggesting the need for enriched or accelerated education. Diagnostic tests generally focus on the assessment of basic reading skills such as decoding and phonemic awareness, reading comprehension, mathematical reasoning, arithmetic calculation, and spelling. Tests such as the Wechsler Individual Achievement Test and the Kaufman Test of Educational Achievement assess multiple academic areas, whereas other tests such as the Woodcock–Johnson Reading Mastery or the Key Math Test—Revised allow for a more detailed examination of a single skill area. Generally, diagnostic achievement tests have excellent psychometric properties, are administered by a psychologist or certified educational specialist, and are part of a comprehensive psychoeducational evaluation. Norm-referenced standard scores, percentiles, and grade and age equivalents describe academic performance relative to the cognitive abilities of the individual or to the skill levels of individuals in a normative comparison group.

### Early Childhood Development and Maladaptive Behavior

Perhaps the greatest change in school-based psychoeducational evaluation in the 1990s was the increasing emphasis on the assessment of early childhood development and on the assessment of behavioral disturbances that influence learning. Focus on these domains reflects changes in federal law and recognition that students often present with difficulties more pervasive or complex than traditional aptitude and achievement constructs can characterize. Reauthorizations of the IDEA have mandated special education for children from birth to the age of 5 years. Special education classifications now include autism as a distinct category. When revised in 1997, the IDEA established new standards for educating seriously emotionally disturbed students. Further, it clarified some issues regarding children with disruptive disorders, such as ADHD who may have special education needs. These revisions in federal law emerged from the combined wisdom of educators, researchers, and policy makers about the diverse and pressing needs of students today.

The advent of mandatory special education for preschool students has demanded the use of psychometric instruments to document developmental delay and the need for special services. Federal guidelines delineate five spheres of development requiring consideration: cognitive, language (including receptive and expressive), adaptive (meaning self-help skills), motor (including gross and fine), and personal and social. Numerous instruments have been developed for assessing these areas of functioning. The Bayley Scales of Infant Development, the Mullen Scales of Early Learning, and the Vineland Adaptive Behavior Scales are the three with the most psychometric rigor and developmental breadth ([Chapter 43](#)). These tests are not designed to assess social, emotional, and behavioral difficulties or problems with attention and state regulation that may adversely affect education. Federal guidelines and the instruments used to document preschool special education needs have been criticized for their emphasis on the criterion of developmental delay ([Meisels and Shonkoff, 1990](#)). At times, preschoolers with significant behavioral, attentional, social, and affective needs do not meet eligibility criteria based on delay. This is an area in which child psychiatry expertise can add essential methods to evaluation in the schools.

The need to assess social, attentional, and emotional disabilities that adversely affect education has increased. Children in educational settings are often identified by teachers and other school staff as having potential social, emotional, and behavioral problems that compromise their adjustment or the adjustment of others. Assessment tools can be useful for providing evidence of disabilities requiring special education. They also can be useful for identifying goals and objectives that may be the focus of a student's IEP. The most commonly used measures for assessing social, emotional, and behavioral difficulties are checklists or survey questionnaires that require informants to rate the frequency or intensity of a large number of specific behaviors. Two comprehensive inventories that are used most often in the school setting are the Achenbach Child Behavior Checklist and the Behavior Assessment Scales for Children (BASC). Extensive studies by researchers have documented the excellent reliability, validity, and functional utility of the BASC and the Achenbach test. Both the Achenbach test and the BASC have versions for parents and teachers. The BASC also has a self-report version for children older than 8 years. Responses on these inventories are converted to standardized scores on numerous different dimensions relating to internalizing, externalizing, and school-related difficulties. These data can help educators and clinicians to determine whether a student's behavior deviates significantly from age norms.

Disruptive behavior is often the presenting problem that leads to a student's referral for psychoeducational evaluation. The most widely diagnosed disruptive disorder affecting a child's academic functioning is ADHD. An integral part of the assessment of ADHD is structured observation of the child's behavior in school with formal rating scales. Parent, teacher, and self-report versions of scales measuring symptoms of ADHD are available. The most commonly used scales are Connor's Scales and the Barkley Scales ([Fennerty et al., 2000](#)). These scales allow the observer to quantify difficulties with attention, activity level, and impulsivity and to compare ratings to normative data. Although clinically significant elevations on behavior ratings are a necessary part of making the diagnosis of ADHD, they are not sufficient in and of themselves. History, clinical interview, and, at times, other tests should be employed to establish this diagnosis. In most communities, school personnel do not make this diagnosis alone. Rather, they provide data to psychologists, psychiatrists, or pediatricians that will help to establish this complex diagnosis. ADHD alone is not usually sufficient to meet the requirements for special education services. Children diagnosed with ADHD often have comorbid learning disabilities or psychiatric disorders that can interfere with their academic functioning. At times, students with severe ADHD meet special education criteria for Other Health Impairment. Thus, ratings of attention are often included as part of a comprehensive psychoeducational assessment.

Children who present with behaviors that indicate broad, atypical disturbances in development or seriously impaired social skills may evidence pervasive developmental disorders such as autism or Asperger's disorder. These children often present unique challenges in school and may puzzle and frustrate teachers and clinicians struggling to assess and remediate aspects of the disorder that compromise learning. Although school-based psychoeducational assessment may be useful for describing behaviors and aptitudes, a thorough assessment by a child psychiatrist or clinical child psychologist who specializes pervasive developmental disorders and social disabilities is essential for these students. Finally, students may present with debilitating psychiatric symptoms or behavioral disturbances that impede their academic progress. Disorders such as anxiety, depression, Tourette's syndrome, obsessive–compulsive, and psychosis may profoundly compromise academic adjustment. Again, school-based psychoeducational assessment is always an essential component of any comprehensive evaluation of a student's functioning. For these students, appropriate psychoeducational evaluation typically includes collaboration with clinical professionals outside of school who may use structured clinical interviews or projective tests to establish diagnosis and treatment goals.

### OTHER ASSESSMENT TECHNIQUES

Discussion of psychoeducational evaluation must include note of assessment techniques that augment, and often substitute for, standardized testing. The current trend emphasizes *functional* assessment ([Reschly, 1996a](#)). Functional assessment requires taking an empirical approach to studying students by intervention interactions. In functional assessment, the educational milieu is as much a subject of evaluation as is the student. Assessment identifies environmental, curricular, and interpersonal barriers to academic progress, as well as student attributes. *Classroom observations* that highlight the relation of antecedents, consequences, and student progress are essential methods of functional assessment, as are structured parent and teacher reports. *Portfolio assessment*, which entails the collection and analysis of work samples and the recording of critical incidents, is an increasingly respected method that can provide essential qualitative data that standardized measures cannot generate ([Black, 1993](#)). In *diagnostic-prescriptive teaching*, different instructional methods, materials, and modifications are tested for success. This procedure has become a staple of comprehensive psychoeducational evaluation. In this approach, assessment and intervention are dynamically linked, with student progress serving as both the outcome measure and the standard by which instruction is judged ([Arter and Jenkins, 1979](#)). Finally, clinical judgment and opinion, based on professional consultation, have important roles in school-based evaluation.

### CONCLUSIONS

Various purposes and influences determine the nature of psychoeducational evaluation in the schools. The urgency of improving outcomes for all, the demand for accountability, and federal and state mandates regarding the special education of disabled students all converge to shape the goals, methods, and philosophies of school-based evaluation programs. Historically, psychoeducational assessment has emphasized psychometric testing of aptitude and achievement. More recently, the



domains assessed have widened to include early childhood development, disruptive disorders such as ADHD, and developmental and psychiatric disorders causing social disabilities and emotional dysfunction. The assessment techniques used have also broadened to include functional assessment, classroom observation, parent and teacher report, portfolio assessment, diagnostic prescriptive teaching, and professional clinical judgment. Child psychiatrists can play an essential role in school-based evaluation. When providing case-oriented consultation regarding individual students, psychiatrists will enhance their effectiveness by considering the unique purposes of evaluation in the schools and the unique societal and legislative influences that have shaped its process and products.

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# 46 AUTISM AND THE PERVASIVE DEVELOPMENTAL DISORDERS

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- Prevalence and Epidemiology
- Clinical Description
- Age of Onset
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## DEFINITION

The *perverse developmental disorders* (PDDs) comprise a group of neuropsychiatric disorders characterized by specific delays and deviance in social, communicative, and cognitive development, with an onset typically in the first years of life. Although commonly associated with mental retardation, these disorders differ from other developmental disorders in that their developmental and behavioral features are distinctive and do not simply reflect developmental level (Rutter, 1978). Although better definitions of the syndrome of *autism* continue to be needed, the validity of autism as a diagnostic category is now well established. The validity and definition of other proposed PDDs are more controversial (Volkmar and Cohen, 1988), but several of these conditions are included in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) (American Psychiatric Association, 1994).

Present definitions of the disorder remain profoundly influenced by Leo Kanner's original and phenomenologic description of the disorder (Kanner, 1943), as well as its subsequent modification by Rutter (1978). Of the various PDDs, autism has been the most intensively studied. Both categorical and dimensional approaches to diagnosis have been used (Parks, 1983; Volkmar and Cohen, 1988). Categorical approaches have typically emphasized three different areas of disturbance: social dysfunction, communicative deviance, and certain unusual behavioral features often subsumed under the rubric of *insistence on sameness*. Typically, the characteristic social and communicative deficits are believed to be aberrant relative to the person's developmental level (Volkmar et al., 1995). The development of truly operational definitions has been hindered by various factors: (a) issues of continuity with other disorders, such as schizophrenia; (b) the broad range of syndrome expression; (c) changes in syndrome expression with age; (d) the frequency of autistic-like symptoms in persons with severe mental retardation; and (e) the relative infrequency of the disorder (Volkmar and Cohen, 1988). Autism was not accorded official diagnostic status in the American Psychiatric Association's DSM until 1980, when it was included within a new class of disorders, the PDDs.

The criteria of the third edition of the DSM (DSM-III) for infantile autism included pervasive social deficits, gross deficits in language development, unusual speech patterns (when speech was present), bizarre responses to the environment, and an absence of the delusions or hallucinations typical of schizophrenia. By definition, autism was apparent by 30 months of age. A "residual" category was included for those persons who had once met criteria for autism but no longer did. Even in the relatively short period of time between the DSM-III's appearance (American Psychiatric Association, 1980) and its revision (DSM-III-R) (American Psychiatric Association, 1987), problems with the DSM-III system were apparent (Volkmar and Cohen, 1988). It lacked a developmental focus, criteria were overly restrictive, and language problems rather than broader *communication* problems were emphasized. In response to the criticism that the DSM-III criteria for autism were too "infantile" (i.e., most appropriate to younger and more severely impaired children), major revisions were made in the DSM-III-R.

The DSM-III-R provided a series of 16 individual criteria for autistic disorder. These 16 criteria were grouped into three categories (impaired social interaction, impaired communication, and restricted repertoire of activities). To achieve a diagnosis of autism, a person had to exhibit at least eight of the 16 criteria, with a specified distribution across categories. Age of onset was no longer an essential diagnostic feature, although onset (before or after 36 months) could be specified. These changes gave the DSM-III-R a greater developmental orientation, but at the apparent cost of a broadened diagnostic concept, that is, in comparison with other systems and with the substantial revisions made in the DSM-IV (Volkmar et al., 1988).

In the DSM-IV (American Psychiatric Association, 1994), the definition of autism was developed on the basis of a very large, international, multisite field trial. The final DSM-IV definition (Table 46.1) retains historical continuity with previous definitions of autism, that is, in relation to the requirements for disturbance in three broad areas of developmental dysfunction. It differs from DSM-III-R in that age of onset is included as a necessary diagnostic feature. More important, this definition is conceptually identical to that employed in the tenth edition of the *International Classification of Diseases* (ICD-10) (World Health Organization, 1994) (Chapter 39), as are the definitions of other disorders in this class; aspects of the historical development of these diagnostic concepts are discussed subsequently.

Table 46.1. DSM-IV Criteria for Autistic Disorder (299.0)

In addition to autism, other diagnostic categories are included within the PDD class in the DSM-IV. Although research on these conditions is less advanced than that on autism, there appears to be sufficient justification for their inclusion as specific diagnostic categories.

In *childhood disintegrative disorder* (also sometimes referred to as Heller's syndrome or disintegrative psychosis) (Table 46.2), children develop a condition that resembles autism but arises only after a relatively prolonged period, usually 3 or 4 years, of clearly normal development (Volkmar, 1994). This condition apparently



differs from autism in the pattern of onset, course, and outcome ([Volkmar and Cohen, 1989](#)).

- 
- A. Apparently normal development for at least the first 2 years, as manifested by the presence of age-appropriate verbal and nonverbal communication, social relationships, skills and adaptive behavior
- B. Clinically significant loss of previously acquired skills in at least two of the following areas:
- (1) Expressive or receptive language
  - (2) Social skills or receptive language
  - (3) Bowel or bladder control
  - (4) Play
  - (5) Motor skills
- C. Abnormalities of functioning in at least two of the following areas:
- (1) Qualitative impairment in social interaction (e.g., impairment in nonverbal behavior, failure to develop peer relationships, lack of social or emotional reciprocity)
  - (2) Qualitative impairments in communication (e.g., delay or lack of spoken language, inability to initiate or sustain a conversation, stereotyped and repetitive use of language, lack of varied make-believe play)
  - (3) Restricted, repetitive and stereotyped patterns of behavior, interests and activities, including motor stereotypies and mannerisms
- D. Not better accounted for by another specific Pervasive Developmental Disorder or by Schizophrenia
- 
- From American Psychiatric Association, Diagnostic and Statistical Manual, 4th ed. (DSM-IV), Washington, DC, American Psychiatric Association Press, 1994, with permission.

**Table 46.2. DSM-IV Criteria for Childhood Disintegrative Disorder (299.10)**

In *Rett's disorder* ([Table 46.3](#)), a very brief period of normal development is usually followed by a period of decelerated head growth, loss of purposeful hand movements, onset of characteristic hand-washing stereotypies, and development of severe psychomotor retardation ([Tsai, 1994](#)). This condition has, to date, been observed only in girls. Although the course of this condition differs markedly from that of autism, there is a period of time, particularly in the preschool years, when confusion with autism may occur.

- 
- A. All of the following:
- (1) Apparently normal prenatal and perinatal development
  - (2) Apparently normal psychomotor development through the first 6 months
  - (3) Normal head circumference at birth
- B. Onset of all of the following between 5 and 48 months:
- (1) Deceleration of head growth
  - (2) Loss of previously acquired purposeful hand movements, with the development of stereotyped hand movements (e.g., handwringing or handclapping)
  - (3) Loss of social engagement early in the course (although often social interaction develops later)
  - (4) Appearance of poorly coordinated gait or trunk movements
  - (5) Marked delays and impairment of expressive and receptive language with severe psychomotor retardation
- 
- From American Psychiatric Association, Diagnostic and Statistical Manual, 4th ed. (DSM-IV), Washington, DC, American Psychiatric Association Press, 1994, with permission.

**Table 46.3. DSM-IV Criteria for Rett's Disorder (299.80)**

Of the various disorders potentially included with the PDD class, the validity of Asperger's disorder as separate from autism has probably been the most controversial. Given the absence of "official" definitions, the term has been used in markedly different ways ([Klin, 1994](#)). It appears that the condition differs from autism in that it is associated with higher levels of cognitive and communicative skills and an absence of signs of central nervous system (CNS) dysfunction. Unusual preoccupations (e.g., with train schedules) and high degrees of egocentrism are common, as are motor delays ([Klin, 1994](#)). Similar traits are sometimes noted in family members. The DSM-IV and ICD-10 definitions of this condition are compatible ([Table 46.4](#)).

- 
- A. Qualitative impairment in social interaction of the type described for autism ([Table 46.1](#))
- B. Restricted, repetitive, and stereotyped patterns of behavior, interests, and activities of the type described for autism ([Table 46.1](#))
- C. Lack of any clinically significant general delay in language (e.g., single words used by age 2, communicative phrases used by age 3)
- D. Lack of any clinically significant delay in cognitive development, as manifested by the development of age-appropriate self-help skills, adaptive behavior (other than in social interaction), and curiosity about the environment
- E. Does not meet criteria for another specific Pervasive Developmental Disorder
- 
- Adapted from American Psychiatric Association, Diagnostic and Statistical Manual, 4th ed. (DSM-IV), Washington, DC, American Psychiatric Association Press, 1994, with permission.

**Table 46.4. DSM-IV Criteria for Asperger's Disorder (299.80)**

The term Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS), also referred to as atypical personality development, atypical PDD, or atypical autism, is included in the DSM-IV to encompass "subthreshold" cases, such as when marked impairment of social interaction, communication, interest, or a stereotyped behavior pattern is found, but full criteria for autism or another explicitly defined PDD are not met. This *subthreshold* category is, thus, defined implicitly; that is, no specific guidelines for diagnosis are provided, and the concept has been broadened compared with the DSM-III-R. Although deficits in peer relations and unusual sensitivities are typically noted, social skills are less severely impaired than in classic autism. The lack of definition for this relatively heterogeneous group of children presents problems for research into this condition.

In addition to categorical diagnostic systems, various dimensional approaches to diagnosis have also been employed ([Parks, 1983](#)). These approaches are based on parent or teacher report or direct observation. Typically, deviant behaviors are rated or sampled with attendant issues of standardization and reliability. Changes in syndrome expression over the course of development and the prevalence of high levels of "autistic-like" behaviors in severely retarded persons also pose problems for the development of such instruments. Some work ([Lord et al., 1991](#)), focused on the development of diagnostic and assessment instruments specifically keyed to diagnostic criteria, offers particular advantages for research studies.

## HISTORICAL NOTE

In the 20th century, various diagnostic concepts were proposed to encompass severe psychiatric disturbances of childhood onset. The history of diagnostic concepts illustrates the pitfalls of simple extension of concepts derived from work with psychotic adults; indeed, children's changing conceptions of reality and normative beliefs in fantasy figures suggest the need for considerable caution in such attempts ([Volkmar et al., 1995](#)).

Diagnostic concepts proposed for childhood "psychosis" are presented in [Table 46.5](#). Of particular note is the early interest in the extension of Kraepelin's description of dementia praecox to children. Early assumptions of continuity between severe childhood psychiatric disturbance and adult schizophrenia were based, in large part, on the severity of the disorders. The term *childhood schizophrenia* became synonymous with *childhood psychosis*. In 1943, Leo Kanner described 11 cases of "early infantile autism" and noted various ways in which this disorder appeared to be distinctive ([Kanner, 1943](#)). These children exhibited an apparently congenital inability to relate to other people (in contrast to an apparent ability to relate to objects); their language (when it developed at all) was remarkable for echolalia, pronoun reversal, and concreteness. Behaviorally, these children engaged in repetitive, apparently purposeless activities (stereotypy), were highly responsive to the inanimate environment, and were intolerant of change. Kanner's use of the term *autism* was meant to convey the unusual, self-centered quality that his cases exhibited, but it was also suggestive of the autism associated with schizophrenia. Although Kanner's description has been remarkably enduring, his speculations about certain

aspects of the condition (e.g., normal levels of intelligence, lack of association with other medical conditions, unusual levels of parental education) proved incorrect (Cohen et al., 1994). The validity of Kanner's concept as distinct from schizophrenia was established over the next several decades only, as various lines of evidence became available.

Diagnostic Concept (Current Terminology)	Originator
Dementia praecox	DeSanctis, 1906
Dementia infantilis (disintegrative disorder)	Heller, 1928
Early infantile autism (autistic disorder)	Kanner, 1943
Autistic psychopathy (Asperger syndrome)	Asperger, 1944
Atypical personality development	Rank, 1949
Rett syndrome	Rett, 1966

**Table 46.5. Development of Diagnostic Concepts: Childhood “Psychosis”**

Heller proposed the term *dementia infantilis* to account for children who develop normally for some period before profound developmental regression (Heller, 1930/1969). His concept, also known as *disintegrative disorder*, is now included in the DSM-IV. In 1944, Asperger proposed a novel concept, *autistic psychopathy*, which resembled Kanner's concept in some ways (Asperger, 1944). This disorder, now usually known as *Asperger's syndrome* or disorder to avoid the confusion produced by the word *psychopathy*, has been described particularly in the European literature; in the United States, such persons are more likely to be termed autistic (Cohen et al., 1994). The continuity of Asperger's disorder with autism remains debated; the disorder is of some interest, to the extent that it may establish areas of continuity between autism and other disorders.

## PREVALENCE AND EPIDEMIOLOGY

Since the first epidemiologic study of autism placed the rate in the population at around four per 10,000 (Lotter, 1966), more than 20 studies have been published in the English language. In a review of these studies, Fombonne (1999) reports prevalence estimates that ranged from 0.7 per 10,000 to 21.1 per 10,000, with a median prevalence estimate of 5.2 per 10,000. Not surprisingly, the most important factor accounting for these differences is the nature of the definition used for autism (Wing, 1993), with broader definitions yielding higher rates. Other factors accounting for differences include differences in screening and ascertainment procedures and the size of target populations. Consistent with widespread views that the rate of autism may have been on the increase since the early 1980s (Fombonne, 1996), prevalence rates have significantly increased with publication year, with a median rate of 7.2 per 10,000 for surveys conducted since 1989 (Fombonne, 1999). However, even though this increase in reported rates may reflect an actual increase in the incidence of autism owing to specific environmental or other processes, data to date indicate that this increase has been primarily related to case definition (i.e., more recent studies are more likely to adopt broader definitions of autism and to include conditions that are similar to autism) and case recognition (i.e., there is increased awareness of autism and related conditions in both the general population and the mental health community). Moreover, estimates of prevalence may be affected by issues of diagnosis in the extreme ranges of the condition, that is, the severely mentally retarded and the nonretarded persons with autism. For example, in a study by Honda and colleagues (1996), about half of the identified sample with autism had intelligence quotients (IQs) of at least 70, a finding contrasting with the rate of about 20% reported in most studies for intellectual functioning within the normative range. Although increased awareness and better detection techniques for identification of cases in the extreme ranges may contribute to the more recent prevalence rates, it is also the case that diagnosis is most difficult, and less reliable, in these cases (Klin et al., 2000). Consistent with gender proportion found in clinically referred samples (Lord et al., 1982), Fombonne (1999) reported that the average male:female ratio across studies was about four males for every female. However, this ratio varied according to the absence or presence of mental retardation: in the moderately to severely retarded range, the median gender ratio was less than two males to one female, whereas the median gender ratio in the normative IQ range was more than six males to every female. Although, over the years, potentially causal medical associations have been reported for autism, the strongest connection appears to be with tuberous sclerosis; approximately 20% of patients with this disorder may also appear to exhibit autism (Baker et al., 1998; Hunt and Shepherd, 1993). Tuberous sclerosis is observed in only a small number of cases of autism.

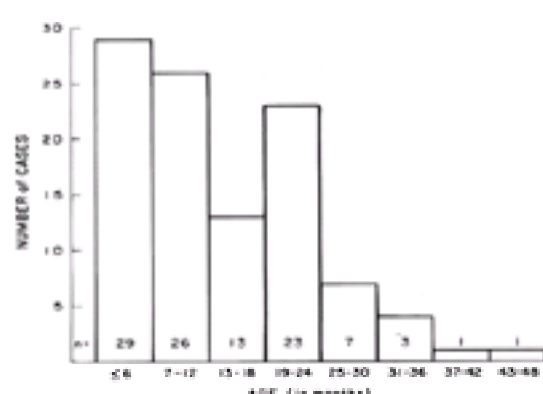
Estimate rates for conditions marked by severe social impairments that fall short of stricter diagnostic criteria for autism (i.e., PDD-NOS) have typically been two to three times higher than the rate of autism (maybe one per 600 or so), although issues of definition and ascertainment become even more acute with this group. Similar methodologic issues question the very high rates reported in a few studies of Asperger's syndrome (Fombonne, 1997). The other PDDs are less common than autism. For example, childhood disintegrative disorder is perhaps 10 times less common than more strictly defined autism (Volkmar and Rutter, 1995), and Rett's disorder or syndrome is similarly uncommon (Leonard et al., 1997). With the exception of Rett's syndrome, which affects female patients almost exclusively, a male predominance has been reported in the other nonautistic PDDs.

## CLINICAL DESCRIPTION

### Age of Onset

In most cases, the apparent onset of autism occurs within the first or second year of life. Age of onset (more properly termed age of recognition) of the PDDs has been important in differentiating these disorders from others (e.g., childhood schizophrenia) and in making distinctions within the PDD class. Studies of large series of cases of “psychotic” children have generally revealed a bimodal distribution of age of onset; children with disorders developing in the first or second year of life are more likely to have autism, whereas those with disorders developing later in childhood, particularly during adolescence, are more likely to exhibit problems more typical of schizophrenia (Chapter 60).

Although Kanner (1943) believed that autism was present from, or shortly after, birth, subsequent work has suggested that the disorder sometimes can be observed after some months, or even a few years, of relatively normal development (Volkmar, 1985). Figure 46.1 illustrates the age of apparent onset (i.e., recognition) of 103 persons with DSM-III diagnoses of infantile autism or childhood-onset PDD and suggests that most cases are recognized in the first or second year of life. Data from detailed parental reports (Volkmar and Cohen, 1988) and from retrospective analyses of videos made during infancy (Osterling and Dawson, 1994) suggest that many children who do not receive diagnoses of autism until later show some differences in social responsiveness and communication, for example, not responding when called by name, at or around their first birthday. Certain factors (e.g., status as a first-born child, parental denial, bilingual household, relatively high intellectual level in child) may act to delay case detection. For example, Asperger's disorder may be detected somewhat later than more typical autism, given the relatively higher intellectual levels and the relative preservation of communicative skills reported to be associated with the disorder (Klin, 1994). In addition, parents of about one-fourth of children with autism spectrum disorders report that their children had a few meaningful words and then stopped talking completely for more than a month, and sometimes for years. Often the loss of words is accompanied by, or preceded by, loss of social skills such as playing patty cake or waving goodbye. This pattern of behavior appears to be quite specific to autistic spectrum disorders, although it is not universal.





**Figure 46.1.** Age of recognition of autism/childhood onset PDD. (From Schopler E, Mesibov G (eds): *Diagnosis and Assessment in Autism*. New York, Plenum Press, 1988, p. 80.)

Children with apparent disintegrative disorder develop an autistic-like condition after a longer period (2 or more years) of unequivocally normal development ( [Volkmar, 1994](#)), which includes the spontaneous use of meaningful phrases, and it often involves loss of toileting, use of utensils, and other adaptive and motor skills. In contrast, in Rett's syndrome, the period of normal development is quite short, usually a few months ( [Tsai, 1994](#)), and it involves the specific loss of directed use of the hands, as well as the development of repetitive behaviors of the hands around the midline.

### Social Disturbance

Autism was initially described by Kanner as a disturbance of affective contact. Social dysfunction in autism is distinctive; it is not explicable in terms of mental age delay in itself and is a, if not the, central defining feature of the disorder. Social features, along with level of language skills and nonverbal intelligence, are the greatest predictors of independence and long-term diagnosis ( [Venter et al., 1992](#)).

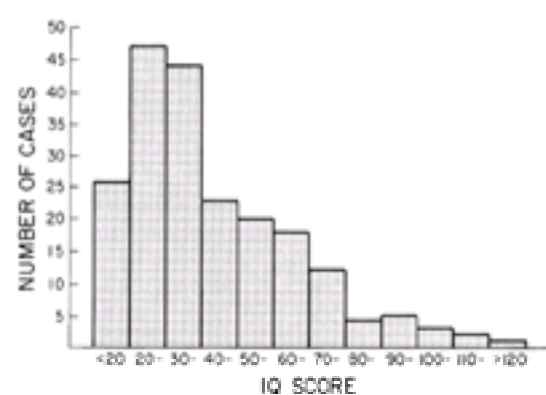
In part, this is testimony to the rapidity and complexity of development of social skills in typically developing children. Normally developing infants are remarkably social, even in the first few months of life. They exhibit an apparent predisposition to form social relationships; this predisposition appears to be an important foundation for the development of other skills ( [Stern, 1987](#)). The social development of autistic children is distinctive in many ways. The human face holds little interest for the autistic infant; lack of eye contact, fewer socially directed behaviors such as facial expressions, vocalizations, or pointing and a lack of interest in other children are typical. Deficits in social interaction in autism change somewhat over the course of development but remain an area of great disability even for the highest-functioning, nonretarded autistic adults. The social deficits in childhood disintegrative disorder and Asperger's disorder are similar to those observed in persons with autism with comparable language and intellectual levels.

### Communicative Disturbance

Deficits in the development of expressive language are the most common source of initial concern for parents of children later diagnosed with autism. Communication problems appear to be a central aspect of the syndrome. Epidemiologic studies have indicated that approximately half the patients with autism never use speech as their primary method of communication, although this number may be decreasing with earlier, more effective intervention ( [Dawson and Osterling, 1997](#); [Rogers, 1998](#)). Those who do speak exhibit language that is distinctive in numerous ways ( [Paul, 1987](#)). The speech of autistic persons is remarkable for the use of stereotyped phrases and delayed echolalia (saying, "Time for bed, honey" as a request to leave the office); pronoun reversal; failure to use appropriate cadence and intonation; impaired semantic development; extreme literalness; and failure to use language for social interaction. Immediate echoing (i.e., repeating right back what someone just said) is observed in normally developing children who are acquiring language, as well as children with autism and those with language impairments, and both types of echolalia may be positive communicative features that can be used to help build a child's language further. Deficits in pragmatic communication, particularly the ability to have a back-and-forth conversation, are common. The language and communicative deficits in autism differ from those seen, for example, in the developmentally language-disordered child ( [Paul, 1987](#)), primarily in the social difficulties that they reflect and in the degree of severity of delay in both receptive and expressive language ( [Lord 1995](#)). The communicative development of persons with apparent Asperger's disorder appears to be less severely impaired than that observed in persons with more classic autism and may represent an area of strength, at least in certain respects ( [Klin, 1994](#)). Children with more fluent, although delayed, language are also more often diagnosed with PDD-NOS or atypical autism. (Towbin, 1997) In Rett's disorder and childhood disintegrative disorder, communication skills, particularly expressive language skills, are usually very limited ( [Tsai, 1994](#); [Volkmar, 1994](#)).

### Cognitive Development

Kanner's initial impression that children with autism exhibited normal levels of intelligence was based on their intelligent appearance and the observation that they performed quite well on certain parts of traditional tests of intelligence; this initial characterization proved incorrect, because most, although not all, persons with autism have since been shown to have significant delays across a range of behaviors concordant with mental retardation. Although attempts were made to account for poor test performance on the basis of "negativism" or "untestability," it became clear that, when developmentally appropriate tests were employed, most (approximately three-fourths) persons with autism scored in the mentally retarded range ( [Fig. 46.2](#)). IQ scores are relatively stable and predictive of outcome ( [Klin and Shepard, 1994](#)). Conversely, marked scatter in performance on tests of intelligence is common and differs from the usual pattern observed in retarded nonautistic children. Islets of unusual ability (e.g., rote memory or block design) may be present ( [Klin and Shepard, 1994](#)). A few autistic persons exhibit truly remarkable abilities, for example, in musical or drawing ability or in exceptional feats of memory, such as the ability to name days of the week corresponding to dates several years in advance ("calendar calculators"). Autistic persons exhibit persistent deficits in abstract thinking and in sequencing and processing information. Lower levels of intelligence are associated both with a greater risk of developing a seizure disorder in adolescence and with a worse outcome. The pattern of verbal versus nonverbal (performance) IQ appears to differentiate persons with autism from those with Asperger's disorder. As may be expected, given that relative preservation of early language skills is the major differential feature of the two conditions, persons with Asperger's disorder often have markedly higher verbal IQ scores, whereas there is more variation in higher functioning persons with autism ( [Klin, 1994](#)).



**Figure 46.2.** Full-scale IQ distribution for 203 autistic individuals, Child Study Center sample. (From Volkmar FR, Cohen DJ: Classification and diagnosis of childhood autism. In: Schopler E, Mesibov G (eds): *Diagnosis and Assessment in Autism*. New York, Plenum Press, 1988.)

Cognitive deficits may be apparent in infancy, and scatter in developmental examination is apparent during the preschool years. In general, autistic children do best with tasks that involve motor and perceptual-motor skills and worst with tasks that involve symbolic information and verbal skills ( [Klin and Shepard, 1994](#)).

### Behavioral Features

The contrast between the response of a child with autism to the inanimate environment (e.g., seeing the credits flash by on television) and the lack of response to social cues (e.g., the voice of a parent) is often quite striking. A child may show relatively little differentiation of his sibling from other children but be particularly attached to an unusual object, such as a spongy puzzle piece. Although parents may initially be concerned that their child with autism is deaf, often the child is quite sensitive to certain nonspeech sounds (e.g., the vacuum cleaner or a jingle on the radio). Interest in nonfunctional aspects of objects (e.g., taste or feel) and stereotyped (purposeless and repetitive) movements are common and include hand flapping, toe walking, spinning objects, and the like. Such activities appear to be preferred modes of behavior and can consume much of the child's time. Unusual affective responses are also common; a child may become panicked in response to new situations, such as having to walk across a porch to a door or regularly recurring stimuli such as singing in church. Play skills are typically quite deviant, accompanied by deficits in imaginative play. These aspects of the disorder are shared with many children without autism who have severe or profound mental

retardation, but they appear to be more common in autism and occur even in children with autism who have normal intelligence ( [Volkmar and Cohen, 1988](#)).

## Developmental Changes

As with other children, significant changes occur over the course of development ( [Rutter, 1970](#)). Typically, preschool children with autism exhibit the more classic syndrome picture previously referred to as infantile autism, with marked lack of interest in other people, absent or severely delayed communication, and cognitive delays. Delays in case detection remain, unfortunately, relatively common because parents are often initially reassured that the child is “just a late talker.” Although parents often report earlier concerns about the child's development, usually delayed language development is the cause for referral. Many children with autism do not show clear repetitive behaviors at the age of 2 years, even though they do show clear social and communicative deficits; behavior may become more obviously stereotyped and unusual as the child enters the later preschool years ( [Lord, 1995](#)). By school age, many children with autism become more responsive socially, develop some response to joint attention (e.g., become able to follow a point), and, in some cases, become more socially directed to familiar people. Language skills and simple gestures may improve considerably, although other skills may be quite deviant. Self-stimulatory and other problematic behaviors, such as self-abuse, also become more common and more difficult to manage. In adolescence, a few persons with autism make marked developmental gains; another subgroup shows very problematic deterioration in behavior. The onset of seizures in adolescence is relatively common, particularly in more severely retarded persons ( [Rutter, 1970](#)). As adults, many of these patients remain severely impaired. Those persons who are able to achieve personal and occupational self-sufficiency still exhibit residual difficulties in interpersonal interaction; typically, these are persons with the highest levels of cognitive and communicative skills.

In Asperger's disorder, the higher intellectual levels, in general, suggest a somewhat better long-term prognosis. Conversely, patients with this condition have been reported to be at increased risk of other psychiatric disturbance, such as psychosis ( [Klin, 1994](#)). The limited data available suggest that the outcome in childhood disintegrative disorder is even worse than that of autism: minimal levels of developmental recovery are typical after the developmental regression. In Rett's disorder, the course of the condition is quite characteristic. Various unusual behaviors (e.g., breath holding, air swallowing) and motor problems (e.g., ataxia, apraxia) are observed, and the patient becomes severely or profoundly mentally retarded ( [Tsai, 1994](#)).

## ETIOLOGY AND PATHOGENESIS

### Biological Mechanism

As the validity of the autistic syndrome became more firmly established, various lines of evidence converged to suggest the importance of neurobiological factors in the pathogenesis of this and similar conditions. Although the variety and consistency of this evidence are impressive, neither specific biological markers for autism nor precise pathogenic mechanisms have been identified. Autistic persons exhibit an increased frequency of physical anomalies, persistent primitive reflexes, and various neurologic soft signs, as well as increased abnormalities on electroencephalograms (EEGs) ( [Golden, 1987](#)). Brain imaging studies reveal various associated abnormalities. Autistic persons are at increased risk of developing seizure disorders ( [Rutter, 1970](#)). The autistic syndrome is observed more commonly (although by no means invariably) in an apparently diverse group of medical conditions, such as tuberous sclerosis, phenylketonuria, maternally inherited deletions (Angelman's syndrome) and duplications of chromosome 15q11-q13, and fragile X syndrome, but much less commonly with others, such as Down's syndrome ( [Cohen et al., 1994](#)). Autism has also been associated with diverse prenatal and perinatal risk factors ( [Tsai, 1987](#)). Similar, although less extensive, data suggest the importance of neurobiological factors in Rett's disorder and childhood disintegrative disorder. The DSM-IV classified pervasive developmental disorder, Rett's syndrome, has been found to result from mutations in a specific gene, *MECP2* ( [Amir et al., 2000](#)). The role of genetic mechanisms in autistic disorder is suggested by the observation that siblings of affected persons are at a 45-fold or greater risk of autism than the general population and are at higher risk of developing various language and cognitive problems; studies of monozygotic and dizygotic twins have shown an increased concordance for autism in monozygotic twin pairs ( [Cook, 2001](#); [Rutter et al., 1997](#)). The apparent genetic component is likely to be more robust in Asperger's disorder ( [Volkmar et al., 1998](#)).

### Family Factors

Kanner's initial report of the autistic syndrome emphasized the apparently congenital nature of autism but also noted the remarkable degrees of occupational success observed in the parents of these first patients, as well as evident deficits in parent-child interaction. These latter observations were subsequently taken to suggest some role of parental psychopathology in syndrome pathogenesis. During the 1950s and 1960s, considerable efforts were made to remediate the effects of deviant caregiving practices of cold (“refrigerator”) parents through, for example, extensive child and parent psychotherapy. Various lines of evidence now make it clear that deviant child-rearing practices do not account for autism. Parents of autistic children resemble parents of children with other developmental problems; they do not exhibit specific deficits in child-rearing practice, nor do they have unusual personality traits ( [Cohen et al., 1994](#)). In retrospect, it appears that early notation of deviant patterns of parent-child interaction may just as well have reflected deviance contributed by the child rather than by the parent; similarly, early studies showing an association of autism with higher social class appear to be artifacts introduced by ascertainment bias; for example, more successful and well-educated families are more likely to seek treatment ( [Schopler et al., 1980](#)). It is clear that the parents of autistic children may be understandably stressed by the experience of caring for a severely impaired child and may suffer from depression and anxiety. In childhood disintegrative disorder, parents often observe some psychosocial or medical event in association with the onset of the condition, such as the birth of a sibling, the death of a grandparent, or hospitalization for elective surgery. It seems likely that such events are simply chance, rather than causal, associations, given their frequency in this age group ( [Volkmar, 1994](#)).

### Environmental and Social Influences

The word *autism* suggests the self-contained quality exhibited by autistic persons. Although evocative, this description tends to minimize the significant impact that the environment can have on persons with autism and related disorders. Early psychodynamic views tended to overemphasize the maladaptive qualities of certain deviant behaviors; for example, poor performance on IQ tests was thought to result from intentional “negativism,” and echolalia was seen as a maneuver used by the child to distance himself or herself from social interaction. Experimental and other data have led to a significant revision of this view. It is clear, for example, that autistic children are not unduly negativistic; they may behave oddly in certain situations, but many aspects of behavioral functioning can be understood as adaptive attempts to cope with a confusing environment. Levels of appropriate behavior are, for example, highest in more highly “structured” situations, and treatment programs that emphasize specific educational goals in the context of highly regulated interactions are more commonly associated with behavioral improvement ( [Lockyer and Rutter, 1969](#)).

## LABORATORY STUDIES

### Biological Studies

Autism and related conditions have been observed in association with certain other conditions ( [Anderson and Hashino, 1997](#); [Golden, 1987](#)). As part of a comprehensive examination, it is important to conduct a careful medical and family history. Genetic screening for various inherited metabolic disturbances is indicated, because some inherited disorders are associated with autism (e.g., phenylketonuria). Chromosome analysis and, possibly, genetic consultation are indicated in the presence of mental retardation or signs of inherited disorders, such as the fragile X syndrome. Hearing tests have often been conducted by the time a child is referred for specialized evaluation; when usual audiologic assessment procedures cannot be employed, brain stem auditory evoked response testing is indicated. Neurologic consultation should be obtained if the child has signs suggestive of overt seizure disorder or other evidence of gross neurologic dysfunction or if unusual features are present (e.g., late onset). EEGs may be helpful in such cases. Computed tomography or magnetic resonance imaging scans may be indicated and sometimes reveal disorders such as tuberous sclerosis or degenerative CNS disease. A history of prenatal or postnatal infections (e.g., congenital rubella) is sometimes elicited. The nature of medical conditions associated with autism and other PDDs remains somewhat controversial. Some investigators suggest a very high frequency of associated medical conditions ( [Gillberg, 1990](#)), but such estimates may be artificially inflated by methodologic and other problems. It appears that the frequency of autism is probably higher than expected in relation to certain medical conditions, although such instances account for only a small proportion of cases of autism ( [Bailey et al., 1993](#)). In general, it seems appropriate to conduct a reasonable search for such conditions, keeping in mind the relative costs of laboratory and other procedures; that is, the choice of laboratory tests or consultations should be guided by the history and examination.

### Psychological Studies

By definition, persons with PDDs have developmental problems in multiple areas of functioning. It is particularly important to approach aspects of psychological assessment, as broadly defined, in a systematic fashion. Problems in assessment are often posed by the difficulties in engaging affected persons, by the need to employ developmentally appropriate assessment methods, and by the degree of developmental scatter commonly associated with these conditions. Commonly, multiple evaluation sessions and the efforts of an interdisciplinary treatment team (including a psychiatrist, a psychologist, and a speech and communication



specialist) are needed. Services of neurologists, as well as physical and occupational therapists, may also be appropriate, such as regarding management of seizure disorder or motor problems.

Intellectual functioning can be assessed by the administration of various standard tests of intelligence or development ( [Klin and Shepard, 1994](#)). To the extent possible, it is helpful to obtain estimates of both verbal and nonverbal or performance IQ. Typically, nonverbal skills are less severely impaired than more abstract verbal skills, which involve sequencing and coding of information. For very low-functioning persons, administration of tests developed for infants and very young children may be appropriate. Adaptive behavior should be systematically evaluated; this is relevant both in terms of documenting degrees of associated mental retardation and for program planning. Speech and communication assessment, not simply limited to evaluation of expressive language or articulation, should be conducted. Psychiatric assessments should include both direct observation and consultation with parents and schools; careful evaluation of reported autistic-like symptoms is indicated. Various rating scales and checklists may aid the diagnostic process but do not replace the need for careful and thoughtful clinical assessment. Associated behavior problems are best evaluated in light of careful developmental assessments. [Table 46.6](#) summarizes evaluation procedures.

1. Historical information (Age development and characteristics of development Age and nature of onset Medical and family history)
2. Psychological and communicative examination (Estimate of intellectual level (particularly nonverbal IQ) Communicative assessment (receptive and expressive language use of nonverbal communication, pragmatic use of language) Adaptive behavior Evaluation of social and communicative skills relative to nonverbal intellectual abilities)
3. Psychiatric examination (Nature of social responsiveness (eye contact, attachment behaviors) Behavioral features (stereotypy/self-stimulation, resistance to change, sensory sensitivities to the environment, etc.) Play skills (nonfunctional use of play materials, developmental level of play activities)
4. Medical evaluation (Associated medical conditions (infectious, genetic, prenatal and perinatal risk factors, etc.) Genetic screen (chromosome analysis and genetic consultation if indicated) Hearing test (if indicated) Consultation (neurologic/pediatric/occupational or physical therapy) as indicated by history and current examination (e.g., EEG, CT/MRI scan)

**Table 46.6. Evaluation Procedures: Autism and Pervasive Developmental Disorders**

## DIFFERENTIAL DIAGNOSIS

Autism and related disorders must be differentiated from other conditions, such as language and other developmental disorders and sensory impairments, particularly deafness. Mental retardation often coexists with autism, and the frequency of autistic-like symptoms increases with more severe retardation ( [Wing and Gould, 1979](#)). Disagreements regarding diagnosis are most pronounced at both ends of the IQ distribution, that is, among very low-functioning and high-functioning persons (Volkmar et al., 1997). Because of the multiple areas of impairment, a multiaxial, developmentally based approach is particularly useful. Specific behavioral features are best viewed in the context of measures of both intellectual and communicative capacities ( [Table 46.6](#)). Individual tests and assessment instruments should be selected as appropriate to the individual patient; measures of adaptive skills in communication and socialization can be viewed in relation to overall cognitive skills and also serve a valuable function in guiding remedial programs. The degree of any associated mental retardation should be noted. Associated medical conditions should be identified; audiologic evaluation is indicated if there are concerns about the child's hearing ability.

Provision of historical information can aid the diagnostic process. The diagnosis of autism is more straightforward when the parents report no history of apparently normal development and when the behavioral deviation is of long standing. Less commonly, some period of apparently normal development precedes the apparent onset of the illness. Questions about specific developmental skills or about development at times of usually well-remembered events (e.g., the child's first birthday) may help to clarify aspects of the early history. Although a few autistic children appear to have had some period of reasonably normal development, such a history suggests that elective mutism, Rett's disorder, childhood disintegrative disorder, language disorder, schizophrenia, and degenerative CNS disorder should be considered in addition to autism. Although some children with histories of severe neglect exhibit deficits in attachment, such deficits typically remit with adequate care, and other features of autism are not typically present. The limited available evidence suggests that children with PDD-NOS probably come to professional attention rather later than is the case with autistic children, and intellectual deficits are less common ( [Dahl and Cohen, 1986](#)). The pattern of onset in disintegrative psychosis is distinctive, although, behaviorally, such patients may be indistinguishable from more typically autistic ones. Both abrupt and gradual developmental regressions have been noted, and intensive neurologic examination does not necessarily reveal a specific cause. Reports of cases of apparent Asperger's disorder suggest that language skills are relatively more preserved, whereas motor delays are more common than in autism. Unusual features (e.g., rapid deterioration in an otherwise normally developing child) suggest the need for thorough medical evaluation. The diagnostician's task is particularly complex when dealing with younger and more severely impaired patients. In some cases, the exact nature of the disorder becomes most clear only over the course of development.

## TREATMENT

Given the severity of these conditions and the relatively poor prognosis, it is not surprising that many varied treatments have been used, including various pharmacologic treatments, somatic treatments (such as electroshock therapy and "patterning"), behavior modification, educational intervention, psychotherapy, dietary change, and the like ( [DeMyer et al., 1981](#)). Unfortunately, until relatively recently, problems in study design and in sample description or selection have made it difficult to assess many treatments systematically. Short-term changes may reflect nonspecific effects and be neither sustained nor clinically significant. It is just such changes, which may be associated with various novel and unproven treatments that are reported, on average, approximately every 6 months in the lay press. Sadly, such reports, usually of amazing successes, are hardly ever rigorously conducted or evaluated. For example, it was suggested that very low-functioning persons with autism were able to communicate through a special modality (facilitated communication) in which the hand of another person (the facilitator) guided that of the person with autism to type out messages on a keyboard or letterboard ( [Cohen et al., 1994](#)). Although the utility of various augmentative communication skills in autism is well established, and although some persons with autism do indeed have particular facility with written (as opposed to oral) expression, systematic study revealed that the alleged communications were most often the product of the facilitator rather than the child ( [Eberlin et al., 1993](#)). Unfortunately, this occurred after some school systems and families had completely reorganized their treatments and goals for these children.

At present, the best available evidence points to the importance of appropriate educational interventions to foster the acquisition of basic social, communicative, and cognitive skills ( [Committee on Educational Interventions for Children with Autism, 2001](#); [Rogers, 1998](#)). Appropriate intensive education not only teaches children with autism spectrum skills that they need to learn and helps them to make more sense of the world, but also it should provide support and training for parents. Behavior modification procedures may be helpful in increasing appropriate and decreasing inappropriate behaviors and may facilitate involvement in educational programming ( [Lovass, 1987](#)). It is clear that early and continuous intervention is highly desirable and has measurable effects on later intellectual and communicative functioning, although initial claims ( [Lovass, 1987](#)) of specific programs resulting in "recovery" from autism in a substantial minority of children with autism have not been replicated ( [Smith and Antolovich, 2000](#)). Educational interventions are best provided over a full day of school (unless a child is very young) on a year-round basis; the usual pattern of summer school vacations is typically not well tolerated by autistic children. Professionals should work with parents to advocate the availability of appropriate educational placements and ancillary services, such as respite care. Because there are often many professionals involved in an evaluation, it is vital that fragmentation of effort be avoided by ensuring that information among professionals and to the parents is conveyed in a timely and responsible fashion. Engagement with persons with autistic spectrum disorders and their families can be lifelong and entails attention to educational interventions, group living situations, and involvement in community-based day and vocational programs, as well as to aspects of family support. Advocacy groups such as the Autism Society of America and similar groups in the United States and other countries have been helpful in this regard; these groups may offer important sources of support to parents as well. Psychotherapy is not usually indicated for children with autism, although it may be useful in higher-functioning adolescents or adults with fluent language, including those with Asperger's syndrome. In such cases, therapy should be carefully focused on specific goals, whether behavioral or emotional and supportive ( [Riddle, 1987](#)). Cognitive-behavioral therapy that is specifically tailored to the individual strengths and difficulties of the person with an autistic spectrum disorder may be helpful with adolescents and adults, as may social groups that provide opportunities for learning and practice of social skills with peers and relevant to work.

Although none of the pharmacologic agents used in the treatment of autism and related conditions have proven curative, certain medications, particularly the major tranquilizers, have been shown to have an important, limited role in the management of certain cases ( [Tanguay, 2000](#)). Careful double-blind studies using haloperidol have demonstrated enhanced learning and improved behavioral adaptation. The major tranquilizers may act to decrease activity levels, increase relatedness and task involvement, and increase accessibility to remediation programs. Patients who receive pharmacologic treatments should be carefully monitored for side effects, and major tranquilizers should be used in the lowest effective dose for the shortest possible period of time. Oversedation should particularly be avoided. Reports suggest the potential usefulness in autism and related conditions of other agents, such as those used in treatment of compulsive behavior ( [McDougle et al., 1994](#)). Studies of

additional agents, such as fenfluramine ([Campbell, 1988](#)) or naltrexone ([Campbell et al., 1990](#)), have not revealed particular benefit. Although considerable interest has centered on the possibility that dietary or vitamin treatments may produce behavioral change, efficacy data from controlled trials are notably absent. The nonspecific increases in activity levels autistic persons sometimes exhibit may be taken to suggest a trial of stimulant medications. In general, stimulants worsen behavioral functioning; this result is not surprising, given that stimulants can induce stereotypes in animals by facilitating the action of the neurotransmitter dopamine. Various somatic treatments have not proven clinically useful. Pursuit of unproven treatments at the expense of educational interventions should be avoided.

## OUTCOME AND FOLLOW-UP DATA

Numerous methodologically sound follow-up studies of autism have been conducted ([Howlin and Goode, 1998](#); [Lotter, 1978](#)). As adults, about two-thirds of persons with autism exhibit very significant limitations in the ability to care for basic personal needs, whereas about one-third of these patients achieve some level of personal and occupational independence, with a smaller number of persons becoming able to live fully independently. The two most important predictors of adult outcome are level of intellectual functioning and communicative competence, even though these do not guarantee a positive outcome because even the highest-functioning individuals continue to exhibit residual and debilitating impairments in social interaction ([Howlin, 2000](#)). Persons with IQs in the moderately and severely retarded range are more likely to have worse outcomes as adults, including the presence of seizure disorders and more severely encompassing language and communication impairments. An important trend has been observed, however; in post-1980 studies, the percentage of persons with better outcomes has significantly increased, whereas the percentage of persons with the poorest outcomes (e.g., living in long-stay institutions) has markedly decreased ([Howlin, 1998](#)), a finding apparently reflecting not only general trends in mental health care but also, and importantly, the fact that older outcome studies focused on persons who had not benefited from any meaningful or systematic intervention. Even more important, with improved earlier identification of children with autism ([Rogers, 2001](#)) and the substantial impact that early intervention services appear to have on the natural course of the condition ([Dawson and Osterling, 1997](#)), the trend toward better outcome overall in autism is very likely to continue as outcome studies are performed on the cohort of children who benefit from empirically proven early, intensive, and structured forms of intervention. It is still the case, however, that curative claims made by proponents of unestablished forms of intervention on the basis of anecdotal or very small studies are unwarranted, and at this time autism is likely to continue to be a lifelong disorder. What is hoped for, however, is the continuation of the current trend suggesting that more and more persons with autism will have a meaningful form of communication and higher levels of independent living skills.

Although fewer follow-up data are available for other forms of PDD, the outcome appears to be worse in the case of both childhood disintegrative disorder ([Volkmar and Rutter, 1995](#)) and Rett's syndrome ([Ellaway and Christodoulou, 1999](#)). In contrast, the outcome for patients with Asperger's syndrome appears to be better ([Howlin, 2000](#); Tantam, 2000; [Volkmar and Klin, 2000](#)), presumably because of better communication and cognitive skills, but possibly also because of great social motivation. The same considerations probably apply to persons with PDD-NOS, although data on these patients are difficult to interpret because of issues of diagnostic definition.

Follow-up studies have illustrated some intriguing aspects of autism that remain poorly understood. For example, it is clear that persons with autism are at higher risk of seizures throughout childhood and particularly in adolescence, a pattern quite unlike that of the normal population, in whom the risk of seizures decreases with age ([Volkmar and Nelson, 1990](#)). A few persons with autism exhibit a pattern of behavioral deterioration during adolescence, whereas another small group appears to improve during this period ([Rutter, 1970](#)). Early outcome studies reported progress in language as "poor" or "very poor." More recently, there have been reports of much greater improvement ([Rumsey et al., 1985](#)), although how much more is difficult to determine, given the variability in measures used in different studies ([Howlin and Goode, 1998](#)). Nevertheless, given the current emphasis on early intervention services and the general focus of these services on fostering communication skills in a very intensive and aggressive fashion ([Dawson and Osterling, 1997](#)), the prospect for better language outcomes in the future is very realistic. As for social competence, despite improvement over the course of a person's life, it is still the case that persons with autism continue to be particularly impaired in this realm, and it is indeed quite rare that such persons are able to achieve romantic or sexual relationships or marriage.

## DIRECTIONS FOR RESEARCH

Considerable progress in understanding the biological basis of autism has occurred since the 1950s. The validity of the disorder has now been established, and attempts to provide more precise definitions have facilitated research studies. Knowledge regarding the validity and definition of other PDDs remains quite limited, however. Continued research is needed to establish their validity, and the development of truly operational definitions for these disorders and autism remains an important research priority. It is now clear that the term infantile autism was, in many ways, a misnomer because autistic infants grow up to be autistic adults. Studies of adults with PDDs remain relatively uncommon. Diagnostic techniques such as positron emission tomography and magnetic resonance imaging scanning may help to elucidate underlying pathophysiologic mechanisms. The study of conditions such as childhood disintegrative disorder may be particularly appropriate to attempt to clarify underlying pathologic mechanisms. At present, it appears that the final behavioral syndrome known as autism may well represent the effects of multiple insults on the developing CNS acting through one or more mechanisms. The explication of underlying CNS substrates for social behavior is particularly important. Similarly, the development of testable hypothesized mechanisms of CNS dysfunction will significantly advance our understanding of these complex disorders.

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### CASE ILLUSTRATION: AUTISM

John was the second of two children born to middle-class parents after normal pregnancy, labor, and delivery. As an infant, John appeared undemanding and relatively placid; motor development proceeded appropriately, but language development was delayed. Although his parents indicated that they were first concerned about his development when he was 18 months of age and was still not speaking, in retrospect, they noted that, in comparison with their older child, John had seemed relatively uninterested in social interaction and the social games of infancy. Stranger anxiety had never really developed, and John did not exhibit differential attachment behaviors toward his parents. Their pediatrician initially reassured John's parents that he was a "late talker," but they continued to be concerned. Although John seemed to respond to some unusual sounds, the pediatrician obtained a hearing test when John was 24 months old. Levels of hearing appeared adequate for development of speech, and John was referred for developmental evaluation. At 24 months, motor skills were age appropriate, and John exhibited some nonverbal problem-solving skills close to age level. His language and social development, however, were severely delayed, and he was noted to be resistant to changes in routine and unusually sensitive to aspects of the inanimate environment. His play skills were quite limited, and he used play materials in unusual and idiosyncratic ways. His older sister had a history of some learning difficulties, but the family history was otherwise negative. A comprehensive medical evaluation revealed a normal EEG and computed tomography scan; genetic screening and chromosome analysis were normal as well.

John was enrolled in a special education program, where he gradually began to speak. His speech was characterized by echolalia, extreme literalness, a monotonic voice quality, and pronoun reversal. He rarely used language in interaction and remained quite isolated. By school age, John had developed some evidence of differential attachments to family members; he also had developed certain self-stimulatory behaviors and engaged in occasional periods of head banging. Extreme sensitivity to change continued. Intelligence testing revealed marked scatter, with a full-scale IQ in the moderately retarded range. As an adolescent, John's behavioral functioning deteriorated, and he developed a seizure disorder. Now an adult, he lives in a group home and attends a sheltered workshop. He has a rather passive interactional style but exhibits occasional outbursts of aggression and self-abuse.

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### CASE ILLUSTRATION: ASPERGER'S SYNDROME

Tom was an only child. Birth, medical, and family histories were unremarkable. His motor development was somewhat delayed, but communicative milestones were within normal limits. His parents became concerned about him at age 4, when he was enrolled in a nursery school and was noted to have marked difficulties in peer interaction that were so pronounced that he could not continue in the program. In grade school, Tom was enrolled in special education classes and was noted to have some learning problems. His greatest difficulties arose in peer interaction; he was viewed as markedly eccentric and had no friends. His preferred activity, watching the weather channel on television, was pursued with great interest and intensity. On examination at age 13, Tom had markedly circumscribed interests and exhibited pedantic and odd patterns of communication with a monotonic voice quality. Psychological testing revealed an IQ within the normal range with marked scatter evident. Formal communication examination revealed age-appropriate skills in receptive and expressive language, but marked impairment in pragmatic language skills.

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### CASE ILLUSTRATION: DISINTEGRATIVE DISORDER

Bob's early history was within normal limits. By age 2 years, he was speaking in sentences, and his development appeared to be proceeding appropriately. At age 40 months, he was noted to exhibit, abruptly, a period of marked behavioral regression shortly after the birth of a sibling. He lost previously acquired skills in communication and was no longer toilet trained. He became uninterested in social interaction, and various unusual self-stimulatory behaviors became evident. Comprehensive medical examination failed to reveal any conditions that could account for this developmental regression. Behaviorally, Bob exhibited features of autism. At follow-up at age 12 years, he still was not speaking, apart from an occasional single word, and he was severely retarded.

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### CASE ILLUSTRATION: RETT'S DISORDER

Darla was born at term after an uncomplicated pregnancy. An amniocentesis had been obtained because of maternal age and was normal. At birth, Darla was in good condition; weight, height, and head circumference were all near the 50th percentile. Her development during the first months of life was within normal limits. At around 8 months of age, her development seemed to stagnate, and her interest in the environment, including the social environment, waned. Her developmental milestones then became markedly delayed; she was just starting to walk at her second birthday and had no spoken language. Evaluation at that time revealed that Darla's head growth had decelerated. Some self-stimulatory behaviors were present. Marked cognitive and communicative delays were noted on formal testing. Darla began to lose purposeful hand movements and developed unusual hand-washing stereotyped behaviors. By age 6, her EEG was abnormal, and purposeful hand movements were markedly impaired. Subsequently, she developed truncal ataxia and breath-holding spells, and her motor skills deteriorated further.

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### CASE ILLUSTRATION: ATYPICAL PERSISTENT DEVELOPMENTAL DISORDER AND PERSISTENT DEVELOPMENTAL DISORDER NOT OTHERWISE SPECIFIED

Leslie was the eldest of two children. She was noted to be a difficult baby who was not easy to console but whose motor and communicative development seemed appropriate. She was socially related and sometimes enjoyed social interaction, but she was easily overstimulated. Leslie was noted to exhibit some unusual sensitivities to aspects of the environment and, at times of excitement, exhibited some hand flapping. Her parents sought evaluation when she was 4 years of age because of difficulties in nursery school. Leslie was noted to have problems with peer interaction. She was often preoccupied with possible adverse events. At evaluation, she was noted to have both communicative and cognitive functions within the normal range. Although differential social relatedness was present, Leslie had difficulty using her parents as sources of support and comfort. Behavioral rigidity was noted, as was a tendency to impose routines on social interaction. Leslie was enrolled in a therapeutic nursery school, where she made significant gains in social skills. Subsequently, she was placed in a transitional kindergarten and did well academically, although problems in peer interaction and unusual affective responses persisted. As an adolescent, she describes herself as a "loner" who has



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# 47 REACTIVE ATTACHMENT DISORDERS OF INFANCY AND CHILDHOOD

Margot Moser Richters, Ph.D., and Fred R. Volkmar, M.D.

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## DEFINITION

It would be natural for a clinician or researcher to assume that the diagnosis of *reactive attachment disorder* refers fundamentally to a disturbance in the child–parent attachment relationship. Not only does the diagnostic label juxtapose the terms attachment and disorder, but also, during the past few decades, the term “attachment” has become virtually synonymous with the infant–caregiver relationship in psychiatry and psychology ( [Bowlby, 1951, 1969, 1980, 1982](#); [Provence and Lipton, 1962](#); [Spitz, 1946](#)). The reactive attachment disorder diagnosis, however, does not necessarily refer to a child's primary attachment relationship. Rather, it applies more broadly to a child's manifestation of disturbances in social relatedness across individuals and contexts.

The abnormal development of social behaviors is presumed to be in response to (hence the term “reactive”) early and profoundly pathological caregiving operationalized as “persistent disregard of the child's basic emotional needs for comfort, stimulation and affection,” “persistent disregard of the child's basic physical needs,” or “repeated changes of primary caregiver that prevent formation of stable attachment” ( [American Psychiatric Association, 1994](#)). The diagnosis therefore is an attachment disorder in the sense that disturbances in the early relationship *between* parent and child are thought to give rise to a diathesis for social dysfunction *within* the child. Thus, although the disorder is thought to arise from a relational context, it differs from V codes and other relational problems in that the behavioral constellation persists over time, is evident across situations, is manifested in contexts well beyond the confines of the child–caregiver relationship, and is therefore a disorder thought to reside within the child.

In its initial incarnation in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) ( [American Psychiatric Association, 1980](#)), reactive attachment disorder was described in general terms as a disorder marked by lack of developmentally appropriate social responsiveness and delayed physical development. With an age of onset *before* 8 months, the diagnostic criteria included detailed descriptions of socially unresponsive behaviors in infants, such as lack of visual tracking, lack of smiling response, lack of alerting and turning toward caregiver's voice, and abnormal physical development such as weak rooting, hypomotility, and failure to gain weight or exhibited weight loss (with no physical explanation).

The diagnosis was essentially a description of failure-to-thrive symptoms, but it was distinguished from this pediatric syndrome by its psychosocial or “nonorganic” cause. Locating the origin of the socioemotional disturbances in the context of the primary attachment relationship also served to differentiate reactive attachment disorder from the pervasive developmental disorders. Whereas abnormal social relatedness is the hallmark of children with these disorders, their defects are thought to be neurobiological, resulting in a lack of capacity for normal social development. Moreover, in contrast to the pervasive developmental disorders, marked improvement in the clinical picture subsequent to adequate caregiving is considered a diagnostic confirmation of the disorder.

The inclusion of reactive attachment disorder in the DSM-III was a significant step toward defining an important clinical phenomenon, but the criteria proved difficult to apply. Some aspects of the definition seemed overly detailed and applicable only to very young infants ( [Rutter and Shaffer, 1980](#)). Additionally, the inclusion of failure to thrive as an essential feature artificially narrowed the diagnostic concept. In response to these problems, the diagnosis underwent major changes in the revised DSM-III (DSM-III-R) ( [American Psychiatric Association, 1987](#)). The transformation of reactive attachment disorder included a dramatic shift from a diagnosis primarily for infants to a disorder more easily applied to young children and a broadening of the diagnostic concept to incorporate the diversity of clinical presentations. The unusual age of onset—before 8 months—was raised to 5 years of age, and the failure-to-thrive symptoms were converted from defining features to associated features documented on Axis III. Rather than a detailed list of symptomatic characteristics, the criteria incorporated two general descriptions of abnormal social relatedness: (a) excessively inhibited, ambivalent interactions, and (b) indiscriminate social behaviors. The psychosocial nature of the disorder was retained through the continued requirement for a history of grossly pathogenic care, diagnostic confirmation by reversed clinical presentation subsequent to exposure to adequate caregiving, and the exclusion criteria of children with infantile autism or mental retardation.

Although the criteria for reactive attachment disorder in the fourth edition of the DSM (DSM-IV) have undergone few substantive changes, they do provide a more accurate interpretation of the research findings. Those findings suggest that the relationship between grossly pathological care and reactive attachment disorder is not as direct as presupposed in DSM-III or DSM-III-R because children's responses to pathological care can be markedly different. The text now clearly states that some children, despite experiencing maltreatment, are able to develop selective attachments to caregivers and do not necessarily develop reactive attachment disorder. In addition, the revised definition deemphasizes the importance of *grossly* pathological care (stating in the text that it is usually but not necessarily present) in recognition that less severe environments can also give rise to the clinical syndrome. The criteria now require the presence of pathogenic care, rather than grossly pathogenic care.

Disturbances in social relatedness continue to be defining features of the disorder, but there are now two designated subtypes: *inhibited* and *disinhibited*. Children manifesting the *inhibited* type are characterized by “excessively inhibited, hypervigilant, or highly ambivalent and contradictory responses” and may exhibit resistance to comforting and “frozen watchfulness” ( [American Psychiatric Association, 1994](#)). The child with the *disinhibited* type, in contrast, exhibits diffuse attachments “manifest by indiscriminate sociability with marked inability to exhibit appropriate selective attachments” ( [American Psychiatric Association, 1994](#)). In addition, mental retardation is no longer an exclusionary criterion. These revisions essentially increase the diagnostic similarities between the DSM-IV and the 10th edition of the *International Classification of Diseases* (ICD-10) ( [World Health Organization, 1992](#)). An enduring difference between the two systems is that the ICD-10 does not require any specific origin, although the presence of pathogenic parental care is implied in the text.

## HISTORICAL NOTE

The recognition that an appropriately nurturing psychosocial environment is a necessary condition for the development of differential relatedness in infants is, surprisingly, a relatively recent phenomenon. Throughout most of history, the emphasis in the “nature–nurture” debate has been on the “nature” side. The presumption that development was largely unrelated to the quality of the psychosocial environment was reflected in institutional care practices for infants and young children and in early work, in the 20th century, that suggested that IQ could be assessed in infancy and was stable into adult life. (See [Boswell, 1988](#), and [Hunt, 1961](#), for interesting reviews of these issues.) The recognition of the profound impact of disruptions in the processes of attachment, by such factors as maternal deprivation, institutional rearing, and maltreatment, signaled a marked shift in the understanding of social developments in infants and young children ( [Ainsworth et al., 1985](#); [Bowlby, 1951](#); [Kempe et al., 1962](#); [Rutter, 1979, 1981](#)). Accordingly, only more recently have efforts been made to develop a diagnostic classification that would capture the clinical picture of children who have experienced pathologic care, particularly in the form of abuse, neglect, institutionalization, or repeated changes in caregivers. The clinical literature proffers an array of diagnostic labels for these children, some describing or emphasizing major signs and symptoms, such as nonorganic failure to thrive, psychosocial dwarfism, and anaclitic depression, whereas others have emphasized the major environmental circumstances of the child,

such as hospitalism, maternal deprivation, and maltreatment syndrome. Reactive attachment disorder is the label selected for the DSM in an attempt to capture both the context of the disorder (“reactive” in response to caregiving) and the sequela of the caregiving (problems in the child’s social relatedness). Diagnostic criteria for reactive attachment disorder incorporate the clinical presentations of children who have experienced various iterations of pathogenic care as they have been described in the maltreatment, failure-to-thrive, maternal deprivation, and institutionalization literature.

What has been curious in the development of the diagnostic category is the relative lack of influence from the field of attachment ( [Zeanah and Emde, 1994](#)). The apparent discontinuity between the DSM-IV definition and findings from the developmental literature is the basis for criticisms proffered by attachment researchers. It is their contention that the name of the disorder is somewhat misleading because the diagnostic criteria include social abnormalities that extend well beyond the infant–caregiver relationship. These developmentalists hold a more circumscribed definition of attachment disorder as residing within the child–caregiver relationship and would like to see the criteria reflect aspects of the attachment behavioral system more directly ( [Zeanah and Emde, 1994](#)).

The conceptualization of attachment disorders as a characterization of child–caregiver relationships has been formalized in a diagnostic classification system developed by [Zero to Three/National Center for Clinical Infant Programs \(NCCIP\) in their manual, \*Diagnostic Classifications of Mental Health and Developmental Disorders of Infancy and Early Childhood\* \(1994\)](#). Similar to DSM-IV, the NCCIP system provides an Axis I diagnosis, Reactive Attachment Deprivation/Maltreatment Disorder of Infancy and Early Childhood, to be applied when a child has experienced persistently inadequate basic emotional, physical, or psychological care and manifests a disturbance in social relatedness. However, the NCCIP diagnostic framework extends beyond DSM-IV by including a classification system for disordered parent–child relationships. Coded on Axis II, there are nine so-called Relationship Disorders in which the child presents with a disturbance specific to a primary attachment figure, but for which a history of grossly pathogenic basic care is not required; these include relationship problems characterized by overinvolvement or underinvolvement; anxiety; hostility; abuse (verbal, physical, or sexual); or mixed types. In contrast to reactive attachment disorder, in which the child necessarily presents with pervasive developmental delays in social relatedness, a child diagnosed with a relationship disorder may manifest symptoms that exist only in the context of the primary caregiving or attachment relationship. Through this classification system, NCCIP system has furnished a meaningful way to understand and address disorders specific to the quality of the child–parent relationship.

Although some applaud the NCCIP’s inclusion of relationship disorders in their classification system, the diagnostic category is still plagued by the same shortcomings as the DSM-IV and ICD-10, namely, the exclusion of criteria that describe disordered infant attachment behaviors, such as disturbances in comfort-seeking behaviors, secure-base behaviors, and exploratory behaviors ( [Zeanah et al., 2000](#)). Furthermore, these investigators suggest that the DSM-IV and its ICD-10 and NCCIP counterparts define a syndrome of nonattachment applicable only to children who have failed to develop selective attachments. The narrow focus of the current criteria, in their view, precludes the diagnosis of children who have formed severely disturbed attachment relationships. Their alternative classification system includes three types of attachment disorders: Disorders of Nonattachment (which are similar to DSM-IV inhibited and disinhibited subtypes), Secure-Base Distortions (which describe children with disturbed attachment relationships), and Disrupted Attachment Disorders (which include children who have experienced the loss of an attachment relationship). These classifications define attachment disorders as a syndrome that exists between individuals rather than within individuals, a significant departure from the theoretical framework of DSM-IV and ICD-10.

Despite these criticisms, research supports the validity of the attachment disorder construct and its viability as a clinical entity ( [Boris, Zeanah, Larrieu, 1998](#); [O’Connor and Rutter, 2000](#); [Volkmar, 1996](#)). Preliminary evidence also suggests that a distinction can be made between the disinhibited and inhibited subtypes, but questions remain about the reliability and usefulness of subtyping ( [Boris, Zeanah, Larrieu, 1998](#); [O’Connor and Rutter, 2000](#); 1994; [Volkmar, 1996](#)).

## PREVALENCE AND EPIDEMIOLOGY

To date, extensive literature searches reveal no epidemiologic studies of the prevalence and incidence of reactive attachment disorder as defined in the DSM-III-R. According to the DSM-IV, the disorder appears to be “very uncommon,” but exactly how uncommon it is and what is the empirical source for this estimate are not specified ( [American Psychiatric Association, 1994](#)). Cases reported in the literature have included children from all socioeconomic levels, although descriptions of children with associated disorders, such as failure to thrive, have reported relationships with variables such as poverty, family dysfunction, and parent psychopathology ( [Richters and Volkmar, 1994](#); [Skuse, 1984a](#) and [1984b](#)). The paucity of epidemiologic data results, in part, from the repeated changes in the diagnostic criteria and the absence of an established protocol for assessing attachment disturbances. To a certain extent, efforts to develop accepted research protocols have been hampered by a limited understanding of the relationship between attachment disorders and attachment classifications. Although researchers have consistently found perturbed patterns of attachment in maltreated children ( [Cicchetti and Barnett, 1991](#)), clearly, some children who are abused or who are institutionalized do form secure attachments to their caregivers ( [Chisholm, 1998](#); [Egeland and Sroufe, 1981](#); [O’Connor and Rutter, 2000](#)). Findings such as these raise questions about the sensitivity of the Ainsworth Strange Situation procedure to attachment disturbances at the level of clinical diagnosis ( [O’Connor and Rutter, 2000](#)). Currently, insecure or disorganized attachment behaviors are commonly understood as risk factors for the development of psychopathology and should not be confused with diagnostic criteria or evidence of clinical caseness ( [Zeanah, 1996](#)).

Given the etiologic requirements for the disorder, it may seem plausible to derive an estimate of affected children from the statistics on maltreated and institutionalized children. Researchers concede, however, that establishing rates of child abuse and neglect is fraught with difficulties. Such estimates depend not only on “reported cases,” the substantiation of reports, and variations in the content, interpretation, and enforcement of reporting laws, but also on our definition of “maltreatment,” which shifts with the prevailing cultural and political winds ( [Barnett et al., 1993](#)). In addition, although the [Panel on Research on Child Abuse and Neglect \(1993\)](#) suggested that 1,000,000 was a *conservative* estimate of the number of children who have been maltreated in the United States, it is not clear what proportion of these children would meet the DSM-IV criteria for experiencing grossly pathogenic care. Finally, there are no available estimates of the number of children experiencing multiple substitutions in caregivers for reasons such as court-ordered custody changes, prolonged parental absences resulting from military deployments, and extensive hospitalizations for illness.

## CLINICAL DESCRIPTION

Because the diagnostic category of reactive attachment disorder is relatively new and has undergone considerable revision, the data available to provide a complete clinical description are limited. Some of what follows, therefore, has been drawn from related literature on failure to thrive, child maltreatment, institutionalization, and attachment. In this context, it should be reiterated that the large body of literature on the development of attachment and, in particular, on attachment types as assessed in the Ainsworth Strange Situation ( [Ainsworth et al., 1985](#)) has only some relationship to attachment *disorder* as such.

### Age of Onset

The literature on maternal deprivation, maltreatment, and institutionalization suggests that children who suffer insults to their early attachment and bonding relationships subsequently exhibit pervasive and persistent defects in their social development. The current diagnostic requirement is that there be marked disturbances in social behavior *before* the age of 5 years (although the diagnosis can be applied as early as the first month of life). The reliability and validity of this specific age of onset have not yet been established, although it seems reasonably clear that, whereas the task of forming attachments continues throughout life, it is during the first years of life that the process is most prone to fundamental disruption ( [Volkmar, 1996](#)).

Empirical questions aside, from a practical standpoint, determining age of onset can be a remarkably challenging task. A review of the clinical literature suggests two common pathways to treatment for children with reactive attachment disorder: (a) pediatric assessments for failure-to-thrive symptoms in infancy and, more recently, for children adopted from international institutions and (b) psychiatric evaluations for behavioral and language delays in early childhood (often subsequent to changes in custody or entrance into school settings). Often it is someone other than the primary caregiver who brings the child in for an evaluation. As a result, ascertaining the quality of early social behaviors becomes almost impossible because the details of the child’s early history are often fragmented and incomplete, particularly for children who are assessed only after permanent removal from the home or after an international adoption. Further research is needed to establish the distribution of apparent onset ages as well as to determine the sensitivity and specificity that the criterion contributes to the diagnosis ( [Volkmar, 1996](#)).

### Social Disturbance

Social dysfunction is the central defining feature of children with reactive attachment disorder. The *inhibited* subtype is marked by the child’s persistent failure to initiate and respond to social interactions in an age-appropriate manner. Instead, the child appears wary and hypervigilant, excessively inhibited, and generally ambivalent in response to caregivers and social interactions. Additionally, caregivers find the child resistant to comforting or prone to exhibit “frozen watchfulness.” In the maltreatment and deprivation literature, these children are described as affectively withdrawn, apathetic, and unresponsive. They are often remarkable for their gaze abnormalities and idiosyncratic or atypical responses to social cues. This pattern of behaviors has been described as a perturbation of the attachment system



characterized by the inhibited development of normal comfort-seeking and social behaviors ([Zeanah et al., 2000](#)).

Children manifesting the *disinhibited* subtype, in contrast, seem to be quite interested in interacting with others but do not appear to make selective attachments. Rather, their relationships are marked by superficiality, “clinginess,” and indiscriminate sociability. In what some call a hyperactivation of the attachment system, these children may demonstrate a preference for a familiar individual but typically engage in diffuse attachments; they seek comfort from unfamiliar adults when they are distressed and willingly, even affectionately, approach strangers ([Chisholm, 1998](#); [O'Connor et al., 1999](#); [Zeanah et al., 2000](#)). Children suffering from the disinhibited subtype also exhibit impaired social behaviors including an inability to detect important social cues, a lack of awareness of interpersonal boundaries, and a failure to engage in social reciprocity ([Rutter et al., 1999](#)). Finally, these children also exhibit excessive aggressiveness and incompetence in their peer relationships ([Alessandri, 1991](#); [Erickson et al., 1989](#); [Hoffman-Plotkin and Twentyman, 1984](#); [Main and George, 1985](#); [O'Connor et al., 1999](#); [Salzinger et al., 1993](#)). For either subtype, the clinical picture may vary depending on chronologic age, developmental level, and early caregiving history.

### Communication Disturbances

Although not included in the diagnostic criteria, indirect evidence from the failure-to-thrive, maltreatment, and deprivation literature and case descriptions of children with reactive attachment disorder suggest that *some* affected children may evidence delays in language development ([Cicchetti, 1987](#); [Coster et al., 1989](#); [Harris, 1982](#); [Oates et al., 1985](#); [Richters and Volkmar, 1994](#); [Rutter and Garmezny, 1983](#); [Skuse, 1984a](#); [Vondra et al., 1990](#)). The linguistic disturbances, when present, appear to range from poor articulation to mild echolalia. The communication impairments among children with reactive attachment disorder appear to be more similar to those of language-disordered children and less similar to the severe forms often seen in children with autism. In addition, language development in these children appears to improve after intervention. Further research is needed to determine the nature and extent to which language disturbances are present among children with reactive attachment disorder.

### Cognitive Development

Because the emphasis of reactive attachment disorder is on abnormal social development, and because mental retardation was an exclusionary criterion before the DSM-IV, there is a paucity of data concerning cognitive impairments among affected children. Reports on children diagnosed with reactive attachment disorder and the indirect evidence from research on children raised in depriving and maltreating environments suggest that intellectual functioning is often below average or delayed at initial assessment. It is clear, however, that children with mental retardation also usually form attachments, albeit at a time commensurate with developmental level ([Berry et al., 1980](#)). [Zeanah and Boris \(2000\)](#) suggest that the diagnostic criteria could be improved by requiring a minimal mental age of 10 months to guard against normal delays in the development of selective attachments resulting from cognitive impairments. Again, reactive attachment disorder differs from other disorders by the presence of marked improvements after therapeutic intervention ([Crittenden, 1985](#); [Erickson et al., 1989](#); [Hoffman-Plotkin and Twentyman, 1984](#); [Richters and Volkmar, 1994](#); [Tizard and Rees, 1975](#)). Postinstitutionalized Romanian children who had experienced severe and global deprivation under the Ceausescu regime were found to have remarkable cognitive gains by the age of 4 years, a finding further supporting the contention that significant improvements in child-rearing conditions can effect dramatic developmental gains.

The cognitive deficits exhibited by some children with reactive attachment disorder are most accurately described as an associated feature. Researchers in the field of maltreatment and deprivation have identified multiple pathways to cognitive impairments, ranging from physical abuse to low economic status. More specifically, in these pathogenic environments intellectual deficits may be the result of brain damage from head injuries, neurologic insults stemming from malnutrition, depressed language development because of inadequate verbal stimulation, or the effects of heavy medication administered to exert behavioral control as seen in some of the Romanian institutions ([Herrenkohl et al., 1984](#); [Kaufman and Cicchetti, 1989](#); [Rutter and the English and Romanian Adoptees Study Team, 1998](#); [Skuse and Bentovim, 1994](#); [Terwogt et al., 1990](#)). Data from the series of studies on Romanian adoptees conducted by Rutter and his colleagues ([O'Connor et al., 1999](#); [O'Connor and Rutter, 2000](#); [Rutter and the English and Romanian Adoptees Study Team, 1998](#)) lead them to conclude that psychological deprivation appears to play a more powerful role than nutritional deprivation in subsequent cognitive development. This finding is consistent with the results of a population-based study in which children diagnosed with nonorganic failure to thrive in infancy were found to have no cognitive or educational disadvantages by the time they reached school age ([Drewett et al., 1999](#)). Moreover, in the studies on the Romanian adoptees ([O'Connor et al., 1999](#); [Rutter and the English and Romanian Adoptees Study Team, 1998](#)), the duration of deprivation seemed to have a more adverse effect on cognitive development than the severity of deprivation.

### Behavioral Features

Clearly, the most striking feature of children with reactive attachment disorder is their atypical social presentations. Yet other behaviors are frequently associated with the disorder. Disruptive, disorganized behaviors, along with poor affect regulation and low frustration tolerance, have been noted particularly among the *disinhibited* type. Some of these children also exhibit distractibility and attentional problems. Studies of the postinstitutionalized Romanian children continue to suggest a link between attachment disorders and the presence of externalizing behaviors, particularly inattention and hyperactivity, but the nature of that link remains uncertain ([Chisholm, 1998](#); [O'Connor and Rutter, 2000](#); [Zeanah, 2000](#)). Although institutionalized children appear to be at risk for developing both attachment and behavioral problems, it is unclear whether these problems share common, overlapping, or discretely different risk factors. Anecdotal clinical descriptions have also cited underdeveloped self-care skills and self-stimulatory behaviors ([Richters and Volkmar, 1994](#)). Among postinstitutionalized children from Romania, [Rutter and colleagues \(1999\)](#) document the presence of both isolated autistic features and what they term overall “quasi-autistic” behavior patterns. These researchers have adopted the latter term because, in sharp contrast to “ordinary” autism, there is almost no gender discrepancy among affected children, head circumference is not elevated, and the children show marked improvement over time. Furthermore, because the children’s behaviors are characterized by indiscriminate friendliness and other social abnormalities typical of attachment disorders, the investigators suggest that the “quasi-autistic” behavior pattern may be indicative of some form of attachment disturbance.

### ETIOLOGY AND PATHOGENESIS

To date, no known primary biological mechanisms underlie the development of the disorder. Evidence of the presence of a biological predisposition may be eventually revealed, given the considerable variation that has been reported among siblings raised together in extremely depriving and maltreating early environments as well as among postinstitutionalized children ([Hinshaw-Fuselier et al., 1999](#); [O'Connor et al., 1999](#); [Skuse, 1984a](#)). In the maltreatment literature, a transactional model has emerged as a way to understand such diversity in outcomes ([Cicchetti, 1987](#)). There may, for example, be biological factors, such as physical anomalies, persistent colic, or difficult temperament, that influence the likelihood that a child will be victimized by a caregiver. Moreover, certain factors, such as difficult temperament, may also influence the likelihood that a child will react pathogenically to maltreatment. Children with these biological risk factors who also experience pathogenic infant-caregiver relationships may consequently experience disruptions in their achievement of, or successful progression along, normal developmental lines. The formation of selective attachment relationships may be one of the normal developmental tasks that falters and thereby sets the stage for the emergence of reactive attachment disorder. More recently, researchers have been developing a psychobiological theory of attachment that looks beyond the natural unfolding of internal biological processes to examine how social experiences affect infant brain development ([Schore, 1997](#); [Zeanah and Boris, 2000](#)). The exact nature and role that the infant-caregiver relationship plays in the development of neural pathways, brain processes, and subsequent behaviors are still under investigation. However, the evidence is clear that the brain of the human child is genetically designed to assemble its own social, emotional, and personality characteristics by taking instruction from its local environment. Early environmental experiences therefore provide the initial scaffolding for social, emotional, and intellectual functioning and help to organize the child's understanding of the world, herself or himself, and others.

By definition, reactive attachment disorder arises in response to pathological caregiving. In fact, the growing body of literature on the effects of maternal deprivation, maltreatment, and institutionalization led to the recognition of reactive attachment disorder as a clinical phenomenon. Several family factors are thought to put attachment relationships at risk, including death of a parent, maternal depression, maternal substance abuse, marital discord, poverty, multiple early hospitalizations, and placement in foster care ([Boris and Zeanah, 1998](#)). Among institution-reared children, the lack of opportunity to form selective attachments appears to be an important contributing factor ([O'Connor and Rutter, 2000](#)). However, to date, there has been no systematic research published detailing significant family factors among affected children. The omission of *grossly* pathogenic care from the formal DSM-IV criteria (it has been replaced with, simply, “pathogenic care”) suggests a continuum of inadequate caregiving that may give rise to the disorder. Unfortunately, the etiologic requirement of adverse caregiving has rendered it impossible to determine whether the condition arises in the absence of such care. In addition, although it is clear in the maltreatment literature that severe abuse or neglect can give rise to disorders of attachment, there is still no definitive evidence on the effects of milder forms of disturbed caregiver-child interaction styles ([Volkmar, 1996](#); [Zeanah and Zeanah, 1989](#)). Researchers in the area of nonorganic failure to thrive describe some differences between mothers with and without infants with nonorganic failure to thrive ([Drotar et al., 1990](#)). In parent-child interactions, mothers of affected infants are observed to have less adaptive social interactions, less positive affect, and more arbitrary termination of feedings. In addition, researchers also report that mothers with nonorganic failure to thrive infants frequently complain of lack of perceived emotional support from family members ([Drotar, 1991](#)). No differences are found, however, on factors such as family size, maternal age, maternal

education, parent marital status, or sex of child.

Preliminary findings with institutionalized children suggest that disordered attachment behaviors are more related to the duration of deprivation than the severity of deprivation ([O'Connor and Rutter, 2000](#); [Rutter and the English and Romanian Adoptees Study Team, 1998](#)). Furthermore, although psychological deprivation and nutritional deprivation often coexist, psychological deprivation appears to have a more profound effect on the presence of disturbed attachment behaviors ([O'Connor et al., 1999](#)). In cases reported thus far, however, it appears that the symptoms of children with reactive attachment disorder are pervasive and are typically manifested across situations and across relationships. Our current understanding of the disorder also suggests that environmental and social influences play an essential role in reversing the clinical picture. In fact, the amelioration of symptoms subsequent to the introduction of adequate caregiving has often been used as a means for confirming the diagnosis.

## LABORATORY STUDIES

No specific laboratory studies exist for reactive attachment disorder.

## DIFFERENTIAL DIAGNOSIS

Various conditions or situations may give rise to problems in infant–parent attachment. For example, the separation of a young child from a primary attachment figure may be associated with the sequence of *protest*, *despair*, and *detachment* outlined by Bowlby. In some such instances, particularly with somewhat older children, depressive symptoms may predominate, and, if the reaction is prolonged, a diagnosis of depression may be indicated. Sometimes blind or deaf children may initially seem to have deficits in attachments, but, usually, once the sensory impairment is identified and the appropriate interventions are made, the apparent social deficit improves markedly.

Infants and young children with mental retardation that is not associated with autism or another pervasive developmental disorder develop attachments that are appropriate to their developmental level; such attachments may, however, be more prone to disruption, for example, as a result of unstable environments, and it is possible that such children are at greater risk of developing reactive attachment disorder. In the pervasive developmental disorders, deficits in reciprocal social interaction are, by definition, present and are usually observed from very early in life. Social deficits may be particularly profound in autistic disorder, especially in very young children. However, over time some selective attachments do develop, although these are usually highly deviant. The presence of other characteristic symptoms (e.g., marked deficits in communication and unusual behavioral responses to the environment, such as stereotypy and resistance to change) usually alerts the clinician to the diagnosis. Because autism and related conditions are usually associated with mental retardation, the child's overall mental age should be taken into consideration. A substantial body of evidence suggests that, in most cases, the psychosocial environment provided by parents of children with autism and related conditions is appropriately stimulating, and the failures in social development observed reflect a fundamental disturbance in the child. As would then be expected, changes in the environment do not typically result in major behavioral improvement or the development of more robust attachments.

Although some children with the disinhibited subtype of reactive attachment disorder may exhibit inattention or hyperactivity and poor concentration, their symptom picture differs from that of children with attention deficit hyperactivity disorder because of their lack of selective attachments. Although it is likely that the two disorders share common risk factors, they are nonetheless considered distinct clinical entities. As suggested in the DSM-III ([American Psychiatric Association, 1980](#)) criteria for this condition, some infants and young children with reactive attachment disorder may also exhibit disturbances in feeding (e.g., feeding disorder of infancy, rumination disorder, or pica) and in growth (e.g., psychosocial dwarfism).

## TREATMENT

Intervention should focus on the totality of the child's situation. Efforts should be made to support the child's development, the adequacy and responsiveness of the parents, and the provision of an appropriately stimulating and nurturing psychosocial environment. Treatment should emphasize the fostering of selective attachment relationships. Provision of appropriate pediatric and early intervention services should be made. Assessment by a pediatrician is particularly important for postinstitutionalized children because they have often experienced malnutrition and inadequate health care. Although foster placement is best avoided if possible, sometimes removal of the child is mandatory, for example, because the child has sustained life-threatening injuries or is in immediate danger. Although the usual presumption is that reunification of child and parents is the eventual goal, it is important to balance attempts at rehabilitation with the child's need for stability. Effective coordination and integration of services are critical for children living in foster care, and clinicians need to be aware of forensic issues such as confidentiality, permanency planning, and custody arrangements. Among children who have been recently adopted, clinicians should be sensitive to goodness-of-fit issues and should assess the degree to which the parent and the child are coping with the new relationship.

The attempt to produce major changes in the child–caregiver relationship often requires sustained, intensive, and highly structured work with the child, the caregiver, and the dyad. Appropriate additional support services should be made available to the caregiver. Pediatric monitoring is particularly critical if eating or growth problems are present.

## NATURAL HISTORY, OUTCOME, AND FOLLOW-UP DATA

As a diagnosis in its own infancy, there are as yet almost no longitudinal data on the developmental trajectory of children with reactive attachment disorder. The reduction or resolution of symptoms probably depends in large part on the nature, duration, and severity of pathogenic care experienced by the child, on the child's own constitutional factors, and on the interaction between these two. In the studies conducted with Romanian adoptees, early and lengthy deprivation appears to play particularly critical roles in the development of attachment disturbances 1999; ([O'Connor and Rutter, 1999, 2000](#); [Rutter and the English and Romanian Adoptees Study Team, 1998](#)). In general, the prognosis is less hopeful the earlier, more severe, and more prolonged the environmental deprivation and the later adequate caregiving and interventions are introduced. From the clinical descriptions and from studies on children from maltreating and depriving environments, a body of evidence suggests that the symptoms appear to be responsive to therapeutic intervention. Improvements in cognitive functioning, language development, and motor development are cited ([Hodges and Tizard, 1989a,b](#); [O'Connor and Rutter, 2000](#); [Rutter and the English and Romanian Adoptees Study Team, 1998](#)). In addition, there are reports of decreases in behavioral problems and increases in age-appropriate self-care skills ([Richters and Volkmar, 1994](#)).

Unfortunately, improvements in the quality of social relatedness are more difficult to measure. There are reports of improved social skills, but the concern is that these changes may be only superficial. For some children, atypical gaze patterns, idiosyncratic interactive styles, and poor integration of social and emotional cues appear to persist ([Richters and Volkmar, 1994](#)). In addition, among institutionalized children, the pattern of indiscriminate friendliness appears to be persist even after the development of preferred attachments ([Chisholm, 1998](#); [O'Connor and Rutter, 2000](#); [Zeanah, 2000](#)). Many investigators have hypothesized that problems with social development may endure throughout adulthood in the form of difficulty in establishing or maintaining meaningful relationships and persistence of idiosyncratic styles of relating. The question remains whether or to what extent social and emotional development in children with reactive attachment disorder will be systematically different from that in other children.

## PREVENTION

Various factors can be used in the prevention of reactive attachment disorders. These include avoiding institutional and multiple foster placements, adequate stimulation of infants and young children, and provision of adequate parental support. Unfortunately, out-of-home placement cannot always be avoided, and repeated foster placements are often made.

## RESEARCH DIRECTIONS

The relative absence of research on this condition is, in some ways, paradoxical, given both the history of work on the effects of maternal deprivation and orphanage rearing and the important social policy problems created by caring for children with reactive attachment disorders. Information on such basic aspects of the condition as clinical phenomenology, course, epidemiology, and response to treatment is lacking. Although the distinction, as in the DSM-IV, of subtypes of the condition seems generally reasonable, research on the potential importance of this distinction, such as for intervention and outcome, remains unclear. Furthermore, whereas preliminary evidence suggests that the construct of attachment disorder has validity and its importance as a clinical diagnosis is supported, the reliability of the diagnostic criteria remains largely untested. The requirement of pathogenic care as an essential feature continues to be widely criticized and warrants further investigation. Whether the sensitivity and specificity of the diagnostic concept could be improved by the inclusion of other symptoms also remains open for future



research.

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## 48 MENTAL RETARDATION

Fred R. Volkmar, M.D., and Elisabeth Dykens, Ph.D

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### DEFINITION

*Mental retardation* (MR) is defined in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) ([American Psychiatric Association, 1994](#)) on the basis of two essential features: subnormal intellectual functioning, characterized by an intelligence quotient (IQ) lower than 70, and commensurate deficits in adaptive functioning (capacities for social and personal sufficiency and independence). The measurement of intelligence is presumed, in most cases, to be based on the administration of an appropriate standardized assessment of intelligence. Deficits in adaptive skills can be measured on instruments such as the revised Vineland Adaptive Behavior Scales ([Sparrow et al., 1984](#)) or a similar scale. DSM-IV criteria are summarized in [Table 48.1](#).

A. Significantly subaverage intellectual functioning, on IQ of approximately 70 or below on an individually administered IQ test (for inform, a clinical judgment of significantly subaverage intellectual functioning)	
B. Concurrent deficits or impairments in present adaptive functioning (i.e., the person's effectiveness in meeting the standards expected for his or her age by his or her cultural group) in at least two of the following areas: communication, self-care, home living, social/interpersonal skills, use of community resources, self-direction, functional academic skills, work, leisure, health, and safety	
C. Onset before age 18 years	
Code based on degree of severity reflecting level of intellectual impairment:	
317 Mild Mental Retardation	IQ level 50-55 to < 70
318 Moderate Mental Retardation	IQ level 35-40 to 50-55
318.1 Severe Mental Retardation	IQ level 20-25 to 35-40
318.2 Profound Mental Retardation	IQ level < 20 or 25
319 Mental Retardation, Severity Unspecified	when there is a strong presumption of mental retardation but the person's intelligence is unmeasurable by standard tests

Data from American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. (DSM-IV). Washington, DC: American Psychiatric Association, 1994.

Table 48.1. DSM-IV Diagnostic Criteria for Mental Retardation

Various levels of MR are specified in the DSM-IV: mild (IQ 50 to 70), moderate (IQ 35 to 49), severe (IQ 20 to 34), and profound (IQ <20). Borderline MR can be noted as a V code. Some flexibility is allowed for clinical judgment. Most persons with MR in childhood are those with mild MR (about 85% of cases); those with moderate (about 10%), severe (about 4%), and profound MR (1% to 2%) constitute the remainder of cases ([Fig. 48.1](#)). In the past, the distinction between educable (IQ 50 to 70) and trainable (IQ <50) was made. Although no longer commonly used, this distinction is important. Persons with mild MR have psychiatric difficulties that are fundamentally similar (if generally more frequent) to those seen in the general population; this is not true for more severely impaired persons. Similarly, specific medical conditions associated with MR are more likely in the group with an IQ lower than 50, whereas lower socioeconomic status is more frequent in the group with mild MR ([Szymanski and King, 1999](#)). The proportion of persons with severe and profound MR is higher than would be expected given the normal curve reflecting the impact of severe medical problems on development ([Dingman and Targan, 1960](#)).



Figure 48.1. Levels of mental retardation.

The tests chosen for assessment of intellectual functioning must be appropriate to the patient, should have reasonable reliability and validity, and should be administered in a standardized way by appropriately trained examiners. (See [Chapter 43](#) for a discussion of psychological assessment.) Unfortunately, in some situations, the selection of an appropriate test can be difficult, such as for a very low-functioning person. Other aspects of assessment can also be problematic, such as when some modification must be made in terms of administration given the specific circumstance. Such modifications may limit the validity of the results obtained. The examiner must make an informed decision in such circumstances depending on the nature of the issue at hand, for example, determination of eligibility for services versus information on levels of functioning that can guide remediation. Particularly in terms of eligibility for services, it is critical that the examiner administer the test in exactly the standardized fashion. Measures of adaptive skills are generally based on parent or caregiver report, although, in some cases, the person may be interviewed directly. In essence, the conceptual notion is that the term *adaptive skills* refers to capacities for personal self-sufficiency on a day-to-day basis.

The inclusion of adaptive skills in the definition of MR rests on the observation that some persons with IQ scores below 70 may, as adolescents or adults, have learned sufficient adaptive skills that they are able to function totally or largely independently as adults and, thus, would not technically meet criteria for MR. This is more typical of persons who, as children, score in the mildly retarded range ([Edgerton, et al. 1984](#)).

The approach to the definition of MR is fundamentally the same in the tenth edition of the *International Classification of Diseases* (ICD-10) (World Health Organization, 1992). However, the [American Association of Mental Retardation's \(AAMR\) \(1992\)](#) definition of MR discards the use of IQ in favor of a "needs-based" assessment relative to 10 different areas of functioning. This definition also gives the clinician leeway to extend the upper IQ bound to 75; this seeming small increase actually

would be the source of a considerable broadening of the diagnostic concept of MR potentially would double the total number of cases ( [MacMillan et al., 1995](#)). The AAMR definition has been much criticized, has had very little empirical support, and has proven difficult to use.

## HISTORICAL NOTE

Interest in MR can be traced to antiquity ( [Trent, 1994](#); [Zigler and Hodapp, 1986](#)). Modern interest in MR began at the time of the Enlightenment and increased greatly during the 19th century; this emphasis occurred at the time of great social upheaval and as infant and child mortality began to decline. There was increased interest in children, in education, and in the role of experience (nurture) versus endowment (nature). The interest in the “nature–nurture problem” is exemplified in the work of Itard with Victor, a child who was thought to be wild or “feral” but who probably had autism ( [Candland, 1993](#); [Simon, 1978](#)). Subsequently, educators such as Sequin began to develop specific educational methods for stimulating children's development. By the latter half of the 19th century, many facilities had been developed for the care of mentally retarded persons. Although the initial goal of such facilities was to provide a period of treatment before the child was returned to the family, these institutions began to be places for custodial care, a problem that has led to a strong counterreaction in recent years and to a renewed emphasis of caring for persons with MR in their homes and communities.

During the 19th century, various distinctions were made between levels of MR by using what now would be seen as rather pejorative terms such as imbecile, cretin, and idiot. Originally, the etiologic basis of any such distinctions was quite limited. On the one hand, there was little systematic information on intellectual functioning that could be used for purposes of categorization; on the other, there were few known etiologic causes of MR. Both these limitations began to be addressed toward the end of the 19th century and the beginning of the 20th century. Binet developed the first test of intelligence, which was translated into English and adapted in the United States by Terman ( [Binet and Simon, 1916](#); [Terman, 1911](#)); the Stanford–Binet test served as a model psychometric assessment instrument for many years. It allowed much more precise characterization of levels of intellectual disability. In addition, Terman had the brilliant notion of taking the mental age and dividing by the child's chronologic age and multiplying this quotient by 100 to yield the IQ. This simplified comparison across children of different ages. Although Binet had originally developed his scale to identify children who were delayed in order to help them, the IQ score quickly became the object of much study. *Faith* in the IQ as a predictor variable led to the extension of developmental testing in infants and young children and a *belief* that, when the test was properly administered, the resulting score from an IQ test was fixed and reflected a person's genetic endowment. This proved not to be correct, as [Skeels and Dye \(1939\)](#) and others began to demonstrate. In his classic study, Skeels demonstrated this practically by transferring infants and young children from an orphanage to a home for the “feeble minded” to make the children normal! This fantastic plan had been prompted by his clinical observation that children in the home for the feeble minded received considerably more stimulation than those in the orphanage. He reported major differences in outcome both in childhood and, later, in adult life ( [Skeels, 1966](#)). By the 1940s and 1950s, there was increased awareness that the IQ score was indeed the product of both experience and endowment, and therapeutic optimism for improving the functioning of mentally retarded children again increased.

In addition to intellectual functioning, it also became apparent that the person's capacities to engage in appropriate self-care or “adaptive” skills was a major determinant of outcome in MR. In the 1930s, the psychologist Edgar Doll developed the Vineland Social Maturity scale in an attempt to quantify such skills. This instrument has been revised ( [Sparrow et al., 1984](#)), and it continues to serve as an important tool in the assessment of children with MR. Deficits in adaptive skills are now required as part of the diagnosis of MR. In addition, and in contrast to IQ, adaptive skills can readily be taught.

Another major line of work centers on the origin of MR syndromes. This was exemplified in the 19th century by the work of Dr. Langdon Down, who reported on a syndrome that now bears his name and is currently recognized as being the result of trisomy of chromosome 21. At the time of his report, Dr. Down, of course, had no notion of chromosomes. His theoretical understanding, which was based on a mistaken idea of racial stereotypes combined with darwinism, was fundamentally flawed, but his clinical observation has been remarkably robust. As time went on, more and more specific syndromes of MR were identified. It became clear that MR could result from a range of risk factors, including problems related to the developing fetus ranging from genetic factors (Down syndrome) to exposure to toxins *in utero* (fetal alcoholism) to maternal infections (congenital rubella), for example. As noted subsequently, advances in genetics have led to an explosion in the recognition of such syndromes, often with a very precise understanding of their cause ( [Szymanski and King 1999](#)).

In recent years, several developments have substantially changed the approach to treatment and prevention of MR in the United States. Beginning in the 1960s, there has been increased emphasis on the care of persons in their homes and communities. This movement has been further stimulated by the mandate of the U.S. federal government for schools to provide appropriate education for all children with disabilities within mainstream settings when possible. The trend toward deinstitutionalization reflects various concerns about the effects of prolonged institutionalization and has led to creation of many community services. The issue of mainstreaming within education settings is the object of some debate. Many mentally retarded students are largely or entirely mainstreamed in the United States, although there are marked state-to-state variations; the benefits of mainstreaming are the focus of some debate ( [Burack et al., 1992](#)). It is clear that students with more severe disabilities are most likely to spend their school time in more restricted settings.

Another major development was the implementation, starting in the 1960s with the War on Poverty, of programs designed to prevent learning and other problems in poor, disadvantaged children. Program such as Head Start have been shown to have an important long-term impact.

One unfortunate aspect of current practice has been the often complete separation of services for those who are mentally ill from those who are mentally retarded. Although administratively useful, this approach has made provision of high-quality psychiatric care even more difficult to obtain for many persons with MR.

## PREVALENCE AND EPIDEMIOLOGY

The use of both subnormal intellectual functioning and deficits in adaptive behavior in the definition of MR has important implications for epidemiology. If only the IQ criterion is used, the expectation, based on the normal curve, would be that about 2.3% of the population should exhibit the condition. This number is significantly decreased, particularly in adulthood, if the adaptive criterion is included.

For example, in the Isle of Wight study, [Rutter and colleagues \(1976\)](#) noted that, in 9- to 11-year-old children, about 2.5 % would be classified as mentally retarded if IQ were the sole criterion but if the prevalence were based only on those receiving services, this rate would be cut almost in half (1.3%) ( [Fig. 48.2](#)). The drop in cases based on inclusion of IQ and adaptive skills is more common among those with mild MR ( [McLaren and Bryson, 1987](#); [Roelevel et al., 1997](#)); these children (and adults) may, however, need services and support at times of stress ( [Edgerton et al., 1984](#); [Granat and Granat, 1978](#)). As children, such persons are more likely to have academic and behavioral problems ( [Rutter, et al., 1976](#)).

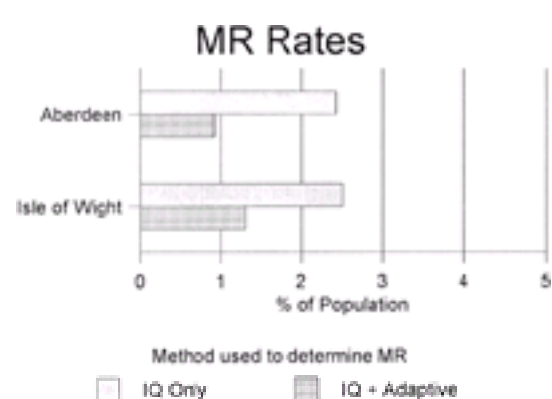


Figure 48.2. Isle of Wight Study.

## CLINICAL DESCRIPTION

### Clinical Features

Associated clinical features vary depending on several factors, most importantly the level of associated retardation. Persons with the severe and profound MR come



to diagnosis at a younger age, are more likely to exhibit related medical conditions, may exhibit dysmorphic features, and have a range of behavioral and psychiatric disturbances (Mundy et al., 1984). In contrast, persons with mild MR often come to diagnosis much later (typically when academic demands become more prominent in school), are less likely to have medical conditions that could account for the MR, and usually are of normal appearance without dysmorphic features. In this latter group, although rates of psychopathology are increased relative to nonhandicapped populations, the range and nature of problems seen are fundamentally similar to those in normative samples (Szymanski and King, 1999). Persons with moderate levels of MR are intermediate between these two extremes. It is well recognized that the nature of associated psychiatric and behavioral disorders undergoes a marked shift between the mild and more severe levels of MR (Sovner, 1986).

### Associated Psychiatric and Behavioral Problems

A growing body of work focuses on psychiatric and behavioral difficulties relative to specific genetic causes (Hodapp and Dykens, 2000). Features have been identified that are unique, or highly frequent, to specific syndromes, and in some cases to very specific genetic or biological processes, such as hyperphagia in Prader–Willi syndrome (Holm et al., 1993), attentional and social problems in fragile X syndrome (Hagerman et al., 1986), inappropriate laughter in Angelman's syndrome (Williams et al., 1996), the unusual cry in 5p- syndrome (Gersch et al., 1995), and the self-hug in Smith–Magenis syndrome (Finucane et al., 1994). In some instances, features are relatively syndrome specific, such as the unusual hand-washing stereotypies of Rett's syndrome (VanAcker, 1997). More frequently, features are shared in various conditions, such as attentional problems are frequent in fragile X, Williams', and 5p- syndromes (Baumgardner et al., 1995; Einfeld et al., 1997; Dykens and Clarke, 1997). In some instances, aspects of syndrome expression have been related to very specific aspects of genetic risk, such as the severity of MR in fragile X syndrome (Dykens et al., 1994; Tassone et al., 1999) and the severity of maladaptive behaviors in Prader–Willi syndrome (Dykens et al., 1999).

Somewhat paradoxically, for many years the diagnosis of MR tended to cause clinicians and researchers to overlook the presence of associated psychiatric and behavioral problems; that is, such difficulties were assumed to be a function of the MR when they were noted at all. In this regard, it was as if a diagnosis of MR "immunized" the individual against other diagnoses, a proposition that is on its face absurd because, presumably, the stresses of having significant intellectual and adaptive deficits should, if anything, make a person more likely to have problems. Historically, persons with MR were not routinely viewed as having comorbid psychopathology or mental illness. This "diagnostic overshadowing" (Reiss et al., 1982) remains a problem in clinical practice (White et al., 1995). Although more clinicians and researchers are being specifically trained to work with this population, the separation of MR and mental health services in most states is a further obstacle to appropriate identification and treatment of mental disorders.

Although rates vary, as many as 25% of persons with MR may have significant psychiatric problems; these rates are much higher if persons with salient behavior disorders are included (Jacobson, 1999). Rates of psychopathology vary considerably, depending on methods used to ascertain subjects. Problems are invariably seen in children who present clinically (Phillips and Williams, 1975), whereas rates are lower, from 10% to 15%, in more population-based studies, including two large-scale medical record surveys of all clients served in New York and California (Borthwick–Duffy and Eyman, 1990; Jacobson, 1982). Rates that fall between these two extremes, from 30% to 40%, are found in other studies based on informant checklists of behavior problems of children or adults in nonreferred samples (Einfeld and Tonge, 1996; Reiss, 1990; Rutter et al., 1976).

Persons with MR experience the same range of psychiatric problems as seen in the general population (King et al., 1994), but prevalence rates for specific disorders vary widely. As previously noted, some of this variability may be associated with different methods for determining "caseness," with common approaches including record reviews, behavioral checklists, and, to a lesser extent, direct interviews. An additional concern is that although some researchers assess DSM-based or ICD-based diagnoses, others identify maladaptive features commonly seen in the general population (e.g., inattention or sadness), whereas still others focus on a narrow range of behaviors seen primarily in persons with MR (e.g., stereotypies or self-injury).

For example, rates of schizophrenia or psychosis in persons with MR range from 1% to 9% among nonreferred samples and 2.8% to 24% in referred samples. Although variable, these rates are much higher than the 0.5% to 1% of the general population with schizophrenia (American Psychiatric Association, 1994). Rates for depression vary from 1.1% to 11% across nonreferred and clinic samples of persons with MR, and rates of attention deficit hyperactivity disorder range from 7% to 15% in children with MR, a finding that contrasts with the 3% to 5% estimate among children in general (American Psychiatric Association, 1994). Patterns of psychopathology also differ across persons with or without MR. For example, relative to the general population, people with MR are more likely to show psychosis, autism, and behavior disorders and are less apt to be diagnosed with substance abuse and affective disorders. (See Moss et al., 1997a for a review of these issues and Chapter 46 for a discussion of autism and other pervasive developmental disorders.)

### ASSESSMENT OF PSYCHIATRIC DISORDERS

As researchers increasingly began to appreciate the scope of problems in persons with MR, they also developed various ways of assessing these problems. Some work has been devoted to the development of specialized rating scales and surveys; most of these measures are geared specifically for persons with MR and have well-developed psychometric properties (Aman, 1991). Among the more widely used are the Aberrant Behavior Checklist (Aman and Singh, 1994), Reiss Screen (Reiss, 1988), and Developmental Behaviour Checklist (Einfeld and Tonge, 1992). A tradeoff in using these scales is that although they are sensitive to the unique concerns of those with MR, they are not necessarily compatible with DSM or ICD psychiatric diagnoses. Further, because each scale has a different set of items and factor structures, these differences may ultimately contribute to inconsistent findings across studies (Dykens, 2000).

At the same time, other researchers have taken issue with the applicability of traditional DSM or ICD diagnoses for persons with MR (Sovner, 1986). Many of these concerns relate to the psychiatric interview itself, including acquiescence bias, and the limited abilities of many persons with MR to answer questions about the onset, duration, frequency, and severity of symptoms (Moss, 1999). In response to these challenges, several groups have adapted traditional DSM or ICD criteria for persons with developmental delay (King et al., 1994; Szymanski et al., 1998), whereas others have designed interview schedules specifically for those with MR, including the Psychiatric Assessment Schedule for Adults with Developmental Disability (Moss et al., 1997b). Direct interviews with both respondents and informants result in fewer cases of missed diagnoses (Moss et al., 1996). Still others shy away from formal diagnoses and advocate a more functional analysis of challenging behavior (Sturme, 1999).

### CAUSES OF INCREASED PSYCHOPATHOLOGY

Although the field has done well with assessment and diagnostic issues, less progress has been made in advancing theories on why persons with MR are at heightened risk of psychopathology in the first place. Many reasons have been discussed over the years and most fall within the "biopsychosocial" spectrum. Yet a comprehensive model of "dual diagnosis" is lacking, in part because researchers cannot simply apply existing risk factors for psychopathology in the general population to the unique characteristics of those with MR. In addition, the causal direction of most risk factors is unclear. Poor peer or social relations, for example, may be a precursor of psychopathology or a consequence of disruptive behavior.

Even so, some advances have been made, and heightened psychopathology in persons with MR has now been linked to specific biopsychosocial problems. Biologically, these include increased rates of seizure disorders (Bird, 1997; Caplan et al., 1998), abnormal neurologic functioning that in most cases is undetected (Peterson, 1995; Robertson and Murphy, 1999), high rates of sensory or motor impairments among persons with MR (Hodapp, 1998), biochemical or neurologic anomalies associated with unusual behaviors such as severe self-injury (King, 1993), and genetic causes that carry higher than usual risks of certain maladaptive or psychiatric vulnerabilities (Dykens, 1999a).

Psychological risk factors include the following: aberrant personality styles, including an outer-directed orientation and being too wary or disinhibited with others (Bybee and Zigler, 1998; Dykens, 1999b; Zigler and Bennett–Gates, 1999); atypical motivational styles or abnormal levels of sensitivity to basic human drives such as the need for attention or acceptance (Reiss and Havercamp, 1998); increased risk of failure experiences, which may lead to learned helplessness, low expectancies for success, and depression (Weisz, 1990; Zigler and Bennett–Gates, 1999); more global and less differentiated self-concepts that may lead to more sweeping negative evaluations of the entire self instead of not liking just one aspect of one's self (Evans, 1998); and reinforcement of negative behaviors, leading to more entrenched maladaptive behavior or interactions (Reiss and Havercamp, 1997).

Finally, specific social risk factors include the following: poor communication or assertiveness skills, which may lead to increased frustration and acting-out behavior (Nezu and Nezu, 1994); social strain or stressful social interactions, more strongly correlated with psychopathology than low levels of social support (Lunksy and Havercamp, 1999; Nezu et al., 1995); social stigma, with a subsequent negative impact on daily living, adjustment, and esteem (Edgerton and Gaston, 1991); peer rejection and ostracism and, among children, atypical patterns of friendship with nonretarded children (Siperstein et al., 1997); compromised "social intelligence," or inappropriate responses to social cues, that may exacerbate stigma and isolation from others (Greenspan and Granfield, 1992); heightened risks of exploitation and abuse, which may worsen behavioral or emotional problems (Ammerman et al., 1994); and family stress, including low levels of emotional, service, or financial support

to families (Minnes, 1998).

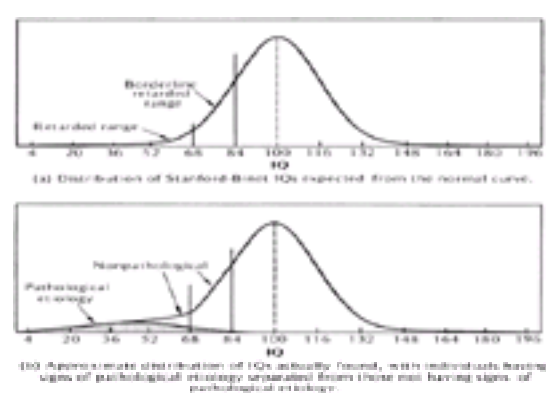
## GENETIC RISK AND PSYCHOPATHOLOGY

As previously noted, a comprehensive model has yet to be developed that identifies the relative importance of these many risk factors. To date, research aimed at doing so has generally relied on heterogeneous groups with MR. Yet each of the factors listed earlier can just as easily apply to those with a genetic diagnosis, and syndrome-specific studies may shed new light on genetic or other mechanisms associated with certain psychopathologic conditions (Dykens, 1995; Holland and Koot, 1998).

## ETIOLOGY AND PATHOGENESIS

Historically, researchers have used two broad categories to classify persons with MR (Zigler, 1967, 1969). One group has organic causes of their MR and consists of people with known prenatal, perinatal, and postnatal insults. Estimates suggest that approximately one-half of people with MR have known “organic” causes (Hodapp, 1994). The second group has no clearly identifiable organic cause of their MR and is postulated to account for most persons with mild MR. In years past, the terms “sociocultural” or “cultural-familial” retardation reflected the view that nonorganic MR stemmed from environmental deprivation. Although impoverished, chaotic environments may indeed be implicated in a few cases, this theory has generally fallen out of favor as an explanation for the population as a whole. Even so, a complicating factor is that disproportionately more persons with sociocultural MR are poor, from minority backgrounds, and of low-IQ parents (Hodapp, 1994).

With increased diagnostic precision and with the discovery of new genetic disorders, many workers speculate that more persons with nonspecific delay will receive specific genetic diagnoses in the years ahead. The complex interplay of organic and genetic (including polygenic) factors with sociocultural and environmental factors has been increasingly recognized (Rutter et al., 1996). Progress has been slower in identifying clear neurologic causes in persons with unspecified MR, because most neuroimaging research is conducted with persons with known genetic or other causes (Robertson and Murphy, 1999). However, some persons with nonorganic causes may simply represent the lower end of the normal, gaussian distribution of intelligence (Simonoff et al., 1996; Zigler, 1967). Assuming that nonorganic MR is the extreme end of the normal IQ distribution, then some persons will always belong to this group, even as progress is made in uncovering genetic or neurologic causes for other persons at the same IQ levels (Fig. 48.3).



**Figure 48.3.** A: Distribution of Stanford–Binet IQ expected from the normal curve. B: Approximate distribution of IQs actually found, with persons having signs of pathologic origin separated from those not having signs of pathologic origin. (From Achenbach T: *Developmental Psychopathology*, 2nd ed. New York, Wiley, 1974, with permission.)

In the organic group as well, many unresolved issues remain. Early researchers grouped people together who had different types of organic causes and often compared these heterogeneous groups with those with familial or nonspecific MR. Even today, mixed or heterogeneous groups, consisting of those with known and unknown causes for their delay, predominate in behavioral MR research (Dykens and Hodapp, 2000). Yet with the remarkable progress in molecular genetics, and improved diagnostic accuracy, researchers are now much better positioned to examine people with specific genetic causes. Indeed, there are now more than 750 known genetic causes of MR (Opitz, 2000), and as many as one-third of all persons with MR have already been diagnosed with a known genetic disorder (Matalainen et al., 1995). Further, although genetic and other organic causes are typically seen in persons with severe and profound delay, high-functioning persons with Down's, fragile X, Prader–Willi, Williams', and other syndromes may comprise 10% to 50% of persons with mild MR (Rutter et al., 1996).

With these advances, research on so-called behavioral phenotypes is gaining momentum, including both between-syndrome and within-syndrome designs (Dykens, 1995; Hodapp and Dykens, 1994, 2000). Between-group studies help to identify possible unique syndromic behaviors that may accelerate our understanding of gene or brain function. Specific examples of these seemingly unique behaviors include hand wringing in Rett's syndrome (Perry, 1991), hyperphagia in Prader–Willi syndrome (Holm et al., 1993), bouts of inappropriate laughter in Angelman's syndrome (Williams et al., 1995), the infantile catlike cry in 5p- syndrome (Gersch et al., 1995), and the self-hug in Smith–Magenis syndrome (Finucane et al., 1994). Further, and as shown in Table 48.2, some syndromes feature unique psychiatric vulnerabilities, including increased rates of obsessive–compulsive symptoms in Prader–Willi syndrome (Dykens et al., 1996), as well as anxiety, fears, and phobias in Williams' syndrome (Dykens, 2000). People without these syndromes can also have these conditions, but they occur much more often in these syndromes. As such, studies on these syndromes hold much promise for differentiating genetic from other pathways to these psychiatric endpoints.

Syndrome	Cognitive features	Cognitive strengths	Behavioral features
Fragile X	Language processing, auditory/visual memory	Visual/auditory attention	Social anxiety, stress, gaze aversion, inattention, hyperactivity, autism/ASD
Prader–Willi	Language processing	Visual/auditory processing	Hyperphagia, reduced daytime competence, symptoms of anxiety, autism
Williams'	Visual-spatial, visuo-motor coordination	Visual recognition, auditory/visual expressive language	Social difficulties, anxiety, fear, inattention, hyperactivity, phobias
Down'	Language processing, expressive language	Visual-spatial processing, expressive language	Autism spectrum, autism, inattention, depression and dementia in adults

**Table 48.2. Examples of Cognitive and Behavioral Profiles in Selected Mental Retardation Syndromes**

Cognitively as well, some syndromes show distinctive profiles of relative strength or weakness that are not typically seen in studies of persons with mixed or nonspecific causes of MR. As summarized in Table 48.2, persons with Williams', Prader–Willi, Down's, and other syndromes often show distinctive patterns of cognitive strength or weakness. Many people with Williams' syndrome, for example, show relative strengths in specific aspects of expressive language, along with pronounced deficits in visual–spatial functioning. Despite visual–spatial deficits, however, many persons with Williams' syndrome have a remarkable sparing of facial recognition and memory. Many, although not all, persons with Prader–Willi syndrome show remarkable skills solving jigsaw puzzles, with performances that exceed those of their chronologic agemates (Dykens, 2000).

However, most syndromic behavior appears to be “partially specific” or shared across one or more conditions (Hodapp, 1997). For example, persons with both fragile X and Prader–Willi syndromes appear to have relative weaknesses in certain short-term memory and sequential processing tasks (Dykens and Cassidy, 1999; Dykens et al., 1994), and inattention and hyperactivity are seen in Williams', fragile X, and 5p- syndromes (Baumgardner et al., 1995; Dykens and Clarke, 1997; Einfeld et al., 1997). These disorders, however, show qualitative differences in symptoms. Inattention in Williams' syndrome, for example, may be associated with heightened anxiety and social disinhibition, whereas in fragile X syndrome, these difficulties may be related to hyperarousal and anomalies in the size of the posterior



cerebellar vermis and caudate nucleus ([Mostofsky et al., 1998](#)).

Although between-group studies help to identify distinctive syndromic behaviors, within-syndrome studies help to explain individual variation in these behaviors. Researchers now need to identify the genetic, environmental, developmental, and psychosocial factors that help to explain individual behavioral differences in people with the same genetic disorder. For example, the level of cognitive delay in fragile X syndrome is associated with both age and molecular genetic status ([Dykens et al., 1994](#); [Tassone et al., 1999](#)). Similarly, in Prader–Willi syndrome, the frequency and severity of maladaptive behaviors such as skin picking appear to vary across genetic subtypes of this disorder ([Dykens et al., 1999](#)).

Whereas research on heterogeneous groups is still necessary, both between-syndrome and within-syndrome studies offer many advantages ([Dykens, 1995](#); [Hodapp and Dykens, 2000](#)). In the long term, work on behavioral phenotypes facilitates the search for gene–brain–behavior relationships, as well as contributing toward a more precise science of MR. In the short term, phenotypic data refine intervention and treatment ([Dykens and Hodapp, 1997](#); [Dykens, 2000](#)). Although many syndrome-specific recommendations for interventions have now been made, the efficacy of these approaches needs to be evaluated, including how they fare relative to more generic interventions ([Dykens et al., 2000](#)).

## DIFFERENTIAL DIAGNOSIS

The diagnosis of MR is based on the appropriate assessment of cognitive abilities and adaptive skills; clinical assessment also includes a careful developmental and family history, physical examination, and laboratory studies as appropriate. The clinician should be alert to any medical or environmental conditions that may be associated with developmental disability. For example, a strong family history or certain dysmorphic features in the child should raise the possibility of an inherited condition; a history of significant birth trauma, of exposure to environmental toxins, or exposure to marked psychosocial adversity are some of the factors that should be considered.

As noted, the age of diagnosis often varies depending on the severity of the disability, so persons with more severe MR present for clinical assessment earlier than those with mild, or borderline, MR. Careful psychological assessment of the child ([Chapter 43](#)) is obviously critical. Various other developmental difficulties, notably language and other specific developmental disorders and autism and related conditions, may be associated with some degree of mental handicap or may be confused with it. Diagnosis can be complicated because persons with MR can exhibit other developmental problems that can complicate the task of both diagnosis and assessment. For example, children with a marked expressive language disorder often do poorly on a test of intelligence test that is highly verbal. In autism and related disorders, social abilities tend to be the area of greatest weakness ([Chapter 46](#)).

## TREATMENT

The treatment of MR has undergone a marked shift in the more developed countries. More persons with MR now reside with the families and in their communities. More children now receive services within regular educational settings, and more services are available to support them and their families. At the same time, it is clear that placement in the community is not sufficient in and of itself, and the provision of adequate and appropriate support is critical. This is particularly important for the growing numbers of older persons with MR, many of whom still reside with their aging parents.

In general, treatment planning begins with a consideration of the underlying cause, if one is known, of the intellectual disability. In many instances, knowing the cause can guide both medical and psychosocial interventions; indeed, many syndrome-specific parent and professional organizations have published “best practice” guidelines across the lifespan (e.g., Prader–Willi, Williams’, and Down’s syndromes.). For rarer disorders, however, such data are lacking.

### Medical Treatment

Medically, laboratory studies should be based on the results of a careful history (including family history) and evaluation. Physical examination should include assessment of growth and developmental status as well as observation for facial features or other physical findings that could suggest a specific medical condition. It is sometimes the case that the evolution of the condition provides important clues to its cause, such as in Rett’s syndrome. Depending on the clinical circumstance, hearing or visual testing may be indicated as are, at times, specific, metabolic studies, chromosome analysis (including fragile X testing), neuroimaging or neurologic assessment, skeletal radiography, screening for organic acidurias, and so forth ([Curry et al., 1997](#); [Szymanski and King, 1999](#)).

Persons with MR may be at increased risk of certain medical conditions ([Ryan and Sunada, 1997](#)). In their review of the Aberdeen cohort of cases, Goulden and colleagues noted that at least 15% of patients developed epilepsy by adulthood; the risk was increased when associated disabilities were present or when there was a history of postnatal injury ([Goulden et al., 1991](#)). To some extent, the associated medical conditions vary depending on the cause of the MR, for example, the cardiac problems in Down’s and Williams’ syndromes and the risk of Alzheimer’s dementia in adults with Down’s syndrome.

### Cognitive and Adaptive Treatment

Regardless of the origin of MR, however, accurate assessment of each person’s intellectual and adaptive strengths and weaknesses is essential, because specific recommendations for intervention depend on the overall level and profiles of cognitive functioning. Although there are many different models of intelligence ([Sternberg and Salter, 1982](#)), investigators generally agree about many of the basic features of intelligence, such as the ability to use conceptual thinking in solving problems and in acquiring knowledge ([Sparrow and Davis, 2000](#)). Various IQ tests are now available, and they may differ in certain dimensions, such as the degree to which they emphasize language-based problem solving or short-term memory. Tests may differ in other ways; for example, some tests include timed tasks, whereas others provide the opportunity for demonstration of tasks by the examiner. As part of the standard administration of test items, the examiner is also able to collect considerable amounts of qualitative information that may be particularly important for treatment.

Some special considerations are involved in assessing persons with MR. Tests should, to the extent possible, be appropriate for the person’s chronologic age as well as her or his levels of receptive and expressive language. Cultural and other factors may need to be considered in some cases. The behavioral problems sometimes associated with MR may pose special problems; these problems can be further compounded if they tend to arise around times of change or frustration.

Assessment of adaptive functioning has the goal of providing a representative picture of the person’s typical abilities in home and school environments. In this regard, the goal is somewhat different from that of the intellectual assessment, in which the aim is to obtain optimal performance in a structured and standardized situation. Large discrepancies between intellectual level and adaptive skills suggest that the treatment should include a major focus on acquisition and generalization of adaptive skills.

Various measures of adaptive functioning have been proposed ([Chapter 43](#)). The most widely used instrument is the Vineland Adaptive Behavior Scales ([Sparrow et al., 1984](#)), which assess capacities for self-sufficiency in various domains of functioning including Communication (receptive, expressive and written language), Daily Living Skills (personal, domestic and community skills), Socialization (interpersonal relationships, play and leisure time and coping skills), and Motor Skills (gross and fine). The Vineland scales are available in three editions: a survey form to be used primarily as a diagnostic and classification tool for normal- to low-functioning children or adults, an expanded form for use in the development of individual education or rehabilitative planning, and a classroom edition to be used by teachers ([Sparrow et al., 1984](#)). Both the survey and expanded editions use a semistructured interview format administered to a parent or primary caregiver. For persons with MR, the expanded form is often particularly useful because it readily translates into goals for intervention planning.

### Psychiatric Treatment

As previously described, persons with MR are at increased risk of psychiatric problems, and these are often a major source of distress to the individual and family and may severely limit opportunities for self-sufficiency and independence ([Szymanski and King, 1999](#)). Yet these mental health problems, and medical ones for that matter, are frequently overlooked. Indeed, [Ryan and Sunada \(1997\)](#) report that up to 75% of persons with MR who are referred for psychiatric assessment have undiagnosed or undertreated medical conditions, and nearly 50% receive nonpsychotropic medications that could have behavioral side effects.

Even though associated psychiatric problems can severely limit personal and social sufficiency, there is often a tendency to neglect or overlook the mental health needs of this population ([Einfeld and Tonge, 1996](#)). Although some of the many rating scales, checklists, and other instruments for assessment of psychopathology in the general population are applicable, other instruments have been developed that are specific for persons with MR. Psychiatric assessment may entail some

modification in usual procedures, particularly for persons with more severe MR, in whom the psychiatric assessment must be comprehensive and multifocal. The presence of associated difficulties (seizures, motor impairments, sensory problems) may further complicate accurate psychiatric diagnoses.

### Other Psychosocial Treatment

General quality-of-life issues are receiving renewed attention in the MR field, with particular emphasis on improving how people with MR live, work, and play in inclusive, community-based settings. Most persons with MR benefit from employment or from structured programs that emphasize vocational, adaptive, or socialization skills, long after formal schooling. Indeed, the transition from school to work is a vulnerable point for many persons and their families. Unlike the school years, when special education and related services (e.g., occupational, physical, and speech and language therapies) are typically provided under one roof at school, services for adults risk being more fragmented. These young adults may particularly benefit from *case coordination*, to avoid becoming isolated or lost between various cracks in services.

### OUTCOME AND FOLLOW-UP DATA

As expected, the course and outcome of MR vary considerably, depending on various factors. These include the level of severity of the MR, associated biological or other vulnerabilities, and aspects of the individual's psychological functioning, family support, and other factors ( [Szymanski and King, 1999](#)). Levels of ability to cope with the demands of daily life (i.e., adaptive skills) are critical in determining adult outcome. It is also clear that the expectations of caregivers and the provision of intervention services and environmental supports are also important. For persons with known medical causes of MR, certain risks may be present, such as the risk of early development of Alzheimer's dementia in persons with Down's syndrome ( [Aylward et al., 1997](#)). Conversely, even when the specific biomedical cause is known, there may be a wide range in ultimate outcome. To simplify the discussion of outcome, we focus the discussion on levels of MR but again emphasize that the outcome in a given individual patient varies considerably, depending on a host of factors.

In mild MR, many children with the condition go on, as adolescents and adults, to make major gains in adaptive functioning and thus may "lose" the diagnosis as they become older. Such persons may be self-supporting, may marry, and may raise families. At the same time, such persons are not without difficulties, because several studies ( [Granat and Granat, 1978](#); [Rutter et al., 1976](#)) show that persons with intellectual deficits who have not required special services in school have higher rates of educational and behavioral problems. In their follow-up study of the Aberdeen cohort of children first seen at 9 to 11 years of age and then followed up at the age of 22 years, [Richardson and Koller \(1992\)](#) report that more than 75% no longer require services as adults, although only about 25% of the entire group are judged to be functioning adequately in all areas. Mild MR is likely to be diagnosed only at the time of school entry, that is, when academic demands increase. As discussed previously, persons from backgrounds of social disadvantage or adversity and from certain minority groups are more likely to be represented in these cases.

As adults, persons with moderate levels of MR (IQ 40 to 55) typically have more serious impairment. It is common for such persons to need services as adults. Moderate MR is often identified in the preschool years. At this, and lower, levels of cognitive functioning, specific medical causes are more likely to be identified, and minority group membership and psychosocial adversity in the family are less frequent. The prognosis for adult self-sufficiency is more guarded, although many persons can live semiindependently or with partial support ( [Ross et al., 1985](#)).

For persons with severe or profound retardation, case identification may occur in infancy or early childhood. Generally, high levels of supervision and support are required during the person's life. Goals for these patients include facilitating self-care and other skills as far as possible. Associated medical problems and behavioral difficulties are frequent. Communication skills may be impaired and are a further source of handicap.

Legislation, legal decisions, and some important social policy changes have markedly changed the provision of remedial programs. The provision of early diagnosis and intervention and the availability of community-based resources and educational interventions within public schools have dramatically improved the care of persons with MR as well as overall outcome.

### PREVENTION

Estimates of recurrence risk vary depending on the situation, such as from instances in which a clear genetic origin can be identified, as in fragile X syndrome, to those in which the difficulties appear to be of nongenetic origin, as in congenital rubella. When no specific cause is identified, estimates of recurrence risk vary considerable, such as between 3.5% and 14% for the siblings of a boy with MR ( [Crow and Tolmie, 1998](#)). For some disorders, such as autism, there has been a growing appreciation of genetic factors, and it now appears that, for parents who have one child with autism, the risk of having a second child with autism is between 2% and 10% ( [Fombonne et al., 1997](#)). Siblings of children with MR who are not themselves affected may be at increased risk of other difficulties, given higher levels of family and personal stress ( [Hannah and Midlarsky, 1999](#)). Support for them, and for their parents, is an important element of treatment planning ( [Crow and Tolmie, 1998](#); [Szymanski and King, 1999](#)).

### RESEARCH DIRECTIONS

Several issues will likely dominate the research agenda for the coming decade. They include the interplay between genetic and environmental (including psychosocial) risk factors in the origin of MR as well as the study of basic neural mechanisms ( [Simonoff et al., 1996](#)). Understanding of basic processes that underlie phenotypic expression, including various forms of psychopathology, offers the opportunity to advance knowledge more generally about mechanisms of disorder. Although many advances in the care and treatment of mentally retarded persons have been made, studies of treatment methods remain an important priority.

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# 49 DISORDERS OF COMMUNICATION

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[Syndromes Involving Communication Disorders](#)

[Mental Retardation](#)

[Hearing Impairment](#)

[Psychiatric Disorders](#)

[Acquired Disorders of Communicative Function](#)

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Language, a unique and characteristic capacity of the human mind, is also one of its most vulnerable faculties. Virtually any disruption in cognitive function, particularly during early development, can affect language acquisition. For this reason, disorders of language development typically accompany a variety of other conditions. This chapter first outlines briefly the most common syndromes in which disorders of communication may play a part. Specific disorders of speech and language development, which are not accompanied by deficits in other areas of mental function, are then discussed.

## SYNDROMES INVOLVING COMMUNICATION DISORDERS

### Mental Retardation

Limitation in communicative skill is often one of the first signs of mental retardation. Children with retardation are often first recognized because of their failure to begin talking at the normal time. The sequence of language acquisition in children with retardation follows, in general, the sequence of normal acquisition, although some differences can be identified. Many children with mental retardation (MR) show communicative skills that are commensurate with their developmental level, but more than half have language skills that are less than what would be expected for mental age (Miller et al., 1999). Productive deficits are common, with some children with MR showing deficits relative to mental age in this area only. Others have both receptive and expressive limitations relative to mental age. Phonologic errors are prevalent in children with MR. These children make similar errors to those seen in normal development, but errors are more frequent ( [Shriberg and Widder, 1990](#)). Pragmatic skills are usually similar to those seen in children at similar developmental levels ( [Lahey, 1988](#)). The two most prevalent syndromes of MR, Down's syndrome and fragile X syndrome, are both very frequently associated with various problems in language development ( [Dykens et al., 1994](#); Miller et al., 1999).

### Hearing Impairment

Children with impaired hearing are vulnerable to language disorders because of their lack of access to the linguistic information in the auditory signal. Still, children with hearing impairments vary greatly in their oral language ability, and as [Boothroyd \(1982\)](#) pointed out, an unaided audiogram is not a child's fate. With amplification via hearing aids, children can be moved from greater to lesser levels of severity of hearing loss. Cochlear implants and tactile aids are also used to provide auditory information to children who would otherwise be considered deaf ( [Roeser, 1988](#)). Language acquisition in children with hearing impairment (HI) follows the same general sequence as it does in children with normal hearing, although it is greatly delayed, and the delays affect all modalities: articulation, receptive and expressive communication, and oral and written language ( [Paul and Quigley, 1994](#)). Children who receive cochlear implants, however, appear to show significant benefits in terms of spoken language ( [Tomblin et al., 1999](#)). [Lahey \(1988\)](#) concluded, however, that use of language for communication is not a major problem area for children with HI but that most of their difficulties lie in acquiring the conventional verbal forms of communication. Reading and writing present particular problems, primarily because of the language basis necessary for acquiring these skills. Average reading comprehension level for adolescents with HI is third to fourth grade ( [King and Quigley, 1985](#); [Paul and Quigley, 1994](#); [Trybus and Karchmer, 1977](#)).

Many children can be taught to bypass the auditory channel through the use of manual sign language. Using this method, children can develop fluency and eloquence in sign that would never be available to them through the modality of speech. There is great controversy within the community of the hearing impaired as to the role of sign language versus oral language instruction for children with severe hearing losses. In general, children taught sign develop higher-level language skills than those taught speech, although their communication may be limited to those in the deaf community who use sign as their mode of communication. The decision whether to use an oral or signed mode of communication with a child who has severe HI must be made carefully in consultation with the child's family. Deaf parents of deaf children may feel it is more important for their child to be fluent in sign and comfortable in deaf culture than to achieve the maximum possible reading level. For these families, instruction in sign may make more sense. Deaf children of hearing parents may have families that feel communication with the general population is among their highest goals. For children in these families, oral instruction may work better. Each family must be presented with all available options in order to make the most informed decision for their child ( [Brackett, 1997](#)).

### Psychiatric Disorders

There is a very high coincidence of sociobehavioral and communicative disorders ( [Giddan and Milling, 1999](#)). [Prizant and Meyer \(1993\)](#) reported that over half of children diagnosed with communication disorders have socioemotional and behavioral problems. Conversely, [Prizant and associates \(1990\)](#) found that two-thirds of children referred to a psychiatric inpatient hospital failed speech and language screening. [Giddan and Milling \(1999\)](#) showed that 65% of children in psychiatric outpatient clinics were similarly impaired. They also reported that a third of children referred for conduct disorders had concomitant speech and language difficulties, and more than two-thirds of children with some form of attention deficit disorder have language disabilities as well. Additionally, as [Prizant and colleagues \(1990\)](#) showed, some children with anxiety and affective disorders fail language screenings. It may be impossible to know the source of this connection. Some writers ( [Redmond and Rice, 1998](#)) hold that a communication problem leads to frustration, creating behavioral or emotional disorders. It may, alternatively, be the case that a behavioral/socioemotional problem leads to decreased motivation to communicate or an inability to "tune in" to learn the rules of communication or use language for self- and other-regulation ( [Paul et al., 1999](#)); or, perhaps some other underlying factor affects both aspects of development. Whatever the answer to these questions, children with language problems are vulnerable to socioemotional difficulties, and children with psychiatric diagnoses show a higher than normal prevalence of language disorders.

The psychiatric disorder most consistently associated with communication deficits is autism, or pervasive developmental disorder (PDD). Communication problems—including severe delays in language, inability to communicate nonverbally, inability to sustain conversation, stereotyped and repetitive use of idiosyncratic language, and abnormal ability to use language for social communication—are included in the diagnostic criteria for autism/PDD ( [APA, 2000](#)). Virtually all children with autism have some form of communication disorder that presents as part of their syndrome. What differentiates autism from a more circumscribed language disorder is the global nature of the child's communication problem. Not only is language affected, but also the ability and motivation to send messages by any means, either verbal or nonverbal, is severely impaired. Although sign and other alternative forms of communication are often tried with autistic patients, it is their underlying deficit in communicative skill and motivation that impedes their use of language; therefore, providing an alternate channel does not usually result in dramatic improvement ( [Lord and Paul, 1997](#); [Tager-Flusberg, 1995](#)).

### Acquired Disorders of Communicative Function

Language disorders can be acquired during the developmental period through four types of neurologic insults: focal lesions; damage associated with seizure disorders; damage resulting from tumors, infection, or radiation; and traumatic brain injury. Focal lesions affect language in children primarily if they are unilateral on the left side. These kinds of lesions are relatively rare in children, and children younger than 10 years tend to recover from the aphasias that follow these insults

nearly completely, although some subtle effects on language and learning abilities can persist ( [Bishop, 1992](#); [Blosser and DePompei, 1994](#)).

[Landau and Kleffner \(1957\)](#) identified a syndrome of expressive and receptive language deficits accompanied by seizures that usually has its onset between 4 and 7 years of age after a period of otherwise normal development. The Landau-Kleffner syndrome has no known cause and, fortunately, is quite rare. Unlike aphasia associated with focal lesions, Landau-Kleffner syndrome usually ends in a permanent aphasia. Cognitive functioning is usually impaired as well. Anticonvulsant medication can help to control the seizures, but does not improve communication. Some behavioral and educational intervention is almost always warranted.

Brain radiation treatment used to treat acute lymphocytic leukemia (ALL) in children has greatly reduced the mortality rate of this disease, but this “cure” sometimes has the unfortunate consequence of causing language and learning problems, loss of developmental skills previously acquired, or seizures ( [Riddle et al., 1991](#) ). Brain tumors can also affect communicative functioning. In addition, brain damage that can affect language development may also result from infectious diseases, such as meningitis. The long-term sequelae of these kinds of insults can be subtle and variable, depending on timing, location, size, and area of the brain affected by the damage. Children with these injuries may retain a great deal of language function but have deficits that surface only during complex tasks, like those required for school. They may have expressive skills that are relatively intact, but poor comprehension. This can result in adults' being fooled into thinking comprehension is on par with production and being frustrated when the child “refuses” to follow instructions. The range of severity of these effects can extend from almost nonexistent to severe enough to result in a diagnosis of mental retardation.

Traumatic brain injuries (TBIs) can be focal in nature, usually involving open head injuries such as bullet wounds. Closed head injuries, such as those resulting from blows or collisions, tend to involve diffuse damage, affecting larger areas of the brain. Like children with acquired aphasia associated with focal lesions, children with TBIs of both types show a great deal of spontaneous recovery. [Russell \(1993\)](#) reported that outcome is generally predicted by the degree of coma, the length of time a child spends in an impaired state of consciousness following the injury, and the length of the posttraumatic amnesia (PTA) period. PTA lasting longer than 24 hours is considered a sign of severe injury. Age at injury does not appear to affect prognosis, and the onset of deficits may be delayed for long periods of time after the injury has taken place. Although some children retain physical disabilities from TBIs, many do not show obvious physiologic damage. However, a substantial minority of these children suffers long-term deficits in cognitive and language function ( [Plante and Beeson, 1999](#) ). [Gerring and Carney \(1992\)](#) reported that, during the acute recovery process, children tend to be mute at first. They may comprehend only simple commands. First language productions often reflect the confused state the child is in and are often dysarthric or dysfluent. Speech may be slow, and prosody may be affected so the speech sounds monotonic and “flat.” Swallowing disorders are also common during this phase of recovery.

### Disorders with Environmental Components

Communication disorders can be associated with prenatal exposure to substances such as drugs and alcohol or with parental behavior disorders such as abuse and neglect. Language disorders are frequently part of the picture seen in fetal alcohol syndrome (FAS) or fetal alcohol effects (FAEs) ( [Shprintzen, 1997](#) ). They include delayed development, poor receptive vocabulary and comprehension, and pragmatic difficulties. The communication disabilities children with FAS and FAE display are related to the level of intellectual impairment. Perseveration and echolalia are often present in children with more severe impairments ( [Sparks, 2001](#) ).

Children with a history of prenatal exposure to cocaine or other street drugs are also described as showing deficits in language development, among other symptoms ( [Sparks, 1993](#) ). However, it is important to remember that many women who abuse street drugs during pregnancy take more than one drug and may mix abuse of both drugs and alcohol. Also, the effects of the mother's drug abuse on her care taking ability are just as important as any biological effects the abuse may have caused before birth. As far as biological effects are concerned, [Sparks \(2001\)](#) pointed out that less than half of children exposed to drugs prenatally experience low birth weight, prematurity, intrauterine growth retardation, or small head circumference. Further, the developmental problems these children exhibit are not much different from those of other children who live in chaotic homes but who did not have prenatal drug exposure. In terms of communicative development, prenatal drug exposure should be considered a *risk* for communication disorder, rather than a cause of it.

Children with communicative and other developmental disorders are, as [Knutson and Sullivan \(1993\)](#) pointed out, more likely than normally developing children to experience maltreatment. [Fox and colleagues \(1988\)](#) suggested that a child with a communication disorder might be less satisfying for a parent to care for and provide less rewarding interactions. These difficulties might predispose a child to abuse.

Maltreatment itself also constitutes a risk for language disorder. [Culp and associates \(1991\)](#) argued that language development is particularly vulnerable in the maltreatment situation because of the disruption in social interaction it entails. [Coster and coworkers \(1989\)](#) showed that maltreated toddlers had more limited expressive language during play with their mothers than peers from nonabusing homes. [Allen and Oliver \(1982\)](#) found that maltreated 4-year-olds had significantly lower language scores than peers of similar socioeconomic level. [Lynch and Roberts \(1982\)](#) showed that children with a history of maltreatment scored significantly lower on verbal IQ relative to nonverbal scores. [Fox and associates \(1988\)](#) found that maltreated children had receptive language deficits, with neglected children suffering greater lags than those shown by children who were abused. In general, severe neglect seems to be a greater risk factor for communicative handicap than does abuse ( [Allen and Oliver, 1982](#); [Culp et al., 1991](#) ). [Coster and Cicchetti \(1993\)](#) point out, however, that it is not entirely clear that the language problems seen in these children are greater than what would be expected on the basis of the generally depressed cognitive levels associated with their maltreatment.

## SPECIFIC SPEECH AND LANGUAGE DISORDERS

Communication disorders can, as we have seen, be associated with a variety of conditions. They can also occur in relative isolation. These more specific disorders of speech and language development can still have broad effects on a child's ability to succeed in social and academic pursuits because communication is so central to human interaction. We examine the two types of specific speech disorders discussed in the DSM-IV-R ( [APA, 2000](#) ), stuttering and phonologic disorders, and the two types of language disorders addressed there, that is, specific language disorders and selective mutism.

### Specific Speech Disorders

#### STUTTERING

Stuttering, according to [Cooper and Cooper \(1995\)](#), is a clinical syndrome involving abnormal and persistent dysfluencies that result in the speaker's perception of a loss of control over speech, which is often accompanied by affective and behavioral reactions. Several types of speech dysfluencies also may be involved, including blocking of sounds, hesitations, and tense pauses (Perkins, 1980). Stuttering usually begins somewhere between 2 and 7 years of age, with a peak between 3 and 4 years. Although normal children often go through periods of dysfluency during the developmental period, these “normal dysfluencies” tend to occur in the larger linguistic units (word, phrases, and sentences). For children who tend to persist in stuttering over time, dysfluencies are more likely to occur in repetitions of syllables (“vi-vi-vi-vi-video”) and sounds (“g-g-g-g-ot”). Other “red flags” for persistent stuttering include sound prolongations (“WWWWWait!”), silent blocks in which the child attempts to speak but no sound comes out, and visible struggle behaviors during speech, such as blinks or grimaces ( [Plante and Beeson, 1999](#) ). If dysfluencies continue to be relatively effortless, there is a good chance of recovery. In addition, [Yairi and associates \(1996\)](#) reported that children who recover from stuttering begin to show reductions in their number of dysfluencies within the first year, whereas those who persist in stuttering are relatively stable in their rate of dysfluency.

When recovery occurs, it usually does so by adolescence, often around the time of puberty. About 1 in 30 children goes through a period of stuttering, but by adolescence, the prevalence drops to 0.8% ( [NIDCD, 1992](#) ). The disorder is much more common in boys than in girls, and the male:female ratio increases with age, suggesting that girls show higher rates of recovery than boys ( [Yairi et al., 1996](#) ).

For individuals with chronic stuttering, the severity of the disturbance varies from situation to situation and is more severe when there is pressure to communicate. Stress or anxiety have been shown to exacerbate stuttering, but are not thought to play a role in the etiology. Reducing stress during speaking can reduce stuttering episodes ( [Van Riper, 1973](#) ), but general treatments for anxiety, including the use of tranquilizing medication, have not been found to be effective treatments ( [Ham, 1990](#) ). In general, speech therapy is used both to shape fluent speech and help the client to stutter with less tension, avoidance, and interruption of the flow of communication ( [Gregory, 1995](#) ). Psychotherapy alone has not been shown to be an effective treatment for stuttering, but counseling is often helpful for overcoming the secondary effects of stuttering on self-concept, thoughts, and feelings ( [Cooper and Cooper, 1995](#) ).

Although stuttering has, at times, been thought to be a learned behavior, most researchers today consider stuttering to have a biological component. People who stutter have been found to show laryngeal behavior different from that in normal speakers, even when their speech is apparently fluent ( [Conture et al., 1985](#) ). Elements of both the central and peripheral nervous system may be involved in stuttering behaviors ( [Plante and Beeson, 1999](#) ). There also appears to be a familial component in stuttering ( [Kidd, 1977](#); [Yairi et al., 1996](#) ). The risk of stuttering among first-degree relatives is more than three times the population risk ( [APA, 2000](#) ).



Stuttering is believed by most researchers today to have a complex multiplicity of causes that include biological vulnerability, environmental demands and expectations, and temperamental characteristics of the speaker ([Adams, 1990](#)).

## PHONOLOGIC DISORDERS

Phonologic or articulation disorders are characterized by impaired production of developmentally expected speech sounds. To diagnose a specific phonologic disorder, it is necessary to ascertain that the problem is not attributable to deficits or abnormalities in intelligence, hearing, or the structure and physiology of the speech mechanism. So a phonologic disorder is one in which, although there is no organic reason for the disability, the child's speech is marked by misarticulations, including distortions of sounds (e.g., /s/ is produced with a lisp), omissions of sounds (e.g., *up* is pronounced "uh," *play* is pronounced "pay"), and incorrect substitutions of one sound for another (e.g., *cat* is pronounced "dat"). Many of these misarticulations represent processes that are typical in the speech of young normal children (e.g., deletions of final consonants, simplifications of consonant clusters), but in phonologic disorders more of them are used, they are used more often, and their use persists beyond the normal developmental period. There may also be idiosyncratic preferences for and/or avoidances of certain sounds or sound simplification processes ([Dunn and Davis, 1983](#); [Ingram, 1976](#); [Weiner, 1981](#)), and/or reversals or misordering of sounds in words ([Trantham and Pedersen, 1976](#)). The age of recognition/onset of the disorder is related to severity. Typically, the disorder becomes apparent around the age of 4 years, when normal children become fully intelligible and eliminate almost all of their normal developmental sound change patterns ([Bernthal and Bankson, 1998](#)). Children as young as 3 years can be diagnosed with this disorder, even if their speech is unintelligible to family members. Milder cases may not be identified until the child is in school.

Phonologic disorders are the most prevalent type of communication problem. [Edwards \(1984\)](#) reported that 80% of speech clinic referrals were for articulation disorder. Six percent of school-age children have phonologic disorders. The prevalence is higher for preschoolers, with estimates ranging from 10% to 15% ([Office of Scientific and Health Reports, 1988](#)).

The characteristics of the misarticulated speech are related to the child's age and severity of the disorder. Younger and/or more severely impaired children may have difficulties involving a wide range of speech sounds, including those like /b/, /p/, and /m/ that are acquired early in the developmental sequence. A variety of sound changes may occur, including dropping syllables ("nana" for *banana*), dropping final sounds (/da/ for *doll*), or leaving sounds out of consonant blends ("bu" for *blue*). These changes, cumulatively, can render the child's speech moderately to severely difficult for others to understand. Older children and/or children who are less severely affected may make only a few kinds of errors, usually substitutions or deletions. Errors in older or more mildly impaired children will occur only on sounds that are acquired later in the developmental sequence (e.g., /s/, /r/, /l/, th). Misarticulations of vowel sounds are rare in this disorder.

Phonologic disorders can, of course, occur with many of the syndromes discussed in the preceding. They can also occur in isolation, but are commonly associated with specific language disorders. [Shriberg and Kwiatkowski \(1994\)](#) found that over 50% of children with phonologic disorders have delays in expressive language, and 10% to 40% have delays in language comprehension. [Shriberg and Kwiatkowski \(1988\)](#) reported that a significant minority of these children, with either speech-only or speech/language delays as preschoolers, required continuing special services throughout their elementary school years. They emphasize the importance of careful assessment and follow-up of both speech-only and speech/language delayed children. Although most of these children do "outgrow" their unintelligible speech, some continue to require services for other aspects of language and academic development.

## Specific Language Disorders

### SPECIFIC LANGUAGE IMPAIRMENT

Some kinds of language disorders have no known concomitants. These disorders have been traditionally defined by exclusion, that is, by the absence of the other factors—mental retardation, sensory disorders, neurologic damage, emotional problems, or environmental deprivation. The terms *childhood aphasia* or *congenital aphasia* were used in the past to describe these disorders. The use of these terms grew out of the conviction of the early neurologists that difficulties in children's learning language were analogous to the loss of language seen in adults who suffered acquired aphasias. This notion evolved from observations that "aphasic" children often appeared bright in other ways and were clearly not retarded. They seemed also to have normal affective bonds to the people around them and were not emotionally disturbed. Their inability to acquire language normally was attributed to some sort of neurologic dysfunction, thought to be comparable to localized brain lesions that resulted in aphasia in adults.

A multitude of studies that attempted to identify these neurologic lesions in children with language deficits ([Paul, 1996a](#)) were unsuccessful in finding brain structure or function differences that were either common to all the children studied or specific to children with language disorders, although there are some indications of reduced hemispheric asymmetry in children with language disorders and their immediate family members ([Jackson and Plante, 1997](#); [Plante et al., 1991](#)). New diagnostic techniques, such as functional magnetic resonance imaging (fMRI) and positron-emission tomography (PET) scanning may be more successful in identifying more specific neurologic markers. However, because language disorders in children do not seem to have the same root in localized brain lesions as do adult aphasias and because no specific, reliable brain differences have been identified to date, the terms *aphasia* or *dysphasia* seem too weighted toward neurologic explanations of dubious value. More descriptive terms are now used to label these disorders. DSM-IV uses *specific language disorder*. *Specific language impairment*, often abbreviated SLI, is also commonly used to label this diagnostic category ([Paul, 2000](#); [Watkins and Rice, 1994](#)).

### Language Development in Specific Language Impairment

Children with SLI are late to begin talking. They may not say their first word until well into their second year. When they do begin to talk, they add new words slowly. They may continue to use single word or telegraphic utterances into their third year. Although expressive vocabularies tend to reach normal size by age 3 in children with mild to moderate handicaps, problems in various areas of syntax, such as verb endings and pronoun usage, persist throughout the preschool period ([Paul, 1993b](#)). Grammatical usage may be characterized by omissions of words (e.g., prepositions, indefinite pronouns, or verbal auxiliaries: "He sitting," "Where we going?") or morphemes (e.g., "Dad wear white shoe," "She sleep"), use of a limited selection of grammatical structures (e.g., only the present tense of verbs), incorrect word ordering (e.g., "Car Mummy have"), or the use of inappropriate combinations of words or morphemes ("They was reading," "two foots"). Phonologic problems very frequently coexist with slow expressive language development during the preschool years. Many of these problems resolve by the early school years in children with mild to moderate disorders. Comprehension of language may appear age-appropriate or mildly delayed during the preschool period. For preschoolers with language problems that appear to be specific to the expressive modality, risk for long-term deficits is about 20% at kindergarten age ([Paul, 1996b](#)). Other positive prognostic indicators include a milder degree of the impairment, few perinatal problems, higher nonverbal IQ, and willingness on the child's part to participate in groups ([Nelson, 1998](#)). Certain measures have been shown to be good predictors of school-age outcome in preschoolers with mild to moderate SLI. These include measures of narrative skills, nonverbal cognition ([Bishop and Edmundson, 1987](#)), and parents' socioeconomic status ([Paul and Fountain, 1999](#)).

There has been a long-running debate in the literature about whether children with language disorders show "deviant" acquisition patterns—patterns unlike those seen in younger normal children—or whether they exhibit a slowed-down version of normal development. Early studies ([Menyuk, 1964](#)) reported that there were qualitative differences in the linguistic systems of normal and disordered children. But recent research, summarized by [Leonard \(1997\)](#), suggests that children with language disorders do resemble younger normal speakers in the general nature of their linguistic systems and in most aspects of the order of acquisition. Although this pattern holds true when we look at one specific feature of development at a time, Leonard pointed out that children may be 1 year below age level in one set of features, 1.5 years below in another, 6 months below in a third, and so on. The result will be that the overall profile of language skills in a child with SLI may not resemble that of a normal child at any point in development. This does not mean that language development is deviant, but rather that it is asynchronous in some ways.

For children with chronic mild to moderate language disorders, problems in the school years tend to narrow in their focus and to be concentrated in subtle difficulties of language organization and efficiency, rather than frank errors ([Nelson, 1998](#)). Word retrieval or "word finding" difficulties, are common. Instead of using correct words (e.g., "chair"), a child with SLI may substitute an incorrect word of related meaning (e.g., "table"); or use functional descriptors (e.g., "thing to sit on"), vague or general terms (e.g., "thing"), or his or her own made-up jargon. Storytelling and discourse problems often persist and affect both oral and written modes of expression ([Paul et al., 1996](#)). These children may lack the ability to elaborate and/or self-correct when needed for clarity in conversation ([Graham et al., 1983](#); [Trantham and Pedersen, 1976](#)). There may be tangential or inappropriate responses to questions, a limited range of communicative functions (e.g., requests, imperatives, questions) expressed, difficulty maintaining and/or changing topics, and difficulty initiating interactions ([Kuder, 1997](#)). Many of these pragmatic problems are caused by a lack of flexibility in language ([Lucas, 1980](#); [Zirkelbach and Blakesley, 1985](#)). These children may sound abrupt, rude, or impolite simply because they do not have access to the full and diverse range of linguistic forms used in normal conversation to encode pragmatic nuance and make language sound appropriate to the social context. In addition, some mild social deficits may also be seen ([Bishop, 1997](#)). Academic problems tend to involve primarily reading and writing. Despite their

persistent problems, however, most of these children do finish high school, some go on to college, and most live independent lives ( [Hall and Tomblin, 1978](#)).

Prognosis is more guarded for children with severe SLI. [Paul and Cohen \(1984\)](#) studied long-term outcomes in children diagnosed as SLI as preschoolers who were not speaking in full sentences by the time they were 6. By the time they were adolescents, these subjects with severe impairments were likely to score in the retarded range on IQ tests, even if they had scored in the normal range at the preschool level. Given intensive intervention, all made steady progress in language skills throughout their school years. Still 90% of these subjects were significantly below the normal range in both areas by adolescence, and all required intensive special education, with most in special classrooms, schools, or residential facilities.

### *Prevalence*

Accurate estimates of prevalence of specific language disorders are difficult to come by, because of methodologic differences across studies (e.g., in the subclassifications and definitions; in the cutoffs and inclusionary criteria; and in the age, sex, and other characteristics of the children sampled). Estimates during the preschool period generally range from 10% to 15% ([Rescorla and Lee, 2001](#)), although some children in this group overlap with those identified with phonologic disorders. Language disorders are the most common type of developmental disability seen during the preschool period.

At school age, the prevalence of primary language disorders is thought to be about 4%, although again there is overlap with related disorders, because some preschoolers with language disorders “grow into” school-age learning disabilities and dyslexia ( [Plante and Beeson, 1999](#)), as discussed in the preceding. Specific language disorders and learning disabilities combined are the most prevalent disorders of school-age children ( [Pore and Reed, 1999](#)). Although disorders of language expression are thought to be more common than those involving comprehension, [Bishop \(1997\)](#) has argued that almost all children with language impairments do have receptive difficulties, although they may be subtle in some cases. It also appears that language disorders are more prevalent in males than in females ([Satz and Zaide, 1983](#)).

### *Etiology*

Although specific biological markers of SLI have not been identified, neurobiological factors are clearly implicated. There is evidence of genetic factors in SLI including higher concordance in monozygotic than dizygotic twins ( [Bishop et al., 1995](#)) and higher than normal risk in family members for language and learning problems if a child has SLI ([Tallal et al., 1989](#); [Tomblin, 1989](#)). The role of environmental factors in these disorders has long been at issue. Although aberrant parental linguistic input has often been suspected as a cause, numerous researchers (e.g., [Conti-Ramsden, 1990](#); [Leonard, 1989](#); [Paul and Elwood, 1991](#); [Whitehurst et al., 1988](#)) have concluded that linguistic input to children with language disorders is adequately matched to the children's language level. Some environmental factors appear to be associated with risk for language delay, however. These include lower socioeconomic status, larger family size, recurrent otitis media, neglectful home environment, and later birth order ([Nelson, 1998](#)). It appears that the operative mechanism behind these factors is deprivation of enriching linguistic input ( [Bishop, 1987](#)), occurring at a critical stage in language development.

How specific are specific language disorders? Children with SLI are at greatly increased risk for attention and activity problems ( [Damico et al., 1999](#)). Other “soft” neurologic signs are also frequently present in children with SLI ( [Benton, 1964](#); [Eisenson, 1972](#)). The involvement of a variety of nonverbal cognitive skills in SLI also has been indicated (Johnston, 1994). These findings have led to the hypothesis that children with SLI may have not just a language problem, but also a general representational deficit, affecting a variety of kinds of symbolic functioning. [Tallal \(1988\)](#) cautioned, however, that in each of these studies there were children with SLI who could perform the nonverbal cognitive tasks adequately, and that sometimes the differences between groups were not qualitative, but a matter of speed of response. [Leonard \(1997\)](#) pointed out that, although some children with SLI fall below age mates on such tasks, they still do better than younger children with comparable language skills.

[Leonard \(1991, 1994\)](#) provided an alternative explanation for SLI. He contended that children who score low on language tests, relative to their scores on other areas of cognition, may no more have a “disorder” than children who cannot learn to play the violin. He argued that some children are just “limited” in their ability to learn language, falling (as someone must) at the low end of the normal distribution of language ability. If this were the case, in his view, it would not be surprising that such children would also be limited in other abilities that related to symbolic function. He referred to [Gardner's \(1983\)](#) notion of “multiple intelligences,” which proposes that there are a variety of somewhat independent spheres of intellectual functioning and that some people have greater abilities in some than others. The tendency to call a language limitation a “handicap” stems, in Leonard's view, from the importance of linguistic skills for academic and vocational success in our society, not from any significant neurologic or neuropsychological pathology in people limited in this way. The fact that language problems tend to aggregate in families ( [Tallal et al., 1989](#); [Tomblin, 1989](#)) could be taken to support the view that some people just have fewer optimal “language genes,” and therefore less talent in language areas, than others.

Many would contest this view, however. They would hold that the frequent co-occurrence of SLI with attention and activity problems and other “soft” neurologic signs raises questions about its relation to normal development. [Aram \(1991\)](#) argued that children with nearly age-appropriate comprehension but expression limited to single words could not be seen as functioning simply at the low end of the normal range, nor could a 3-year-old who can read but can not produce spontaneous speech ([Aram and Healy, 1988](#)). Clearly, some children with SLI do have pathologic factors involved. The question for researchers and theoreticians is whether these children are the exception or the rule.

An alternative explanation has been offered by [Bishop \(1997\)](#) and [Tallal and associates \(1996\)](#). These writers discuss the possibility that SLI is related to deficits in the processing of auditory information, particularly the rapid, short-lived information contained in speech. Tallal and associates have developed an intervention program, known as Fast ForWord, based on this theory. The program trains children to discriminate auditory stimuli on the basis of increasingly brief acoustic cues. Although they have claimed dramatic success in their program, controversy still surrounds these claims at the present writing ( [Gillam, 1999](#)).

### *Subtyping*

We have already mentioned another controversy in the field of SLI; that concerning subtypes of the disorder. Several schemes have been proposed ( [Aram and Nation, 1975](#); [Fey, 1986](#); [Naremore, 1980](#); [Rapin and Allen, 1983](#)). The most common subtyping systems concern the division between disorders restricted to expressive language and those that involve both reception and expression. This is the categorization method used in DSM-IV-R ( [APA, 2000](#)). Still this scheme is not universally accepted. [Curtiss and Tallal \(1985\)](#), for example, reported that patterns of performance on language testing over time in a longitudinal study were the same for children with SLI regardless of the subgroup in which they had been placed. [Bishop \(1997\)](#), too, has argued that virtually all children with SLI have some difficulties in language comprehension. Currently, most clinicians describe children as SLI without a specific subtype given.

### **Selective Mutism**

Selective mutism is a failure to speak in one or more particular definable communicative environments. Historically, stuttering, phonologic disorders, and language disorders have been considered the domain of speech pathology and psycholinguistics, whereas selective mutism has been considered the domain of psychiatry and psychology. This division remains, to some extent, today, as exemplified by the placement of selective mutism outside the communication disorders section in DSM-IV-R. Still selective mutism does involve a form of communication disorder and is important in the differential diagnosis of other forms of communication impairment. Because this is the case, a brief discussion of this disorder is presented here.

As with other communication disorders, accurate estimates of the prevalence of selective mutism are difficult to find, although the condition is known to be rare. DSM-IV-R ([APA, 2000](#)) estimates the prevalence at less than 1% of children seen in mental health settings, a very small proportion of the population in general. Unlike all the other communication disorders discussed here, selective mutism is thought to be somewhat more common in females than males. Selective mutism is associated with the other speech/language disorders at greater than chance rates.

The major variations in symptomatology are unclear because the disorder is so rare. It appears, however, that the most common manifestation is a refusal to speak in school and to adults outside the home despite speaking normally within the home to siblings and at least one parent ( [APA, 2000](#); [Browne et al., 1963](#); [Kratowill, 1981](#)).

The child's silence seems to represent a refusal to talk rather than an inability to talk, although, as noted, language disorders are associated with this syndrome more often than would be expected by chance alone. Although these other speech/language impairments are sometimes present ( [Cantwell and Baker, 1985](#); [Kolvin and Fundudis, 1981](#)), they are not sufficiently severe to account for a lack of speech. Intellectual functioning generally is found to be average or above ( [Browne et al.,](#)



1963; [Hayden, 1980](#); [Hesselman, 1983](#)), although one study found slightly lower performance intelligence levels ([Kolvin and Fundudis, 1981](#)).

Despite their “refusal to talk,” selectively mute children usually appear interested in communication and, in the situations where they will not talk, they may attempt to communicate by using gestures, pantomimes, drawings, nods or shakes of the head, whispers, or monosyllabic utterances ([APA, 2000](#); [Cantwell and Baker, 1985](#); [Harris, 1996](#)). In those settings where they are not mute, these children may be quite talkative ([Hesselman, 1983](#)).

The disorder usually has its onset between 3 and 8 years, although the most frequent point of onset is school entry; however, some cases have been reported with onset after 12 years of age ([Kaplan and Escoll, 1973](#)). The majority of patients with selective mutism recover from their symptoms within months or years ([APA, 2000](#); [Harris, 1996](#); [Kolvin and Fundudis, 1981](#)). Improvements usually occur before the age of 10 years ([Kolvin and Fundudis, 1981](#)); the prognosis is poorer for children older than 12 years ([Hayden, 1980](#); [Kaplan and Escoll, 1973](#)).

Biological factors are usually not thought to play a significant role in the etiology of selective mutism. Still it has been found that children with selective mutism are at a somewhat increased risk for other developmental disorders (including speech/language disorders, enuresis, and encopresis) and electroencephalogram (EEG) immaturity. This suggests that a biologically based maturational disorder may play some role in the etiology of selective mutism ([Kolvin and Fundudis, 1981](#)).

Family and interpersonal dynamics have long been suspected to play a major role in the etiology of selective mutism. Some studies have found that selectively mute children have increased rates of psychiatrically ill parents and/or abnormal family dynamics ([Browne et al., 1963](#); [Hayden, 1980](#); [Hesselman, 1983](#); [Kaplan and Escoll, 1973](#); [Kolvin and Fundudis, 1981](#)). Anecdotal reports mention isolated family situations; at least one very shy or uncommunicative parent; broken families; hospitalization ([Browne et al., 1963](#)); significant separations from family ([Kolvin and Fundudis, 1981](#)); physical traumas ([Hayden, 1980](#)) such as child abuse, rape, or mouth injuries; and an overly strong attachment to the mother. Still, careful controlled research on this disorder is scarce, and larger studies with appropriate contrast groups are needed before definitive conclusions on the role of family dynamics can be drawn.

## DIFFERENTIAL DIAGNOSIS

When seeing children suspected of specific speech and language disorders, the main diagnostic task is to rule out other syndromes with which speech and language problems are frequently associated. These syndromes include the ones discussed in the first section of this chapter, that is, deafness or significant hearing loss, mental retardation, pervasive developmental disorder, autism, psychiatric disorder, and organically based communication disorders (e.g., cleft palate, apraxia, cerebral palsy, or childhood-acquired aphasia).

The absence of significant hearing impairment or deafness must be established by audiometric testing by a certified audiologist. Because subtle deficits in hearing can affect language acquisition, it is important to get complete and accurate results in order to assure that hearing deficits do not play a role in the disorder. Although chronic middle ear pathology is sometimes associated with speech and language problems, recent research ([Roberts et al., 1997](#)) suggests that chronic otitis media alone does not significantly increase the risk of language disorder in otherwise normal children. If parents report chronic otitis in a child who presents with a communication disorder, attributing the problem entirely to the middle ear problem is not justified, and treatment for the otitis will not necessarily alleviate the language disorder.

Mental retardation must be diagnosed by means of an individually administered, standardized test of intelligence, as well as by a standardized measure of adaptive behavior. For children suspected of language disorders, it is necessary to use nonverbal intelligence tests to assess intellectual ability, so as not to penalize the child for the language deficit and to get an estimate of intelligence unbiased by language performance. [Table 49.1](#) provides a sample of nonverbal intelligence assessments that can be used in the determination of cognitive level in children with communication disorders. A child must score significantly higher on the nonverbal intelligence assessment than on the language measure to be diagnosed with a specific speech or language disorder.

Instrument	Age Range	Key Feature	Comments
WISC-III Nonverbal Reasoning & Block Design Subtests (Wechsler, 1974)	6:0-17:0	Comprehension, perceptual	Best for screening language comprehension; also assesses spatial, auditory, and motor development
Cognitive Assessment Battery for Young Children (CAYC) (Fisher et al., 1989)	18-36 mos	Figure-objects	Used for children with motor impairments
Cognitive Behavior Scale (Carroll, 1975)	18-36 mos	Concept development	Used for establishing cognitive skill level
Developmental Scales Screening Inventory (DSI) (Peters and Longley, 1988)	18:0-3:0	Memory, auditory, sequencing, and problem-solving	Used to screen children with hearing impairment
Bayley Scales of Infant Development (BSID) (Bayley, 1973)	1:0-18:0	Concept formation	Used for determining whether child is functioning at language level
Bayley Infant Scales of Infant Development (BSID) (Bayley, 1973)	1:0-18:0	Classification, memory	Used for determining whether child is functioning above 2;0 and 3;0
Bayley Infant Scales of Infant Development (BSID) (Bayley, 1973)	1:0-18:0	Spatial behavior	Used for determining level of representational thought
Bayley Infant Scales of Infant Development (BSID) (Bayley, 1973)	1:0-18:0	Nonverbal skills	Used for determining level of representational thought
Bayley Infant Scales of Infant Development (BSID) (Bayley, 1973)	1:0-18:0	Developmental delay or mental retardation	Used to determine if child is functioning at language level
Bayley Infant Scales of Infant Development (BSID) (Bayley, 1973)	1:0-18:0	Developmental delay or mental retardation	Used to determine if child is functioning at language level
Bayley Infant Scales of Infant Development (BSID) (Bayley, 1973)	1:0-18:0	Developmental delay or mental retardation	Used to determine if child is functioning at language level

**Table 49.1. Instruments for Assessing Nonverbal Cognition**

Organic disorders that can affect the speech mechanism, such as dyspraxia, dysarthria, and motor deficits associated with cerebral palsy, can be ruled out by physical and functional assessment of the speech mechanism (oral peripheral examination). A speech-language pathologist typically examines the morphology, symmetry, and alignment of the facial features. The functional integrity of the larynx, lips, tongue, and velopharyngeal structures, as well as the respiratory support for speech are also assessed. The quality of oral volitional movements also may be examined if an apraxia is suspected. If physical limitations to speech production are identified in this examination, an alternative system of communication—such as a picture board or electronic communicative device—may be indicated ([Glennen and DeCoste, 1997](#)).

Psychiatric/behavioral assessment can rule out pervasive developmental disorder, autism, and other psychiatric disorders. Features associated with autism/PDD include lack of (nonverbal) social interactions, absence of imaginative activity, stereotypic behaviors, self-injurious behaviors, odd responses to sensory input, and mood abnormalities. Language features that characterize these disorders include abnormal use of imitation (echolalia); idiosyncratic use of words or phrases for “private” meaning; pronoun reversals (saying “you” when the speaker means “I”); persistent, obsessive preoccupation with one topic of conversation (such as bus schedules reiterated, elaborated on, and questioned about incessantly); and repetitive, inappropriate use of questions. Forty to fifty percent of children with autism are mute. What distinguishes these children from those with motor or intellectual limitations that lead to lack of speech is a dearth of nonverbal communication. That is, mute children with autism not only do not speak, but also do not use other means, such as pointing, gesturing, or shifting gaze, to convey their thoughts ([Lord and Paul, 1997](#)).

The differential diagnosis among stuttering, phonologic disorder, expressive language disorder, mixed receptive-expressive language disorder, and selective mutism requires detailed speech/language testing. Some examples of the most commonly used language tests are given in [Table 49.2](#). To diagnose stuttering, a clinician should ascertain, by means of standardized testing, that phonologic, receptive, and expressive language skills are age-appropriate. Many children go through periods of developmental dysfluency, and children with language disorders may persist in developmental dysfluency longer than normal because their developmental period is longer than normal. If dysfluency coexists with other speech and language disorders, it may be appropriate to delay making a diagnosis of stuttering until some of the other problems resolve, then reevaluate to determine whether the dysfluency has persisted.

**Table 49.2. Some Frequently Used Communication Assessment Instruments**

Phonologic disorders are diagnosed by means of standardized testing of phonologic production, usually using procedures that ask children to name pictures or objects and transcribing the child's rendition of the target word for comparison with adult production standards. Because phonologic disorders typically coexist with other language disorders, it is acceptable to confer a concurrent diagnosis of phonologic disorder even if other language disorders are present. However, it is necessary to rule out hearing impairment, mental retardation, and speech mechanism limitations before diagnosing a specific speech disorder.

To diagnose a specific expressive language disorder, it is necessary to demonstrate—by means of individually administered, standardized tests—that both nonverbal intelligence and receptive language skills are significantly better than expression. Again, because phonologic disorders so frequently coexist with expressive language deficits, both conditions can be diagnosed concurrently.

To make a diagnosis of mixed receptive-expressive (R/E) language disorder, it must be shown—again using individually administered, standardized tests—that nonverbal intelligence is significantly higher than *both* expression and comprehension of language. As with expressive disorder, phonologic deficits can coexist with R/E disorders and would be diagnosed according to the criteria given earlier.

## TREATMENT OF SPEECH AND LANGUAGE DISORDERS

It is important to make a careful differential diagnosis of any communication disorder in order to decide whether the problem is specific to speech and language or is part of a larger syndrome. In the case of communication disorders associated with syndromes such as hearing impairment, mental retardation, and autism, treatment must address all aspects of the child's problem, not just those of speech and language.

Still, once a communication problem has been identified and the broad range of interventions necessary to address all the child's needs has been instigated, treatment for the communicative aspect of the disorder is quite similar, regardless of whether the problem is specific to speech and language or part of a bigger picture of developmental disorder.

The treatment of choice for all the disorders discussed here, except for selective mutism, is individual or small group therapy administered by a certified speech/language pathologist (SLP). Because associated educational and/or psychiatric problems are common with these disorders, educational tutoring, social skills training, and/or psychiatric intervention also may be indicated, even if the disorder is specific to communication.

Methods of intervention are essentially behavioral. Some clinicians use strict operant procedures, whereas others favor more child-centered approaches such as indirect language stimulation, or “whole language” intervention that involves a rich communicative environment with opportunities for incidental learning. Many SLPs take a middle ground between these extremes, using structured play opportunities and focused stimulation to provide examples of desired forms and elicit language targets. Although all these methods have been shown to be effective in small studies (Fey et al., 1995), much more research is needed on the efficacy of particular approaches to intervention and variables that can be used to best match the method to the child.

Because selective mutism is seen more as an anxiety disorder than a developmental disorder, treatment methods differ somewhat for this syndrome. Although some authors have suggested psychodynamic therapy for selective mutism, the most convincing literature pertains to behavioral modification approaches (Giddan et al., 1997). Contingency management (positive reinforcement of verbalizations and nonreinforcement for nonverbal responses), stimulus fading (gradually extending the number of people with whom and environments wherein verbalization is rewarded), shaping (rewarding gradual approximations to speech, such as mouthing and whispering), and response cost procedures (losing money or tokens for not speaking) have all been reported to be successful in eliciting and maintaining speech in selectively mute children (Labbe and Williamson, 1984).

Caution should be exerted when treatments make claims of dramatic improvement, particularly when the improvement is claimed for a broad range of disorders. In recent years, several “miracle cures” have been advanced for communication disorders. Facilitated communication (Biklen, 1990), Fast ForWord (Tallal et al., 1996), and auditory integration training (Tharpe, 1999) are just a few examples. Clinicians who work with families of children with communication disorders need to maintain a healthy skepticism regarding these programs that make extravagant claims. If something sounds too good to be true, it probably is. Any intervention, whether familiar or innovative, must be shown to meet the particular needs of the client receiving it. Communicative interventions need to be evaluated with the same kind of rigor that would be used to evaluate the efficacy of a medical or surgical treatment. Although double blind, placebo controlled trials are difficult to design for behavioral interventions, clinicians must strive to achieve some degree of objective evaluation before deciding that a treatment is appropriate for an individual.

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# 50 DEVELOPMENTAL LEARNING DISORDERS

Larry B. Silver

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Developmental learning disorders are listed in the *Diagnostic and Statistical Manual*, fourth edition (DSM-IV and DSM-IV-TR) under Axis II. These disorders are characterized by inadequate development of specific academic, language, speech, and motor skills and are not caused by demonstrable physical or neurologic disorders, a pervasive developmental disorder, mental retardation, or deficient educational opportunities. There are four subgroups:

1. Reading disorder
2. Mathematics disorder
3. Disorder of written expression
4. Learning disorder not otherwise specified (NOS)

## DEFINITION

The DSM-IV conforms to the international classification system; thus, the term learning disorders with skill-related subtypes. The public school systems in the United States use the term learning disability. Thus, clinicians use the term *learning disorder* when completing medical records but use the term *learning disability* when interacting with school systems. The DSM-IV definition of a learning disorder relates to the individual's ability in specific academic areas. The disorder exists if the ability in question is "substantially below the expected given the person's chronological age, measured intelligence, and age-appropriate education." No clarification is given on the extent of the disability needed to be "substantial."

The federal guidelines used in public school systems uses a definition based on a specific level of discrepancy between potential and performance. Thus, a child or adolescent might meet the medical criteria for the diagnosis of a learning disorder but not have the extent of discrepancy to meet the educational criteria for being identified ("coded") with a learning disability. This is a critical issue, because a student must be identified with a disability to receive services. Another difference is that the federal definition states that the student must be of at least average intelligence or higher. The DSM-IV definition for a learning disorder does not state such a criterion.

The public school definition of a learning disability is based on Public Law 94-142, Education for Handicapped Children. This definition persists in the revisions of this original law now called the Individuals with Disabilities Education Act (IDEA), or Public Law 101-476. This federal definition describes a learning disability as

a disorder in one or more of the basic psychological processes involved in understanding or in using language, spoken or written, which may manifest itself in an imperfect ability to listen, think, speak, read, write, spell, or to do mathematical calculations.

This public school definition notes specific exclusionary criteria.

This term does not include children who have learning problems which are primarily the result of visual, hearing, or motor handicaps, or mental retardation, or emotional disturbance, or of environmental, cultural, or economic disadvantage.

Public school systems have specific guidelines for assessing the level of discrepancy. This level varies with each school system in this country. The average is a 1.5 to 2.0 standard deviations difference between potential (usually based on an IQ test) and performance (usually based on a battery of achievement test). In addition to this discrepancy requirement, testing must document the presence of a central nervous system processing deficit. (This concept of a processing deficit is discussed afterward in this chapter.)

## HISTORICAL BACKGROUND

Prior to the 1940s in the United States, children who had academic difficulties were considered mentally retarded, emotionally disturbed, or socially and culturally disadvantaged. A fourth possibility for these difficulties was introduced between 1940 and 1949. Children might have problems with academic performance for neurologic reasons. That is, the cause of the difficulty was related to brain function. Two different research approaches explored these presumed to be neurologically



based learning disorders.

One group of researchers noted that these children had the same types of learning difficulties often found in individuals who have known brain damage. They concluded that these children also had brain damage; however, it was so subtle or minimal that it could not be confirmed clinically; therefore, the term minimal brain damage was introduced (Strauss and Lehtinen, 1947; Werner and Strauss, 1941). Other researchers explored the possibility that the learning difficulties reflected a physiologic dysfunction. The brain functioned differently because it was “wired” differently rather than damaged. The term minimal brain dysfunction was used to reflect this concept (Clements, 1966). Recent research, to be discussed later in this chapter, confirms the concept of brain dysfunction rather than brain damage.

Initially, the types of academic difficulties were described based on the primary skill disorder (Farnham-Diggory, 1978). Reading disorders were called *dyslexia*, writing disorders were called *dysgraphia*, and arithmetic disorders were called *dyscalculia*. Later, efforts shifted from focusing on the presenting problem to the underlying brain processes responsible for the reading, writing, and math difficulties. The term *learning disability* was introduced. This is now the preferred term within the fields of education.

## PREVALENCE

The true prevalence of learning disorders is not known. In the DSM-IV, reference is made to the difficulty of obtaining data on learning disorders. In this text, the suggested prevalence rate for reading disorder is 4%, mathematics disorder 1%, and disorder of written expression 4%.

The best data come from the public school systems and can range from 1% to 30% of the school population. The number of children and adolescents identified as having learning disabilities depends largely on the criteria used to determine eligibility for services. The more stringent the discrepancy criteria, the lower the prevalence rate—the more lenient the discrepancy criteria, the higher the prevalence rate.

The only non-school system prevalence data is from the Centers for Disease Control (1987). The focus of this study was on learning disability. In the absence of standardized, consistent criteria, it was estimated that between 5% and 10% of the school-aged population was a reasonable estimate of the percentage of persons affected by learning disabilities.

The public school system uses data based on the number of children who were identified as having a learning disability and who were being serviced by the school system. No data existed before Public Law 94-142 was passed in 1975. The first year the law was implemented (1977 to 1978), fewer than 800,000 children (1.8% of the enrolled school population) received services under the category of learning disabilities. The number of students meeting the criteria for having a learning disability and receiving services has increased dramatically each year. The more recent data show that 5.27%, or over 2.4 million, receive services. These figures do not take into account the many children with learning disabilities who are not identified and diagnosed or who are evaluated but do not meet specific school systems' discrepancy criteria.

This increase in numbers reflects the greater awareness within public school systems of this disability, improved procedures for identifying and assessing learning difficulties, and increased social acceptance of this problem. Another factor is the number of court decisions forcing school systems to address the needs of individuals with learning disabilities.

Studies of children with learning disabilities have consistently noted an increased prevalence in males. The ratio reported has ranged from 3:1 to 5:1 and higher (Ackerman et al., 1983; Finucci and Childs, 1981; Rutter et al., 1976). Recent studies suggest that this increased prevalence in males over females may be explained in part by referral bias. Males are more likely to be referred for evaluation because they are likely to act out their frustrations (Berry et al., 1985). Females may become anxious or depressed more than disruptive when frustrated and may not be recognized as quickly as having difficulty. Johnson and Blalock (1987) found no specific cognitive differences or patterns of problems between the sexes.

Recent studies suggest that there may be no sexual differences in the prevalence of learning disabilities (Shaywitz et al., 1990, 1992). Any differences in statistics appear to be secondary to referral bias and the possible bias of the assessment test used that appear to identify more male than female students with learning disabilities. In this study, all children in a specific grade were evaluated for a possible learning disability. These data were compared to the school system data, based on the number of children identified and serviced. The school-identified population showed a 3:1 ratio of boys to girls. The true data based on assessing all children showed a ratio of 1:1.

## ASSOCIATED DISORDERS

Individuals with a learning disorder (disability) might have emotional or behavioral problems. For many, these emotional and behavioral disorders are secondary to the frustrations and failures experienced because the disabilities either were not identified or inadequately treated (Silver, 1989). For others, these difficulties may be another reflection of a dysfunctional nervous system. Each theme is discussed. In addition, many students develop social skill and social competency problems. These, too, are discussed in this chapter.

We now better understand a continuum of neurologically based disorders that show a high comorbidity pattern. Because these new data on brain function and dysfunction are so critical to understanding children and adolescents with learning disorders, they are discussed first.

It is critical that the clinician be aware of these associated disorders. Each must be considered and, if present, diagnosed and treated. Unless all of the problems the child or adolescent faces are addressed, there will be less than optimum clinical success.

### Continuum of Neurologically Based Disorders

Comorbidity studies suggest that there is a continuum of neurologically based disorders often found together. The common theme appears to be that something impacted on the developing brain, possibly early in pregnancy, causing some of the “wiring” to be laid down differently. For some of these individuals, we find a familial pattern and suspect a genetic theme (Cardon et al., 1994; Grigovenko et al., 1997). For others, we do not yet understand the reasons for these changes. There is an increasing concern with the impact of environmental developmental and neurologic toxins on the developing brain (discussed in the following). If something impacts on the developing brain, then more than one area is usually involved.

#### Cortex

This faulty wiring might involve the cortex, areas below the cortex, or both. If the cortex is involved, this impact might be in the areas critical to language, motor, cognitive, or executive functioning.

#### Language Difficulties

If the area of the brain that is wired differently relates to language functioning, the person will have a problem with language called a *language disability*. (These disabilities are covered in Chapter 49, Communication Disorders.) The first clue often is a delay in language development. The child is not speaking by 2 years, or by 2½ or 3 years is using only a few words. Some may be no better by 4 years old. If a speech and language therapist works with this child, it might be possible to speed up the development of language. There is a sigh of relief. Then, by 4 or 5 years old another problem becomes clear. This child may have difficulty processing and understanding what is being said (called a receptive language disability) or might have difficulty organizing thoughts, finding the right words, and speaking in a fluid and clear way (called an expressive language disability). More help is needed. Another problem might become apparent as this child enters the early elementary grades. The first task in reading is language based. The child must recognize units of sound (called phonemes) and connect this sound to the correct unit of symbol (called graphemes). There are 44 phonemes in the English language. Each letter has a sound; vowels have two sounds (a short and long sound); certain combinations (sh, th, ch, etc.) have their own sounds. There are 36 graphemes in the English language (a through z and 0 through 9). To learn to read, a child must learn to “break this code” by learning what sounds go with what symbols and sounding out words. Many children with a delay in language development and later with receptive and/or expressive language problems have difficulty learning to read in first grade. Spelling is the reverse process. One must start with the language in his or her brain and connect it with the right symbol by writing on the page. Thus, many children with reading problems also have spelling problems.

### Motor Difficulties

If the area of the brain that is wired differently relates to the use of our muscles, we see what is called a *motor disability*. For some, the primary problems relate to the ability to coordinate and use teams of large muscles (gross motor skills) and have difficulty with running, jumping, or climbing. Others might have difficulty coordinating and using teams of small muscles (fine motor skills). They have difficulty learning to button, zip, tie, color within the line, use scissors, use eating utensils, and, later, using a pencil or pen to form letters and write. Still others might have a broader pattern of motor problems called *sensory integration disorder*. Now, in addition to gross and fine motor planning functions, they might have difficulty making sense out of information coming from nerve endings in the skin. They might be very sensitive to touch or misread temperature or pain. They also might have difficulty processing information from the vestibular system, information needed to know where the body is in relation to gravity. They have difficulty with movement in space or position in space. Which of these many possible motor problems are present depends on the areas of the brain involved.

### Cognitive Difficulties

We call it a *learning disability* if the area of the brain that is wired differently relates to the processing of information for learning. In some ways, this division of the cortex is artificial. If an individual has a learning disability, then more than one area of the brain is involved.

### Executive Function Difficulties

The most recent addition to the cortex is the frontal cortex. This sophisticated area of the brain acts like the chief executive officer in a company. This area carries out what is called *executive functions*. It orchestrates behaviors. This is the area that assesses a task or problem, decides how to tackle or solve the task, orchestrates the necessary activities or functions, continually makes mid-course changes or corrections, and eventually reaches a successful conclusion. If this area of the brain is involved, then we see people who have difficulty with organizational planning and carrying out tasks successfully.

### Attention Deficit Hyperactivity Disorder

If the areas of faulty wiring involve a set of interrelated systems involving the frontal cortex, limbic system, basal ganglia, and reticular activating system, the child or adolescent may have difficulties regulating motor activity, attending, and reflecting before speaking or acting. He or she might be hyperactive, inattentive, or impulsive. This cluster of clinical problems is called attention deficit hyperactivity disorder (ADHD) ([Chapter 52](#)).

Clinical studies suggest a comorbidity rate between 15% and 30% between ADHD and learning disabilities. The difference in percent is based on the definition used to establish this diagnosis. If the discrepancy required is based on 1.0 or 1.5 standard deviation, then the rate is higher than if 2.0 standard deviations are required ([Biederman et al., 1992](#); [Halperin et al., 1984](#); [Shaywitz et al., 1992](#); [Silver, 1981](#)).

### Modulating Disorders

With the dramatic new methods for studying the brain and new data on clinical comorbidity, we have a better understanding of related clinical problems. There appears to be another area of the brain that might be called the “modulation” area. This is the area that maintains a balance or equilibrium, avoiding extremes. Studies of this area show that there are many functions that need to be modulated. If any specific area is involved, there will be a problem with modulation of a specific function. Some people have problems modulating *anxiety*. These children have a history since an early age of being high strung or anxious. Over the years, the focus of the anxiety may change, but the central theme is a high anxiety level. They might be afraid to go to sleep alone at night. Later, it might be a fear of being in part of the house alone, or bees, or something else. As these children move into later childhood and adolescence, they might develop an *anxiety disorder*. Some may have so much difficulty regulating anxiety that the level gets too high and triggers off a sympathetic discharge and a panic attack, called a *panic disorder*.

Another regulating problem relates to the ability to modulate *anger*. These children may have been more irritable and angry since early childhood. They have always had tantrums. As they get older, they show a specific form of difficulty regulating anger called *intermittent explosive disorder*. When they get angry, they do not just have a tantrum or pout or slam doors. They have a “melt down.” What is characteristic of this child or adolescent (or adult) is that they have a very short fuse. Sometimes they explode so fast that it is not clear what caused it. Once they pass over their threshold, they lose their temper. They yell, scream, curse, hit, throw, and threaten. They act in an irrational way and cannot be reasoned with. Sometimes they seem paranoid, saying people are trying to hurt them. This episode lasts minutes, sometimes up to 15 or more minutes. Then, it ends almost as abruptly. Once it is over, they may be tired and want to rest or sleep. They usually do not want to discuss what happened and may seem confused about their behavior. Later, they might feel remorse about what they did.

Some may have difficulty modulating *mood*. They seem to have been unhappy or sad much of their life. They are moody or depressed some of the time or much of the time. Some will go on to develop a *depressive disorder*. A few may show difficulty modulating not just the down side (depression) but the up side (excitement, manic behavior). They might evolve into a *bipolar disorder*. Here the mood swings are rapid and include both depression/euphoria cycles and calm/anger-rage cycles.

Another pattern of behavior that seems to relate to these modulating disorders relates to the ability to regulate thoughts and behaviors. They develop *obsessive-compulsive disorder*. They might show difficulty controlling their thoughts and obsess over a thought or thought pattern. Others might have difficulty regulating behaviors. They feel they must do certain things, or they will get too anxious. They know “it is silly” but cannot stop. They might need to touch things a certain way or number of times. They might need to check and recheck things (e.g., see if the front door is locked or the stove is off). They might need to perform certain patterns or rituals.

There is another area of difficulty with modulation that relates to this pattern of comorbidity. Some children and adolescents experience motor tics. Others may develop vocal tics. These individuals have a *motor tic disorder*. If they have a chronic history of both motor and oral tics, the disorder is *Tourette's disorder*.

Thus, the continuum of neurologically based disorders with a high incidence of comorbidity includes:

1. Cortical problems: language disorder, developmental coordination disorder, learning disorder, executive function disorder
2. Attention deficit hyperactivity disorder
3. Modulating disorders: anxiety disorder (possibly panic disorder), mood disorders (possibly bipolar disorder), anger-regulation disorder (intermittent explosive disorder), obsessive-compulsive disorder, and tic disorders

Many of the studies of comorbidity relate the modulating disorders and ADHD. Few specifically refer to these disorders and learning disorders. However, given the reported relationship between ADHD and learning disabilities, it might be appropriate to infer that learning disabilities, ADHD, anxiety disorders, mood disorders, anger-control disorders, obsessive-compulsive disorders, and tic disorders have a high level of comorbidity.

An excellent review of these patterns of comorbidity can be found in a recent publication edited by [Brown \(2000\)](#). Several references for these patterns of comorbidity include:

1. Anxiety disorders ([Anderson et al., 1987](#); [Biederman et al., 1991](#); [Bird et al., 1988](#); Tannick, 2000)
2. Mood disorders ([Anderson et al., 1987](#); [Biederman et al., 1991](#); [Bird et al., 1988](#); [Spencer et al., 2000](#))
3. Obsessive-compulsive disorder ([Curry and Murphy, 1995](#); [Geller et al., 1996](#))
4. Tic disorders ([Comings et al., 1990](#))

### Emotional and Behavioral Disorders

Some children and adolescents under stress internalize this tension. They might present with a clinical picture of depression, anxiety, or somatic difficulties. Others might find the stress too unpleasant to handle and externalize the conflicts. These individuals might present with a clinical picture of oppositional defiant disorder or conduct disorder ([Chapter 53](#)).



Many of the clinical studies on comorbidity relate primarily to ADHD and oppositional defiant/conduct disorder ( [Caron and Rutter, 1991](#)); however, there are studies relating to learning disabilities. Studies of youth diagnosed as having a conduct disorder show that about one-third have unrecognized or recognized but poorly treated learning disabilities ( [Forness, 1981](#); [Hunt and Cohen, 1984](#); [Rutter et al., 1970](#)). Similar findings have been observed with adolescent boys in juvenile detention centers ( [Berman and Siegal, 1976](#); [Keilitz et al., 1979](#); [Lewis et al., 1979](#); [Lewis and Balla, 1980](#); [Mauser, 1980](#); [Robbins et al., 1983](#)).

### **Social Skills and Social Abilities Difficulties**

Some individuals with a learning disorder (disability) have social problems because the specific learning disability interferes with specific social skills ( [Silver, 1989](#)). For example, a child with a receptive and expressive language problem might have difficulty making small talk or following what peers are saying. A child with visual perception, visual-motor, and motor skills problems might have difficulty playing sports that require quick eye–hand coordination (e.g., catching, hitting, or throwing a ball).

It may be that for some children and adolescents with learning disabilities, the inability to read social cues (facial expressions, body movements, tone of voice, etc.) might reflect another type of a perception problem. Their difficulty reading social cues and mastering social skills also may be neurologically based ( [Hazel and Schumaker, 1988](#)).

## **SPECIFIC DEVELOPMENTAL LEARNING DISORDERS**

Before describing the specific developmental disorders related to reading, writing, and arithmetic, it is helpful to review the concept of specific learning disabilities. The presenting problem, difficulty with reading, written language, or math, should lead to an assessment. This evaluation process focuses on the specific processing problems resulting in learning disabilities. Knowledge of specific learning disabilities and abilities is useful in explaining problems to parents, children and adolescents, and others. Evidence of a processing disorder is necessary to have a student identified and serviced by the school system.

A psychoeducational evaluation is the assessment process used to explore the possibility of a learning disability. These studies use a processing model. Learning is evaluated based on the steps for processing information in the brain. The first task is to receive the information and record it in the brain (input). Once recorded, this information must be handled in such a way that it can be understood (integration). The third process is storage and retrieval (memory). Finally, information must be communicated from the brain (output).

Learning disabilities are defined based on this input-integration-memory-output model. Each area is described briefly.

### **Input Disabilities**

This central brain process is called perception; a person might have a visual or auditory perception disability. Visual perception disabilities might be reflected in distinguishing subtle differences in shapes (misperceiving a “d” and a “b,” or “p” and a “q,” or a “6” and a “9”). One might have difficulty with figure-ground tasks, that is, not being able to focus on the relevant stimulus in a field of vision. Judging distance or depth is another visual perceptual task. A child or adolescent might misjudge depth, bumping into things or falling off a chair.

Auditory perception disabilities might be in the area of distinguishing subtle differences in sounds (phonemes), thus leading to misunderstanding of what is being said. Many words sound similar, such as “hair” and “air” and “ball” and “bell.” Some individuals have difficulty with auditory figure-ground, confusing what sounds to listen to when there is more than one source. Some children and adolescents cannot process sound inputs quickly. They have to think about what is heard briefly longer than normal before understanding what is said. This is called an auditory processing problem or an auditory lag. They appear to be lost or confused when too much is said and may appear to be preoccupied.

### **Integration Disabilities**

At least three steps are needed to understand what is recorded in the brain. Individual incoming stimuli must be placed in the correct order (sequenced), then understood in the context used (abstractioned), and finally integrated with all other incoming stimuli plus all relevant memory tracks into a concept (organized). One can have an integrative disability in either of these areas: sequencing, abstraction, or organization. Such a disability might be more for visual or auditory inputs. Thus, one can have visual-sequencing or auditory-sequencing disabilities, visual-abstraction or auditory-abstraction disabilities, and so forth.

Sequencing disabilities can result in an individual's confusing inputs, for example, writing a “21” rather than a “12,” “dog” rather than “god,” or mixing up parts of words and mispronouncing the word. A child might try to explain something but start in the middle, then go to the beginning, and finally the end. These individuals often have difficulty using sequences. They can memorize the months of the year, for example, but be unable to say what comes after any month without starting with January and working their way up.

The inability to derive the correct meaning of a word based on how it is used can result in an abstraction disability. These individuals may have difficulty generalizing out from specific words or concepts. They might miss the meaning of jokes, puns, or idioms. They might appear to be paranoid because they interpret what others say in a more concrete or literal way than intended.

Organizational disabilities can result in difficulty pulling multiple parts of information into a full or complete concept or breaking down whole concepts into its parts. Such individuals may show this “dys-organization” in other aspects of their life. Their notebooks or desk or their room may be totally disorganized. Organizing time or making plans might be difficult. They lose things, forget things, or do their homework but forget to turn it in.

### **Memory Disabilities**

Children or adolescents with learning disabilities usually have excellent long-term memory. They can retain information once stored. They might have difficulty, however, in short-term memory—the ability to concentrate on information and store it. They might learn information well while attending to it (spelling list, math concept, etc.), yet they will not retain this information a short time later. These students need many more repetitions to process information into long-term memory than the average. They can learn, but they must work on the process over time. One might have a visual short-term memory disability or an auditory short-term memory disability.

### **Output Disabilities**

One may have difficulty getting information out of the brain through oral communication (i.e., a language disability) or muscles (i.e., a motor disability). The individual may have a disability in one or in both of these areas.

#### *Language*

Most individuals have little difficulty with spontaneous language. In this situation, the person initiates the conversation. There is the opportunity to organize thoughts and find words before speaking. Some individuals might have difficulty with demand language. If such people are asked to produce language, they must organize their thoughts and find the right words as they speak. They might speak with ease when they initiate the conversation; however, they might not be able to respond when asked a question or requested to speak. In this situation, they might struggle for thoughts and words, ramble, or say the wrong word and appear to be disorganized.

#### *Motor*

Some people with learning disabilities have trouble coordinating the use of large or small muscle groups. Gross motor disabilities result in being clumsy or having difficulty with such activities as running, climbing, or skipping. The most frequently found fine motor disability relates to writing. This skill requires the coordination of teams of small muscles. These individuals have difficulty getting their dominant hand to write in a fast and legible way. In addition to having poor handwriting, they might have difficulty with writing tasks such as spelling, grammar, spacing, or punctuation (called a written language disability). Other fine motor problems might be in

buttoning, zipping, tying, coloring, cutting, or controlling eating utensils.

### **The Learning Disability Profile**

Each individual with a learning disability has his or her own profile of learning abilities and disabilities. Each has one or more of the previously described disabilities. There is no stereotyped individual. Each must be assessed and understood individually.

These learning disabilities might result in what would be classified under the DSM-IV as a learning disorder, communication disorder, or developmental coordination disorder.

## **DEVELOPMENTAL READING DISORDER**

### **Definition**

There are three criteria for establishing this disorder in the DSM-IV: (a) Reading achievement, as measured by an individually administered standardized test of reading accuracy or comprehension, is substantially below that expected given the person's chronological age, measured intelligence, and age-appropriate education. (b) The disturbance in achievement significantly interferes with academic achievement or activities of daily living that require reading skills. (c) If a sensory deficit is present, the learning difficulties are in excess of those usually associated with it.

The skills required to learn to read are usually taught in first and second grade. The child first learns to connect the proper sounds (phonemes) to letters (graphemes); thus, "decoding" the word. After a child has decoded a word frequently enough, he or she recognizes the word as a whole unit, called sight-reading. Finally, recognition of words is so automatic that children can scan lines and thoughts come into their heads, that is, there is reading comprehension. These basic reading skills should be in place by third grade. The shift is to not, can you read, but what have you read? Thus, if a child has a reading disorder, he or she might struggle with reading in first and second grade but not have major difficulties until third grade when he or she can not move on to rapid, automatic reading.

### **Etiology**

Most reading disorders involve an inability to segment the written word into its underlying phonologic components ( [Shaywitz, 1996](#); [Tallal, 1985](#)). This deficit in phonologic awareness impairs decoding, preventing word identification. This basic deficit in what is essentially a lower-order linguistic function blocks access to higher-order linguistic processes and gaining meaning from text. Thus, the child has difficulty with reading fluency and text comprehension ( [Shepherd and Uhry, 1993](#)). It is not uncommon to find a spelling disability associated with a reading disorder because spelling requires the same phonologic skills as reading.

Studies of the brain using functional magnetic resonance imaging clearly shows a difference in brain function between individuals who have no reading problems and those with this language-based reading disorder (dyslexia). Brain activation patterns differ significantly between the groups of dyslexic readers, showing relative underactivation in posterior regions (Wernicke's area, the angular gyrus, and striate cortex) and relative overactivation in an anterior region (inferior frontal gyrus). These results are seen as confirming that the impairment in dyslexia is phonologic in nature, and these brain activation patterns may provide neural proof for this impairment ( [Shaywitz et al., 1998](#)).

### **Diagnosis**

First establishing a discrepancy between reading skill and intelligence and then systematically eliminating all other explanations for the discrepancy make the diagnosis.

### **Treatment**

Direct instruction in reading (and spelling and writing) is considered the essential treatment for a student with a reading disorder. A person trained to use appropriate remedial methods provides these efforts. These methods emphasize explicit instruction in letter-sound associations. Stress is placed on improving phonologic awareness. Most instructional models use a multisensory approach. Children see a letter and hear its name and sound, they trace the letter saying its name and sound, and then they write the letter repeating its name and sound. Sounds and letters are blended to form words. Reading, spelling, and writing are taught simultaneously. Instruction involves extended practice and is supplemented by speech segmentation training and study skills instructions. Parents are asked to read to their children to enhance appreciation of reading and to give their children access to knowledge normally obtained by reading. Early recognition and intervention are critical. There is a good possibility that the child will succeed in reading if a reading disorder is identified before the fourth grade and appropriate phonologically based interventions are provided. The level of success with intervention is less if the difficulties are not identified and treated until a later grade or if the reading difficulties are noted but not addressed.

The ability to read is an essential part of education. If the student does not master this skill not only will educational progress be limited, but also occupational and vocational opportunities are limited and self-esteem and self-confidence suffer. It is critical that these children be identified early and receive appropriate educational interventions.

## **DEVELOPMENTAL DISORDER OF WRITTEN EXPRESSION**

### **Definition**

There are three essential criteria for establishing this disorder in the DSM-IV: (a) Writing skills, as measured by an individually administered standardized test (or functional assessment of writing skills) are substantially below that expected given the person's chronologic age, measured intelligence, and age-appropriate education. (b) The disturbance in skills significantly interferes with academic achievement or activities of daily living that require the composition of written texts (e.g., writing grammatically correct sentences and organized paragraphs). (c) The learning difficulties are in excess of those usually associated with it if a sensory deficit is present.

It is helpful to distinguish between a fine motor problem that results in difficulty with the mechanics of writing and a language-based learning disability that results in problems with the language of writing. Individuals with a disorder of written expression might have a fine motor problem but always have a language-based disorder, resulting in difficulty with spelling, grammar, punctuation, capitalization, and/or composition.

### **Etiology**

There must be an automatization of most of the lower level mental activities for skilled writing. These lower level mental activities guide handwriting, spelling, word choice, and the construction of sentences that conform to the conventions of written language. Lower-level mental activities also guide the construction of textual connections, specifically connections between sentences. The writer's attention can focus on the content, organization, and clarity of the task if these activities are functioning. One can shift attention between levels of mental processing without losing control of the text. Difficulties with lower-level mental activities appear to be the source of the problem in expressive writing disorder ( [Uhry and Shepherd, 1993](#)).

Research focuses on the relationship between writing and reading disorders ( [Johnson, 1988](#)). They may be linked in two ways: First, the same linguistic deficits that impede learning to read (phonologic awareness) could impede learning to write and spell. Second, slow progress in learning to read might deprive the child of knowledge about writing that is gained by reading (e.g., about sentences, punctuation, and spelling).

### **Diagnosis**

First establishing a discrepancy between writing skill and intelligence and then systematically eliminating all other explanations for the discrepancy make the diagnosis.



## Treatment

Treatment for a writing disorder might involve a skills or holistic approach ( [Uhry and Shepherd, 1993](#)). Skills programs are often used with younger children and focus on letter–sound associations, focusing on reading and spelling. Children might be asked to listen carefully for the sounds in words and then to represent these sounds with written letters, saying each letter aloud as it is written.

The holistic approach to writing begins with the student's ideas. It involves a series of highly structured steps for narrowing ideas to one topic, writing a first draft, reading it aloud to an audience of peers, and then refining organization and language. The final step involves working on mechanics in preparation for “publishing” a draft for peers to read.

Most efforts combine these two approaches. Children with a writing disorder need direct, sequential instructions in letter–sound associations and spelling rules as well as sentence structure, and the connections between sentences and paragraphs that make text cohesive. Even with good remedial interventions, writing requires enormous effort because this disorder is not “cured” but compensated for and this compensation must continue into adulthood. Appropriate accommodations, such as using a computer, may be needed throughout the individual's education and career. The use of the computer is an example of using strengths to compensate for weaknesses. Writing requires detailed fine motor planning using one's dominant hand. Typing requires the use of the lower arm muscles to lower and raise the fingers and, thus, is predominantly a gross motor planning task. Individuals who write slowly and with difficulty might be able to type with great speed and accuracy.

## MATHEMATICS DISORDER

### Definition

Three criteria are used to establish this diagnosis in the DSM-IV: (a) Mathematical ability, as measured by an individually administered standardized test, is substantially below the expected given the person's chronological age, measured intelligence, and age-appropriate education. (b) The disturbances in ability significantly interfere with academic achievement or activities of daily living that require mathematical ability. (c) The learning difficulties are in excess of those usually associated with it if a sensory deficit is present.

Children and adolescents with a mathematics disorder may have a wide range of symptoms, including delays in the acquisition of basic spatial and number concepts, problems learning and using number words and number facts or writing numbers correctly (and in correct alignment when doing computations), and difficulty in applying arithmetic skills when solving everyday problems. Because math achievement is highly dependent on the quality of instruction offered students, it may be that a significant number of those students who are coded as having a learning disability by the school system do not have intrinsic math disorders but have not had appropriate instruction.

### Etiology

Research suggests that there may be four primary factors involved in mathematics achievement: language, conceptual, visual-spatial, and memory ( [Johnson, 1988](#)). In addition, knowing how to use the correct strategy is important ( [Fleishner and Garnett, 1993](#)). Information-processing capabilities research suggests that one area of difficulty might be in visual-spatial processing deficits. Another problem might relate to the ability to use the necessary pattern of strategic behaviors associated with arithmetic learning. Proficiency in mathematics requires more than computational skills. The difficulty might be in an inability to develop a systematic plan for problem solution. Memory is essential to the ability to do mathematics. One must retain basic number facts. In addition, it is necessary to remember specific equations or other steps necessary to solve a problem. Once a problem is started, students must remember where they are in the process as they proceed from step to step.

### Diagnosis

First establishing a discrepancy between arithmetic skill and intelligence and then systematically eliminating all other explanations for the discrepancy make the diagnosis.

### Treatment

Treatment involves general concerns and specific interventions ( [Fleishner and Garnett, 1993](#)). Initially, the problem of anxiety, withdrawal, defeatism, or other responses to repeated failure must be addressed. Much of the teaching of math is based on criticism of failure. The emphasis is on getting the right answer rather than problem solving. Children may develop performance anxiety or feel they are stupid in math because of this focus on getting the right answer. For some, the premature pressure for speeded answers can short-circuit reasoning and foster impulsivity, resulting in the belief that math has little to do with thinking. Students who have difficulty with math memory may be deprived of compensatory techniques, only adding to the problem. They are told that it is shameful to use their fingers, mark down intermediate calculations, or use a calculator. These are considered “cheating.” Sometimes the difficulties reflect poor teaching in math. Further, math learning is very instruction-bound. Absenteeism, missed lessons, and inconsistency of instruction can leave gaps that result in persistent confusion. Those students with learning disabilities in the areas that result in deficits in short-term memory, language, attention, or spatial perception and those with ADHD are especially susceptible to weak instruction. Appropriately focused and systematic instruction in improving math abilities may be all that is needed.

Specific interventions focus on the underlying difficulties. Some students have problems relating to acquiring the conceptual underpinnings of the subject and some have problems relating to procedural learning, recall of discrete information, and self-monitoring. These remedial efforts must take into account the student's areas of learning abilities and disabilities.

## NEUROLOGIC BASIS FOR LEARNING DISORDERS

For many years, learning disabilities were presumed to be neurologically based. Newer brain studies confirm this concept; therefore, the word presumed is no longer needed. Gross and microscopic studies of the brain as well as newer imaging techniques have focused on finding the biological substrate for the learning disorders (disabilities). In addition, familial and genetic studies and data on environmental developmental and neurologic toxins contribute to our new knowledge.

[Galaburda and associates \(1985\)](#) showed on detailed microscopic analysis of the brains of individuals known to have had dyslexia strongly support the concept of miswiring. Each case studied demonstrated the same microscopic findings: neurons in places where they are normally absent (ectopias), cortical cellular disorganizations (dysplasias), and polymicrogyria. These cortical anomalies were located predominantly in the left hemisphere and mainly around the left sylvian fissure. These findings have been interpreted as anomalies of neuronal migration that are most likely to have occurred between the 16th and 24th weeks of gestation.

As noted, imaging studies of the brain clearly show a neurologic difference between the brain of individuals without a reading disability and those with a language-based reading disability ( [Shaywitz et al., 1998](#)). Increasing research focusing on the functional differences in brain function between individuals with learning disorders and those with specific types of learning disorders will result in even more evidence of the neurologic basis for these disorders.

Recent findings suggest a possible connection between environmental toxins and the increased incidence of developmental, learning, and behavioral problems ( [Physicians for Social Responsibility, 2000](#) ). The National Academy of Science ( [2000a,2000b](#) ) has released two studies on developmental and neurologic toxins. U.S. companies reported to the federal government that in 1998 they released 1.2 billion pounds of chemicals into the nation's air and water that have the potential to affect the way a child's body and brain develops. More than half (53%) of all toxic chemical emissions reported to the Federal Toxics Release Inventory are known or suspected to be developmental or neurologic toxins. There is much to learn about the impact of these environmental toxins on the increased incidence of developmental, learning, and behavioral problems.

## DIAGNOSIS OF DEVELOPMENTAL LEARNING DISORDERS

Children and adolescents with learning disorders (disabilities) are often first referred for an evaluation because of behavioral difficulties. Thus, when a mental health professional evaluates a student with emotional, social, and/or family problems who also have academic difficulties, it is essential that one differentiate between cause and symptom ( [Silver, 1997](#) ). Are the emotional, social, and/or family problems causing the academic difficulties or are they a consequence of the academic difficulties

and the resulting frustrations and failures? The emotional, social, and/or family problems may have started from the unrecognized and untreated learning disabilities and then evolved into emotional or behavioral disorders. Each must be addressed. However, if the clinician does not address the underlying cause of emotional and behavioral problems (possibly the learning disabilities), little progress might be made.

The clinical history helps to raise the suspicion of a learning disability. The clinician should ask about school performance and ability with each academic area. A suggested "system review" would include the following ([Silver, 1993](#)):

Start with questions relating to basic skills.

### **Reading**

Is reading something you like to do or have to do? How well do you think you read? Do you ever make mistakes because you misread questions or instructions?

Do you ever find yourself skipping words or lines or reading the same lines twice?

Do you find that you can read each word but that when you finish the page or chapter you do not remember what you have read?

### **Writing**

How is your handwriting? Do you prefer to print or use cursive?

Do you find that you cannot write as fast as you are thinking?

Tell me about your spelling? grammar? punctuation? capitalization skills?

Do you have difficulty copying off of the blackboard or overhead quickly enough?

Do you have difficulty getting your thoughts onto the page?

### **Math**

Do you know your times tables?

When you do math do you make silly mistakes like write "21" when you mean to write "12," or do you mix up your columns or add when you mean to subtract?

Do you have difficulty with word problems where you have to read and understand then change the words into formulas?

Do you sometimes start a math problem but halfway through forget what you are trying to do?

Next, ask questions relating to the processing skills needed to learn.

### **Sequencing**

When you speak or write, do you sometimes have difficulty getting everything in the right order?

Ask the child to name the months of the year. Then ask what comes after May. Can he or she answer or is it necessary to go back to January and then count forward?

Do you have trouble using the dictionary and remembering the order of the alphabet?

### **Abstraction**

Do you understand jokes when your friends tell them?

Do you sometimes get confused when people seem to say something yet they tell you they meant something else?

### **Organization**

What does your notebook look like? How about your binders and papers?

Is everything in the wrong place or falling out? What about your desk? backpack? locker? bedroom?

Do you lose or forget things? Do you do your homework but, somehow, it never gets turned in?

Do you have difficulty organizing your thoughts or the facts you are learning into a whole concept so that you can learn it?

Do you have difficulty planning time so that things get done?

### **Memory**

Do you find that you can learn something at night and then go to school the next day and forget what you have learned?

Do you learn best by listening to people (such as discussion groups) or by being alone and reading or writing?

If a learning disability is suspected, a psycho-educational assessment is needed. This should be done by the student's school system but might also be done privately. These studies include three areas of assessment: First, an assessment of the level of intellectual function; second, an assessment of academic skills in reading, writing, and math; third, a comprehensive study of processing skills.

The results of these evaluations should show if there is a discrepancy between ability and performance and establish the presence or absence of a learning disability. These findings along with the psychosocial evaluation clarify if other disorders are also present.

Some of the currently used tests used in the United States include:

1. Intellectual assessments
  - a. Stanford-Binet Intelligence Scale (ages 2 to adulthood)
  - b. Wechsler Pre-School and Primary Scale of Intelligence (WPPSI) (ages 4 to 6 1/2)
  - c. Wechsler Intelligence Scale for Children (WISC) (ages 6 to 16)
  - d. Wechsler Adult Intelligence Scale (WAIS) (ages 16 and over)
2. Achievement tests



- a. Woodcock-Johnson Psycho-educational Battery
- b. Metropolitan Achievement Tests
- c. Peabody Individual Achievement Test
- d. Stanford Diagnostic Achievement Tests
- e. Wide-Range Achievement Tests
- 3. Educational/processing abilities tests
  - a. Woodcock-Johnson Psycho-educational Battery
  - b. Detroit Tests of Learning Aptitude
  - c. McCarthy Scales of Children's Abilities
  - d. Slingerland Screening Tests

## TREATMENT OF DEVELOPMENTAL LEARNING DISORDERS

The treatment of choice for developmental learning disorders (disabilities) is special education. The specific approaches to treatment were described for each disorder. In general, there are three approaches used. Remediation is an effort to teach skills by using different approaches than the traditional models. The teaching of compensatory strategies is another valuable approach. Given the student's strengths and weaknesses, what is the best way for him or her to read and retain what is read, to write in an organized way, to learn material, and so forth. The third approach is accommodations. Teachers are asked to modify the material, teaching approach, assignments, or test designs to maximize the student's ability to communicate what is known.

If emotional, social, and/or family problems are present, they must be addressed in a multimodal treatment plan. When providing individual, group, family, or behavioral therapy, it is important to be aware of the impact the learning disabilities have on the individual's life as well as the impact of these disabilities on the treatment method itself (Ostrander and Silver, 1993). There are no psycho-pharmacologic treatments for learning disabilities. If, in addition to the learning disabilities, the individual has ADHD, medication should be used to address this disorder. Many of the psychiatric disorders found in children and adolescents with learning disabilities would benefit from the use of medication as well.

## OUTCOME

For most, learning disorders (disabilities) persist throughout life. The child with learning disabilities becomes the adolescent with learning disabilities and the adult with learning disabilities. Thus, the clinical interventions change with each developmental stage, including adulthood. Services might be needed in college, graduate, and professional schools as well as vocational and job training programs. Appropriate accommodations may be needed throughout this individual's career.

Most individuals can overcome or learn to compensate for their disabilities with early recognition and appropriate special education intervention. Additional strategies might have to be learned and mastered at each stage of education, as well as for the demands of work.

The outcome can be poor without recognition or treatment. These children and adolescents experience frustration and failure, resulting in a poor self-image and low self-esteem. They are at risk for emotional and behavioral problems. Early recognition and intervention are the best preventions for these serious problems.

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# 51 PSYCHIATRIC EVALUATION OF PERCEPTUALLY IMPAIRED CHILDREN: HEARING AND VISUAL IMPAIRMENTS

Mayu P.B. Gonzales, M.D. and Stella Chess, M.D.

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## GENERAL CONSIDERATIONS

Sensory impairments do not cause psychiatric illness per se but may affect symptom presentation. Misdiagnosis of psychiatric disorders, particularly mental retardation in sensory-impaired children, may occur if the diagnostician is unaware of the adaptive differences and, hence, the variability in timing and acquisition of developmental milestones owing to the sensory impairment. Troubled behavior may represent independent psychiatric conditions or consist of acquired maladaptive compensatory behaviors. Sensory impairment influences the acquisition of skills, self-concept, and interaction with environment. In response to the impairment, the child's environment can shape the sequential acquisition of skills and the developmental process. The influence of impairments on the developmental process and presentation of psychiatric signs and symptoms and the context in which the child functions are pivotal to the psychiatric evaluation of perceptually impaired children. The objectives of a psychiatric evaluation are accurate diagnosis and development of an appropriate treatment plan that address the multiple and complex problems identified ([American Academy of Child and Adolescent Psychiatry Practice Parameters, 1997](#)). Fundamental considerations for evaluation of a sensory-impaired child include the tremendous range and heterogeneity of the individual impairments, frequent occurrence of multiple impairments, complex interdependence of the impairments with the environment and caregiver response, and differing potentials among children with similar impairments. The nature and extent of impairments differ with etiology, age of onset, and other complicating factors.

High frequency of additional impairments and comorbidities result from autism, attention deficit hyperactivity disorder (ADHD), mental retardation, neuropsychological disorders of learning, central nervous system syndromes, and traumatic brain injuries. Many children and adolescents have comorbidities that do not fit neatly into a simple diagnosis, but categories are identified in a full psychiatric evaluation through multiaxial diagnoses. Those who evaluate and work with sensory-impaired children may lack exposure to profoundly impaired individuals who lead successful and constructive lives. This is important because professional attitudes and expectations subtly shape the evaluation.

Consistent language is helpful in communicating findings to patients and their families. Impairment is an identifiable defect in the basic functions of an organ system and for our purpose concerns hearing and vision. Disability refers to limitation, restriction, or disadvantage imposed on an individual's functioning. The term handicap is not currently favored. The term challenged has been used in its place, as in visually challenged, reflecting the outcome of an actual or perceived disadvantage in performing normal life functions because of personal and/or societal expectations and attitudes toward the impairment.

Legally, "learning disability" as defined by the Individuals with Disabilities Education Act ([IDEA, 1970, 1997](#)) includes students with "perceptual handicaps" resulting from visual, hearing, motor disabilities and/or mental retardation [20 USC sec. 1401(26)(B)(C)]. There is a need to be familiar with the definition of disability as provided by the IDEA:

'a child with disability' means a child (i) with mental retardation, hearing impairments (including deafness), speech or language impairments, visual impairments (including blindness), serious emotional disturbance . . . , orthopedic impairments, or specific learning disabilities; and (ii) who, by reason thereof, needs special education and related services [20 USC sec. 1401 (3)].

An infant or toddler with a 'disability' means an individual experiencing developmental delays, in areas of cognitive, physical, communication, social or emotional, and adaptive development; or has a diagnosed physical or mental condition with a high probability of developmental delay . . . [20 USC Sec. 1432 (5)].

When the IDEA, originally passed in 1970, and revised in 1997, it replaced the Education for All Handicapped Children's Act of 1975. These federal definitions have been adapted and incorporated into state and local education laws.

## THE ROLE OF A CHILD AND ADOLESCENT PSYCHIATRIST

The role of the psychiatrist as liaison to interface and interact with a multidisciplinary medical or rehabilitation team and the family, school, and community is unique. The child psychiatrist's training permits integrating biological, developmental, family/environmental, learning/education, and psychodynamic/psychiatric issues in order to develop a diagnostic formulation. In both evaluation and treatment, the consulting psychiatrist needs to establish a collegial relationship with the parents and school

staff. Frequently, the school or rehabilitation service requests a consultation in order to assess risk of suicide, psychopharmacologic management, change in level of care, or educational program. Current educational trends for sensory impaired persons include early intervention assessments and therapeutic interventions for infants and toddlers exhibiting developmental delays and integration of sensory impaired children into regular schools. These trends increase the probability that child psychiatrists lacking specialized experience may be asked to evaluate sensory-impaired youngsters. Referrals also come from schools where children are mainstreamed. In special schools, the need is for initial triage, assessment of complex biopsychosocial issues, and monitoring outside psychiatric care. Most sensory impairments are life long; hence, the initial involvement of the child psychiatrist often evolves into long-term assistance. Child and adolescent psychiatrists must accommodate comfortably to specialized settings and adapt innovative interviewing techniques in both assessment and treatment.

Learning manual sign language is very helpful but difficult to master. An interpreter for hearing-impaired children and utilization of play with a blind child are feasible alternatives. Familiarity with the use and purpose of various assistive devices can facilitate rapport with the child and family. The child psychiatrist who is best able to approach the evaluation is knowledgeable in child development; looks for adaptational, problem-solving, and functional strengths; is aware that a sensory impairment does not routinely result in psychological impairment; and seeks to identify the strengths and weaknesses within the family. The questions, "How does the impairment interface with the developmental process and/or the environment and what have been the adaptive responses?" are very helpful. The goals of the examination should include looking for the child's level of independence, cognitive abilities, functional abilities, social adjustments, and psychological adaptations. Further, the examiner would want to consider why this case has been referred at this time; who made the referral; what is happening in the home, family, or school; for example, does the child need a new school or is the child aging out of the current school; has the child grown and developed to the point where previous management techniques are no longer possible and medications are being requested; and is there an underlying purpose for the referral, such as expediting removal of the child from the home, school, or changing the level of care? Assessment of the various domains of development is indicated in infants and toddlers. Further, the examiner would want to assess the child's ability to understand cause and effect relationships to modify his or her behavior. Reviewing various records and looking for the longitudinal patterns in addition to associated impairments answers some of these questions.

It is important for the evaluating psychiatrist to ascertain the parents' expectations and from what sources they have received advice. The normal parental wish is for a happy, unimpaired child and parents may disregard or deny the reality of their child's circumstances, pursuing unrealistic treatments and goals. The authors have repeatedly seen that the level of the parental involvement and advocacy for their child are important predictors of the child's success. In many cases, the "validity" of the parental expectations can be determined only after the fact, retrospectively once the child has grown. Assessment of the environment and family of the sensory-impaired child should consider the increased demands in time and energy required of parents. The demand of caring for a sensory impaired child can drain the emotional and physical resources of families, causing strife and parental dysfunction. Family structure and alliances can become skewed, and the full dimensions of family life may be curtailed. Financial costs can further tax family resources. Mothers more frequently than fathers attend rehabilitation services, and childcare support for the sensory impaired child or other children in the home can further drain resources. A gulf can grow between parents as polarized reactions occur. Siblings may feel excluded, jealous, or cheated while conflicted with the relief that they themselves are not impaired. Many families are able to form healthy and constructive responses. The task of communicating one's findings and engaging the family and school toward recommended treatment and intervention is made more difficult if they have contributed to maladaptive behaviors and stunted development. Clinical sensitivity and expertise is required to help parents and other caregivers including school personnel recognize both their positive and negative influence on the process and outcome of treatment.

## HEARING IMPAIRMENTS

### Definition

Hearing-impaired children are divided into hard of hearing and profoundly hearing-impaired or deaf groups. Traditionally, the distinction was based on those youngsters who could utilize their diminished auditory perceptions for communication and those who could not. The development, improvement and increased use of hearing-assistive devices including cochlear implants and expanded rehabilitation services has permitted an increasing number of hearing-impaired children with significant hearing loss to effectively use their residual hearing. Hearing-impaired children are also classified by the age of onset of hearing loss. "Prelingual" refers to onset of hearing impairment before the use of spoken language, and "postlingual" to impairments occurring after spoken language has been acquired. Generally, a loss of hearing before 2 years of age is considered prelingual. The age of onset of hearing impairment is a significant factor in subsequent language outcome even in children with similar levels of hearing loss.

### Historical Note

Hearing deprivation has existed throughout history. Educators and religious figures who have taken on the task of instruction of deaf pupils have influenced Western European attitudes and practices. In some Western European cultures, the right to inherit property was dependent on the ability to confess to God; therefore, oral speech took on a special significance. Early teaching combined variations of oral and sign communication. The controversy among proponents of manual, oral, and total communication, which is a combination of both, have waxed and waned over time and are detailed graphically by [Lane and Fischer \(1993\)](#). The deaf culture movement has challenged the rights of parents to remove a child from the culture by assistive hearing devices. Further, it has questioned the obligation of larger systems and even society's role in maintaining the integrity of the deaf culture. The deaf culture movement and the promotion of American Sign Language (ASL) have influenced implementation of IDEA. With the introduction of Public Law 94-142 in 1975, deaf and hard-of-hearing students have increasingly been taught and educated in regular schools largely populated by hearing children, although the extent of academic integration varies. The IDEA revision further supported the movement of deaf and hard-of-hearing students into mainstream education where opportunities for bilingual (sign and oral language) education and an accommodation for various communication devices (language boards, computers, etc.) is an expectation. PL99-457, which established early intervention programs, has done the same for children ages 0 through 3. The recommendations of the Commission on the Education of the Deaf also represent significant steps on behalf of the deaf.

### Prevalence and Epidemiology

The survey conducted by the Gallaudet Research Institute, now in its 32nd year, represents the largest ongoing database of information on deaf and hard-of-hearing children in the United States and provides the best epidemiologic estimates of that population. The most recent completed report for the 1997 to 1998 school year with a total of 50,629 cases represents more than 12,000 schools and is estimated to cover about 60% of identified deaf and hard-of-hearing children receiving any special services. The shift in demographics of the completed survey reflected a doubling of the Hispanic constituency over the past 20 years. There has been a dramatic decline of maternal rubella cases from 21% in 1977 to 1978 to 1% in 1996 to 1997. Over the same period, the age constituency of the survey reflected the movement of the rubella bulge, which is the cohort of children impaired secondary to the rubella epidemic in 1964. The proportional representation of other common known etiologies such as heredity, prematurity, meningitis, and otitis media remain fairly constant. However, the number whose etiology is cytomegalovirus, which was first added in the survey in 1986, has climbed steadily, and now accounts for 2% of the population. Some sociocultural changes with respect to maternal drug abuse are also reflected in the survey. Reported emotional and behavioral problems, uncorrected visual problems, mental retardation, and learning disabilities accounted for additional disabilities in a third of the population, whereas the remaining 66% reported only hearing impairment. An increase in reported learning disabilities from 2% in 1977 to 1978 to 9% in 1996 to 1997 and a decrease in emotional/behavioral problems from 7% to 4% during the same period many reflect systemic changes from IDEA implementation as students were reclassified from emotional/behavioral disability to learning disability. Critics point out under reporting of children with mild hearing impairments, thereby skewing the survey.

The degree of hearing impairment traditionally has been stated in terms of volume or decibel loss over the speech frequencies (250 to 8,000 Hz). The testing procedure measures the volume threshold at which a youngster perceives a pure single tone at a particular frequency. Such testing excludes associated frequency harmonics; the capacity to distinguish subtleties of sounds when frequencies are mixed or auditory input competes with background noise. Vowel sounds group in the low- to mid-range frequencies; consonants and sibilants tend toward the higher ranges. Thus, a child with partial loss might comprehend some words but guess at others ([Northern and Downs, 1991](#)). There is broad agreement on the need for early identification and intervention with hearing-impaired children. Universal neonatal audiologic screening to detect deafness has been suggested ([Sutton, 1999](#)), and auditory brain stem response screening for at risk newborns is seen as cost effective and clinically efficient ([Van Riper, 1999](#)). Assessments that measure specific areas of functioning may provide a more accurate picture of hearing impaired children ([Karchmer, 1999](#)). Audiologic assessment of preverbal and nonverbal children requires different evaluation methods. Periodic and repeated audiologic evaluations, particularly of young children, provide information about developmental strides. The generally accepted audiologic classifications for hearing impairments are given in [Table 51.1](#).



Hearing Loss Level	Character	Major Cause	Effect on Language Development	Developmental Effects on School Achievement	Major Needs
0-20 dB	Normal range	Conductive hearing loss	All speech sounds clearly heard	None	None
20-40 dB	Mild hearing loss	Conductive hearing loss	Speech sounds heard but may be distorted	None	None
40-70 dB	Moderate hearing loss	Conductive hearing loss	Speech sounds heard but may be distorted	None	None
70-90 dB	Severe hearing loss	Conductive hearing loss	Speech sounds heard but may be distorted	None	None
90-120 dB	Profound hearing loss	Conductive hearing loss	Speech sounds not heard	None	None
0-20 dB	Normal range	Sensorineural hearing loss	All speech sounds clearly heard	None	None
20-40 dB	Mild hearing loss	Sensorineural hearing loss	Speech sounds heard but may be distorted	None	None
40-70 dB	Moderate hearing loss	Sensorineural hearing loss	Speech sounds heard but may be distorted	None	None
70-90 dB	Severe hearing loss	Sensorineural hearing loss	Speech sounds heard but may be distorted	None	None
90-120 dB	Profound hearing loss	Sensorineural hearing loss	Speech sounds not heard	None	None

**Table 51.1. Handicapping Effects of Hearing Loss in Children**

Many states set the criterion of an average 80-dB loss in the better ear for admission to a state school for the deaf. Many children with a greater than 80-dB loss can learn to utilize their residual hearing and function in a mainstream hearing school, whereas others with a loss less than 80 dB cannot. A child with intermittent middle ear conductive impairment may vary in hearing ability from day to day. Further, a loss of more than 15 dB, especially following several episodes of otitis media during critical periods of language acquisition and education, results in diminished attainment, potential, and IQ scores ( [Northern and Downs, 1991](#) ).

### Etiology

The relative etiologies of childhood hearing loss have varied historically based on the presence of epidemics, pandemic diseases, and preventive measures available, whereas genetic inheritance has persisted in stable numbers. Deafness was a frequent consequence of ear infections and systematic diseases. Congenital deafness includes intrauterine etiologies and more than 50% of cases of congenital or early onset deafness have a genetic cause. Unknown etiologies have accounted for as much as a third of the cases of hearing impairment, although genetic sources may be responsible. There are many distinct genetic forms of hearing loss interfering with normal conversation. Mutations in the GJB2 gene are the most common cause of inherited congenital deafness ( [Green et al., 1999](#) ). The effects of the impairment may range from delays in the development of speech to lifelong impairments, although the timing of the etiologic event results in differing presentations and associated complications. Viral fetal infections interfere with other organ development, and multiple impairments are found, including visual impairments (e.g., congenital cataracts), cardiac malformations, neurologic impairments, seizure disorders, and mental retardation. The increase in cytomegalovirus infections demands a combined approach for universal screening for hearing as well as screening for cytomegalovirus in order to identify sensorineural hearing loss early ( [Fowler et al., 1999](#) ). Autism has been seen in disproportionate numbers in rubella and several other viral intrauterine etiologies ( [Chess et al., 1971](#) ). Deafness associated with prematurity and other intrauterine insults is often complicated by other disorders. Temporary, fluctuating, or permanent hearing impairment may complicate postnatal ailments such as meningitis, mumps, measles, middle ear infections, and allergies. Deaf children with hearing parents comprise the majority of the pediatric deaf population.

### Pathogenesis in Development

The basic deprivation of deafness is language ( [Meadow-Orlans, 1987](#) ). Deafness and its consequences on developing language acquisition have a profound effect on other domains of development given its influence on cognition and social behaviors. During the developing preschool years, when hearing children gain sufficient mastery of spoken language to express needs, desires, and emotions, the hearing-impaired children of hearing families are confronted with tremendous obstacles. The diverse influence and interrelationship of sound and language to the developmental processes is ubiquitous. The effects of profound prelingual hearing impairment can be grouped into language development and acquisition, cognitive development and information acquisition, and social development and behavioral difficulties.

### Language Development

Language is the tool for formulating thoughts and is differentiated from speech (articulation) or communication (which is an interactive and reciprocal exchange). A deaf baby babbles at the same age as a hearing baby. The deaf baby ceases to babble when babbling becomes social and infants begin to model the sounds they produce on the sounds they hear. There are wide variations in the milestones of the onset of speech in hearing children. The deaf child's lack of response to sounds might be rationalized away. For the hearing parents, the delayed and startling diagnosis of deafness requires a dramatic reorientation of their conceptualization of their child. The parents are faced simultaneously with a major decision as to the type of language acquisition training—manual or sign language, oral language, or total communication, which encompasses both, often in the face of conflicting professional opinions. The campaign against the use of manual language was based largely on the misconceptions that oral language is necessary for abstract thought and interacting with the hearing world, and that sign language is not a true language. [Noam Chomsky's \(1998\)](#) suggestion of an innate preprogramming for the process of language has been widely supported. Spontaneous sign systems observed in children of different cultures contribute to the understanding of the innateness of language in human infants ( [Goldin-Meadow, 1998](#) ). Observations that the syntactic structures and processes of language development of deaf and hearing children are similar lend support to the expanding use of sign. [Stokoe \(1976; Stokoe et al., 1978\)](#) demonstrated sign language to be a true language through linguistic analysis of ASL and published the first dictionary of ASL that was based on linguistic principles.

The hearing child with hearing parents learns spoken language through environmental interaction. The deaf child learns communicative language by visual means. The position of oral language proponents is that the deaf child will be unable to interact with the dominant hearing population without some spoken communication. Support for early auditory amplification and training is encouraged, so that the process of vocalization and babbling can be preserved and the concept of sound for communication and shaping of the vocalizations can be facilitated. The manual argument asserts that the vast majority of deaf people ultimately enter deaf culture for social and personal needs; therefore, they should feel comfortable with and accomplished in the language of their culture. The potential psychological conflicts in this dichotomy, which confront the consulting psychiatrist, are highly emotionally charged. This controversy is not dissimilar to the debates on bilingual education and accommodations to diversity and multiculturalism. Oral training is demanding; its proponents state that signing hinders its successful mastery. There remains great heterogeneity of achievement within both the oral and total communication groups and varying degrees of success in formal language acquisition. The tragedy of this politically charged controversy is that parents are required to make a lifelong decision for their child while facing emotional upheaval. Child psychiatrists' greatest value during the child's crucial cognitive and social development period may be to help parents teach acceptable behavior, including rules, compromise, and delayed gratification. This may be particularly difficult when the parent is dependent on only a "yes" or "no" response in exclusive oral communication.

The eventual language preference among deaf young adults balances the nature of the residual hearing, quality of early training, usefulness of communication, and cultural orientation of the individual toward the hearing world or deaf culture social groups. Many adults develop bilingualism in varying degrees but the issue of language and social isolation persists with varying psychological consequences. The deaf child in a hearing family tends to be isolated irrespective of communication. Deaf adolescents trying to function within the hearing environment particularly complain about their difficulty at parties and dances, where lowering of lights and background dance music makes communication and flirting almost impossible. To conceptualize such difficulties, consider the child who encounters any number of situations where vision and hearing are used and coordinated simultaneously, such as class note taking, talking at the dinner table, or honking cars warning of danger. Many children who later prefer to use sign but whose parents do not, often view the parents' lack of communication skills as evidence that they do not love them. Sibling interactions with the hearing-impaired child also affect self-perceptions and social skills. Adolescents often try to fake hearing and discard aids with frequent negative consequence in self-concept.

### Cognitive Processes and Theories

Cognition is linked to language acquisition. Areas of exploration, including memory, visual-motor and perceptual-motor functions and problem-solving strategies are being pursued with advanced technologies, an increased knowledge in the basic neuroscience, neuropsychology, and rehabilitation therapies. Reported intelligence quotient (IQ) scores and academic achievement are concerns for the child psychiatrist but scores can be misleading. The most commonly used tests among deaf children are the Wechsler Intelligence Scale for Children, 3rd ed (WISC-III, 1991), Leiter International Performance Scale, Wechsler Adult Intelligence Scale, 3rd ed (WAIS-III), Hiskey-Nebraska, Goodenough Draw-a-Person Test, Merrill-Palmer, and Ravens Progressive Matrices. The Wechsler scales were not designed for hearing, visual, or motor impairments. As stated in the WISC-III Manual, although one may prefer to place greater weight on the performance subtests to estimate a hearing-impaired child, the WISC-III was not standardized with modifications. If sign language and other visual aids are necessary to give instructions to a deaf child,

the evaluator and presumably the clinicians who are likely to use the tests scores should recall such alterations to have an impact on scores. For infants and preschoolers, developmental measures such as the Bayley Scales of Infant Development, 2nd ed (BSID-II), Gesell Developmental Schedules, or Cattell Infant Intelligent Scales are used. The following considerations about psychometric testing of hearing-impaired children include: The instrument should be nonverbal, because a verbal instrument can reflect language rather than cognition; there is greater likelihood that the instrument will report a falsely low score than a falsely elevated score; there is increased potential error when the test is administered by an examiner not familiar with hearing impairments, unable to communicate instructions in the child's preferred mode; personality tests are difficult to interpret because subtlety of instructions might not have been communicated and responses may be limited or unintelligible; and testing of preschool children at whose age communication skills are only beginning to develop is vulnerable to error and underestimation of potential. [Slate and Fawcett \(1995\)](#) have investigated the relationship of the WISC-III Performance scale with the WISC-R Performance scale and the WRAT-R subscales. Students communicating via total communication exhibit higher performance IQ means than students communicating orally. The WISC-III performance IQ was moderately related to the WRAT-R subscales having the strongest association with the arithmetic subscale. [Braden \(1989\)](#) found little correlation of the nonverbal, WISC-R Performance Scale k(PS) IQ to academic achievement using the Stanford Achievement Test-Hearing Impaired Edition. This suggests that the criterion of academic achievement fails to be an accurate reflection of the deaf child's innate cognitive abilities. Currently, many schools for the deaf use total communication where the teacher articulates and uses sign. The educational outcome of impaired children who have been mainstreamed is still uncertain, as is the long-range achievement results for deaf children taught by total communication. However, it is most likely that the psychiatrist evaluate a child with academic delays or unevenness in achievement.

### **Social Development and Behavioral Difficulties**

Language status influences social development and subsequent behavioral difficulties. The Vineland Scale reports show a lower social quotient among deaf children. The impact of deafness on child development can be readily observed in infants' orienting behaviors. Hearing children can hear their mother coo to them and orient toward their direction. They learn that crying attracts their mother's attention even out of sight. This experience is not available to the deaf child. Teaching social rules and concepts or right and wrong cannot be done easily by explanations, and the child often perceives limit setting as capricious. The child's developing theory of mind, as an interconnected network of beliefs, desires, and feelings influencing behavior, can be helpful in understanding social emotional and cognitive development. [Peterson and Siegal \(1995\)](#) have developed a theory of mind from studies of prelingual signing deaf children. The influence of deafness on development can be understood in light of various theories of development, for example, Eriksonian developmental stages, Bowlby's attachment theories, and so on. [Vaccari and Marschack \(1997\)](#) have explored the role of effective parent-child communication in the social and emotional development of deaf children.

The prevalence of reported emotional and behavioral problems among deaf children is three times that of the general pediatric population. In summarizing various reports, [Meadow \(1980\)](#) finds deaf children to be described as hyperactive, immature, and impulsive. There is a general impression that a typical "deaf personality" exists. Personality features usually included are characteristics such as impulsivity, hyperactivity, rigidity, suspiciousness, and immaturity. Following other investigators who have challenged these suppositions, [Chess and Fernandez \(1980\)](#) used a sample of 248 children with congenital rubella followed longitudinally from age 2.5 to 14 years. They concluded that deafness does not confer "typical personality"; it is the behavioral symptomatology of those with neurologic damage that is largely responsible for such a stereotype in the rubella-deaf. Application of stress and coping models in studying adjustment indicate that social support is predictive of maternal adjustment and that maternal problem-solving skills are significant predictors of child adjustment ([Calderon, 1999](#)).

Adolescent development poses different difficulties because information comes primarily from peers and the environment outside of the home, at the mall, pizza parlor, or street. By this age, signers have a social network within the deaf community, obtaining information not accessed from hearing parents or other adults. [Charlson and associates \(1992\)](#) have reported an in-depth evaluation of successful deaf teenagers. All of their subjects reported some degree of isolation from peers or family that was associated with communication difficulties. Their sample included deaf teenagers with deaf parents, and residential schools. Most of the students had developed a strategy for coping with the isolation, further raising the impact of parent's signing skill on adolescent social development, highlighting deafness as a significant variable in the task of developing sense of autonomy and competence. In a study of hearing-impaired youths and their hearing parents, parental ratings of adolescent behavior has been linked with parental symptomatology and low family adaptability ([Watson, 1990](#)). Gathering social information is particularly difficult for oral adolescents in a mainstream program.

### **Psychiatric Evaluation of a Hearing-Impaired Child or Adolescent**

The evaluation, treatment, and management of deaf children with psychiatric disorder is intimately related to the child's communication ([Roberts and Hindley, 1999](#)). Beyond the general techniques of child psychiatry interviewing, the examiner should focus on the preferred mode of communication for the child and be prepared to function within that modality. Assessments should include the parent's ability to communicate in the child's preferred mode, the impact of the parent-child communication and alternative, nonverbal interactive strategies of parents and their deaf children ([Vernon and Alles, 1986](#)). For preverbal and nonverbal children, a behavioral analysis with detailed information from long-term caregivers helps to interpret the child's behavior. The oral child relies on lip-reading cues; hence, the examiner should provide a continuous unobstructed view. To communicate with a deaf child a gentle touch or waving the hand in the child's visual field is acceptable. The examiner should not sit with the sun or a bright light behind the back, creating shadows and eyestrain. If the child's preferred mode of communication is sign, a certified sign interpreter is required for an examiner with sign skills no greater than those of the child or adolescent. A family member is unacceptable, because the child is not free to communicate confidentially. The interpreter is best situated next to the examiner, allowing the child an unobstructed view of both examiner and interpreter.

The syntax and grammar of ASL, as with other languages, are completely different from those of English. American Sign Language is a distinct language, not a codified form of English. When ASL is translated word for word, the language form appears fragmented and disorganized, and has been confused with psychotic processes ([Evans and Elliott, 1987](#)). Formal language defects are often similar in sign language to those found among hearing populations, with changes in rate of production, echolalia, perseveration, and neologisms. The assessment of language and communication is fundamental to any psychiatric evaluation and particularly vulnerable to errors in assessing the hearing impaired. Even good speech readers understand a limited percentage of the mouth and lip formations without cues. Effective communication requires constant concentration, guesses, adequate vocabulary level, quality training, and experience. The common use of idioms and slang in verbal conversations can present problems when working with oral (speech-reading dependent) youngsters. For example, an oral deaf adolescent boy considered to have excellent speech-reading skills responded with an inappropriate response to the quotation, "when push comes to shove." After a prolonged effort to understand and explore the bizarre response, it appeared that the youngster had speech read, "put it on the shelf" and that his response was congruent to that reading. Proverbs that are used in psychiatric examination for the hearing are misleading. Many proverbs have no meaning to the deaf, such as, "The squeaky wheel gets all the grease." Many words and phrases routinely used in mental health settings adapted from physical medicine assume a mental health, context-dependent meaning. For example, "How are you feeling?" has elicited the response, "Nothing" from a crying deaf adolescent because she had no physical pain. A common vocabulary is important with children and adolescents; and a determination whether they know the sign "emotion" and its connotations is critical. The examiner might list for the child the signs of happy, sad, and the like, interspersed with some simple nouns to find if the patient can distinguish which are emotions. Deaf individuals have hallucinations, look into space, and even sign into space toward the source, but it is not helpful to pursue the question, "Are you hearing voices?" Some may report that people or sources such as loudspeakers are talking about them or commenting specifically to or about them. This must be distinguished from prevalent beliefs often true within deaf culture that hearing people talk about them behind their backs. Schneiderian first-rank symptoms can be elicited, but ideas of reference must be teased away from cultural belief systems and experience. The psychopathology of paranoia poses similar problems. Drawings can be helpful in finding psychodynamic material. The Goodenough Draw-a-Person Test can provide an estimated intelligence level. Squilly drawings or Family Kinetic Drawings are helpful in gathering projective material and exploring relationships. It is best to interpret the drawings based on a story obtained about the drawing. Questions about what is happening, what the subject of the drawing is thinking, feeling, and hopes for the future prove richer than the usual theoretical interpretive formulations of symbolic meanings. Psychometrically sound functional scales specific to this population may further clarify symptoms and adaptive functioning.

### **Differential Diagnosis**

Mental retardation needs to be ruled out in a deaf child or an adolescent with problem behavior. Pervasive developmental disorder, autism, childhood schizophrenia, attention deficit hyperactivity disorder (ADHD), depression, conduct disorder, and parent-child conflict need careful consideration. ADHD is a common school problem not fully examined in the deaf population. Delays in academics or simple knowledge about the world may present as immaturity, dependency, or educational delay. Deaf children are accustomed to routine sequences of actions because their parents and mentors find it difficult to explain changes. They themselves may later insist on sameness, leading to a misdiagnosis of childhood schizophrenia, autism, or obsessive-compulsive personality disorder. In a deaf child, alternative causes must be considered for all these diagnoses. Differential diagnosis of behavior disturbances among sensory-impaired children requires the clinician to consider unusual presentations of conditions that exist in nonimpaired children. Tinnitus that occurs among hearing-impaired children may be mistakenly diagnosed as hallucinations. The assessment of multiply disabled deaf children requires a large team of highly specialized evaluators. The position paper on recommended assessment procedures for these youngsters adopted by the Conference of Educational Administrators Serving the Deaf provides a useful and comprehensive listing of all the areas that need to be considered. They consider deafness to be the primary disability. It is not realistically possible to go beyond that position when generating



information ([Johnson, 1989](#)) because of the lack of theoretical knowledge about the interactions of the differing impairments. IDEA and PL99-457 that establishes Early Intervention for children 0 through 3 with developmental delays and handicapping conditions identify areas that need consideration. In evaluating a deaf multiply impaired child, the psychiatrist is cautioned from automatically placing sensory impairments as the most important in the hierarchy. This could inadvertently underestimate other impairing conditions such as mental retardation, psychosis, central nervous system syndromes, and so on, which might more accurately dictate functional capacity, treatment, and prognosis.

## Treatment and Management

Deaf professionals have a vital role in providing mental health services to deaf children and adolescents. Intervention strategies utilizing competency-based approaches always should be considered. Offering services to support parents, enhance their coping skills, and develop their own support system is an integral part of providing services to this population. A developmental approach in providing services requires recognizing the parents' and children's varying needs at different ages and family stages. Management of the different developmental trajectories and unevenness in various developmental domains needs a wider array of viable modalities of communication, such as sign, play, art, movement, and so on. Prioritizing treatment and subsequent management is like that for other children presenting with psychiatric disorders. Effective communication, both prelanguage and language-based, is a high priority for primary prevention of emotional-behavior disorders. The selection of total communication or oral training depends on parental ability to sustain a demanding communication therapy program (either for oral training or to learn sign language), along with the child's intelligence, temperament, and associated impairments. The authors' general recommendation is a total communication approach but decisions should be made on a case-by-case basis. It also may be necessary to accomplish this over time and in stages. As child psychiatrists, we place a high priority on social/emotional development, psychosocial group identification and support, and a sense of self-identity, which is the core of deaf culture based on sign language. Many of the referrals are orally communicating older children, where problems have already developed. Referrals of the manual communicators come from dysfunctional families, particularly those with multiply handicapped children. We tend to see unsuccessful youngsters and their families; it is unusual to be called into a case where the impairment is newly discovered. There is a tremendous heterogeneity among hearing-impaired children, and the clinician is best advised to remain flexible in recommendations. Initial oral training is important, both prior to and after surgery, for successful use of cochlear implantation. Any 2- or 3-year-old child in a purely oral program not making prompt and significant progress should be transferred to a total communication program. Delays beyond the fourth birthday leave a child ill-prepared for school; the absence of a practical communication mode may be the largest contributor to the problem, should the child be brought for treatment of a behavior problem at a later age. The Rubella Longitudinal Study found that mainstreaming with oral education is helpful, although not mandatory if postsecondary education is used as a criterion of success. In addition to addressing the long-term consequences of the impairment, unrealistic parental expectations, and a wish to deny the impairment and diminish its impact should be discussed.

Treatment focuses on the acquisition of acceptable behaviors for the developmental period, if the behavior problem is not owing to independent psychiatric conditions. Behavior modification has been successful with deaf children, as group psychotherapy has been with deaf adolescents. Group psychotherapy provides an avenue to share feelings and behavioral strategies, particularly around maturational and adjustment issues. Studies proving efficacy of individual psychotherapy in the language used by the child are limited only by the availability of therapists. Clinical reports on the value of psychotherapy tend to be single-case studies or highly selected samples. Hearing therapists are most successful if they use a transcultural model and acquaint themselves with the cultural beliefs of their clients. As in psychotherapy of the nonimpaired, the therapist is advised to convey, "Teach me what your experience is like" rather than, "I know how you feel."

Evaluating psychopharmacologic agents and monitoring their desired and adverse effects is similar to hearing children with the same symptomatology or disorder, but with a warning. Psychoactive medications, especially neuroleptics, can be used successfully with deaf children for management, but the common anticholinergic side effects of psychoactive medications must be made clear, although they are difficult to communicate in sign language. The blurry vision often experienced with these drugs is intolerable for an individual dependent on vision and limits the usefulness of such medications. Stimulant medications can be used effectively for ADHD in carefully diagnosed cases. Hyperactivity in hearing-impaired children may be confused with a reaction to poor communication or learning social cues and appropriately responding to them. The determination is complicated by reported impulsivity among deaf children. Cases referred for stimulant medications have identified overwhelmed parents lacking communication skills, having trouble setting limits, and overusing physical punishment to establish their authority. [Kelly and colleagues \(1993a\)](#) evaluated a state residential school for the deaf and obtained ratings on 238 students using the Conners Parent Rating Scale and the Attention Deficit Disorder with Hyperactivity (ADD-H) Comprehensive Teacher Rating Scale. They found that the Conners ratings statistically differed from normative numbers for hearing children. Among those youngsters who were considered positive for ADD-H, hearing loss tended to be acquired (e.g., bacterial meningitis or congenital rubella). A wide range of management interventions, including stimulant medications, also was reported ([Kelly et al., 1993b](#)). The decision to use stimulant medication should consider that the chronic nature of the deafness results in lifetime use of medications to maintain the child in the least restrictive environment. This dilemma can be addressed only on a cautious, case-by-case basis with careful monitoring and periodic assessments. Repeated failures at nonpsychopharmacologic intervention warrant a trial of medication. Treatment goals and a time frame for the medication trial, and periodic review of the medications should include a risk-benefit analysis of optimum medication dosages.

Treatment and management includes counseling and supportive measures for the family. Emotional reactions of parents to a deaf child can interfere and/or limit their capacity to respond to their child's needs. An impaired child's temperament and the concept of goodness of fit should be considered ([Chess et al., 1980](#)). Many parents reexperience painful emotional feelings and new sets of problems with each developmental transition. Successful adaptation to the complex and ever-changing challenges of parenting a deaf child can be integrated in bereavement models. [Kampfe \(1989\)](#) adapted the House Social Stress Model for transitions into her work with parents whose child has recently been diagnosed as deaf. Conditioned variables of the parents include social status, experiences, personal resources, social support, which interact with the various responses, influencing the central core of transition to adaptation and the parent's perceptions of deafness. She has found working with these perceptions a helpful supplement to the more simplistic mourning and grief structure ([Kampfe, 1989](#)). Siblings are a sadly neglected group requiring intervention and education. Family therapy with a hearing family and deaf child should use interpreters, even if the therapist can sign ([Harvey, 1982](#)). Counseling for both the parents and siblings include teaching technical information in terms that can be understood clearly. The importance of genetic counseling services for both parents of deaf children and deaf adults cannot be overlooked ([Arnos, 1992](#)).

The first experimental cochlear implants on adults were performed in the early 1960s and clinical trials began in 1973. In 1984, the Food and Drug Administration (FDA) approved the devices in adults. The first implant trial on children was initiated in 1980 at the House Ear Institute in Los Angeles, California. By 1990, over 500 children, ranging in age from 2 to 17 years, had received the device when the FDA gave approval for implantation in children. The Subcommittee on Cochlear Implants of the American Academy of Otolaryngology makes clear that pediatric candidate selection requires more than a failure to benefit from a full trial of appropriately fitted hearing aids and other devices. Medical history must be considered in addition to psychosocial factors. The psychosocial factors for successful selection include the absence of severe organic brain damage, psychosis, mental retardation, personality traits that may interfere with training, and/or unrealistic expectations. Parent-child interaction that permits follow-up rehabilitation is a requirement for long-term speech therapy training following surgery, and frequent readjustment of the device ([Kveton et al., 1991](#)). [Kampfe and associates \(1993\)](#) review the literature on the impact of parental expectations as a contributing factor to successful procedures and suggest that parents in early stages of either shock or denial in regard to their child's deafness are not in a position to manage successful cochlear implantation. Cochlear implantation requires active long-term participation for parents and children. The experience with adult recipients of the device is more extensive, whereas the experience with children is growing. The Cooperative Veterans Implant Study showed that all recipients were able to hear sound with their implants but there was a varying degree of effective meaningful auditory perceptions, although being able to hear warning sounds (e.g., horns) is considered beneficial. Of all postlingually deafened with a working knowledge of spoken language, only 61% showed improvement in "open-set" speech recognition, that is, where the listener cannot be aided by speech reading ([Cohen et al., 1993](#)).

Cochlear implants hold out the possibility of enabling profoundly prelingually deaf children to hear. The decision to use this prosthesis is neither easy nor obvious ([Crouch, 1997](#)). [Waltzman and associates \(1992\)](#) have reported success in implanting multichannel implants in prelingually deaf children. Fourteen prelingually profoundly deaf children (2.6 to 5.1 years old) were given implants following intensive screening evaluations. The children had varying means of communication, including sign, oral, and total communication, as well as no demonstrable means of communication. Follow-up data on a variety of testing instruments showed benefit in auditory reception and the acquisition of speech and language expressive skills. Children receiving only sign language training prior to implantation did not progress as rapidly as the oral youngsters, but all of the youngsters continue to require rehabilitation services. The ultimate level of open-set auditory receptive and expressive communication these youngsters will achieve is unclear at this time. Informal observations from clinicians suggest that the youngsters are also expanding their social interactive potential and the initial reports are encouraging. Cochlear implants are not a "bionic" ear or cure for profound hearing impairments. The decision to choose hearing aids, communication devices, or medical technologies for their child can be stressful for parents. Controversies on the ethics of using medical intervention through cochlear implantation on the deaf culture, and thereby altering the relationship of social conditions, adds a moral dimension to this dilemma ([Enerstvedt, 1999](#)). The concept that deafness is not a disability requiring intervention is a position held in deaf culture ([Tucker, 1997, 1998](#)). The opposition to the use of medical technologies and intervention may distract from the purpose of evaluation. The timing and trials with hearing aids and assistive devices requires thorough evaluation and training ([Chute, 1997](#)). The benefit of cochlear implants is clear in patients who had acquired speech and language before hearing loss or who had a shorter duration of deafness. The gradual and steady improvements in speech production and perception that have occurred in prelingually deaf children are promising ([Loizou, 1999](#)). In a study of 23 prelingually, profoundly deaf children with cochlear implants, there was no significant difference between the language



performance of children who use oral versus total communication ([Robbins et al., 1999](#)). Studies show that bringing up deaf children without sign language results in deficient ability to communicate, whether they have an implant or not ([Enerstvedt, 1999](#)). In this context a decision to withhold sign is tantamount to denying language.

## Outcome and Follow-up Data

The human havoc consequent to the worldwide epidemic of congenital rubella of 1964 to 1965 is important to record, not only for its contribution to sensory and other infant impairment, but also because repeated epidemics should be anticipated owing to a large pool of unvaccinated young women and children, the ease of worldwide travel, and constantly evolving new viral infections with possible teratogenic effects. Prospective studies provide the most reliable data. In 1967, Stella Chess initiated the Congenital Rubella Behavioral Study with a cohort of 243 children whose physical status was under study at Bellevue Hospital in New York City. Initially evaluated at ages 2.5 to 5.5 years, follow-up studies were done at ages 8 to 9 and 13 to 15 by [Chess and colleagues \(1971\)](#) and at age 22 by [Kasen and colleagues \(1990\)](#). The major impairments were hearing loss (177, 72%), with 61 severely to profoundly deaf. Visual loss, mainly caused by congenital cataracts, cardiac anomalies, and neurologic impairments, also were present, often as multiple events. Fifty children with congenital rubella diagnosed on the basis of successful culture of the rubella virus were free of any deficit. These mothers' infections had been in the third trimester of pregnancy, and the children became the control population of the study.

There was an inverse correlation between the number of impairments and behavioral normalcy both on initial evaluation and into adolescence. The intelligence quotients of the rubella deaf children ( $N = 177$ ) ranged from superior to mentally retarded ( $N = 30$ ). The prevalence of autism ( $N = 18$ ) was equivalent to 741 per 10,000, several hundred-fold greater than expected. This study was ongoing at a time of great and contentious argument over the legitimacy of sign language, when oral training and lip reading were dominant. By adolescence, only a minority of the severely and profoundly deaf had been profitably mainstreamed. When evaluated at young adulthood, Kasen and Cohen ( $N = 46$ ) found that although better employment status characterized the mainstreamed group ( $N = 24$ ), the majority nevertheless felt more comfortable in the deaf community. The New York Longitudinal Rubella Study covered 22 years, during which time public attitudes and awareness concerning the special needs of impaired children and their families changed dramatically. Future research in the area of language education or English as a second language might result in different conclusions on optimal educational methods for hearing-impaired children. The complexities of culture, socioeconomic status, parental education and expectations for children, educational issues, the heterogeneity of hearing capacity and associated other impairments present daunting research challenges. This complexity and heterogeneity of the population attest to the inadequacies of simplistic approaches, based on a unitary theoretical formulation, or dogmatism, as to the (correct) communication modality for hearing-impaired children.

Data are sparse on employment of deaf individuals. An important report by [Vernon and LaFalce-Landers \(1993\)](#) presents discouraging data. Forty-seven deaf and hard of hearing individuals for whom Vernon had found a gifted IQ score (130 to 150) were followed up after a period ranging from several months to 36 years. Most had attended or graduated from college or graduate schools for the deaf or were mainstreamed, receiving liberal arts or technical/vocational training, but there was only a 52% graduation rate. The dropout rate resulted in large part from the lack of sufficient deaf support services in colleges. At the time of follow-up, one-third were in professional or supervisory work, almost all within the deaf community, whereas 30% were unemployed. The rest were underemployed with respect to their training. Forty percent had received psychiatric therapy for emotional problems or mental illness and 9% had been hospitalized.

## Research Directions

Research into sign language as a true idiom has already been done. The close relationship between gesturing and speaking in the human brain has to be pursued. The efficacy of alternative therapies in the outcome of sensory impairment is a major research area. Spirituality and its contribution to resiliency and adjustment also require serious attention. The pros and cons of the use of Signed Exact English (SEE) or ASL in schools need further examination, although most deaf children learn to read and write English as a second language. Research to improve language, reading skills, and overall literacy is most urgent.

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### CASE ILLUSTRATION

Brian, a 13-year-old hard of hearing boy, lives in a group home for deaf and hard of hearing adolescents. Diagnosed with attention deficit hyperactivity disorder (ADHD) and conduct disorder, he has never been able to communicate effectively with oral language and relies heavily on American Sign language (ASL). Brian has consistently tested in the mildly deficient range on performance tests of the Wechsler Intelligence Scale for Children, revised (WISC-R), which is generally surprising to those who know him well, because of his apparent strengths in social skills and ability to mask his deficits. His current treatment plan includes methylphenidate for his ADHD symptoms, individual therapy, and an integrated behavior modification program in the group home and school. Brian's therapist advises staff in the group home and school and works directly with Brian using cognitive and supportive techniques to help Brian better understand the consequences of his behavior and achieve appropriate social behavior. Target behaviors have included disrespect toward authority figures, lying, refusal to do schoolwork, physical aggression, inappropriate sexual touching of others, lack of remorse or empathy for others, and blaming others for some of his problems.

On one occasion, Brian presented unusual symptoms during a routine visit to his psychiatrist. The interpreter during this visit was primarily fluent in Signed Exact English (SEE) not ASL, and was not familiar with Brian's use of language. During the previous week, group home staff described Brian as more agitated and impulsive than usual. Brian tried to explain his behavior to his psychiatrist, stating that peers were picking on him, that they "preferred the devil," commenting that, "The devil is picking on me." He preferred time alone, away from his peers. Brian reported the voice of a popular singer in his head when asked about possible auditory hallucinations. He also complained of a "machine" in his stomach. Alarmed that Brian might be developing psychotic symptoms, paranoid ideation, auditory hallucinations, and somatic delusions, the psychiatrist discontinued methylphenidate and considered treatment with an antipsychotic after a period of observation. Brian was seen a week later with an interpreter who was fluent in ASL and familiar with Brian's language and the structure and activities of the home. The interpreter learned that several teenagers in the group home, including Brian, had been attending religious education classes and had begun to express bad behavior as being an influence of the devil. Brian, who particularly lacks significant abstract reasoning skills, had meant that he was misbehaving when he said the devil was picking on him. Saying that his peers preferred the devil meant that they were misbehaving. With this interpreter, Brian clarified that he had been listening to music with headphones and had heard the voice of a popular singer in his head. Finally, Brian had been complaining the previous visit of feeling "weak" in his stomach, not that there was a "machine" in his stomach. The ASL signs for weak and machine are similar. Thus, all of the suspected psychotic symptoms were the result of communication problems! In this case, the communication problems were exacerbated by Brian's cognitive and language limitations, which included idiosyncratic signs, as well as an interpreter who was not proficient in ASL, Brian's language of choice.

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## VISUAL IMPAIRMENT

The general considerations and role of the child and adolescent psychiatrist in the beginning of the chapter applies to visual impairment as well.

### Definitions and Historical Note

Blindness was legally defined by the American Medical Association in 1934, for the purpose of determining eligibility for federal insurance programs. This definition as central vision of 20/200 or less in the better eye with corrective glasses, and a visual field subtending an angular field of no greater than 20 degrees in the better eye has endured and is used in PL94-142, 1975. There are alternative definitions that focus on functional capacities, rehabilitative potential, and the nature and quality of the residual vision on educational perspectives; however, there is no consensus about which criteria to use and how to measure them, and the legal definition varies worldwide. The term visual impairment includes both partial sight and blindness. Partial sight is defined as visual acuity above 20/200 but worse than 20/70 in the better eye with correction. Approximately 20% of legally blind children receive no visual input and are totally blind, whereas the remaining 80% have some form of visual stimulus of tremendously varying quality and usefulness. A child who is legally blind may have considerable vision, able to read large-type materials and learn by the efficient use of residual vision. There are differences in the prevalence of blindness among the developed and developing countries, where blindness secondary to infectious processes and malnutrition continue to be epidemic. The World Health Organization (WHO) defines blindness as visual acuity of less than 3/60 (0/05), corresponding to loss of walk-about vision. The WHO definition of low vision as visual acuity of less than 6/18 (0.03) overlaps with the legal definition of blindness in the United States ([Lawrence et al., 1992](#)). Worldwide, there is increased interest in low vision, generally associated with the aging process. The nature of the visual pathology and quality of vision can result in varying perceptions and is often dependent on lighting conditions. The legal definition is essentially a distance definition, and although there is usually a correlation between near and far vision, this is not consistent. Attempts at a functional definition usually involve a reading task and are most helpful for educational purposes ([Scholl, 1986](#)).

### Prevalence and Epidemiology

The collection of statistics on incidence and etiology of visual impairments is vulnerable to the definitions of blindness. Unlike some other countries, the United States does not keep a national register of blind or visually impaired people. There is no one definitive study that includes all the relevant information owing to confusion in the field about which definition and therefore which statistics to use. The National Center for Health Statistics (NCHS) stated in 1990 that there are 95,410 "severely visually impaired" children between the ages of 0 and 17 years old in the United States. The definition of severely visually impaired differs from that of legal blindness and is not based on visual acuity but perceived vision problems generally based on reading. This number does not include institutionalized individuals ([Nelson and Dimitrova, 1993](#)). The 1992 survey of the American Printing House for the Blind reported 51,813 students who were registered as visually impaired. Of that group, 27% were classified as visual readers, 9% were in residential schools, and 4% were in programs for multihandicapped individuals ([American Printing House for the Blind, 1992](#)). [Kirchner and Schneider \(1997\)](#), in a United States demographic update, pulled together items from various perspectives. The prevalence of people 15 years and over, who have a functional to severe functional limitation, defined as those who cannot see words and letters in ordinary newsprint, is 9.7 million with a prevalence rate of four per 100. By clinical measures, the estimated prevalence of the legally blind population of the United States is 1.1 million, with a prevalence rate of 4.5 per 1,000. The estimate for the prevalence of total blindness with light perception or less is 220,000, which was calculated from 20% of the legally blind. Of



that, half or 110,000 are estimated to have no light perception at all.

## Etiology

Etiologies of visual impairments have varied through time, reflecting changing medical ability to treat infections. Vision loss in childhood caused by congenital, genetic, or prenatal congenital conditions represents approximately 50% of cases. Optic atrophy is a common diagnosis among the severely mentally retarded with visual loss. Lymphocytic choriomeningitis causing chorioretinitis *in utero* is believed to be responsible for some (Mets, 1999). Other causes include optic atrophy, retinitis pigmentosa, optic nerve hypoplasia, cataracts, foveal hypoplasia, persistent hypoplastic vitreous, and microphthalmos. Uveitis potentially leading to blindness can develop in children with juvenile rheumatoid arthritis (Foster and Nguyen, 1998). The improved management of premature infants has reduced the incidence of retinopathy of prematurity (ROP), previously referred to as retrolental fibroplasia. There is a higher incidence of handicapping conditions and blindness among lower socioeconomic and very low birth weight children. Cortical visual impairment caused by a disturbance of the posterior visual pathways in the occipital lobe is most commonly caused by perinatal hypoxia, cerebral vascular accident, meningitis, and acquired hypoxia. Most children with cortical visual impairment have other associated neurologic abnormalities (Hero et al., 1999).

## Clinical Description and Developmental Approach

Visually impaired children suffer the full range of clinical psychiatric syndromes. Developmental delays and maladaptive, troublesome behavior among visually impaired children result from failure to learn adaptive skills, or to correct maladaptive behaviors reflective of gaps in the teaching of social norms and behaviors. Commonly observed behaviors in blind children, such as stereotypic rocking, passivity, and immobility, do not signify psychiatric illness, nor is late achievement of motor milestones reflective of cognitive problems. Such behaviors may be owing to the visually impaired child's reliance on caregivers for stimuli and insufficient environmental response. Substantial developmental data on visually impaired children are accumulating with the establishment of early intervention programs. Differences are emerging between children with visual impairment alone and those with multiple handicaps, in the acquisition of sequential motor milestones between the visually impaired and nonimpaired groups. Vision provides the infant and young child with stimulation and observation to permit imitation and modeling, and to encourage exploration necessary for further development. This includes all domains of motor, cognitive, social, and language skills (O'Donnell and Livingston, 1991); the blind child is at a particular disadvantage with imitative learning.

## Conceptual Approach to the Evaluation

The psychiatric evaluation of the troubled, visually impaired child is aided by the observation that blindness affects the cognitive functions of children, the range and variety of their experience, their ability to get about, and their control of their environment and self in relation to it (Lowenfeld, 1981). Developmental assessment of a blind child needs to be integrated with personality development. The effect of visual impairment in the range and variety of experiences is a major consideration and a potential psychiatric pitfall. A blind hearing child can locate a bird by its sound but not describe its shape, size, or flight. Although the tactile sense can describe concrete objects that can be held in the hand, it does not help to describe clouds, stars, or colors, nor can the child experience a very large or small object. This affects cognitive developmental processes. In comparing 40 congenitally blind and 40 sighted children for fantasy-reality distinctions between real and imagined objects and their development of certain abstract concepts, Ittyerah and Goyal (1997) found that blind children's knowledge that contents of fantasy are not real may be obtained through interpersonal relationships. Utilizing sensory information is important in forming ideas and developing concepts of space, size, weight, and object classification. Language development for the blind and the sighted infant are essentially equivalent through the babbling stages but can be stunted without visual stimuli. The visually impaired child must rely on verbal descriptions provided by the sighted. Iverson and Goldin-Meadow (1997) compared sighted and congenitally blind children and found blind children producing gestures resembling those produced by the sighted. Both groups used similar gestures in explaining particular processes, suggesting that gestures serve a communicative function for the speaker, and that the processes of gesturing and speaking are connected. However, without the experience of knowing through sight, verbalized descriptions repeated by the blind child can seem shallow or meaningless and are referred to as "verbalisms." Because development of vocabulary and the fund of general information can lag, intelligence testing results, especially the verbal portions of the WISC-III, should be interpreted cautiously. It may be more informative to look for problem-solving strategies than to compute the IQ score. Groenvelde and Jan (1992) analyzed the subtests of the WISC-R and the Wechsler Preschool and Primary Scale of Intelligence (WIPPSI) and were able to distinguish whether errors are the result of vision, understanding sequential relationships, or the interpretation of spatial relationships.

The early motor developmental milestones of the blind infant are similar to those of the sighted child until the age of 4 months (Warren, 1984). After this period, the lack of visual stimulation as motivator for the child to explore and interact with the environment can lead to delays in crawling, walking, reaching, grasping, and bilateral hand coordination. The blind child tends to lag behind the sighted in motor tasks where visual stimulation is a motivator, whereas there is little difference when the task is purely neuromuscular (e.g., sitting or standing alone). Prolonged period of immobility and limited environmental stimulation for the blind infant can result in poor development, especially in the areas of initiative and self-confidence.

Lowenfeld's observation about control over immediate environment and the self in relation to it has a subtle but profound influence on the child's development. In the sighted child, seeing overcomes distances and provides a quick way to evaluate new or changing situations or emergencies. Vision provides the means for observing the behavior of others, especially parents or peers, and learning by imitation while obtaining immediate feedback through observing facial expressions or other nonverbal cues. Early face recognition and social smile by the infant along with eye-to-eye contact are important both to the infant and caretakers in establishing and developing affective bonds. The blind infant's muted response to the caretaker's approach can discourage parental affective response and diminish reciprocity of nonverbal communication. Nonverbal social cues and body language are not available to the visually impaired child, complicating the development of peer and social relationships. The socially isolated child who is only around adults has no way to learn age-appropriate social behaviors. An interesting study of self-esteem, body size, and parental views in 9- to 11-year-old blind children discovered positive views about self-presentation. Low self-esteem was found in children who were thought too thin by their parents, but being appraised as fat or believing their parents thought of them as fat, had no effect on self-esteem (Pierce and Wardle, 1996).

Mainstreamed blind children tend to be more socially mature than visually impaired children in a residential setting, but they usually have more residual vision and thus greater potential to observe behaviors. Despite trends toward mainstreaming of children, most schools lack teachers with the necessary training to meet the children's needs. Special schools provide necessary supports for attaining skills that complement those learned in mainstream schools. The developmental tasks of adolescence are particularly vulnerable to troubled behaviors. Visually impaired adolescents cannot tailor their clothing or appearance to conform to their peer group and may worry about how acceptable they look, nor can they compare a peer's body language with verbal statements to pick up important cues. The concern among the blind that the sighted are looking at them critically may lead to self-consciousness. Blindness and serious visual impairment have considerable economic implications, leading to loss of productivity and social dependence.

## Personality Development and Psychiatric Issues

There is no typical blind personality and personality research comparing the visually impaired and the sighted have resulted in inconsistent findings (Warren, 1984). The Vancouver study of 86 visually impaired children reports that 18.6% of these children were mentally retarded, 3.5% psychotic, and 5.8% suffered organic brain syndromes (Jan et al., 1977). Although some children carried multiple diagnoses, 43% of the children were considered to be free of psychiatric diagnoses, and the remainder had a range of difficulties, including adjustment reactions and neurotic and behavior disorders. The report makes note of several significant points for personality development. The totally blind children in the study had averaged 50 days in the hospital during the first year of life, as opposed to 20 days for the partially sighted and only 2 days for the unimpaired controls. In incubator infants, breast-feeding was markedly reduced and the parents showed increased worries about their child's health status in comparison with the control group parents. Difficulties with clinging and separation problems, prolonged bottle-feeding, and chewing problems associated with delay in offering solid foods and self-feeding were noted later. Activity levels varied and there was increased use of pacifiers. Toilet training was essentially on the same time schedule as for the control children. Of the visually impaired children, 9% had a major problem with fears, but 28% were reported not appropriately fearful. A review of the literature regarding fears noted that the average number of fears among the children was high and differed in content from reports among sighted children. The blind children's most common fear was that someone they loved would be hurt, followed by fears of physical danger and harmful situations, whereas among sighted children there tends to be a higher percentage of fears about being hurt psychologically. "Severe" fears reported by the school counselors included getting a shock from electricity; getting lost; and fear of snakes, fire, and guns. The children also reported fears of falling from high places, strangers, walking alone in the night, dying, bombing attacks, and getting or losing a boyfriend or girlfriend (Wiemer and Kratochwill, 1991).

Visually impaired children are reported to have restrictive, unimaginative play patterns. Findings from the Vancouver study suggest that play might serve a different function for blind children. The inability to imitate inspired reluctance on the part of caretakers to introduce a wide variety of toys to the child, hence, social skills are hampered owing to the visually impaired child's paucity of interactive play experience. Adults' interventions to promote social contacts may focus the child's attention toward the adult and not the playmate. In adolescence, dating arranged by sighted friends or adults reduces privacy and inhibits acquisition of social skills. Despite this, many visually impaired children and adolescents are skillful at interpersonal manipulation. Lowenfeld (1981) describes four major concerns that present difficulty

to the visually impaired adolescent: sexual curiosity, dating, mobility, and concern for the future. The inability to gather sexual information from observation or magazines might compel the sexually curious adolescent to behave in a manner that would violate cultural and social taboos. Dating is an important arena in which one develops self-confidence and asserts a gender role, but ability to date is dependent on meeting another person and engaging in rituals of flirtation and personal appearance. The visually impaired must rely on introductions and arrangements that diminish flexibility and self-confidence while invading privacy. Limited mobility reduces opportunities for the separation and individuation process to occur, which further limits autonomy. Concerns for the future that involve leaving a protective family or school setting, accepting vocational challenges, ensuring economic stability, worrying about marriage, and the genetic transmission of the visual impairments pose significant hurdles.

### Psychiatric Evaluation of a Visually Impaired Child or Adolescent

As a general principle, the child and adolescent psychiatrist is cautioned against using a “deficit model,” assuming that damage is inevitable with a perceptual impairment, or at best only partially compensated, and always results in personality and/or psychiatric disorder. The examiner should be aware of myths about the blind such as innate helplessness and total dependence, blindness as a punishment for sins, etiologic association with venereal disease, and belief of hyperacuity of other senses. Prior to the interview, the examiner should have knowledge of the child’s medical history, degree of independence and cognitive level, events in the home, school adjustment, purpose of the referral, and the child’s ability to modify behavior. The age of onset of blindness is a major consideration.

The child psychiatrist evaluating a visually impaired child must be prepared to touch the child more than he or she would touch sighted children, asking for the child’s permission when appropriate. Talking softly to orient the child toward the examiner before even touching or approaching the child helps sets the tone for the evaluation. The child should be led to the examining room, and the examiner may initially remain in continuous physical contact by holding his or her hand or touching the shoulder. The interview space, its layout—including where the child sits and all of the toys available to the child—should be described, so that valid conclusions can be drawn about activity selection. The child will often ask seemingly impertinent personal questions of the examiner, including appearance and marital status, some of which a sighted child or adolescent determines by observation. Whether answered or not, a friendly tone is essential. It is important to note that with sensory impaired children, and more so with the blind and multiply handicapped, any interaction presents an opportunity to teach and engage the child.

The vast majority of legally blind children are better described as low vision, thus lighting in the examining room is important. There are no fixed rules, but the examiner must be alert to the unique nature of the child’s vision and maximize the residual capacities. Glare, background light and shadows, along with placement of objects in the visual field are considerations. A gooseneck lamp on the table enables the child to maximize vision while playing or drawing. Creating nonglare contrast can be helpful to increase visual perceptions. If drawing tasks are to be part of the evaluation, then the table surface should be free of glare. A dull light blue-gray table surface works well, contrasting both white and dark drawing paper or covering a white table with brown wrapping paper is a satisfactory solution. If white paper is used for drawings, then the standard pencil often does not provide sufficient contrast and a black felt-tipped pen or marker would provide better contrast. Some children prefer and respond better to dark drawing paper and a light-colored or white marker.

Play therapy can be used in the evaluation of blind children, but often the child must be taught how to play. The process of learning play through imitation often is not available to the blind child. The clinician can make some valuable observations about the child in the process of teaching a particular type of play. Blind children respond best to very realistic objects or toys. Play is better with utensils that the child routinely uses, such as real fork and spoons, rather than small toys, which have no meaning to the child. Similarly, dolls close to life size with molded realistic facial features and hair elicit more meaningful response as the child fingers the face than would the soft, more stylized, cloth dolls. Dollhouses with realistic figures of people and furniture can elicit helpful information about the home life of the child. The clinician might first have to teach the child how these small objects symbolize real people or furniture. In general the child plays with toys that are for a younger age group. It is best to start with real props to develop “pretend” imaginative play as part of therapy. The children pretend play verbally by telling stories as opposed to acting out behaviors. The clinician can elicit psychodynamic material and dreams in a verbal form, but the content may seem to be lacking in affect. This lack of apparent substance or affective content or “verbalism” seems more related to the language acquisition process than to the potential for affective content and the alert clinician can piece together the affective web that binds the meaning for the child. The child’s total body and other nonverbal responses including gestures as a communicative device need to be considered with the caveat that the child’s facial expression may reveal less than he or she understands.

The visually impaired child may be slower to warm up than the nonimpaired. Long silences should be avoided and the child should be notified of the examiner’s movements. Anticipated shifts of topic in the course of the evaluation should be announced so as not to confuse or contribute to disorientation. Echolalic language is relatively common and rarely pathologic. Self-stimulating behavior and stereotypic movements are frequently seen and not diagnostic of specific psychiatric conditions. The examiner should speak directly to the child and accept as an indication of relatedness that the child faces him or her with the ear and not the eyes. In an interesting protocol, [Raver-Lampman \(1990\)](#) asked 50 subjects who were informed about blindness and in contact with the blind, to evaluate videotapes of two children without gaze direction and two with gaze direction as they answered questions from examiners sitting on either side of them. The tapes were presented in a random order and opposite recording positions. The results revealed that, when a visually impaired child used gaze direction toward the questioner, the child was evaluated by the responding subjects as being more intelligent and socially competent than when the child did not use gaze direction. This serves as a caution to the examiner to be alert to his or her personal bias and unconscious judgments, especially involving the child’s body posture and direction of gaze during the evaluation.

It is helpful to indicate that several meetings with the child are normally required when accepting the referral. The clinician should initially be ready to teach the child tools necessary for a psychodynamic evaluation; for example, pretend play and drawing tasks. In effect, the assessment is a functional protocol looking at the child’s strengths, learning, and problem-solving styles, and what if any, interfering psychiatric processes are present.

### Differential Diagnosis

Differential diagnoses include mental retardation; pervasive developmental disorder; attention deficit disorder; childhood schizophrenia; and conduct and personality disorders, including immaturity, overdependency, and obsessiveness. Repetitive rocking, swaying, or eyeball pressing and other “blindisms” are also seen in other child psychiatric entities, especially mental retardation and autism. When these behaviors are found among blind children, they should be differentiated from a symptom of other conditions. There are conflicting research data as to the etiology of these behaviors among blind children. [Warren \(1984\)](#) summarized the data and concluded that currently these repetitive behaviors constitute compensatory self-stimulation for the child in the absence of visual stimulation. [Brown \(1997\)](#) compared a group of congenitally blind children with IQ over 70 to a matched group of sighted children, and found that the blind children exhibited more autistic-like features. A group of blind children with IQ below 70 and a matched group of autistic children shared many clinical features. [Brambring and Troster \(1992\)](#) looked at a stability of stereotyped behaviors in both infants and preschool blind children over 1 year using questionnaires to the parents. Children were divided into two groups, those under 24 months of age and those over. It is noted that the age of onset and frequency of the behaviors influenced the stability of the behaviors. More frequent behaviors and later age of onset increased the probability of the behaviors remaining in the child’s repertoire; however, the brief follow-up period was limited only to preschool age.

Blind youngsters may not acquire factual information or develop learning skills without educational opportunities. Early social interaction may have been muted for blind youngsters, and care should be taken in assessing diminished interpersonal affective responses as indicating clinical psychiatric conditions. Superficial symptoms suggesting autism in a blind child often abate when meaningful personal interaction is offered. For reasons of convenience and safety, many parents and caretakers tend to provide unchanging sequences of activity to their children. An insistence on sameness or a severe negative reaction to a change in patterns and activities may be a nonpathologic adaptation to visual impairment and past learning.

### Treatment

Early intervention programs with blind infants and preschoolers teach social skills and promote attainment of developmental milestones while supporting the family structure, thereby preventing psychiatric problem. Providing a means of communication and socialization is the first consideration for preventing most conflicts. The blind child learns to speak in the usual manner, but the meaning of words that the youngster cannot experience is difficult to convey and may cause frustration. Color and the contours of large objects or very small ones are most difficult to describe. Checking self-appearance for neatness and simple rules of social behavior, such as chewing with lips closed, cannot be learned by imitation and must be demonstrated. Alternate strategies of adaptation to an invisible, barely discernible or at best blurred world must be taught so as to avoid confrontation, tantrums, and shame. A psychiatrist treating a blind child or adolescent may be involved in consultation and serve as a liaison to provide advice on management to parents, family, and the school. The examiner’s familiarity with specific technologies to help the visually impaired may be useful in guiding the family and school to meet the child or adolescent’s developmental needs. Computerized magnification, Braille computer, and other literacy devices have been increasingly available to school age children. Current advances in Global Positioning System via satellites and its application to cane technology have helped increase safe mobility, autonomy, and self-esteem. These new technologies have alleviated concerns and reassured parents and facilitated skill development. Behavior modification has been useful for both deaf and blind children. Changing specific behavior in order to gain acceptance is successful for motivated children. Sharing experiences, feelings, and problems in group therapy and counseling can be very helpful. [Erin and coworkers \(1991b\)](#) have provided a



helpful review of literature regarding teaching social skills to the blind and visually impaired. They emphasize the value of social skills for a visually impaired child to be successful in mainstream programs. Studies strongly support success in teaching social skills using different approaches. There is the interesting question of whether the goal of social skills training should be to teach the blind behaviors of the sighted or rather than other skills more appropriate to the blind. Individual psychotherapy can be invaluable for a visually impaired child or adolescent. The therapist must constantly be alert to the possibility of confusion in the use of language. Although blind individuals cannot follow a description of color, they do use many vision-related idioms, such as "I see" for "I understand."

Medications can be employed with the same criteria as for nonhandicapped children and adolescents. Medications with the potential of interfering with residual vision should be avoided especially when some vision remains. Use of psychoactive medication among the visually impaired for symptom relief should be used cautiously. Stimulant medications can be used for attention deficit disorder, giving both child and caregiver an opportunity for better communication, education, and other interactions. Even more than in the nonperceptually impaired, psychopharmacologic intervention should be used judiciously as an adjunct in addition to other psychosocial interventions.

### Outcome Study

The Vancouver, British Columbia group led by Jan and Freeman studied a group of 92 legally blind children during 1973 to 1974. Freeman reevaluated the cohort in 1987 to 1988. The information from the first evaluation compared to follow-up resulted in varying outcomes, with many of the subjects defying negative predictions. On reevaluation of 75% of the original subjects 14 years later, there was deterioration of vision among 29% of the partially sighted, which created a higher rate of psychiatric disorder (68.7%) than with those with stable vision. The presence of hard neurologic signs without mental retardation was a nonpredictor of psychiatric outcome. The authors found the original narrative to be more helpful than the early IQ numbers themselves. About half of the cohort had romantic relationships, about a fifth with sighted partners. An alarming number, especially women, reported sexual molestation or abuse. Of particular note was that stereotyped mannerisms had almost disappeared. Several participants revealed that they perform the mannerisms at home and alone but understand that they are socially unacceptable and control them in public. They remembered their mainstreaming school experiences as being very difficult, with 73% being teased in a mean way. Most (73%) traveled independently and only three used dogs, but 40% reported having been hit by a car. For partially sighted persons, there was a strong tendency to avoid the negative stereotype of blindness and try to "pass." They also found deplorable levels of unemployment among the subjects who were qualified for competitive jobs ( [Freeman et al., 1991](#)).

### Research Directions

A central issue at this time is the future direction of education for blind children. The passage of Public Law (PL) 99-457 has provided educational services for impaired children, including infants, along with encouraging educational integration of blind with sighted children. An increasing number of blind children are currently mainstreamed. There has been a long-standing concern that the development of social skills among visually impaired children is delayed in a segregated school for the blind. It is believed that these skills are better developed in an integrated environment as preparation for adult roles in the sighted world. Conversely, with the push for mainstreaming, literacy in Braille has declined. There is a controversy between mainstreaming to improve social skills versus specialized schools to increase learning and literacy skills. The outcome of using the new technologies in both settings is an open question. Consulting child psychiatrists as the child and family advocate must remind school boards about the broad heterogeneity of the blind pediatric population, to forestall decisions based on monolithic assumptions about the visually impaired, which may overlook the best interest of each child. There is great need for reconciling clinical definitions and therefore statistical studies worldwide if an impact is to be made in improving outcomes.

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#### CASE ILLUSTRATION

Lucy is a 14-year-old blind girl, who lost her vision at the age of 1 year following bilateral enucleation for retinoblastoma. She was referred for uncontrollable outbursts of crying, screaming, and striking out wildly at people. She was diagnosed with borderline personality and started on neuroleptic medications in fairly substantial doses. Both of her parents were professionals with graduate degrees.

Lucy's early developmental milestones were within normal limits, and as an infant was easy to care for. Following her surgery and numerous prolonged hospitalizations, her development was delayed and she was remembered as a very "whiny" crying child, clinging to her mother. Her family situation included several geographic moves. A sister was born when Lucy was 4 years old. Lucy had been enrolled in a special program and later in a school for the blind. She used a Braille typewriter skillfully. She did well academically, and was described as clinging to her mother, who was described by teacher reports as "too protective and enmeshed." She had no friends or social skills in school and was described as shy and slow to warm up. Several school changes, resulting from family relocations, disrupted her education and friendships. Her parents' marital strains had increased several years prior to Lucy's referral.

On psychiatric examination, Lucy had difficulty separating from her mother, but came into the consulting room alone. She sat with her head down and her right ear turned toward the examiner as she gently swayed back and forth. Her speech was clear and vocabulary age-appropriate, although she spoke loudly with a broadcasting style. Her concepts were immature, and she repeatedly wanted reassurance that her mother would take care of her and would not leave her. She then described how her father would hit her on the head when she made a mistake, bumped into something, or knocked something over, calling her "dumb." She then said, "I couldn't see him when he hit me. I never knew which way he was hitting me from. I couldn't protect myself or get out of the way."

The parents decided to separate following a trial of family therapy. Lucy entered supportive psychotherapy, and medications were reduced. A behavior management program was introduced to increase her independence.

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## DEAF-BLINDNESS

All that has been said in the introductory sections under the heading of general considerations and role of the child and adolescent psychiatrist is even more pertinent in the case of deaf-blindness and should be referred to. In particular, the effects of a handicapped child on family dynamics are exponential in a dually impaired child or adolescent.

### Definition

Deaf-blindness is defined in PL 94-142 as

concomitant hearing and visual impairments, the combination of which causes such severe communication and other developmental and educational problems that they cannot be accommodated in a special program solely for the deaf or blind child.

The definition is educational and does not focus on specific clinical measures. It implies that the two concomitant impairments generate specialized educational needs in combination. As with auditory or visual impairments, the child might be classified as deaf-blind while still retaining some usable residual hearing and/or vision sufficient to permit education, self-sufficiency, and support or independent living in some cases. The term "deaf-blind," although professionally widely used, might not accurately convey the individual client's circumstances. The imprecision in definition highlights the very nature of the clinical work, when the clinician's cumulative experience and knowledge is called to bear on a unique child or adolescent and his or her specific circumstances and needs. As indicated earlier in the chapter, the words "deaf" and "blind" have strong connotations and the combination can be overwhelming. Familiarity with resources and supports for sensory impaired children and their families is essential.

The data from the 1990 survey of the U.S. Department of Education reported 7,297 deaf-blind children enrolled in educational programs under various assistance programs. Children receiving no services or those remaining at home or institutionalized were excluded. One difficulty in gathering accurate numbers is that many jurisdictions classify the deaf-blind as multihandicapped. The University of Arkansas Rehabilitation group's work suggests that the mean expectation is 9,805, based on statistical data. The report shows problems in definition, identification of cases, and demographic issues (Watson and Taff-Watson, 1993). Although the total numbers are relatively small, the number of dual sensory impaired children will rise with the increasing number of children born into poverty, improved neonatal intensive care unit success with very low birth weight infants, and the increasing number of high-risk pregnancies. In addition, changing social and sexual mores, increased incidences of sexually transmitted neonatal diseases, and congenital infections with the herpes simplex and cytomegalovirus are contributors to increased risks for sensory impairment.

The literature on deaf-blindness is limited. Workers in the field of deafness view the deaf-blind child as being deaf with an associated visual problem and vice versa for visual impairments ( [Warren, 1984](#) ). Few centers are equipped to provide the comprehensive assessment that a deaf-blind child requires. The psychiatric evaluation has three potential pitfalls: failure to recognize visual or auditory impairment when evaluating a child who is known to be either deaf or blind, but ascribing the behavioral difficulties to a clinical psychiatric illness, most commonly mental retardation or autism; diagnosing a child as deaf-blind when only one sensory impairment exists concurrently with a major clinical psychiatric syndrome; and diagnosing mental retardation, schizophrenia, autism, or organic brain syndrome in a deaf-blind child, based on an unusual presentation, without being fully aware of the child's potential strengths or problem-solving and adaptive abilities. Tragically, many deaf-blind children have spent years or a lifetime in custodial institutions, based on inaccurate diagnosis. The age of onset of deaf-blindness is a crucial

consideration (other factors, such as intelligence, degree of sensory loss, and associated impairments being equal).

Estimates of independent function for deaf-blind individuals vary and depend on the degree of residual vision and hearing available to the individual. Fortunately, the historical evolution in societal attitudes has changed from warehousing in large public institutions to actively seeking placements with habilitation services if they cannot be maintained at home. Home support services with respite care are a desirable option for many families ( [Gonzales and Fontana, 1998](#)). A large public investment in programs has failed to perform longitudinal follow-up of the efficacy of various programs, and thus predictions and prognoses are uncertain (Watson, 1993). [Bjorling \(1981\)](#) summarizes the findings to estimate that probably no more than 3% to 5% of deaf-blind individuals are considered to function independently, and the vast majority of this small group comes from adventitiously impaired groups. There have been strong advocates for early infant intervention programs and these have been enacted in law. The uniqueness of the dual sensory impairment requires a systematic conceptual approach in evaluating the acquisition of various skills in a developmental sequence specific to the child.

## Etiology

Etiologies, especially of adventitious onset, include trauma, infections, diabetes, tumors, and injudicious use of antibiotic medications (aminoglycosides). Congenital conditions, including maternal rubella and cytomegalovirus infection, are causes of multiple sensory impairment. Usher syndrome is congenital deafness with progressive visual impairment of varying degrees secondary to retinitis pigmentosa, the latter starting usually during adolescence. The incidence is approximately three per 10,000 in the general population but is reported to be between 3% and 6% of the congenitally deaf population ( [Hicks and Hicks, 1981](#)). The etiology of a case of deaf-blindness influences the incidence of associated handicapping conditions. Further, the degree of sensory loss is quite variable and influences outcome. Congenital rubella has a 50% to 71% reported rate of associated neurologic complications among deaf-blind children with a high incidence of mental retardation. [Stein and associates \(1982\)](#) reported a high frequency of abnormal head size (40%), neuromuscular disorders (40%), and abnormal electroencephalogram (55%). It was remarkable that in their population of 141 "deaf-blind" children referred to their statewide research institute for special evaluation, 38 (27%) proved to have normal or near-normal hearing, and one had normal vision. This high degree of misdiagnosis is alarming because the combination of dual sensory impairment results in a profoundly different environment for the child's development. Language and general information development have been reported to be significantly below expectations for 75% of these children.

## Psychopathology

Difficult as it is for a hearing parent of a deaf child to communicate, teach social skills, and impart learning, it is even more demanding with a deaf-blind child. It is not only the addition of blindness that adds to the difficulty, but also that the environmental experience of the dynamic neurologic processes are as yet not fully understood. Techniques for teaching age-appropriate rules of society and imparting knowledge to a blind child assume common conceptualizations about the environment and society between teacher and child. A formal evaluation of adaptive behaviors and functional activities and strengths is helpful in order to formulate recommendations. Psychiatric evaluation of multiply handicapped children reveals a direct correlation between the number of impairments and the incidence of clinical psychiatric syndromes, including autism and organic brain syndromes, with an increase in mental retardation associated with dual sensory loss ( [Chess et al., 1971](#)). [Smithdas \(1980\)](#) and [Yoken \(1979\)](#) have reviewed personal experiences of deaf-blind children who experienced the world as "shrinking in space" and the isolation, dependence, anger, and societal rejection. There are often reactive feelings of anxiety, even progressive isolation, mourning the loss of function and changing self-image.

## Psychiatric Evaluation of a Deaf-Blind Child or Adolescent

When a clinician is asked to evaluate a hearing or visually impaired child, one should always consider that dual impairment could mislead the examiner. Conversely, the clinician might encounter a child who is diagnosed as deaf-blind and might be deaf-autistic, blind-autistic, or sensory impaired and mentally retarded. The diagnostic process is a collaborative team effort with psychologists, educators, and habilitation and rehabilitation specialists. Techniques for eliciting information, especially physical contact, lighting, and realistic props for toys, are all similar to those described for blind children. Evaluation requires time, patience, and the use of an interpreter if a system of manual sign language has been developed. [Wheeler and Griffin \(1997\)](#) examined a movement-based approach to development of language that assists deaf-blind children learn complex concepts by emphasizing gestures.

## Differential Diagnosis

The common differential psychiatric diagnoses in deaf-blind individuals include mental retardation, childhood autism, childhood schizophrenia, organic brain syndrome, and reactive behavior disorder. One of the sensory impairments may be missed when a diagnosis of schizophrenia or autism already has been made. The converse might also occur if the clinician attributes behavior to the sensory impairments. Ritualistic acts are common in severe or profound mental retardation as well as in childhood autism, and both rigidity of behavior and perseveration may characterize deafness or blindness, as well as mental retardation or autism. Normal deaf-blind individuals easily stop the rituals if offered human contact.

## Treatment

Data on treatment of deaf-blind individuals with reactive or more serious psychopathology are woefully lacking. Behavior therapy, psychotherapy, and counseling may each be useful. The use of medication that could affect residual hearing or diminish visual perception should be avoided, but can be useful for symptomatic treatment to assist in daily living.

## Outcome and Follow-up Data

The remarkable achievement of Helen Keller is instructive and inspiring but is not a good model because she acquired a language foundation of both sight and sound prior to the onset of her impairments. Some individuals who are deaf and blind fulfill their potential and lead competent lives. Deaf-blind youngsters who are aging out of the educational system lack appropriate supportive services, including housing. There has been a shift in focus for adolescents to establish greater independence rather than an abrupt transfer to adult rehabilitation facilities when they reach 21. The Arkansas Center's Model Program is one of a number of reports in the literature and is a helpful place for the clinician to review developments in community-based approaches to service delivery (Watson and Taff-Watson, 1993).

## Research Directions

Alternative treatments, and the role of spirituality and its contribution to outcome are areas needing further studies. Recent advances in the basic neuroscience, cognition, and its applicability in understanding learning processes in the deaf-blind continue to be a frontier of research, as are relational studies, including movement-based communication. In general, there needs to be greater awareness about the burden of impairment in order to support sensory-impaired children and families in their communities.

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### CASE ILLUSTRATION

David is a deaf-blind 18-year-old youth referred for neuroleptic medication and "sedation" by the residence where he lived. He is profoundly hearing impaired and legally blind, and he understands simplified and exaggerated sign language. He is unable to use finger spelling and has a usable vocabulary of approximately 50 formal signs. The resident staff is concerned that when upset he swings his arms and bangs chairs on the floor. The etiology of his impairments is possible congenital rubella. Early in life, he suffered physical abuse from his mother and was subsequently placed in an institution for the mentally retarded at age 4.

Mental status examination revealed David to be a very large, neatly dressed African-American male. He frequently jumps from his chair pantomiming his responses. He rapidly moves his arms in excited gestures as he expresses himself with affect-laden signs and pantomime. Yet, when his meaning is comprehended, David immediately sits down in his chair, satisfied that he has communicated. His use of language is restricted and, at times, perseverative, but his response to limit setting is appropriate. He is alert, and no hallucinations or signing into space were observed. He picks up the furniture and bangs the floor and then returns it to the same position. There are periods when he becomes agitated, goes to his room, and "bangs around" a little.

The resident staff responds to the consultant's interpretation of the communicative nature of David's behavior and how he has never crossed the line to violence while responding to firm limit setting. The staff acknowledges that he intimidates people because of the difficulty in communicating with him, as well as his size and race. They agreed to record his baseline behaviors if they initiate a full course of medications and try to correlate his behavior with precipitating events over a 3-month period. They accepted a plan to initiate medication and record what initiated his tantrum, with neuroleptics if needed. On follow-up, David was successfully maintained in his residence for 4 years without any untoward events, and only on three occasions was he given a single dose of medication.

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## 52 ATTENTION DEFICIT HYPERACTIVITY DISORDER

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A popular German children's book written in 1863 by Heinrich Hoffman, entitled *Strüwel Peter*, colorfully described the characteristics of attention deficit hyperactivity disorder (ADHD). In the English translation, one verse reads:

Phil stop acting like a worm  
The table is no place to squirm  
Thus speaks the father to his son  
Severely say it, not in fun.  
Mother frowns and looks around  
Although she does not make a sound.  
But Phillip will not take advice  
He'll have his way at any price.  
He turns and churns  
He wriggles and jiggles  
Here and there on the chair.  
Phil these twists I cannot bear.

Phil's antics would not describe children who have attention deficit hyperactivity disorder (ADHD) inattentive subtype. For these children the refrain might go something like this:

Jane was dreaming, looked spaced out  
Which made her teacher want to shout  
Pay attention, start right now.  
Keep your mind on task somehow.  
You knew your spelling words last night  
And yet today there are none right.  
Your attention flits to every sound.  
Homework lost and never found.  
Your books are always out of place.  
Jane, these are problems you must face.

Phil and Jane's difficulties are seen in normal children at times, but in those who have the disorder, they are more severe and enduring, more pervasive, and are not a reaction to an immediate stressful event.

This syndrome has captured the interest of researchers and clinicians for four decades. It is now the most intensively studied syndrome in child psychiatry and possibly the most controversial. For example, between 1957 and 1960, 31 articles were published on hyperactivity in children, whereas since 1996, there have been about 400 articles a year on ADHD. The increase in the rate of publication is continuing and, as a result, it is no longer possible to write a comprehensive chapter on all aspects of the syndrome. This chapter emphasizes an update of key clinical issues and recent research findings.

Children with ADHD-combined type (ADHD-C) have inappropriate restlessness, behavioral and cognitive impulsivity, and difficulty paying attention. These problems are present at home and school and cause the children significant impairment. The children have a hard time controlling their impulses and regulating their activity, attention, and social interactions to a degree consistent with relevant age and cultural norms. This leads to their being frequently in trouble with adults and unpopular with peers. They underachieve at school (or do not achieve at the level expected for their intelligence), and most have learning difficulties—owing, mostly, to poor attention and organization, and impulsive cognitive styles. In addition, some have specific learning disabilities, such as reading disorders. Children with ADHD may also have other disruptive behavior disorders, such as conduct disorder or oppositional defiant disorder, which further compound their impairment.

Children with ADHD-inattentive type (ADHD-IA) are those who meet DSM-IV diagnostic criteria for inattention but who fail to meet criteria for hyperactivity-impulsivity. The children have difficulty with many aspects of attention such as focusing, sustaining attention, and organization. In class, they have difficulty getting started on a task, sustaining attention to that task and completing the task unless their teacher supervises them. Their work is often messy and poorly organized, and assignments are not handed in. As a result, they underachieve for their intelligence. They may seem to lack motivation, perhaps because their past attempts at success have always failed.

Children who have ADHD-hyperactive impulsive type (ADHD-HI) do not meet the diagnostic threshold for symptoms of inattention. Because these children are often younger when they present (Lahey et al., 1994), it is not yet clear if this is because they are not yet being asked to function in a classroom setting. Similarly, children with ADHD-IA often present at a slightly older age, and this may be because they have simply outgrown hyperactivity from their younger years.

## HISTORICAL NOTE

### Identification of the Syndrome

One hundred years ago, the British pediatrician [George Still \(1902\)](#) described a group of 20 children who had problems very similar to those we now diagnose as having ADHD combined type along with conduct disorder. He wrote that the children had “a defect of moral control,” which he defined as the control of activity in conformity with moral consciousness. The children lacked inhibitory control and required immediate gratification without regard to others. They were described as restless and impulsive, and had attention problems, as well as intense affect. They were dishonest, destructive, broke the law, and were cruel. Many had specific learning disorders and physical anomalies. Still believed environmental as well as organic factors were causal. This was the first published paper to identify a disorder of impulse control and inattention as a syndrome or disorder in itself, not necessarily as a result of brain injury or retardation.

Still considered whether a lack of inhibitory control or attention problems was the primary difficulty of the disorder. He concluded that poor attention was primary because it did not allow children to hold onto a course of action, thus putting them at the whim of stimuli promising immediate gratification.

It is interesting to observe that the controversies first raised by Still continue to be actively debated. Still moved difficulty with “attention” and “impulsivity” from the moral and religious sphere (laziness, badness) into a medical paradigm. The public continues to question whether or not this is appropriate because they fear that normal human struggles with laziness and disobedience will be dealt with as diseases and treated with drugs rather than discipline. Still described the syndrome with great clinical acumen. However, as noted in the discussion of the subtypes, we continue to debate what symptoms belong to the disorder, what symptoms are associated features, and what symptoms truly belong to comorbid conditions such as conduct disorder (CD). Still described a broad group of children, many of whom would be considered in today's diagnostic nomenclature to be comorbid. The extent to which problems with conduct, mood, or sleep represent associated features of the disorder or comorbid conditions is an active area of research. Finally, Still asked the question as to what is “the core deficit.” This more theoretical approach to questions about the disorder has currently been the source of much heuristic discussion and psychological experimentation ( [Barkley, 1997](#)).

There was a significant historical shift in our understanding of these children during the early 1920s. At that time, the syndrome began to be viewed as organic in origin. This was owing, in part, to the influenza pandemic following World War I and the epidemic of encephalitis lethargica that occurred as a result. Children who survived frequently developed a severe behavior disorder ( [Hohman, 1922](#)), similar to that described by Still, and were described as being “organically driven” ( [Kahn and Cohen, 1934](#)). [Lewin \(1938\)](#) in Great Britain, was also influential in that he described a link between severe brain damage and restlessness in retarded children and adults, as well as a similar correlation in laboratory animal studies. Finally, in the United States, [Strauss and Lehtinen \(1947\)](#) emphasized the organic etiology hypothesis; they described retarded children who had hyperactivity, distractibility, impulsiveness, perseveration, and cognitive defects. Strauss considered these retarded children to be brain damaged even when the latter could not be demonstrated. Strauss termed these children as having “minimal brain damage syndrome.”

Some clinicians at the time recognized the circular reasoning that this implied and suggested that a child should not be labeled as brain damaged solely because of his or her behavior, unless the damage was demonstrated. In an attempt to compromise between the idea that some disorder appeared to be present in the brains of these children, and the idea that there was a purely environmental etiology, [Clements and Peters \(1962\)](#) coined the term minimal brain dysfunction (MBD). The term achieved widespread popularity and was endorsed, but with a warning, by MacKeith at the conclusion of an international conference held in Oxford in 1962. In an article entitled “Minimal Brain Damage: A Concept Discarded,” [MacKeith \(1963\)](#) warned that the new terminology of MBD, although an improvement over the older one, included a heterogeneous group of children who would have to be subclassified in the future. Nevertheless, the term MBD was held onto tenaciously in spite of criticisms of overinclusiveness.

### Development of Formal Diagnostic Criteria

Efforts to create a more scientifically valid and reliable classification began with the International Classification of Diseases-9 (ICD) ( [World Health Organization, 1965](#)) and the DSM-II ( [American Psychiatric Association, 1968](#)), when the terminology was changed to hyperkinetic syndrome of childhood. The term hyperkinetic disorder is still in use in the United Kingdom, and retarded and/or brain-damaged children are included in the diagnosis in ICD-10. However, if conduct disorder and a hyperkinetic disorder co-occur, the term hyperkinetic conduct disorder is used. Differences in diagnostic criteria between the two countries have made comparisons of research more difficult.

The work of [Douglas \(1983\)](#) was influential in distinguishing specific learning disabilities from the core problems of ADHD, which had traditionally been an integral part of the diagnosis of MBD. Furthermore, she postulated that the basic deficit of the disorder lay not in hyperactivity, but in a failure to regulate attention, arousal, and inhibitory control, deficits that she felt were more fundamental to the disorder than restlessness. The DSM-III ( [American Psychiatric Association, 1980](#)) recognized this emerging opinion and renamed the disorder attention deficit disorder with hyperactivity (ADHD) or without hyperactivity (ADD).

DSM-III ( [American Psychiatric Association, 1980](#)) represented an advance in that a task force was created consisting of a group of experts who were committed to using available research data as the basis for changes in diagnostic criteria. Three core constituents of the disorder were described, namely inattention, impulsivity, and hyperactivity. Five or six symptoms were listed under these three headings and more than two or three symptoms in each area were arbitrarily designated as the diagnostic cutoff point. ADHD required that all three constituents were present, whereas ADD required that only inattention and impulsivity were present. The requirement of impulsivity distinguished ADD from ADHD-IA type, so that these two diagnoses are not identical. Again, this makes it difficult to compare studies done on ADD without hyperactivity and ADHD-IA. In the years following the publication and use of DSM-III, interest began to be focused once more on the lack of inhibitory control as the cardinal difficulty of these children ( [Barkley, 1994](#); [Quay, 1988](#)). Factor analytic studies of parent and teacher rating scales indicated that impulsivity correlated highly with hyperactivity, and in DSM-IV these symptom clusters form a single dimension.

The distinction between the three core problems of the syndrome was short-lived, and DSM-III-R ( [American Psychiatric Association, 1987](#)) changed the terminology to attention-deficit hyperactivity disorder (once again making restlessness more primary to the disorder) ( [Porrino et al., 1983](#)) and ADD—without hyperactivity—was omitted. DSM-III-R listed 14 symptoms, of which eight were required for a diagnosis.

In DSM-IV ( [American Psychiatric Association, 1994](#)) the terminology remains the same, ADHD, and the disorder is once again described under the category of disruptive behavior disorders ( [Table 52.1](#)). In this classification, however, a patient would obtain the diagnosis of ADHD-IA type by scoring on six out of nine items related to inattention (that have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental norms), or ADHD-HI type by scoring on six out of nine items related to hyperactivity/impulsivity. A patient is said to have ADHD-C when he or she scores above the cutoff on both the inattentive and hyperactive-impulsive items.

**Table 52.1. Diagnostic Criteria for Attention-Deficit/Hyperactivity Disorder**

This new system of classification has had a profound influence on practice in the last 5 years. This change in the definition of ADHD has led to an increase in prevalence, a broadening of the disorder to include more girls, preschoolers, and adults, influencing patterns of comorbidity, impacting on educational practices and



various forms of treatment. The use of the DSM-IV diagnostic system increased the prevalence of ADHD by more than 50% (Wolraich et al., 1996), which, in turn, contributed to a large increase in the use of stimulant medication. The specifics of these changes and how they are reflected in each of the three ADHD subtypes are reviewed in greater detail in the following.

From an historic point of view, it is interesting to observe how the DSM-IV classification scheme ( [American Psychiatric Association, 1994](#)) reflects the latest chapter in those issues first raised by [Still \(1902\)](#). DSM-IV suggests that both inattention and disinhibition may be important in ADHD but reflect different subtypes. The subtypes may also represent a compromise between the trend to make the disorder quite specific, and a need to extend diagnostic criteria to include a wider number of individuals who are impaired and suffer from various of the associated features of ADHD.

The DSM-IV diagnostic criteria are also unique in the stringent methodology of the field trials ( [Frick et al., 1994](#); [Lahey et al., 1994](#)), in which 380 children from 10 sites were evaluated systematically. The field trials confirmed the hyperactive and impulsive symptoms as one factor or dimension, and that individuals who mainly suffered from inattention were nonetheless impaired. All of these changes reflected the growing concerns of the academic community of researchers from a variety of disciplines and the subsequent emphasis on measurement and more rigorous methodology that has characterized the past 5 years.

### History of Treatment Studies

In 1937, Bradley discovered that Bensedrine (*α*- and *β*-amphetamine) resulted in marked improvement in a group of children who were inpatients at the Emma Pendleton Bradley Residential Treatment Center, many of whom had behavior problems. Treatment with this stimulant resulted in reduced restlessness, and better concentration and motivation. It was 30 years later that [Conners and coworkers \(1967\)](#) used dextroamphetamine in a double-blind study to treat a group of inpatient adolescents with behavior problems. Eisenberg helped to establish the placebo-controlled, double-blind clinical trial as the gold standard to test pharmacotherapy treatment for children. With the help of newly standardized rating scales, particularly by [Conners \(1969\)](#), study after study confirmed the effectiveness of stimulant treatment. From 1965 until 1995, over 350 clinical trials were conducted with as many as 3,000 children, demonstrating unequivocally the short and intermediate term effectiveness as well as the safety of stimulant treatment for ADHD ([Spencer et al., 1996](#)).

Empiric studies of treatment have branched into three new directions since 1996.

1. Studies looking at younger, older, and comorbid populations
2. Studies of new stimulant treatments, and other types of medications
3. Studies looking at multimodal treatment versus treatment with stimulant alone. This has been the subject of the most expensive clinical trial ever conducted by the NIMH, the Multi-modal Treatment Assessment project, described more fully in the following ([Arnold et al., 1997](#); [Greenhill et al., 1996](#); [Hinshaw et al., 1997](#)).

These new research directions have resulted in the major developments in our knowledge of ADHD that are summarized in this chapter.

In summary, a chapter on ADHD in 2001 looks at an area that has become highly publicized and researched and that is the subject of controversy. The areas of debate today are not too dissimilar from those marking the inception of the disorder. ADHD is now a complaint very much in the public awareness, with an industry of books, magazines, Websites, and support groups behind it. It is a disorder that has recently been the subject of a major National Institute of Health Consensus Conference ([http://odp.od.nih.gov/consensus/cons110110\\_intro.htm](http://odp.od.nih.gov/consensus/cons110110_intro.htm)).

Practice guidelines for treatment have been developed based on current practice and research. Despite all of these efforts by the scientific community, a sizable proportion of treatment in use is still based on alternative therapies that have not been subject to empiric study or any other type of external control. In this context, the present chapter represents an attempt to place these new developments into a historic context, and to provide both a clinical and research guide to practice.

## CLINICAL DESCRIPTION OF THE DISORDER

### Attention Difficulties

Children with ADHD-inattentive type (ADHD-IA) are mainly impaired in school. Although they may have some difficulty with social and family relationships, the presenting problem typically is academic underachievement. Teachers complain about these children being chronically late, forgetful, disorganized, losing things, daydreaming, off task, unable to finish their work, and procrastinating.

Attention is a clinical construct that has been used to embrace a wide range of difficulties that may overlap. These include difficulties getting on task, staying on task, and finishing things. Attention problems may also include being “scatterbrained,” “spaced out,” or “not listening.” They may also include problems with motivation, variability in performance, and difficulties with understanding sequenced commands or following instructions.

With such a broad sweep of clinical presentations, it is not surprising that attention has not been successfully captured by any single psychological or electronic test. It is also notable to observe the difference between the meaning of “attention” as used by professionals, and the meaning of “attention” as used in common parlance. Parents may come into the clinic stating that their child “pays excellent attention; they play video games for hours.” For the clinician, a child who is stuck on attention relieving activities such as playing video games, using construction sets, or watching television may be the very child who has an attention deficit.

At the present time, attention is most often measured by obtaining a four-point rating (ranging from “never” to “very often”) of the nine DSM-IV items chosen to operationalize the concept. These nine items are *sensitive* to attention deficits, but they are by no means a comprehensive account of these deficits. Further, we do not know the extent to which the nine symptoms of inattention defined by DSM-IV are *specific* to attention disorders. The functions associated with “attention” or executive function represent some of the most sophisticated pathways in the brain, and also perhaps those most vulnerable to injury. We know that most serious psychiatric disorders are associated with problems with attention, executive function, and concentration. We know further that for many of these disorders, attention symptoms are the first to be evident and the last to return to health. Therefore, it is important to characterize what (if any) items used to define attention deficits are specific to ADHD.

### Hyperactivity

[Rutter and associates \(1982\)](#) pointed out that hyperactivity is not a unitary dimension and that the fidgety child is different from the child who is always on the go. Furthermore, the restlessness of the hyperactive child is more than excessive activity, although the latter has been demonstrated to be present during structured in-seat activity ([Barkley et al., 1999](#)), as well as in sleep ([Porrino et al., 1983a,b](#)). Rather, hyperactivity is activity off task, out of seat, and disruptive. The activity is developmentally inappropriate and is not goal directed or purposeful to the task on hand. [Klein and Young \(1979\)](#) hold the view that it is the combination of high activity with high disruptive behavior that distinguishes the ADHD child from the normal child. The difference lies in the quality, as well as in the quantity of activity.

At the first visit of a child to a physician for evaluation of possible ADHD, hyperactivity may not be observed, even when described as severe at home and school. The increase in anxiety, newness of the situation, and amount of adult attention may improve the inhibitory control over activity. However, preschool children are likely to be restless and succeed in messing up the office and appear to be driven, even at the first visit. In older children, the syndrome should not be ruled out simply because restlessness was not observed in the office.

Restlessness can be measured in different ways, such as standardized parent–teacher rating scales, direct observation, and devices that measure activity (actometers). These measures relate to different facets of activity and are unlikely to correlate. More perplexing is the poor correlation between two teachers rating one child, and between parent and teacher ratings. Hyperactivity is clearly situation-dependent varying with the type of activity, interest in the activity, amount of adult attention available, and relationship of adult and child. [Whalen and Henker \(1991\)](#) concluded that the level of hyperactivity was a product of a given child, the situation, and the observer. Furthermore, hyperactivity varies unpredictably in any one child from day to day.

In a preschool study of 3- to 5-year-old children in an experimental nursery, children with ADHD and normal children were rated on hyperactivity by direct observation in structured play and in free play situations. Differences between children with ADHD and normal preschoolers were observed in structured but not free play when all preschoolers were active. Rule-enforced activity was differentiating ([Schleifer et al., 1975](#); [Weiss et al., 1985](#)).

Hyperactivity decreases over the course of development, and frequently children change from being on the go all the time in preschool to being able to sit in a chair fidgeting in elementary school. Hyperactivity may disappear in adolescence, although the adult may "feel restless" even when this is not observed ( [Weiss and Hechtman, 1993](#)).

## Impulsivity

Impulsivity refers to the inability to delay a response despite the anticipation of negative consequences for the behavior. The difficulty in controlling impulses is rated in comparison to others of the same age and gender. The manifestation of this difficulty varies across the life span.

In the preschool years, the following complaints are common:

"He just grabs other kids' toys if he happens to want them. Kids now avoid him."

"On the playground he shoves and pushes other kids if they are in his way: He hits when he is angry."

"He darts across the street if I am not holding his hand. He could have been hit by a car the other day."

"She burned herself on the stove yesterday. I can't believe she'd do it again today but that's what happened."

There is a relative failure to learn from experience. Impulsive preschoolers are at risk for accidents in general and accidental poisoning in particular. They have to be supervised more carefully than others the same age to ensure safety because they are often unaware of danger.

Impulsivity shows itself in behavioral and academic difficulties in elementary school. In the classroom these include interrupting or blurting out answers without waiting to hear the questions and difficulties waiting in line. Impulsive ADHD children are more daring than their peers. Therefore, accidents continue to be more common. Peer relationships are marred by being a poor loser, wanting "his way or no way," having to be the boss or leaving the game, and getting into physical fights if feelings are hurt. Procrastination becomes an increasing problem because responsibility has now increased. Assignments are not completed or handed in; homework may become a daily battle and affect family functioning.

Cognitive impulsivity refers to a cognitive style characterized by short reaction time. Schoolwork is replete with errors, detail is missing, writing is messy, and the answers are written without reading the question carefully.

Many of the preceding difficulties continue in adolescence. In addition, at this age, the risk for adolescent maladaptive behaviors increases, including early drug use, drinking, smoking, and unwanted pregnancies. A higher incidence of reckless driving and car accidents has been documented in adolescents with ADHD. Adolescents are better self-reporters than younger children and complain about losing friends because of impulsive comments or acts.

Insight into the difficulties improves in adulthood. Procrastination is frequently complained about and causes daily minor or major difficulties. Failure to answer phone calls or letters or to do any boring or difficult task is common.

A marriage may be broken without careful thought or a job may be left without other work being available. Reckless or unwise spending may lead to debts. Increased car accidents ([Weiss and Hechtman, 1993](#)) may result in loss of license or injury. Parents with ADHD may lose their temper and physically punish their children when provoked. This can be serious and may result in physical abuse.

Impulsivity across the life span has been considered to be among the most serious aspects of ADHD in causing functional impairment and also among the most difficult to treat.

## Associated Features

Areas of impairment that are not part of the hyperactive, impulsive, or inattentive symptoms listed in DSM-IV and ICD-10 accompany ADHD. These associated features, nonetheless, can cause significant difficulty, or even be the main reason for referral. These associated features fall into behavioral, cognitive, affective, and social dimensions.

### BEHAVIORAL

Children with ADHD function better in one-to-one versus group situations; this causes them great difficulty in peer relations and group situations, such as extracurricular activities, camps, sports, and group play, as well as classroom proceedings. They often respond quite differently to their father and mother, being more responsive to the parent who is less familiar. They do much better in novel versus familiar situations. By the same token, they have difficulty functioning in situations in which consequences are not immediate, consistent, and salient, or situations without adequate supervision. This implies that individuals with ADHD require a great deal more supervision at school and parenting at home, even with appropriate medication. Often this presents a crisis at the beginning of high school, when it is more difficult to provide this type of supervision.

Individuals with ADHD have problems with persistence all through the life cycle. This is more than "not finishing things," and more than difficulty with remaining on task. They describe taking up new projects with much more readiness than they show for persisting with an old project, for example, taking up a new musical instrument before mastering the last. They complain of "losing interest." They also have much more difficulty with situations in which there is delayed gratification, so that they fail to pursue opportunities in which they would have to "hold out" before receiving an anticipated benefit.

Individuals with ADHD show marked variability in performance. This makes academic assessment difficult in childhood. Teachers can neither reliably judge a child's achievement by how they do at their worst (in which case they would never graduate), nor by how they do at their best (because they cannot rely on optimal functioning being consistent). Even as adults, marked variability of performance leads to difficulty in areas of employment that demand consistent output, so that many adults seek self-employment or occupations that reward peak performance. Variability of performance leads to considerable fragility in the patient's sense of self, as well as frustration. Many individuals with ADHD describe that they have friends who are "smarter" than they are and wonder if they are "smart" or "dumb." They see that their skills do not match their performance.

Individuals with ADHD often find themselves clashing with others. They may be insensitive to some of the emotional or social nuances of those around them. On the other hand, they may also be very reactive to mood or tensions around them, escalating those tensions and precipitating crises. Although fewer than half of individuals with ADHD also meet diagnostic criteria for oppositional defiant disorder, many patients with ADHD do have difficulty with rule-governed behavior. Where this may be driven by the symptoms of ADHD itself, it appears that rule resistance is sometimes a characteristic of ADHD in its own right. Finally, the lack of sensitivity to reinforcement, and the lack of awareness of consequences from the past or consequences that may ensue in the future make it difficult for patients with ADHD to engage effectively with others toward a goal.

### COGNITIVE

Psychological ([Seidman et al., 1997](#)) and electronic testing ([Conners, 1985](#)) have not been effective ways to define or measure the cognitive characteristics of ADHD. This is partly because the testing situation itself mitigates the very qualities it intends to measure. The testing situation is structured, novel, uses short and interesting tasks, is carefully supervised, and presents a challenge that serves as an intrinsic motivator. As one mother put it while listening to a sophisticated neuropsychological report on her son in which there were no remarkable findings, "But where is the test that tells me why he comes downstairs an hour late for school, with socks that don't match, and is unaware that there's a problem?"

On the other hand, we are beginning to understand some of the associated cognitive impairments of individuals with ADHD on the clinical and theoretical levels ([Barkley, 1997](#)). Barkley has shown that individuals with ADHD have a decreased sense of time ([Barkley, 1994](#)) and therefore show difficulty in tasks that are time



dependent. This not only impacts on test performance, but also on the emotional development that grows out of connecting past, present, and future. The lack of sense of time may lead these individuals to live exclusively in the present. For example, many adults with ADHD are underinsured because they find it difficult to understand the imperative to spend money now to protect against future events that are not a present reality. A poor sense of time leads to problems with planning, waiting, and playing games.

Patients with ADHD often state that they have superb long-term memory, even for eccentric details, but very poor short-term memory. What they then go on to describe in their examples are often grouped together as difficulties with "working memory." These problems include various areas of difficulty. They may include short-term registration of a list of items, inability to remember and follow instructions, difficulty holding things in their head, and doing mental manipulations of concepts or numbers ([Denckla, 1996](#)). Most interesting and also most difficult to measure is the lack of ability to access information that has been present in the past. These are children who can know something one day, but are unpredictable in their ability to access that body of knowledge at other times. This could be described as difficulty with memory activation, like a light switch with a loose connection that works intermittently.

## EMOTIONAL

Attention deficit hyperactivity disorder is often associated with dysregulation of affect, in addition to the hallmark dysregulation of activity, speech, or activities of daily living. Children with ADHD, and those who live with them even more so, complain of temper outbursts, and mood lability and reactivity. Not only do moods change dramatically and out of tune with environmental stimuli, but also moods are often explosive, intense, and infectious. They begin almost instantaneously, but may disappear as dramatically as they appear, leaving close ones in shock, whereas the impact is unprocessed by the children themselves.

## SOCIAL

Children with ADHD are unpopular with peers ([Pelham and Bender, 1982](#)) and often at odds with siblings, parents, and teachers. Enduring friendships are rare, and may result in social isolation later in life ([Weiss and Hechtman, 1993](#)). Social impairment is complex ([Whalen and Henker, 1992](#)) and can include difficulty understanding what is appropriate, and difficulty with appropriate social performance even when the skill is established ([Barkley, 1998](#)).

Parents may comment that their child is bossy, intrusive, or insensitive to the personal space needs of other people. Children with ADHD find it difficult to cooperate with other children, respect social hierarchies, and follow rules when playing games. As they experience increasing social rejection they may also emulate the negative social teasing they receive, and become verbally or physically aggressive. Children with ADHD have trouble sharing and taking direction from other children, and trouble with impulsive pushing and touching. They may overreact to situations, have trouble with transitions, and be unable to let go of an argument or stay out of trouble between other children. Parents often complain that their child is "attracted like a magnet to other children just like him." They also complain that their child holds onto a rigid sense of justice, needing to "make it fair." The most heartbreaking complaints of a child or parent may be the observation that the child has never been invited for a play date, that invitations were sent for a birthday party and no one came, or that the child spends each recess alone because he is not asked to play.

It is interesting to observe that some of these difficulties are less pronounced in other types of social situations. Children with ADHD often love and may be good with animals that provide the "unconditional love" and companionship they crave. Children with ADHD function better one to one with a calm and patient adult, who is able to minimize excessive stimulation, and tune his responses to the child's needs and level of processing. Sometimes this type of interaction is obtained during time alone with a parent, grandparent, or childcare worker. Children with ADHD also may do better playing with children either younger or older than themselves. Adolescents with ADHD often seek out Internet chat groups, where their difficulties with impulsivity, timing, and hyperactivity are not evident. The therapist may discover that the patient's so-called best friends or boyfriends are virtual relationships. It may be possible to optimize the time that a child with ADHD spends in positive social situations and minimize time spent in rejecting social situations.

The socialization difficulties we have described involve problems with doing what is right, not doing what is wrong, knowing what to do, and then doing what you know. They involve difficulties in modulating all aspects of behavior, and require that the child do so consistently, all at once, and in overstimulating situations. Given this, it is no wonder that social deficits have been described as the "most intervention resistant domain of ADHD" ([Hinshaw, 1992](#)) and have required multimodal and specialized treatment approaches ([Piffner et al., 2000](#)).

## THE ASSESSMENT

### Parental Interview

This is the core of the assessment process, because the diagnosis of ADHD is made on clinical judgment. The child's current difficulties at home, school, and in the community are explored with respect to ADHD and its associated features. The majority of children given this diagnosis have comorbid disorders; these must be investigated because they may simulate ADHD or be true comorbid conditions. A careful history is obtained of the child's physical and emotional development from birth to the present, including the age when presenting difficulties were first seen and how they developed.

Family functioning is important to assess. Have family stresses affected the child? Is there psychosocial adversity or past trauma? What punishments do the parents use? Has the child been abused? The marital relationship is evaluated, and the effect of the child on the marriage and the family is explored. Do the parents agree on parenting issues? A history of psychiatric disorders in the family is obtained particularly with respect to ADHD, conduct disorder, learning disabilities, and depression. Does one of the parents have a disorder (e.g., depression in the mother, alcoholism in the father, or ADHD in either)?

### EVALUATION OF THE CHILD IN THE OFFICE

It is often difficult to confirm the diagnosis by interviewing the child because the child generally behaves normally at the first visit. Children often do not know why they are visiting the doctor and cannot accurately report their difficulties. Certain simple pencil and paper games can often show cognitive impulsivity. The child interview is important to understand how the child views himself or herself and the world and to evaluate comorbidity. Answers to questions such as, "Do you sometimes feel sad or mad?" "What kind of things make you feel this way?" and "Do you worry about things?" may provide windows to the child's inner world. Having the child identify three magic wishes "for things money cannot buy" can elicit unexpected and revealing answers.

### DIRECT OBSERVATION OF THE CHILD IN THE SCHOOL

Parents often request an observation, believing that this is the most accurate part of the assessment. In practice, it is time-consuming and may be inaccurate because of behavior variability. Direct observation is more useful in evaluating medication changes.

### INFORMATION FROM THE SCHOOL

Is the child in an appropriate learning situation? If not, what changes are required? It is important for the child's teacher to complete a DSM-IV rating scale to evaluate ADHD symptoms in the school (where attentional difficulties are best seen) and assess comorbidity. Ideally, the treating physician will follow this up by a telephone call to the child's teacher. Previous and current report cards are helpful in determining the onset of the disorder and present difficulties. Lack of contact between the treating physician and school was noted to be a major difficulty in assessment and treatment of ADHD by the NIMH consensus meeting (<http://odp.od.nih.govconsensus>).

### PSYCHOEDUCATIONAL ASSESSMENT

Evaluation of the child's intelligence and academic achievement is indicated. Often these resources are not available or are scarce. However, a psychoeducational assessment will determine the presence of specific learning disabilities and their nature. This is particularly important for children with the inattentive subtype, who frequently have associated learning disabilities or a language disorder. Inattention can be secondary to these; it can also result from the classroom teaching level being too advanced for children with low IQ or not sufficiently interesting for bright children.

## ELECTROENCEPHALOGRAPH AND NEUROLOGIC EVALUATION

These are indicated if seizures are suspected or if there is a history of head trauma or focal neurologic symptoms or signs or if the history is suggestive of a neurologic disorder.

## MEDICAL EVALUATION

Visual and hearing deficits must be ruled out. The child requires a complete medical history and physical examination. A history of the mother's pregnancy (including smoking and drinking patterns) is important. Impaired sleep is assessed because this may result in ADHD-like symptoms.

## AUXILIARY EVALUATIONS

These are sometimes required for school-age children who come for an ADHD assessment and may include a language assessment when indicated or an occupational therapy assessment to evaluate such problems (among others) as poor fine and gross motor skills when the history suggests impairment in these areas.

## TESTS

Parents frequently ask for a "test" for ADHD and have to be told that this does not exist. Computerized tests of attention (CPT) are not useful in the diagnostic process because of their low specificity and sensitivity ( [Lovejoy and Rasmussen, 1990](#)) and poor correlation with behavior in the classroom ( [Abikoff and Klein, 1992](#)).

Commercial actometers exist that work like a watch, are simple to use, and measure the child's total amount of activity; however, they have limited value. The quantity of activity is not generally the main difficulty; rather, it is in the quality or appropriateness of the activity ( [Pinto and Tryon, 1996](#)).

## RATING SCALES

Ratings scales play an important role in clinical practice, psychoeducation, and research. In clinical practice rating scales provide the clinician with information about a wide range of different symptoms, reported by different observers, in different environments and at varying points in time. The information that is obtained helps the clinician assess the child's symptom severity against well-standardized norms. They facilitate communication of these findings with parents and teachers, and allow measurement of change with treatment.

In research, ratings scales insure common definition of the samples being studied, measurement of efficacy and safety, and permit cross study comparison. Thus, treatment of ADHD has advanced partly as a result of the development of reliable, well validated, normed, sensitive, and specific symptom measures. The use of one of the common measures has become a standard of clinical care, meant to augment (but never replace) the clinical assessment.

Two different types of scales can be employed. Broadband scales assist with the assessment of comorbid symptoms and functioning, whereas narrow band rating scales assist with measurement of symptoms and treatment response of the primary diagnosis. Two of the best-known and commonly used rating scales are the Achenbach ( [Ostrander et al., 2000](#); [Vaughn et al., 2000](#)) or Child Behavior Checklist, and the Conners Rating Scales. A revised and updated version of the Conners Rating Scales was issued in 1999, and a new version of the Achenbach in 2001. Both have recently been revised to be comparable to DSM-IV. Thus, both scales now provide separate ratings of attention and hyperactive/impulsive symptoms. This is going to permit clinicians and researchers to rate improvement on these dimensions separately, and refine the assessment of outcome for ADHD-IA. It is pointless to measure outcome on an ADHD rating scale that emphasizes primarily hyperactive/impulsive symptoms if the child is diagnosed with ADHD-IA.

[Conners and associates \(1999\)](#) developed rating scales that were normed specifically for use with children ( [Conners, 1997](#)), adolescents, and adults ( [Conners, 1994](#)). The scales include up to seven different factors that have been empirically derived and can provide information on anxiety and oppositional symptoms, as well as cognitive, social, and somatic problems. The scale provides an ADHD index, DSM-IV symptom criteria, and a dimensional score for attention, hyperactive-impulsive symptoms, or both. The scales are sensitive (> 90%) and specific (77% to 98%), with correct classification rates of 84% to 96%.

Broadband rating scales often are used in conjunction with more specific symptom rating scales because they serve a different purpose. These scales provide a way of assessing the presence of other disorders, and symptoms in other areas. Therefore, they are useful initially to aid in diagnosis. They are also useful outcome measures in that they allow the clinician to assess how ADHD and non-ADHD symptoms change with treatment, and how changes in symptoms are correlated with actual change in functioning. The Achenbach scale described in the preceding is among the best known rating scale of this type. The Achenbach is a dimensional scale, and it provides severity ratings based on population norms on a variety of empirically derived factors. The Child Symptom Inventories ( [Gadow and Sprafkin, 1980, 1997](#)) group symptoms by DSM-IV diagnostic categories, and have been validated against semistructured interviews ( [Grayson and Carlson, 1991](#)). They are designed for a clinician to obtain a diagnostic screen based on report from the patient, parents, and teachers. There are preschool, child, adolescent, and adult versions available. The outcomes can be read easily without complex scoring procedures ( [Achenbach, 1991](#); [Biederman et al., 1993](#)).

## DIFFERENTIAL DIAGNOSIS AND COMORBIDITY

### Differential Diagnosis

The following summarizes some of the key clinical issues in differential diagnosis:

1. Physical causes of inattention, such as hearing or visual deficits, poor sleep, chronic illness, seizure disorder, and tic disorder
2. Excessive anxiety or depression may be comorbid or may be the main cause of behavioral and attentional difficulties. In the DSM-IV, four out of six listed symptoms for generalized anxiety disorder are similar to ADHD (restlessness, difficulty concentrating, irritability, sleep disturbance). Posttraumatic stress disorder can also simulate ADHD, but in such cases there is a traumatic event that precedes the onset of ADHD symptoms.
3. Specific learning disabilities: Language disorders, other learning disabilities, and borderline IQ can result in secondary attentional problems and in restlessness out of boredom because the child is not able to understand what is being taught. In ADHD-IA comorbid with the preceding problems, the cause of the attentional problems is difficult to determine.
4. Child abuse and/or a chaotic home: Although the existence of a dysfunctional environment does not rule out ADHD, and although the presence of ADHD is a risk factor for abuse because the child is so provocative, the presence of abuse or a chaotic home may result in a behavior disorder that mimics ADHD and oppositional behavior.
5. Oppositional disorder or conduct disorder: Although these may be comorbid with ADHD, they may also simulate ADHD. In teacher and parent ratings, there may be a halo effect onto ADHD symptoms.
6. Bipolar disorder or hypomania: These can be comorbid or simulate ADHD. Distinguishing features may be found in the family history, in earlier onset of ADHD, in the latter's chronic course and the absence of an elevated mood. ADHD and hypomania are both characterized by excessive activity, impulsive behavior, and poor judgment.
7. High level of activity: Some parents do not know what level of activity to expect, particularly in a boy. The toddler age is characterized by high activity and not staying long in one place; this activity may be mistaken for restlessness.

### Comorbidity

Patients are said to be 'comorbid' when they have more than one psychiatric disorder. This is true, whether or not one disorder caused the other or exists independently.

Attention deficit hyperactivity disorder is comorbid in a majority of children, both in clinics and the community ( [Biederman et al., 1991b, 1992b, 1993b, 1996c](#)). Comorbid ADHD is often more treatment resistant, and the child is less able to function than with ADHD on its own. It is also true that treatment of a child with comorbid ADHD often requires treatment of both conditions to be successful.



Comprehensive reviews are available that describe the details of the body of research that has been developed on the clinical presentation and treatment of ADHD comorbid with each of the other major disorders (Brown, 2000a; Pliszka et al., 1999). These include many disorders such as learning disability, central auditory processing, developmental coordination disorder, neurologic disorders, and medical disorders, which are less commonly included in discussions of comorbid psychiatric illness.

#### ATTENTION DEFICIT HYPERACTIVITY AND DISRUPTIVE BEHAVIOR DISORDERS

More than half of children with ADHD also meet the diagnostic criteria for another disruptive behavior disorder. The relationship among ADHD, oppositional defiant disorder (ODD), and conduct disorder (CD) has been the subject of considerable research in the last 5 years (Biederman et al., 1996e). Younger children with CD almost always have ODD and/or ADHD as well (Szatmari et al., 1989); however, only some children with ODD go on to have CD.

Children who have both ADHD and CD tend to have a lower socioeconomic status (Lahey et al., 1988b), higher prevalence of learning problems (McGee et al., 1984), and stronger family history of antisocial behavior (Faraone et al., 1997). Comorbid CD in addition to ADHD worsens the long-term prognosis, and leads to greater antisocial behavior, substance abuse, and aggression in adulthood (Mannuzza and Klein, 2000; Mannuzza et al., 1989; Weiss and Hechtman, 1993). Nonetheless, children with ADHD-CD can respond to stimulants, with a decrease in both ADHD symptoms, as well as a decrease in antisocial behaviors (Klein et al., 1997). Caution is warranted, however. In using stimulants to treat ADHD-CD, the clinician needs to insure that there is indeed comorbid ADHD, that neither the child nor others will sell or abuse the drug, and that the individual child does not suffer a worsening of aggression or CD symptoms when the medication is wearing off. It is also important to insure that parents understand that the prognosis of stimulant treatment of ADHD-CD is more guarded, especially for the CD symptoms, than the treatment of ADHD alone. This is particularly problematic when there is also substance use or abuse.

#### ATTENTION DEFICIT HYPERACTIVITY DISORDER AND DEPRESSION

Attention deficit hyperactivity disorder and depression-major depressive disorder (MDD) is common, both in children presenting for ADHD and in children presenting to the clinic for depression. There is a referral bias, so that there is a higher prevalence of ADHD-MDD in clinics than in the community. In addition to referral bias, this difference may also reflect differences in diagnostic practices in community and tertiary centers. Rates of diagnosis of depression in ADHD children vary markedly depending on whether the assessment is based on interview of the child, interview of the parent, a combination of both, rating scales, semistructured interviews, or clinical impression. Clinicians may dismiss depressive symptoms as “demoralization,” insisting that the child is reacting appropriately to rejection. They may also be reluctant to identify symptoms such as insomnia, irritability, mood lability, suicidal threats uttered in anger, decreased appetite, and poor concentration as evidence of depression, because many of these symptoms also can be understood as associated features of ADHD. There continues to be a lack of agreement regarding these issues and how they should be managed. As a result MDD is said to be present in anywhere from % to 75% of children with ADHD (Biederman et al., 1991b). Using a strict DSM-IV definition of depression and very rigorous diagnostic assessment, 6% of the children in the MTA study met criteria for a major depressive disorder (Pliszka, 2000).

Despite these caveats, there are children who have longstanding ADHD and also develop depressed mood of at least several hours duration on a daily basis and for at least 2 weeks. At times when such children also suffer psychomotor retardation, they may become lethargic and quiet, so that paradoxically when the depression lifts and they “bounce back” (literally), their teacher may describe their behavior in the classroom as worse. Children with ADHD-MDD may be tired, their attention may be even worse than their baseline, and they may have “given up.” Although it is not uncommon to see the emergence of such depression during developmental transitions, they cannot be dismissed as situational. In working with ADHD children who also become depressed, one becomes impressed with just how resilient many ADHD children are despite the difficulties they face every day.

Familial studies (Biederman et al., 1992a,b) found elevated rates of depression in both the first-degree relatives of children with ADHD and the first-degree relatives of children with ADHD-MDD. This suggested that there is a common familial link between ADHD and depression. Clinicians often find themselves treating a depressed parent when treating a child with ADHD. Conversely, it is not uncommon to find ADHD among the children of depressed mothers (Beardslee et al., 1983). Failure to treat depression in a parent could contribute to comorbid depressive symptoms in the offspring.

In treating children who have a longstanding history of ADHD and who are also clinically depressed, clinicians may use a stimulant, antidepressant, or both. Some clinicians prefer to use an antidepressant with some evidence of efficacy for both depression and ADHD such as bupropion (Casat et al., 1987; Conners et al., 1996) or venlafaxine (Olvera et al., 1996). If the ADHD does not respond, these medications can still be augmented with a stimulant. Another option is to combine stimulants with serotonin reuptake inhibitors (Gammon and Brown, 1993). Opinions differ as to whether or not in such circumstances the depression or the ADHD should be treated first. Some clinicians recommend treating the depression first (Weiss et al., 1999), noting that many depressed children with decreased appetite, insomnia, headache, and irritability will not tolerate stimulants; however, a recent practice algorithm recommended treatment of the ADHD first (Pliszka et al., 2000a,b). Research in this area is quite limited, with only two studies that demonstrate that depression in children and adolescents is responsive to SSRIs (Emslie et al., 1997, 1998, 1999; Keller et al., 1998).

#### ATTENTION DEFICIT HYPERACTIVITY AND BIPOLAR DISORDERS

This is an area of heated and unresolved controversy (Biederman et al., 1996d, 1998a; Carlson, 1997, 1999). Manic illness was classically understood to be episodic, euphoric, and distinct from periods of depression. In the DSM-III-R, and even the DSM-IV, the definition of bipolar illness and bipolar II illness evolved to include states that were chronic, of early onset, primarily irritable, and with labile mood as opposed to clear mania or depression. The differential between this difficult, stormy condition and ADHD continues to cause confusion among both clinicians and patients (Steele, 1998). This is particularly true because three of the symptoms of mania are identical to their counterpart symptoms of ADHD (distractible, talkative, hyperactive); the remaining symptoms are considered by many clinicians to be associated features of ADHD, and only three symptoms are required for a diagnosis of mania.

Attention deficit hyperactivity—bipolar disorder is described in epidemiologic studies as being quite rare (Costello et al., 1996a,b), whereas some clinical studies suggest that up to 16% of children presenting for ADHD also have mania (Biederman et al., 1996d; Wozniak and Biederman, 1997; Wozniak et al., 1995). Family genetic studies of ADHD versus ADHD-BP seem to indicate that ADHD-BP cosegregates (i.e., first-degree relatives are likely to have ADHD and BP). This literature as well as studies that look at depressed children and adolescents or young adults who go on to have bipolar illness (Akiskal et al., 1995; Strober et al., 1995) seem to strongly suggest that there is a group of ADHD children and adults who have explosive outbursts, severe comorbidity, and require multiple hospitalizations. These patients are labile, irritable, aggressive, and have difficult temperaments. Further study of this population, including long-term follow-up into adulthood, would be of great benefit in terms of better understanding these children and their management. Whatever the prevalence of this disorder, it can certainly be considered demanding of treatment resources and expertise.

There is a paucity of empiric research to guide treatment of ADHD-BP. Lithium, anticonvulsants, antidepressants, stimulants, and atypical neuroleptics have all been widely used in this population of children. Whether or not the clinician prefers a diagnosis of severe ADHD or ADHD-BP, there is good agreement that these children are extremely ill, at risk, and hard to help. They often end up in the hospital. They demand aggressive treatment, but at the same time this population is at risk for overdoses and substance use. We also know very little about the long-term side effects of mood stabilizers, such as weight gain or polycystic ovarian syndrome from valproate.

In the meanwhile, clinicians often treat these patients empirically with careful, sequential, intensive, and combination therapies. Further research may provide more information as to whether stimulants exacerbate the mood cycling of these patients, or whether the disinhibiting and activating effects of serotonin reuptake blockers exacerbate their ADHD (Findling, 1996; Hoehn-Saric et al., 1990, 1991; Riddle et al., 1991; Strober et al., 1995). Guidelines for clinical management of ADHD-BP are available elsewhere (Pliszka et al., 1999; Weiss et al., 1999).

#### ATTENTION DEFICIT HYPERACTIVITY AND ANXIETY DISORDERS

The Multimodal Treatment of ADHD (MTA) study (The MTA Cooperative Group, 1999a) found that one-third of the children referred with ADHD met criteria for an anxiety disorder. Family genetic studies indicate that anxiety and ADHD are separate disorders and that they segregate independently (Biederman et al., 1991a, 1992a; Perrin and Last, 1996). Consistent with this hypothesis, children with ADHD-ANX share the social problems associated with anxiety disorders (Tannock, 1994).

Several investigators have looked at whether comorbid anxiety impacts on the effectiveness of stimulants. Clinicians have had the impression that the presence of anxiety in addition to ADHD often predicts poor stimulant response, and more side effects, and this was demonstrated in a series of clinical trials of stimulants with ADHD-ANX children ([Pliszka, 1989](#); [Taylor et al., 1987](#)). [Tannock and colleagues \(1995, 1996\)](#) found that comorbid anxiety predicted less behavioral improvement and the behavioral improvement that was seen declined over a 1-year follow-up. ADHD-ANX also showed less improvement in working memory when treated with stimulants, as compared the ADHD without anxiety. Finally, Tannock found that children with ADHD-ANX had more side effects with stimulant treatment than children with ADHD only. These results led Tannock to suggest that ADHD-ANX represented a meaningful subtype, with a different stimulant response profile to ADHD alone ([Tannock, 1994](#)).

It is interesting that neither the MTA nor a carefully designed study from Tannock's group ([Diamond et al., 1999](#)) replicated these findings. What the MTA did show was that ADHD-ANX children benefited more from medication and psychosocial treatment than medication alone. This result supports Tannock's hypothesis that ADHD-ANX represents a distinct subtype, with a different treatment response profile, and that treatment of this subtype may require both behavioral treatment of the anxiety and medication treatment of the ADHD. At the time of writing, systematic clinical trials are in process to look at the response of children with anxiety disorders. As we learn more about the treatment of anxiety disorders in children, we may also become more adept at treating child anxiety disorders when comorbid with ADHD ([Birmaher et al., 1994](#); [Fairbanks et al., 1997](#); [Geller et al., 1996](#); [Rocca et al., 1997](#)).

#### ATTENTION DEFICIT HYPERACTIVITY DISORDER AND LEARNING DISABILITIES

Learning disability (LD) has been defined in various ways. For example, it has been defined as a discrepancy between learning potential (IQ) and learning achievement on formal psychoeducational testing. The disparity may be defined as greater than 1.5 or 2 standard deviations from the mean ([Semrud-Clikeman et al., 1992](#)). The more conservative the definition, the lower the prevalence of children who will qualify as having learning disability. It also needs to be said that schools and government may use these definitions to define access to special education resources.

Children can underachieve academically for many reasons, such as lack of motivation, ill health, poor teaching, inappropriate educational placement, or language barriers. If one defines LD as disparity between IQ and achievement, then children may be precluded from early intervention, which could prevent more serious LD later. Use of this definition by schools therefore runs the risk of precluding children from being eligible to receive educational resources until they are so far behind that the resource is no longer likely to be effective.

A second definition of LD assumes that the child must be both underachieving for his or her potential and also achieving poorly. This definition of LD excludes very bright children, who are underachieving relative to their own potential, from being eligible for resources. Definitions of LD also vary as to whether they refer to more global learning impairments (overall underachievement) or only specific learning impediments such as dyslexia, dyscalculia, or dysgraphia. Again, this distinction is quite important because problems with executive function in the absence of other psychiatric symptoms would be considered a learning disability by broader definition.

The prevalence of comorbid LD with ADHD varies depending on the definition used, anywhere from 10% to 90% ([Semrud-Clikeman et al., 1992](#)). Research on reading disability (RD) and ADHD indicate that these are two distinct disorders that can be inherited independently ([Faraone et al., 1993](#); [Gilger et al., 1992](#)). Both research and clinical expertise suggest that RD alone does not respond to stimulants, but that ADHD-RD responds to stimulant treatment of the ADHD combined with special education remediation of the RD. Similarly, treatment of the ADHD alone will not lead to improvement of the RD, unless the child also has the opportunity to receive appropriate educational remediation ([Kupietz et al., 1988](#); [Richardson et al., 1988](#)).

Stimulant medication has been shown to have a specific effect on some aspects of LD. There is evidence to suggest that stimulants improve working memory ([Tannock et al., 1995](#)) and handwriting ([Schain and Reynard, 1975](#)). These effects may impact on written output, math computation, examination skills, and various other aspects of academic performance.

#### ATTENTION DEFICIT HYPERACTIVITY DISORDER AND MENTAL RETARDATION

ADHD exists together with mental retardation (MR) in rates higher than the general population. Research in this area is inconsistent as to whether symptoms of disruptive behavior should be measured against the child's developmental age, rather than chronological age ([Pearson and Aman, 1994](#)). Some measure of clinical judgment is required. Adults with MR are not expected to behave as young children, nor are young children with MR expected to have attention and behaviors consistent with their chronological age. Children with ADHD-MR have been shown to respond well to stimulants in double-blind trials ([Mayes et al., 1994](#)).

It is also imperative that children with a borderline IQ have the opportunity to receive both psychological testing and appropriate educational placement. Certainly, it would be inappropriate to diagnose a child as having ADHD-IA on the basis of not paying attention to material that he or she cannot understand. We have found that this differential is becoming increasingly important and common in the face of greater awareness of ADHD-IA by teachers, and in the face of limited special education resources and psychological testing ([Aman et al., 1991](#)).

#### ATTENTION DEFICIT HYPERACTIVITY DISORDER AND TOURETTE'S SYNDROME

A recent study looked at over 3,500 children referred for treatment of Tourette's syndrome (TS) and found that ADHD was the most common comorbid condition ([Freeman et al., 2000](#)). ADHD accounted for much of the anger, sleep, and behavior difficulty associated with TS. It should be noted that by contrast in a study of a nonreferred community sample, only one in 12 subjects who met diagnostic criteria for TS also met diagnostic criteria for ADHD ([Apter et al., 1993](#)). The implication is that patients with TS are more likely to be referred for psychiatric assessment if they also have comorbid behavior problems, and the comorbid symptoms account for significant aspects of their impairment. A family genetic study ([Apter et al., 1993](#); [Pauls et al., 1993](#)) indicates that TS and ADHD are genetically independent; therefore, ADHD and TS cannot be considered variant phenotypes of a common gene, as was previously hypothesized ([Comings et al., 1990](#)).

Stimulant treatment of ADHD in the presence of tic disorder or TS can be beneficial in helping to ameliorate impairment from ADHD ([Freeman, 1997](#)). Recent research strongly suggests that stimulants do not worsen tics, and are not contraindicated ([Gadow et al., 1995a,b](#); [Nolan and Gadow, 1997](#)). Gadow's findings were confirmed in an independent clinical trial conducted by [Castellanos and associates \(1997\)](#). He found that although usual doses of stimulants were not statistically correlated to an increase in tics, unusually high doses were. He also noted that children who show worsening of tics on one stimulant might not show this effect with a different stimulant. It has become our clinical practice to provide patients with written summaries of this research in order to assure their informed consent, and carefully evaluate outcome of both ADHD and TS with each child. Treatment strategies may be directed at either condition alone, or both conditions in combination.

#### ATTENTION DEFICIT HYPERACTIVITY DISORDER AND DEVELOPMENTAL COORDINATION DISORDER

Developmental coordination disorder (DCD) is defined in the DSM-IV as motor coordination markedly below developmental norms, which causes impairment with activities of daily living. Specific screening tests are available, such as the Test of Motor Impairment and the Movement Assessment Battery for children ([Gillberg and Kadesjo, 2000](#)). Many studies indicate that there is a strong relationship between DCD and ADHD, such that about half of the children diagnosed as having one of the disorders also meet the diagnostic criteria for the other. This association is clearly much stronger than would be expected by chance alone, suggesting that difficulty with inhibition ([Barkley, 1997](#)) may impact on motor output as well as behavior and attention. The association between DCD and ADHD has been of particular interest in the Nordic countries, where a unitary diagnostic category, Deficits in attention, motor control, and perception (DAMP) is commonly used to describe these children. Deficits in attention, motor control, and perception have been well validated and subjected to epidemiologic ([Kadesjo and Gillberg, 1998](#)) as well as clinical ([Hellgren et al., 1994](#)) follow-up studies. The clinical implication of this work is to alert clinicians to appreciate that clumsiness may be associated with severe impairment, and should be referred to an occupational therapist. Treatment should involve provision of educational and psychological support, as well as individually designed training programs and school accommodations.

#### ATTENTION DEFICIT HYPERACTIVITY AND NEUROLOGIC OR MEDICAL DISORDERS

Several conditions are either likely to be encountered in clinical settings, or are likely to be raised by families as relevant to ADHD. A brief comment and reference is included to summarize current clinical and research expertise in these areas. It should be noted that although the clinician will encounter these comorbid presentations, they do not account for the majority of ADHD cases, most of which remain constitutional in origin.



## Epilepsy

Epileptiform discharges can be found in up to 3.5% of otherwise normal children and do not by themselves indicate epilepsy ( [Pliszka et al., 2000](#)). Children with ADHD have a somewhat higher prevalence of nonspecific electroencephalogram abnormalities ( [Kuperman et al., 1996](#)), but this has not been found to be predictive of clinical correlates or outcome. Children with ADHD and a seizure disorder are as responsive to treatment with stimulants as children without a seizure disorder. Stimulants do not lower the seizure threshold, and they do not affect anticonvulsant blood levels. Nonetheless, cautious clinicians may elect to do a prestimulant and poststimulant EEG in the individual child.

## Head Injury

There is no empirical evidence that mild head injury leads to ADHD, although it would not be surprising to find that children with ADHD have more head injuries because they are more accident prone ( [Bijur et al., 1990](#)). Severe head injury is associated with ADHD at 2-year follow-up, and up to 20% of such children show cognitive and behavioral effects ( [Gerring et al., 1998](#); [Max et al., 1998a,b](#)). These children often show features of disinhibition or a frontal lobe syndrome and should be diagnosed as having a personality change secondary to a medical condition. A trial of stimulant treatment is warranted, and has been found to be helpful in many but not all cases ( [Gualtieri, 1991](#)).

## Perinatal Complications

Studies of the relationship between perinatal complications and ADHD are inconsistent, some showing an association ( [Milberger et al., 1997c](#)) and some not ( [McGee et al., 1984](#)). ADHD may be a long-term outcome of very low birth weight, with up to half of these children showing difficulty with attention deficit or hyperactivity at age 5 ( [Astbury et al., 1987](#)). Infants who are small for gestational age (SGA) may also be at risk for later ADHD ( [Schothorst and van Engeland, 1996](#)).

## Thyroid

Attention deficit hyperactivity disorder may be more prevalent in patients with generalized resistance to thyroid hormone (GRTH) ( [Hauser et al., 1993](#)), although replication of this study did not confirm the finding. The converse is not true: GRTH is exceedingly rare as a cause of ADHD ( [Elia et al., 1994](#); [Weiss et al., 1993](#)), so that general screening for thyroid problems is only recommended in the presence of symptoms of thyroid illness.

## Diet, Allergy, and Asthma

Research has not confirmed any association between diet ( [Gross et al., 1987](#)), food additives ( [Conners, 1989](#); [Conners et al., 1980](#)), sugar ( [Wolraich et al., 1994](#)), allergy ( [McGee et al., 1993](#)), or asthma ( [Pliszka et al., 1999](#)) and ADHD over that expected by chance. Patients often come in with strong convictions that these disorders are associated with worsening behavior. Unfortunately, this has had the effect of submitting children and families to expensive and onerous treatments that are not effective, and preventing access to treatments that are effective.

## Exposure to Toxins In Utero

Fetal alcohol syndrome (FAS) and fetal alcohol effects (FAE) include craniofacial abnormalities and behavior symptoms. Many children with these syndromes also meet diagnostic criteria of ADHD ( [Coles et al., 1997](#); [Oesterheld and Wilson, 1997](#); [Steinhausen et al., 1993](#)). There are no studies comparing treatment response and dose response with stimulants in this population, but stimulants are commonly used in treatment. It has also recently been demonstrated that even low doses of alcohol may also lead to later difficulties with attention and behavior ( [Olson et al., 1997](#); [Schneider et al., 1997](#)). This implies that children with FAE and/or children who do not have FAS craniofacial abnormalities but were exposed to alcohol *in utero*, could still have neuropsychiatric sequelae of alcohol exposure.

A study on outcome of children exposed to cocaine *in utero* found deficits on attention but not behavior ( [Richardson et al., 1996](#)).

There is research supporting the idea that exposure to nicotine *in utero* also contributes to risk of ADHD in offspring ( [Milberger et al., 1996](#), [1997b](#), [1998](#)).

## Lead

Lead poisoning has been associated with cognitive and behavioral disturbance in children but only accounted for 1% of the variance after the psychosocial variables were accounted ( [Fergusson et al., 1988a,b,c](#)).

## IMPLICATIONS OF COMORBIDITY

[Peter Jensen and colleagues \(1997\)](#) proposed seven criteria that would define whether ADHD in the presence of a particular comorbid condition should be considered distinct from ADHD alone. These criteria included the presence of a distinct clinical picture, demographics, psychosocial factors, biological factors, genetics, family environment, course and outcome, and response to specific treatments. Applying this definition to a complete review of the literature led them to conclude that ADHD-CD and ADHD-ANX should be considered distinct syndromes.

Study of comorbidity is expected to be of major clinical and research importance in the future. Clinically, it is apparent that clinicians need to be aware of the treatment implication of particular comorbid presentations. Families need to be aware that functional improvement for a child with ADHD and a serious comorbid condition may be limited by the impact of the comorbidity. Clinical practice needs to provide for assessment of a wide range of symptoms, both at assessment and outcome evaluation. Strategies for management of comorbid conditions need to include practice guidelines for evaluation of the impact of one change at a time, and changes in symptoms, functioning, and development. These strategies need to be based on clinical trials that are specifically designed to determine outcome for a comorbid presentation, with treatment for the first disorder, second disorder, both, and the sequence of treatment.

Research on ADHD is complicated by the presence of various comorbid conditions in a majority of ADHD children. Neuroimaging studies and genetic studies have strengthened our understanding of ADHD and comorbid conditions. As illustrated, knowledge of whether ADHD and a comorbid condition segregate independently (as in ADHD-ANX; ADHD-LD) or together (as in ADHD-BP) has significant implications for understanding etiology and treatment. Conversely, it may well be that further research on the neurobiology of ADHD is going to require identification of distinct clinical populations. For example, studies of the D4 or DAT1 gene or neuroimaging studies looking for anatomic or metabolic differences in children with ADHD versus controls may find stronger effects if they look at a "pure" ADHD population. On the other hand, a study of environmental toxins might find greater effects if they identify a group of children who do not carry any of the ADHD genetic markers and do not have a strong family history.

## THEORETICAL CONCEPTS OF ATTENTION DEFICIT HYPERACTIVITY DISORDER

### Historical Conceptualizations of Attention Deficit Hyperactivity Disorder

Still originally argued that the children he described in 1902 had a deficit in volitional inhibition and a defect in moral control. It was almost three-quarters of a century later that Paul Wender began the work on minimal brain dysfunction that laid the foundation for contemporary work. Wender's work is a landmark that comprised major innovations, such as:

1. Describing the syndrome as a developmental disorder
2. Publishing the first easy to understand patient material
3. Recognizing that the disorder extends through the life cycle and into adulthood in some patients
4. Conducting clinical trials of various medications at a time when clinical trials in child psychiatry were in their infancy
5. Conceptualizing MBD as a neuropsychiatric syndrome in which symptoms per se would be unlikely to remit with psychotherapy
6. Recognizing the importance of developing firm age-appropriate diagnostic criteria and rating scales

## 7. Delineating the phenomenology of the core syndrome and its associated features

In this regard, Wender's historical position is of considerable interest, as is his theoretical understanding of the disorder. Wender argued that MBD comprised six dimensions of dysfunction: motor, attention and perception, learning, impulse control, interpersonal relations, and emotional dysregulation. He suggested that these children were active, extroverted, and not easily conditioned by normal consequences. Wender went on to develop diagnostic criteria for adult ADHD that included such difficulties as hot temper, emotional reactivity, and affective lability (Wender, 1995). The result was an accurate description of the *breadth* of ADHD symptoms. By contrast, DSM focuses on those symptoms, which operationalize the concepts of attention, hyperactivity, and impulsivity while carefully excluding items that overlap with mood disorders, aggressive items that overlap with conduct disorder, cognitive difficulties that overlap with a thought disorder, or aspects of interpersonal difficulty associated with personality disorders.

Douglas (1972) was among the first of the great founders who tried to design experiments to test a theory. Her experimental work led her to believe that a *dysregulation* of attention was fundamental to the difficulties of these children. It was partly as a result of her work that the disorder was then renamed attention deficit disorder. She further described difficulty with previously undescribed aspects of cognitive functioning such as problems with effort and motivation, poor modulation of arousal, and a tendency to seek immediate gratification. She grouped all of these difficulties under one basic concept: ADHD represented an impairment of self-regulation. She is one of the pioneers who then pushed interest in ADHD into the arena of neuroscientists exploring the impact of difficulties with executive function.

### Barkley's Theory of Attention Deficit Hyperactivity Disorder

Barkley (1999c) argues that the fundamental impairment of ADHD is a deficit in delayed responding. Barkley developed a schema in which he illustrated how a deficit in behavioral inhibition then leads to the other problems associated with ADHD (Fig. 52.1). Behavioral disinhibition is seen as fundamental to problems with working memory, self-regulation of affect, motivation and arousal, internalization of speech (and in turn capacity for rule governed behavior, moral reasoning, and reflection), and finally reconstitution. Reconstitution includes problems with verbal fluency, goal-directed behavior, analysis, and synthesis. These ideas represent a landmark in that they represent the first self-conscious attempt to enunciate a theory of ADHD that could be tested, and to present that theory to the public at large.

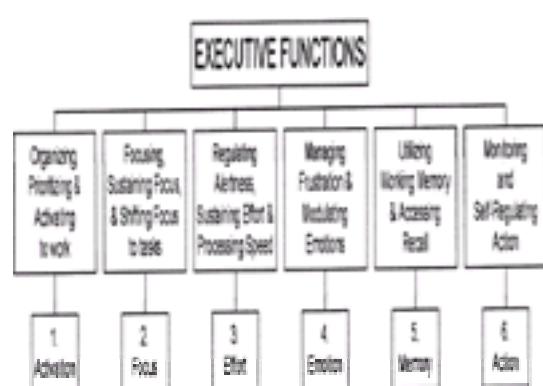


**Figure 52.1.** A model of the impairments in executive functions predicted to be associated with the deficits in behavioral inhibition that characterize ADHD. From Barkley RA: *ADHD and the Nature of Self-Control*. New York: Guilford, 1997. Copyright by Guilford Publications. Reprinted with permission.

Barkley's (1999c) theory is meant to describe children with ADHD-combined type. Barkley argues that children with ADHD-IA type who are lethargic rather than hyperactive and withdrawn rather than disinhibited may have a distinct disorder.

### Other Current Concepts of Attention Deficit Hyperactivity Disorder

Barkley's (1999c) work has also been heuristic in inspiring debate and discussion with other clinician researchers. Brown (2000a), Douglas (1999), and others (Sergeant et al., 1999) have become interested in problems with attention and difficulties with executive function as a primary source of difficulty. Brown (1996, 2001) has developed rating scales for children, adolescents, and adults that include various empirically derived dimensions of executive function. His six dimensions are illustrated in Fig. 52.2.



**Figure 52.2.** Brown's dimensions of executive function. *Brown Attention Deficit Disorder Scales, Manual*. Harcourt Brace and Company, San Antonio, TX, 1996. Permission granted by Psychological Corporation.

This work has generated new interest in adults with attention difficulties in the absence of overt hyperactivity, and raised questions about whether ADHD may lead to language and reading problems independent of learning disabilities (Purvis and Tannock, 2000).

Brown's model of ADHD differs from Barkley's in that behavioral inhibition is not considered to be primary or superordinate over other aspects or components of executive function. His model is meant to be inclusive of all the ADHD subtypes, where Barkley's model is specific to the combined type. Barkley argues that not all problems with attention are ADHD, and that it is incumbent on the clinician to demonstrate the specificity of an attention deficit in the absence of problems with delayed responding by ruling out the presence of any other learning or psychiatric disorder (Gordon and Barkley, 1999). This debate has important clinical implications. We now turn to what is known about the subtypes of ADHD.

### EPIDEMIOLOGY OF THE SUBTYPES

The change to the DSM-IV diagnostic system with the creation of the three subtypes increased the prevalence of ADHD from 3% to 5% (DSM-III-R) to about 12% (Baumgaertel et al., 1995; Wolraich et al., 1996). This is partly because of the addition of ADHD-IA, which itself has a prevalence of between 5.4% (Wolraich et al., 1996) and 9% (Baumgaertel et al., 1995). The DSM-IV subtypes permitted inclusion of more females, preschoolers, and adults who would have been excluded by previous DSM diagnostic criteria but who were found to have significant impairment.

Since the DSM-IV criteria came into use, it has become increasingly evident that further work is necessary to determine whether these two new ADHD subtypes represent changes in the presentation of ADHD at different developmental stages or unique disorders. There have been six clinic-based studies (Eiraldi et al., 1997; Faraone et al., 1998b; Lahey et al., 1994; McBurnett et al., 1995; Morgan et al., 1996; Paternite et al., 1996) and three community-based studies (Baumgaertel et al., 1995; Gaub and Carlson, 1998; Semrud-Clikeman et al., 2000). A metaanalysis of these nine studies (Carlson et al., 1999) found that there are clear differences in



the clinical correlates that characterize the various subtypes.

ADHD-HI is more common in preschoolers, and represents only about 2% of clinic ADHD populations. It is unclear at this point whether these children are simply a younger variant of the combined type (ADHD-C) who do not meet the inattentive diagnostic criteria because they have not yet encountered demands for attention in classroom settings. Children who are just hyperactive may improve with time because hyperactivity alone tends to remit with age. It is difficult to study this population because the prevalence is low.

The clinic-based studies show some consistent differences when compared with the community studies (Table 52.2). Clinic studies (Lahey et al., 1994) show a pattern in which the children with ADHD-HI are of preschool or early elementary age, children with ADHD-C are of intermediate elementary school age, and children with ADHD-IA are of late elementary or early high school age. This is not found in community studies (Gaub and Carlson, 1998). The discrepancies in age between community and clinic studies may reflect differences in when a child is referred for evaluation, rather than differences in age of onset. The age of referral may be a marker for when a particular group of symptoms presents the maximal developmental challenge. Overt hyperactivity, for example, decreases with age, but inattention does not (Lahey et al., 1988a). The mean age of a diagnostic group, therefore, may reflect referral bias based on whether the child is causing problems for others, as opposed to experiencing academic delays.

Clinical	Community
Age is older for IA	Age of IA and CB is the same
Prevalence of IA: 27%	Prevalence of IA: 50%
Gender	Gender
IA 2:1 boys/girls	IA 2:1 boys/girls
C 5:1 boys/girls	C 3:1 boys/girls
Comorbidity	Comorbidity
IA more internalizing problems than C, and more language problems	IA less internalizing problems than C
References	References
Erkild, 1997; Faraone, 1998; Lahey, 1994; McBurnett, 1995; Morgan, 1992; Faraone, 1996	Baumgaertel, 1995; Gaub and Carlson, 1997; Wolach, 1996

**Table 52.2. Clinical versus Community Samples**

Only in clinic populations, and only in some studies (Lahey et al., 1984), are children with ADHD-IA found to have more internalizing problems than children with ADHD-C. This suggests that children who have difficulty with attention in the absence of any other psychiatric problems are unlikely to be referred for psychiatric help or are referred later. Clinic and community studies also differ on whether depression and anxiety were identified by an interview of the child, or parent and/or teacher report on rating scales.

Clinicians may be making the assumption that because ADHD-IA children are “ADHD,” that their difficulties, prognosis and response to treatment are the same as those of children with ADHD-C; however, given the distinct clinical correlates of ADHD-IA, this should be subjected to empiric study. Children who only have attention problems or problems with hyperactive/impulsive behaviors may be quite different from children who have difficulty in all three domains. They may require different psychosocial treatments, different doses of medication, and have a different medication response profile. Further, we have never studied, and know little about, the developmental course of ADHD-IA or ADHD-HI.

The DSM-IV field trials did not attempt to determine the specificity of the ADHD-IA diagnosis, that is, whether these children might have secondary difficulties with attention as a result of other problems, such as learning disabilities, poor educational placement, language problems, depression, and so on. It may be that the diagnostic criteria of the DSM-IV are sensitive, but not specific. Clinicians may be using the inattentive-type diagnosis as a diagnosis of exclusion, that is, a diagnosis made only in the absence of other conditions that might also predict attention problems.

## TREATMENTS

### Psychosocial Treatment

#### INTRODUCTION

##### Interventions

These form a broad-based range of interventions that include all interactions between the therapist, child, and family, other than medication. The following interventions are summarized:

- Psychoeducation
- Parent training in behavioral management skills
- Classroom interventions
- Contingency management
- Cognitive behavior therapy
- Social skills training
- Individual psychotherapy of the child

Of the preceding treatment modalities, parent training, classroom interventions, contingency management, and recently social skills training have demonstrated efficacy (Pelham et al., 1998; Pfiffner et al., 2000).

Psychosocial treatments are generally combined with medication because alone, they have only a weak effect on the core symptoms of the disorder. However, medication alone cannot address parental concerns around child management issues such as the daily battles over homework, getting to bed, how to deal with critical neighbors or exasperated teachers, or how to help the child with peer rejection. Nor does medication alone address chaotic family functioning, disorders in parents affecting the family or child abuse. Medication use requires a discussion of parental fears about harm to the child and ongoing discussion about dose adjustments, side effects, and how the child feels about taking pills. Attention deficit hyperactivity disorder is a complex disorder affecting every area of functioning and requires a comprehensive treatment program.

#### PSYCHOEDUCATION

Psychoeducation is an essential component of the treatment of ADHD. It is treatment based on attention to the whole family unit and intensive support and education of the parents, over the years. All interventions are aimed at developing an ongoing therapeutic alliance between therapist, the child and his or her family, in an atmosphere that is nonjudgmental and calm.

Psychoeducation begins at the first assessment interview, when the purpose of the evaluation is made clear to the child and parents. A diagnosis of ADHD has great impact on both the child and his or her parents, usually arousing positive and negative feelings. On the positive side, there is nonjudgment and a better understanding of past difficulties in the light of the new information. On the negative side, the child is now labeled and “different.” Each family experiences the impact of the diagnosis differently, and psychoeducation provides the necessary support and information required. All uncertainties about the nature, causes, treatment, and prognosis of the disorder are discussed. The treatment plan is outlined in the context of active child and parent participation. This is an ongoing activity as treatment response is assessed and interventions are modified. The use of medication generates fears in many parents; these are aired and discussed. Accurate information of ongoing treatment studies is provided. Whether children interpret the effects of their medication as being the result of the drug, or a change they have directed (attribution) is a concern. Charlotte Johnston and colleagues (2000) have actively researched this area in recent years. It is important that the physician explains to the child and his

or her parents that the medication gives the child the potential to improve, but that the effort must still come from them.

The therapeutic alliance becomes increasingly one of mutual respect and equality as information is shared by the parents about themselves and their child, the child about his or her world, and the therapist about all aspects of the disorder. Psychoeducation forms the background for all other interventions, and for many, together with supportive interviews and ongoing information from teachers, may be the only treatment other than medication.

### SCHOOL INTERVENTION

School intervention has received considerable attention and only highlights can be discussed in this chapter. Contact with the school is a required part of the diagnosis but may be limited to the teacher completing a rating scale. Physicians may lack the time to establish adequate contact with the child's teacher. Ideally, the therapist should meet the child's teacher initially and at periods when this is helpful. These meetings have the purpose of insuring that the teacher understands the disorder, feels positive toward the child despite the behavior, understands the pros and cons of medication, and becomes an active participant in treatment. The child needs to be situated in a seat where he or she can be more intensively supervised, which is usually in the front of the classroom and some distance from other disruptive children. A psychoeducational assessment is required for some children, leading to specialized and individualized learning assistance, one-on-one tutoring, peer tutoring, or use of a computer.

Parents need help to learn how to be advocates for their children and avoid a negative feedback system with the teacher because they are disappointed with the school system. When such a conflict exists between parents and teacher, the therapist may be helpful as a mediator.

A simple to use behavioral tool is the daily report card (DRC). This is an intervention in which the teacher observes the child's academic output and behavior throughout the day. The child is rewarded; for example, after the morning and at the end of the school day by receiving tokens such as a card or star. These are brought home and exchanged for a tangible reward by the parents. An additional star can be given daily for the child accurately assessing him or herself as to work completed and behavior. This method is particularly useful because it promotes increased supervision from the teacher and positive reinforcement for the desired work habits and behavior. It also leads to more positive emotional interaction between the teacher and child. When teachers object that they do not have the time because they have so many other children in their class, they are asked to try it out because by reducing classroom disruption, it may save them time in the end. If positive reinforcement is not sufficient to produce the required results, then the parents may add negative consequences (mild punishments such as withdrawal of privileges) ([DuPaul and Stoner, 1994](#)).

Trained behavior therapists may set up a token management program of the child in the classroom with a similar program being set up at home.

The reader may refer to a recent review by [Hoffman and DuPaul \(2000\)](#) for more details on types of school interventions.

### PARENT TRAINING IN TECHNIQUES OF BEHAVIOR THERAPY

This may be carried out with groups of parents or individual parents. Parent groups are more efficient and provide group acceptance and support, but techniques learned in the group may need to be reinforced in individual parent sessions in order to be adequately implemented in the home environment.

Parent groups are usually set up with about eight sets of parents, and are held weekly for eight to 20 sessions. Parents are assigned readings and homework to practice specific skills ([Pelham and Waschbusch, 1999](#)). In our experience, the more active the parent's participation is in the group, the better the results ([Cousins and Weiss, 1993](#)). It must be remembered that for some parents didactic teaching is not useful, particularly for those parents who themselves have ADHD ([Weiss et al., 2000a](#)). These parents have a long history of "tuning out" in a classroom situation. Their difficulties with following routines, organizational skills, poor attention to detail, make the carrying out of a behavioral program difficult or impossible unless they too are treated.

Most parents are aware that their current way of handling their child is not working and are eager to learn new skills. In the group meetings parents are taught that reinforcements of desired behavior and lack of attention or negative consequences of undesired behavior can change behaviors. Positive reinforcement, for example, a later bedtime or token leading to later reward, may be used alone or together with negative consequences (mild punishments). Rewards and consequences are usually given daily. The parents are taught to "catch the child being good" and to recognize that angry, prolonged attention to the negative behavior with inconsistent consequences may actually reinforce the behavior.

Rewards and negative consequences are carefully selected with the child as an active participant. Many children choose time alone with the parent of choice, for example, 10 minutes doing an activity chosen by the child without receiving any criticism as the positive reinforcer. Access to screening activities (computer games, TV, Nintendo, etc.) has recently become a popular reinforcer. "Time out" is a common consequence that may be impossible to carry out with older children.

Behavior modification is an effective treatment ([Pelham and Waschbusch, 1999](#)), but the effects gradually dissipate when therapy stops and it has no additional effect on the core symptoms of the disorder when the child is on stimulants. For this reason, behavior therapy is usually combined with medication, and is particularly helpful for younger children. [Pelham and Fabiano \(2000\)](#) provide a recent, more comprehensive description of behavior modification.

### CONTINGENCY MANAGEMENT

Contingency management is a form of behavior therapy used in special programs such as special classroom settings, summer camps for children with ADHD, such as those run by [Bill Pelham and associates \(1997\)](#) and inpatient units. Treatment in these settings is intensive and extensive. The behavioral component is based on a token point system with reward and cost components for a range of behaviors implemented in every activity throughout the day. In these programs, contingency management is combined with medication and other psychosocial treatments such as parent training and social skill enhancement. The efficacy of these programs has been demonstrated ([Pelham and Fabiano, 2000](#)).

### COGNITIVE BEHAVIOR THERAPY (OR SELF-INSTRUCTION INTERVENTIONS)

It was hoped that teaching children how to self-regulate their behavior and modify their self-talk would lead to better rule-governed behavior. Programs were designed to teach children techniques of problem solving, dealing with anger and frustration, persistence, and social skills ([Braswell et al., 1991](#)); however, the gains made during the program were not maintained at follow-up, confirming various previous reports that little benefit remained when training was discontinued ([Abikoff, 1985](#)). The reasons for this are probably complex, and include insufficient attention being paid to the generalization of the learned skills and the immaturity of executive verbal functioning of ADHD children.

### SOCIAL SKILLS TRAINING

Impaired social functioning is one of the most debilitating aspects of children with ADHD and also the most resistant to treatment ([Whalen and Henker, 1992](#)). Social skills training (SST) programs have been refined over time and increased emphasis has been placed on the need to incorporate ways to ensure generalization of learned skills into the natural environment of the child by incorporating teachers and peers. As a result, there is now evidence of treatment efficacy ([Piffner et al., 2000](#)).

Social skills training programs have generally been carried out on small groups of school-aged children using both cognitive behavioral and behavioral interventions. These programs may include modeling, didactic instruction, symbolic play (e.g., with puppets) *in vivo* practicing, role-play, and behavioral rehearsing. Children are taught to evaluate their own behavior in the group and school yard. Prosocial behaviors are taught and practiced, and contingency management is used to increase prosocial behaviors and eliminate unwanted responses.

Linda Piffner and coworkers use six modules, related specifically to the social difficulties of ADHD children, including good sportsmanship, for example, following the rules, not leaving the game, accepting consequences gracefully, assertiveness, ignoring verbal and nonverbal provocation, problem solving, and recognizing and identifying feelings in self and others. Gains lasting 3 to 4 months after treatment stopped were recorded ([Piffner and McBurnett, 1997](#)).



## INDIVIDUAL PSYCHOTHERAPY OR PLAY THERAPY

No empiric data exist on the effectiveness of this form of treatment and it would not be expected to improve the core symptoms of the disorder. [Greenfield and colleagues \(1992\)](#) described modifications useful for treating secondary difficulties commonly seen in children with ADHD, such as intense sibling rivalry, low self-esteem, and feelings of alienation. Feeling understood and confiding in a helpful nonjudgmental adult and developing the hope of change may result in clinical improvement, although this has not been demonstrated ([Stubbe and Weiss, 2000](#)). The authors have found that brief individual psychotherapy with the child can establish the foundation of a long-term therapeutic relationship, which contributes to good long-term management of the child with ADHD and his or her family.

## Medication Treatment

### PHARMACOEPIDEMOLOGY OF MEDICATION USE

There has been a continuous and dramatic rise in the use of stimulants in North America, representing at least a doubling in the number of prescriptions every 5 years, or as many as 1.5 million children receiving medication for ADHD ([Safer et al., 1996](#)). This has raised concern in the public and the media about whether or not this increased use represents an inappropriate use of medication for behavioral control, although the prevalence statistics for use of stimulants do not extend beyond the prevalence of ADHD itself. Up to three-quarters of children with a diagnosis of ADHD are currently prescribed medication. This means that there are many children with ADHD who are not treated with stimulants, and also children without ADHD who do receive treatment ([Angold et al., 2000](#)). This is not entirely surprising because the indication for stimulants is a group of symptoms, and not necessarily the diagnosis.

The rise in the number of stimulant prescriptions can be attributed to an increase in both the population of patients treated, and the way in which medication is being prescribed. At the present time there is a significant increase in use of stimulants to treat preschoolers ([Rappley et al., 1999](#)), adolescents ([Safer and Krager, 1994](#)), and adults ([Morrow, 1997](#)). In addition, there has been a rise in the rate of stimulant treatment for patients with a diagnosis of ADHD-predominantly inattentive type from 7% (1970s) to 20% (1990) ([Safer and Zito, 2000](#)). This trend has probably continued to grow in the last decade, even though clinical trials demonstrating efficacy are still needed. There has also been a considerable increase in the use of stimulants to treat children who have diagnoses of ADHD and comorbid disorders such as bipolar disorder, anxiety and depression, autism, Tourette's syndrome, other disruptive behavior disorders, and mental retardation ([Brown, 2000b](#)).

### PRINCIPLES OF STIMULANT TREATMENT OF ATTENTION DEFICIT HYPERACTIVITY DISORDER

Two stimulant medications commonly prescribed for the treatment of ADHD are methylphenidate (Ritalin) and dextroamphetamine (Dexedrine). Both of these stimulants are also available in a long-acting preparation. The benefits and side effects of both these medications are similar, but some patients may do better on one or the other ([Elia et al., 1991](#)). The short-term effects of stimulants are well researched. There are more than 350 double-blind placebo controlled studies (on over 3,000 children) consistently demonstrating that stimulants lead to improved impulse control, attention, academic, social, and family functioning ([Spencer et al., 1996](#)). Similar studies have been done demonstrating the same degree of improvement in adults ([Wilens et al., 1995](#)).

In practice about three-quarters of children respond to the first medication tried ([Barkley, 1999b](#)), although for children who do not respond, most (90%) respond with alterations of dose or a trial of a different medication ([Elia et al., 1991](#)). About 80% of adults respond well to methylphenidate ([Spencer et al., 1995](#)). Preschool children have a lower probability of response and a higher incidence of side effects ([Spencer et al., 1996](#)). Adolescents may benefit from particular accommodations in the way in which medication is used ([Weiss et al., 2000b](#)), but otherwise show a good response ([Spencer et al., 1996](#)).

It is recommended that medication be initiated at a low dose and increased slowly. This gives the body a chance to adapt, and minimizes nuisance side effects. There are few differences in the titration of medication for children, teens, or adults. Individuals vary widely in their sensitivity to stimulants, so that some people require higher doses, and others are very sensitive to lower doses. Doses in excess of 1 mg/kg per day or 60 mg of methylphenidate (Ritalin) or 0.5 mg/kg per day or 30 mg of dextroamphetamine (Dexedrine) should be used with caution. When titrating stimulant medication, consideration should be given to 7 day per week dosing during the titration in order to ensure that the family or significant other can observe the benefits or adverse effects. This also makes it possible to assess the effects of medication in different environments. Compliance should be assessed at each visit. Dosage is optimal when the patient has obtained significant benefit with minimal side effects.

Many patients experience difficulty when the medication is wearing off; therefore, it is very important to establish the timing of side effects, aggressive episodes, or other adverse events. It is important to know if side effects are worse during periods of peak medication, or periods in which medication is wearing off. It is important to assess whether the patient is adequately medicated during critical transition periods (e.g., recess for a child or the drive home at the end of the day for an adult). If not, it may be necessary to change the timing or frequency of medication administration, increase the dose of medication, or switch to a longer-acting formulation.

A placebo trial may be helpful if a family member, teacher, or clinician is unsure of medication response or if side effects are caused by medication ([Johnston and Fine, 1993](#)). This means that someone observes the patient and rates their symptoms, without knowing if they are on medication. There are several ways to do this. In a true double-blind placebo trial, neither the patient nor observer knows if they are receiving the active medication or the placebo with no active medication. This requires that the pharmacist encapsulate the medication because it has a strong taste. Alternatively, a single-blind trial can be done by having either a teacher or a spouse rate the patient's symptoms, without their being told if the patient is on medication or not. The observer is told that the patient will be on or off medication at different times over the month, without being told exactly when. In this way, the observer can provide blind ratings, while still being aware of the trial, so that the placebo trial can be interrupted in the event of severe deterioration.

Outcome of medication is best evaluated with a rating scale that provides separate ratings of attention, disruption, and oppositional behaviors. It is educational to the patient to compare symptom ratings before medication and after medication. It is also a useful follow-up tool that can be used by patients to determine whether the medication response should be reevaluated by the clinician. Self-ratings of ADHD symptoms often underestimate difficulty; therefore, an outside observer is critical.

### SHORT-ACTING STIMULANT MEDICATIONS

#### Methylphenidate

Methylphenidate (Ritalin) comes in 10-mg tablets. The medication starts working 30 to 45 minutes after it is taken. The maximum effect is seen 1.5 to 2.5 hours after it is taken. Then the effect decreases and is usually gone in 3 to 4 hours. It is necessary to start the medication at a low dose and increase it slowly in order for the child to obtain optimal benefit and minimal side effects. A typical titration schedule would look like this:

	Morning (mg)	Afternoon (mg)	Noon (mg)
Week 1	5	5	—
Week 2	10	5	—
Week 3	10	5	5
Week 4	10	10	5
Week 5	10	10	10

#### Stimulant Enantiomers

Methylphenidate is a complex racemic drug with two different isomers. Each enantiomer may affect different populations of ADHD individuals and has a unique side-effect profile. The  $\alpha$ -isomer (dextro-methylphenidate or  $d$ -MPH) continues to harbor the bulk of clinical efficacy of the compound. Recent research has made it possible to isolate  $d$ -MPH as a separate compound, with possible unique pharmacokinetic properties and side effects ([Quinn, 2000](#)).

#### Dextroamphetamine

Dextroamphetamine (Dexedrine) is available in 5-mg tablets. For convenience, a 5-mg tablet of dextroamphetamine is seen as roughly equivalent to a 10-mg tablet of methylphenidate. The short-acting dextroamphetamine lasts about 5 to 6 hours, and sometimes this can be an advantage over using methylphenidate. This time

difference may be quite significant in that the increased time between doses may allow for some patients to make it through critical transitions, which might otherwise have been problematic. It may also facilitate compliance. A typical titration schedule for dextroamphetamine might look like this:

	Morning (mg)	Lunch (mg)	After School (mg)
Week 1	2.5–5	—	—
Week 2	5	2.5–5	—
Week 3	5–10	5	—
Week 4	5–10	5	5

### *Adderall*

Adderall is a mixture of dextroamphetamine/amphetamine salts available in 5-, 10-, 20-, and 30-mg tablets. It contains 25% each of dextroamphetamine saccharate, amphetamine aspartate, dextroamphetamine sulfate, and amphetamine sulfate. This mixture of salts is felt to prolong the duration of action, so it will last 6 to 8 hours. Most children do well with either a morning dose, or a morning and lunch dose. Its side-effect profile and dosing is similar to that of dextroamphetamine. Adderall is not available in Canada.

### LONG-ACTING STIMULANTS

Long- and short-acting forms of a given stimulant can be combined. For example, medication with Dexedrine Spansule may cover the patient for most of the day, with a “topper” of the shorter-acting Dexedrine in the mid-afternoon to carry the patient through the evening. There is no research on combining methylphenidate with dextroamphetamine.

### *Pemoline*

Pemoline (Cylert) carries a risk for hepatotoxicity. As a result, it is now only used as a third-line medication in the United States and is unavailable in Canada and the United Kingdom. The use of pemoline requires monitoring liver enzymes.

### *Long-Acting Formulations*

Both dextroamphetamine (Dexedrine Spansule) and d,l-amphetamine (Adderall XR) are available in long acting formulations that last all day. Clinicians should be aware that long acting medications minimize rebound and provide continuity of effectiveness but may also prolong side effects such as appetite suppression and insomnia.

Ritalin SR was the first marketed longer-acting methylphenidate preparation. It is limited by difficulties with erratic release, and was somewhat less effective than methylphenidate in clinical trials. As a result its use has been superseded by other long-acting formulations. Various new release technologies have been explored, such as an osmotic pump, and others are under investigation. These modified release mechanisms have led to the development of medications with 8–12-hour duration of action. These new long-duration stimulants (LDS) have been the subject of rigorous pivotal trials that have done much to develop new methodologies to establish LDS pharmacokinetics and pharmacodynamics ([Greenhill et al., 2001a](#)). These trials differ from earlier studies on Ritalin SR in that they have a larger number of subjects, use parallel rather than crossover designs, are multisite, include open label follow-ups, and involve careful measures of multiple domains of impairment. Pharmacokinetic and dynamic information has been obtained by using simulated classrooms, of which there are now several. It is anticipated that these new medications will offer practical solutions to the stigma associated with asking children to take medication at school during the lunch hour. They are also likely to markedly increase compliance, especially in adolescents.

### ADVERSE EFFECTS

Stimulants are associated with loss of appetite, headache and stomachache, and insomnia ([Corkum et al., 1998](#); [Spencer et al., 2000](#)). Recent research suggests that growth suppression seems to be associated with a developmental delay associated with ADHD itself, rather than a direct effect of medication ([Spencer et al., 1996](#)). Caution should be exerted in children who are medicated for many years without drug holidays or used in children with short stature. Recent research (see the preceding) suggests that stimulants are not correlated with precipitating the onset of Tourette's syndrome or exacerbating tics ([Gadow et al., 1995a](#)). Careful assessment of tics prior to and following treatment as well as appropriate education of parents regarding the risks and benefits of the use of stimulants in the presence of tics is advisable. Most somatic side effects can be managed effectively ([Spencer et al., 2000](#); [Wilens and Spencer, 2000](#)).

Equally important are psychiatric side effects such as obsessive-compulsive behaviors, social withdrawal, appearing ‘zombied,’ rebound irritability, and dysphoria. Most of these psychiatric side effects are more apparent in the long than the short run ([Schachar et al., 1997](#)) and may be misinterpreted by parents as stemming from the disorder rather than its treatment. They are a significant source of distress, nonetheless, and may be associated with significant morbidity or noncompliance.

### THE BENEFITS AND LIMITATIONS OF STIMULANT TREATMENT

Stimulants are associated with a robust (effect size equal to 0.8 to 1.0) short-term effect on behavior ([Greenhill et al., 2001b](#)), and up to 62% of children who improve meet criteria for successful treatment ([Swanson et al., 2001](#)). Methylphenidate also leads to improved academic productivity and attention, although this is a less powerful effect. It is interesting that this short-term benefit has not been demonstrated to lead to a long-term improvement in academic achievement ([Schachar and Tannock, 1993](#)). It has been demonstrated that stimulants are associated with an immediate and substantial improvement in the quality of social interactions with peers, family, and teachers; however, it should be noted that not all social deficits respond. Problems with aggression, processing of social information, social withdrawal, and dysphoria may be less responsive. Most children experience improved self-esteem as their capacity to function normally improves. This may be less evident where the child has profound negative attributions around medication.

Stimulant treatment is helpful to a majority of children with ADHD; however, it offers significant palliation, not a cure. The effectiveness of stimulant medication can be limited by its duration of action, somatic and psychiatric side effects, worsening of a comorbid condition, noncompliance, lack of full normalization, periods of time off medication, tolerance, rebound, and social stigma. For all of these reasons, medication is often combined with psychosocial treatments for residual problems ([Schachar and Ickowicz, 1999](#)).

### OTHER MEDICATION TREATMENTS

Other types of medication are used as second-line treatments for ADHD ([Biederman et al., 1999](#); [Schachar and Ickowicz, 1999](#); [Wilens and Spencer, 2000](#)). There are clinical trials demonstrating an effect on ADHD for tricyclic antidepressants ([Altman et al., 1994](#); [Biederman et al., 1989](#)), bupropion ([Barrickman et al., 1995](#); [Conners et al., 1996](#); [Murphy and Barkley, 1995](#)), tomoxetine ([Spencer et al., 1998](#)), and moclobemide ([Myronuk et al., 1996](#); [Trott et al., 1991](#)). There is weak evidence of efficacy of alpha-adrenergic agonists such as clonidine ([Hunt, 1987](#); [Hunt et al., 1985](#)) and guanfacine ([Hunt et al., 1985](#)). In general, these treatments have not been found to have as robust an effect on the cognitive symptoms of ADHD but may be more effective for associated features, comorbid conditions, or where round-the-clock coverage is imperative.

### Multimodal Treatment

#### THE MULTIMODAL TREATMENT STUDY OF ATTENTION-DEFICIT HYPERACTIVITY DISORDER

The NIMH sponsored MTA (1999a,b) is the most comprehensive long-term treatment study for child psychiatric disorders, the results of which will guide clinical practice, public policy, and research in the years to come ([Boyle and Jadao, 1999](#)). Unique aspects of this study include the large number of children studied and the 14-month duration of the study, making it the first study to evaluate treatment results for a longer period. Assessment and outcome measures of the children were comprehensive and included a wide range of symptoms and areas of functioning, using rated and objective measures. All treatments used had prior established efficacy and were manualized. Titration of medication was carried out systematically according to a documented protocol. Finally, the principal investigators of all six sites from which children were recruited were experienced researchers who had made significant contributions to the field and who collaborated in the design of the study and its execution. The investigators compromised in design between the objectives of efficacy (maximum control) and effectiveness (maximum generalizability),



to enhance the feasibility and likelihood of the MTA protocol being used in clinical practice.

The aim of the study was to compare the effectiveness of medication combined with intensive and broad-based psychosocial treatment with medication management or psychosocial treatment alone, and to compare these treatment programs with treatments in the community.

The sample included 579 children 7 to 9 years old recruited from six sites, who met criteria for ADHD-C. Of these, 34% had comorbid anxiety disorders, 40% comorbid oppositional defiant disorder, and 14% had comorbid conduct disorder, 20% were girls, and 19% of the parents were on welfare. Assessments were carried out at baseline and repeated at 3 and 9 months and at the end of treatment, 14 months. These assessments spanned five domains, core ADHD symptoms, aggressive and oppositional behaviors, anxiety and depression, peer and family relations, social skills, and academic achievement. A maximum of eight emergency sessions were provided for every family.

Children in the medication management (MM) group were started on Ritalin (or alternative medication if they could not tolerate it) over a 4- to 11-day warm-up period followed by a 28-day double-blind medication placebo controlled trial. The active medication was given in different doses in the double-blind period to determine optimal benefit for a given child. Medication was adjusted on a monthly basis with monthly teacher feedback, and each family received a half hour of time each month with a pharmacotherapist. Children assigned to the MM group required a higher dose of Ritalin (mean 38.1 mg) than did children in the CT group (mean 30.1 mg). A small percentage of children (12%) did not do well on Ritalin, and half of these responded well to Dexedrine ( [Greenhill et al., 2001b](#)).

Children who received psychosocial interventions participated in an 8-week, all-day summer treatment program, STP, which utilized contingency management and included social skills training. Parents attended 27 group sessions of carefully designed training and eight individual sessions. School intervention included 10 biweekly sessions of classroom behavior management consultation with the child's teacher. The child's counselor from the STP acted as the child's classroom aide on a half-time basis for 2 months with the aim of generalizing gains made in the STP into the classroom. Seventy percent of children treated in the community control arm of the study were treated with stimulants.

Results from this comprehensive study are still being published and conclusions are being refined ( [Cunningham, 1999](#)). Findings to date are that for most ADHD symptoms, children in the combined treatment and medication management groups showed significantly greater improvement than those given intensive psychosocial treatment alone or those treated in the community. On direct comparison of ADHD core symptoms, combined treatment was not superior to medication management alone and medication management was also superior to behavioral treatment alone and treatment in the community. For non-ADHD areas of functioning, the combined treatment program offered modest advantages over single component programs ( [Jensen, 1999](#)). For some difficulties, however, such as oppositional and aggressive symptoms, internalizing symptoms, teacher-rated social skills, parent-child relations, and reading achievement, combined treatment was superior to psychosocial treatment alone and to community care.

There have been recent attempts to reanalyze the original data using different measures with the aim of clarifying small group differences in treatment outcome. [Conners and Epstein \(2001\)](#) used a "composite score" of outcome, which provided a more reliable index of change with greater statistical power, than the original analysis of multiple paired comparisons over time, which lost statistical power associated with the Bonferroni correction. This single composite score was internally consistent, reliable, and correlated with clinician global judgment. A small but significantly better outcome was found in the CT group versus the MM group using this outcome measure, whereas large differences were found between the CT group, which had the best outcome compared to the other three groups.

[Swanson and associates \(2001\)](#) also reanalyzed the data using a single composite score, the percentage of children in each group who fell into the "successful treatment" category. Sixty-eight percent in the CT group, 56% in the MM group, 34% in the BT group, and 25% in the CC group fell into this successful treatment category. Here again the outcome in the CT group was statistically significantly better than the MM group. It remains to be demonstrated if these differences are clinically significant, and if so which kinds of children with ADHD respond best to which kinds of treatment.

Recent studies utilizing the continuous performance test (CPT), have suggested that children who have ADHD only, and those with ADHD comorbid with externalizing disorders, ODD-CD or with anxiety disorder (AD) or both may be sufficiently distinct to warrant classification as subtypes. [Newcorn and colleagues \(2001\)](#) found that CPT-rated inattention, impulsivity, and dyscontrol were high in all ADHD groups; however, children with ADHD and ODD-CD were more impulsive than inattentive, whereas children with ADHD and AD were relatively more inattentive than impulsive. Girls were less impaired than boys on most CPT ratings.

[Jensen and coworkers \(2001\)](#) also found that the preceding subgroups based on comorbidity differ on baseline variables, as well as response to treatment. He suggested that these findings have clinical implications with the following rule of thumb: Children with ADHD without comorbidity and children with ADHD comorbid with ODD-CD respond best to (MTA) medication management, and behavior therapy alone is contraindicated. Those children who have ADHD and anxiety respond well to all the MTA treatments. However, combination treatment (MTA-CT) may offer significant advantage for children who have ADHD comorbid with both anxiety and ODD-CD.

It is interesting to speculate on the better outcome of the children in the medication management program compared to the children treated in the community. Perhaps entry into a prestigious study creates optimism for families. Another important point is that the doses used in the medication management program were titrated for each child to ensure maximum benefit and the average dose was higher than in those treated in the community, and even more important is the fact that dose adjustments were made monthly after feedback from the child's teacher, and the family was given half an hour with the psychopharmacologist. These monthly visits with regular teacher feedback may have been an important adjunct to the medication itself.

As pointed out by [Jensen \(1999\)](#), some erroneous conclusions have been made about the results of the MTA including the notion that behavior therapy does not work or that the treatment protocols, all of which have been well described in treatment manuals, can never be carried out in the real world. Certainly, the protocol for the medication management program is a model for medication treatment for clinicians in the community that treat children with ADHD.

The conclusion is that all four treatment programs were highly effective over the 14 months of treatment for children with ADHD. Medication management and psychosocial treatment may impact selectively on different target symptoms. The findings are optimistic given the range of problems of children with this disorder, treatment preferences of parents and variations in professional expertise and professional availability in different geographical areas.

#### *MTA FOLLOW-UP 14 TO 24 MONTHS*

Results of this 10-month follow-up (after treatment termination in the study) have been presented at the American Academy of Child and Adolescent Psychiatry Meeting in New York in 2000 but have not yet been published, and some of the findings have not yet been submitted for statistical analysis.

At the end of the 14-month treatment period, all families met with their clinician who discussed recommendations for future treatment with them based on the finding of the MTA committee. Compliance with treatment recommendations was lowest in the families who had received behavior therapy. Seventy percent of children in this group were given recommendations for initiating stimulant therapy and only 44% complied versus 88% of the children in the combined treatment and medication management groups. It was found that prior experience with a treatment was a strong predictor of compliance. Families in the medication management group sought out more child individual, family, and group therapy. Excellent responder status in the treatment period predicted compliance with recommendations.

The following results were obtained when "excellent responder" status was measured at 14 and at 24 months. Combined treatment children had 65% of excellent responders at 14 months, which fell to 44% at 24 months. Medication management children had 55% of excellent responders at 14 months, which fell to 25% at 24 months. Behavior therapy children had 25% of excellent responders at 14 months, which fell to 10% at 24 months.

The preceding pattern was obtained also by other measures of ADHD and ODD. The combined treatment group best upheld the gains made during the intensive treatment period whether or not they continued on medication. Improvement declined more steeply in the medication management group, but was better than in the community control or behavior therapy group. In general, the use of medication in the 14- to 24-month period reduced deterioration. We can conclude that the pattern of treatment response seen at 14 months was maintained at 24 months. The MTA-NIMH researchers are planning to do a 10-year follow-up with several consecutive assessments during the period. They will be able to assess individual trajectories, growth patterns, side-effects of stimulants, effects of initial and later treatments, and the possibility of the emergence of sleeper effects.

It is interesting to speculate on the possible reasons why, particularly those children in the combined treatment group but also those in the Medication Management group, maintained their gains better than the children in the behavior therapy group or those treated in the community, 10 months after their intensive treatment

ended. Although it is possible that the children in the former two treatment groups received a higher dose of stimulants in the follow-up period, this is probably not the only reason for their better outcome, even at 24 months.

## DEVELOPMENT AND OUTCOME

The adolescent, and where available, the adult outcome from four major prospective controlled follow-up studies—the Montreal, New York, Milwaukee, and Biederman Studies—are reported on. Their methodologies are summarized in the following.

### Prospective Studies

#### THE MONTREAL STUDY

Weiss and coworkers followed 91 hyperactive children 6 to 12 years old whose primary difficulties were hyperactivity, impulsivity, and attentional problems. DSM diagnoses did not exist when this study was initiated and the children were rated on a Werry Weiss Peters Scale of Hyperactivity. Children whose primary difficulties lay in conduct disorder problems were excluded, as were psychotic children, children with autism, children with an IQ below 80, and those who were not living with their family. Weiss and coworkers assessed these children at five yearly intervals at a mean age of 13, 18, and 24 years. Psychiatrists who were not blind as to the status of probands and controls assessed psychiatric status.

#### THE NEW YORK STUDY

Gittelman and colleagues conducted a 9- and 16-year follow-up on 115 cross-situationally hyperactive children 6 to 12 years old and a matched control group. The methodology of this study was similar by design to the Montreal Study but “blind” observers (versus psychiatrists) rated the groups at follow-up. A replication study of 111 ADHD children seen in the same clinic and controls were subsequently followed using the same criteria and measures as in the first study. Subjects were 18 years of age at the 9-year follow-up and 25 years of age at the 16-year follow-up. Exclusion criteria were identical to the Montreal Study.

#### THE MILWAUKEE STUDY

Barkley and colleagues studied 123 hyperactive children and 60 matched controls and compared their outcome 8 years later when their mean age was 14 years and 15 years later when their mean age was 21 years. Children were included in the study if they scored one and a half standard deviations above the mean of the control group on several well-standardized rating scales of ADHD symptoms. Unlike the Montreal and New York Studies, children who had ADHD comorbid with conduct disorder were not excluded.

#### THE BIEDERMAN STUDY

Biederman and colleagues studied 138 children with ADHD diagnosed by DSM-III criteria, their siblings, and 120 normal controls. Exclusion criteria were sensory motor handicaps, IQ below 80, psychosis or autism, and severe social adversity. The assessors were blind as to proband, sibling, or control status. The following outcome measures were used: Kiddie SADS, Gilmour Reading Test, subtests of the WISC-III, WAIS, DSM-III-R, GAF, and the Social Adjustment Inventory for children and adolescents (SAICA). The groups were assessed at baseline and 4-year follow-up, when 91% of the groups could be assessed. There were no statistical differences between the groups with respect to age and intact families at intake. Small differences in social class were corrected for statistically.

### Adolescent Outcome

Unless otherwise stated, references for adolescent outcome are as follows: Montreal Study ([Weiss et al., 1971, 1979](#)); New York Study ([Klein and Mannuzza, 1989](#)); and the Milwaukee Study ([Barkley, 1999a](#); [Barkley et al., 1990](#); [Biederman et al., 1996b](#)).

#### EDUCATIONAL ACHIEVEMENT

Poorer grades, more grades failed, and a lower level of education achieved was found in all four studies between the ages of 13 to 18 years. The adolescents previously diagnosed with ADHD (probands) performed more poorly on achievement tests of reading and arithmetic and had more learning disabilities ([Piacentini et al., 1997](#)). We can conclude that the academic difficulties, which characterized ADHD children in elementary school, are compounded in high school.

#### PSYCHIATRIC STATUS

In all four studies, probands were given more psychiatric diagnoses than controls, namely about 50% versus 20%. Probands compared to controls had an increasing number of comorbid disorders at adolescent follow-up including oppositional defiant disorder (ODD), conduct disorder (CD), major depressive disorder (MDD), psychoactive substance abuse disorder (PSUD) ([Biederman et al., 1997](#)), anxiety disorders, and learning disabilities (LD).

#### ANTISOCIAL PERSONALITY DISORDER OR CONDUCT DISORDER

A risk for the development of antisocial personality disorder (APD) or conduct disorder (CD) was found in all four studies. In the New York Study, persistence of ADHD symptomatology into adolescence (with or without hyperactivity) was highly correlated with the development of both APD and PSUD. The risk for APD varied from 23% to 40% in probands versus 1% to 8% of controls in the different studies. The risk of developing APD was greatest in those who had ODD or CD at baseline whose ADHD symptoms persisted into adolescence and for those who came from antisocial families ([Faraone et al., 1998a](#)).

#### Major Depression

In the Montreal Study at a mean age of 13 years, probands showed that they had strong feelings of discouragement or demoralization and had mood lability. DSM-II diagnoses (diagnostic nomenclature at the time) were not given.

In the Biederman study, probands were at risk for the development of MDD in midadolescence by DSM-III-R criteria. Those who had MDD at intake were most at risk for MDD at follow-up. No risk was found for the development of MDD in late adolescence in both the Montreal and New York studies. These differences may be partly explained by the inclusion of children with more comorbidity in the Biederman Study. Biederman's findings also suggested that MDD is a distinct disorder and not just demoralization secondary to ADHD.

#### CIGARETTE SMOKING

At 14 years, probands and their siblings were more likely to smoke cigarettes, and started smoking earlier ([Barkley et al., 1990](#); [Biederman et al., 1996c](#)). Cigarette smoking was associated with MDD, drug abuse, and less treatment received ([Milberger et al., 1997a](#)).

#### CAR ACCIDENTS

Probands in late adolescence had more car accidents with bodily injuries ([Barkley et al., 1993](#); [Weiss et al., 1985](#)).

#### CRIMINALITY

In the New York Study at an average age of 18 years 39% of probands versus 20% of controls had been arrested, 28% versus 11% had been convicted and 9% versus 1% jailed. Twenty-five percent versus 7% of these had been charged with a felony. An important finding was that the presence of APD almost completely accounted for the increased risk of criminality ([Mannuzza and Klein, 1999](#)). These findings were lower than the rates of criminality in Satterfield's study in California,



but higher than those in the Montreal Study ([Hechtman et al., 1984](#); [Satterfield et al., 1982](#)).

#### *FAMILY FACTORS*

In the Milwaukee and Biederman Studies, the families of probands had less stability, higher divorce rate, moved more frequently, and changed jobs more often. The fathers had a higher rate of antisocial disorders than the fathers of controls. In the Biederman Study, the families showed less cohesion and more family conflict (i.e., more evidence of family adversity) and those probands whose parents had antisocial behavior had more anxiety, MDD, antisocial behavior, and aggression at follow-up.

#### *GROWTH*

Adolescents were evaluated for possible growth deficits in the Biederman Study. Small but significant deficits in height were found in midadolescence, which usually normalized by late adolescence, and which were unrelated to the use of stimulants ([Spencer et al., 1996](#)).

#### *CONTINUED SYMPTOMS OR PRESENCE OF FULL ATTENTION DEFICIT HYPERACTIVITY DISORDER SYNDROME IN ADOLESCENCE*

In the Biederman Study, 85% of probands still met full or subthreshold evidence of the disorder, as well as evidence of impairment at a mean age of 14 years. Half of the 15%, which had remitted, did so before age 12 years (early remitters) and half after age 12 years (late remitters). It was found that early remitters and controls did not differ with respect to exposure to parental pathology and family conflict (i.e., on measures of social adversity), but family adversity was markedly elevated in late remitters. Similarly, the incidence of comorbidity and impaired psychosocial and academic functioning was higher in those adolescents whose ADHD had not remitted (Biederman et al., 1996d, [1998b](#)). In the Milwaukee Study, at a mean age of 14 years, 71% of probands versus 3% of controls still met DSM-III-R criteria for the full ADHD syndrome. In the New York study, at a mean age of 18 years, 31% of probands versus 3% of controls had the full syndrome, and 40% had two symptoms of the disorder.

In general, these results indicate that ADHD continues into early adolescence in the majority, 71% to 85%, declining somewhat to about 31% in later adolescence. The studies suggested that persistence of ADHD symptomatology was associated with more comorbidity including CD and APD.

#### **Outcome in Adulthood**

As might be predicted from the nature of the outcome in adolescence, the outcome in adulthood shows impairment in several important domains of functioning. Only three prospective controlled follow-up studies have followed children with ADHD into their twenties: the Montreal study ([Weiss and Hechtman, 1993](#)); two cohorts of the New York Study ([Mannuzza et al., 1993, 1998](#)); and the Milwaukee Study. The Milwaukee Study evaluated their subjects 15 years later at a mean age of 21 years and was able to report on 91% of their original sample. Adult outcome from these three studies are described.

#### *EDUCATIONAL ACHIEVEMENTS*

All three studies found that even in early adulthood children once diagnosed as having ADHD had completed 2 to 3 years less education than matched controls ([Mannuzza et al., 1993, 1998](#); [Weiss et al., 1979](#)). More probands dropped out of high school and less were enrolled in or had completed a graduate degree.

#### *WORK HISTORY*

All three studies had similar findings. Probands at 24 to 25 years had significantly lower occupational status than matched controls ([Mannuzza et al., 1993](#); [Weiss and Hechtman, 1993](#)). In the Montreal Study, employers rated probands significantly worse than controls on work competence, working independently, completing tasks, and getting along with supervisors; and there was a trend suggesting employers would not hire them again ([Weiss and Hechtman, 1993](#)). In the Milwaukee Study, employers rated probands as scoring worse on ADHD and ODD rating scales. The New York Study did not evaluate employer ratings. The most common occupations for both cohorts of the New York Study were skilled workers, such as plumbers, painters, machinists, and mechanics. The Montreal Study found that a greater number were fired or laid off, and they changed jobs more frequently. It is interesting that in spite of lower educational status in all three studies, there were no differences in the percentage currently working or in their annual income.

#### *SELF-ESTEEM AND SOCIAL SKILLS*

The Montreal Study found that self-esteem (Davidson and Lang checklist) and social skills (Situation Social Skills Inventory) were found to be impaired in adolescence, and social skills had deteriorated further in adulthood ([Hechtman et al., 1980](#)).

#### *PSYCHIATRIC STATUS*

##### *Personality Disorders*

All three studies, the Montreal Study, the two cohorts of the New York Study ([Mannuzza et al., 1993, 1998](#)) and the Milwaukee Study, showed that children with ADHD are at risk for the development of antisocial personality disorder (APD). In the Montreal Study, figures were 23% versus 2% ([Weiss et al., 1985](#)); in the New York Study, 12% to 18% versus 2% to 3% ([Mannuzza et al., 1993, 1998](#)); and in the Milwaukee Study, 21% versus 4%. An important question is whether conduct disorder in the childhood of ADHD accounts entirely for APD at adult follow-up. We do not have the answer to this question but both the Montreal and New York Studies excluded children at intake whose main problems lay in antisocial or aggressive behavior. However, some of the probands in the Montreal and New York Studies may have developed early onset conduct disorder after they were included into the study. In contrast, in the Milwaukee Study, the presence of conduct disorder or aggression was not an exclusion criterion for study entrance. It is possible from the overall findings that ADHD in childhood may be a risk factor for APD even in the absence of conduct disorder at baseline; however, the risk for developing APD is higher once conduct disorder develops.

It is interesting that the Milwaukee Study found that the probands also were at risk for other personality disorders, namely passive aggressive (19% versus 8%), borderline (14% versus 2%), histrionic (11% versus 0%), and narcissistic personality disorder (5% versus 0%), as measured on the semistructured interview for DSM personality disorders (SCID II).

##### *Mood Disorders*

Both the Montreal and New York Studies failed to find more major depression (MDD) in probands than in controls at age 24 to 25 years. This is in contrast to the finding in the Milwaukee Study, where 27% of probands versus 12% had MDD at follow-up. This confirmed the finding of increased MDD in the Biederman adolescent outcome study. There was a higher level of comorbidity in probands than controls in both the Montreal and Milwaukee Studies.

As mentioned in the section on adolescent outcome, the difference may be in whether or not children with conduct disorder were included in the study and used different measures of MDD outcome.

##### *Polysubstance Use Disorder*

Findings are inconsistent with respect to polysubstance use disorder (PSUD) at the time of adult reevaluation. The Montreal Study failed to find significant differences between probands and controls with respect to PSUD, but there was a trend for probands to have tried heroin and sold drugs more often. There was also a trend for more probands than controls to fall into the abuse category with respect to amount of alcohol consumed in the past 12 months ([Hechtman et al., 1984](#)).

In both cohorts of the New York Study, PSUD was significantly more prevalent in probands than controls (12% to 16% versus 4%) ([Mannuzza et al., 1993, 1998](#)). This is in agreement with the findings of the Milwaukee Study ([Milberger et al., 1997d](#)) in which probands were at increased risk for cocaine (37% versus 18%), and amphetamine use (29% versus 8%). Variations in cultural factors among sites, as well as variations in the point of time in which follow-up assessments were done, could have influenced the variations of outcome with respect to PSUD. For example, 8% of the control group in the Milwaukee Study used amphetamine; in contrast,

only a single control subject had tried amphetamines in the Montreal Study, and none had used heroin versus 3% in the Milwaukee Study.

### *Sexual Behavior*

The Milwaukee Study found that probands had intercourse at an earlier age than controls (15.4 versus 16.5 years) and had had more sexual partners (18.5% versus 16.5%). They were less likely to use birth control (76% versus 91%), and had more pregnancies (38% versus 4%). Most importantly, they had more sexually transmitted diseases (17% versus 4%) and more subjects had been tested for HIV, 21% versus 5.4%.

### *CONTINUING ATTENTION DEFICIT HYPERACTIVITY DISORDER SYMPTOMS OR ATTENTION DEFICIT HYPERACTIVITY DISORDER IN ADULTHOOD*

The Montreal and New York Studies differed significantly in their findings. The two cohorts in the New York Study ( [Mannuzza et al., 1993, 1998](#)) found that 4% to 11% of the probands met criteria for ADHD in adulthood by DSM-III criteria. The [Weiss Study \(1985\)](#) found that 66% still had disabling ADHD symptoms, but this study did not use the maintenance of the full syndrome as an outcome measure, reflecting the fact that different assessment methodologies result in different findings.

Only the probands themselves were evaluated at follow-up in the New York Study, whereas parents were included as part of the follow-up assessment in the Montreal Study. The Milwaukee Study highlighted how different ways of assessing outcome in adulthood changes the findings and also gave evidence of how parents rate probands as having a poorer outcome of the syndrome than did probands themselves. When they used the Milwaukee Young Adult Interview Self-Report, on which eight of 14 symptoms are required for diagnosis, only 3% now met diagnostic criteria. When they used 1½ standard deviations from the mean of the control group, 25% still met diagnostic criteria. However, on the parent version of this scale, parents reported that 58% still met diagnostic criteria. When DSM-IV criteria were used with parents, 42% of probands still met diagnostic criteria. The Milwaukee Study concluded that by self-report 2% met diagnostic criteria at a mean age of 21 years, or 25% if two standard deviations from the mean were used as a criterion. However, on parent reports the rate of persistence of the syndrome was 58% to 68%. This figure is quite similar to that in the Weiss Study.

The Milwaukee findings help to clarify differences between the Montreal and New York groups, because persisting symptoms were included and parents were interviewed in the former, whereas the latter relied on self-reports, did not include parent ratings, and did not count disabling continued symptoms (if they did not meet full diagnostic criteria).

The Biederman Study (unpublished) found that at 18 to 20 years, 65% had achieved full syndrome remission, 27% had achieved symptomatic remission (less than half of the symptoms required for the full disorder), and only 10% had achieved functional remission (measured as being higher than 65 on the GAF). In this study, hyperactivity had greatly improved by 9 to 11 years, and impulsivity by 12 to 14 years, but attention problems persisted.

It is clear from the preceding that how remission is measured (full syndrome, symptomatic remission, or functional remission) or 1½ to two standard deviations from the mean on a rating scale changes the findings. The figures for remission also vary according to whether adult probands report on themselves or their parents' reports are taken. No gold standard has been found to measure outcome, and it is hardly surprising that differences are found.

### **FUTURE DIRECTIONS**

The summary of research and clinical investigation reviewed in the preceding opens up questions of future research opportunities. We close by summarizing some of the developments that we anticipate may characterize the next 5 years.

The broadening of the diagnostic criteria for ADHD has made it evident that we need to learn more about the impairment suffered in preschool, adolescence, and adulthood, as well as the reliability and validity of our diagnostic criteria for these populations. We need to carry out specific treatment studies that focus on each of these developmental stages. By the same token, we also need to explore the various diagnostic subtypes. How specific are diagnoses of ADHD-HI subtype in preschool? How specific are diagnoses of ADHD-IA in adolescents and adults? Do males and females present differently?

This in turn raises the issue of how we assess attention in general, and the relationship between difficulties with attention and broad deficits in executive function. Hopefully, we will learn more about how treatments initially developed for ADHD-combined type help or do not help patients who suffer from problems with attention and executive function.

The last 5 years have witnessed an explosion of new pharmaceutical interest in medication development for ADHD, and have led to the first new treatments for ADHD in half a century. We anticipate that many issues around compliance will be addressed by better long duration stimulants, and many issues of management of rebound, tolerance, and comorbid anxiety/depression will be addressed by new and more effective designer antidepressants specifically created for round the clock coverage of ADHD. These medications need short- and intermediate-term clinical trials, but functional longer-term assessment of the impact of medication also is critical. Some of this information may come out of the 10-year follow-up of the MTA.

The majority of patients with ADHD also suffer from other conditions, and often it is the other disorder that determines outcome. Specific clinical trials on treatment of ADHD with other disorders may allow us to develop treatments specific to comorbidities, as opposed to trying to apply treatment strategies developed for simple ADHD cases to children with more complex presentations. The MTA has led to more specific knowledge of ADHD with other disorders and has opened new methodologies for exploring this further.

Finally, we conclude by noting the critical importance of extending the education of all health care specialties around this disorder because it presents at different stages of the life cycle and in different environmental contexts. Recent attempts to formalize clinical knowledge in practice parameters have already begun to lay a foundation for such educational initiatives.

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## 53 CONDUCT DISORDER

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Conduct disorder is a diagnostic category that is so broad and all-encompassing that it requires more deliberation to avoid the diagnosis than to make it. It is, consequently, one of the most frequently made diagnoses in child psychiatry ( [McDermott, 1996](#)). Experienced clinicians, however, know that nearly every other psychiatric diagnosis of childhood and adolescence may, under certain circumstances, be manifested by disordered, obnoxious, aggressive behaviors. Conduct disorder, as currently defined, includes such diverse behaviors as staying out late, fire setting, and sexual assault. By covering such a broad spectrum of behaviors, the diagnosis catches in its net a multitude of sins, as well as a multitude of different kinds of neuropsychiatric and psychosocial conditions and combinations thereof. Children have a limited verbal capacity as well as a limited repertoire of behaviors with which to express their discomfort, misery, and confusion. Therefore, the same kinds of maladaptive behaviors in different children can be indicators of very different kinds of problems.

Potentially treatable conditions such as attentional problems, brain damage, mood disorders, psychoses, and dissociative states frequently present with disruptive behaviors that also meet the diagnostic criteria for conduct disorder. Moreover, hidden physical, sexual, and emotional abuse play important roles in the genesis of certain kinds of aggressive behaviors and inappropriate sexual behaviors. The failure to recognize such vulnerabilities can have devastating consequences. This chapter familiarizes the clinician with some of the most common treatable conditions that underlie the antisocial, often aggressive behaviors of children and adolescents diagnosed with conduct disorder.

### HISTORICAL NOTE

Conduct disorder, listed in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) under Disruptive Behavior Disorders ( [American Psychiatric Association, 1994](#)), is the latest term used to designate children whose signs, symptoms, and behaviors impinge more on others than on the child or adolescent with the condition. Although many of its childhood manifestations are very similar to the behaviors of adults diagnosed with antisocial personality disorder, conduct disorder has been separated from that adult diagnosis to acknowledge what psychiatrists believe to be a greater potential for change in the young. The diagnosis harkens back to a period in the history of psychiatry antedating the very existence of the field of child psychiatry, a time in which adult psychiatrists struggled to understand the behaviors of their recurrently antisocial but not overtly psychotic patients. Terms such as *manie sans delire* (mania without delirium) ( [Maugh, 1941](#)) and *moral insanity* ( [Prichard, 1837](#)) were used to designate patients with recurrently antisocial behaviors who were not blatantly psychotic.

In 1952, the diagnosis of “Sociopathic Personality: Antisocial Reaction” for adults was codified by the American Psychiatric Association in DSM-I ( [American Psychiatric Association, 1952](#)). Individuals so diagnosed were described with pejoratives: immature, disloyal, hedonistic, irresponsible, and unable to benefit from experience or punishment. In DSM-II ( [American Psychiatric Association, 1975](#)), under the category of Behavior Disorders of Childhood and Adolescence, the diagnosis for behaviorally maladapted children became “Unsocialized Aggressive Reaction of Childhood (or Adolescence).” The words used to delineate this syndrome were more judgmental than scientific (e.g., quarrelsomeness, vengefulness, disobedience).

In 1980, DSM-III ( [American Psychiatric Association, 1980](#)) was published, and with it came the new category, conduct disorder. Like its predecessor, conduct disorder was used to designate children with behaviors ranging from truancy and substance abuse to rape and assault. Conduct disorder was divided into four different categories: aggressive and nonaggressive, socialized and undersocialized.

In DSM-III-R ( [American Psychiatric Association, 1987](#)), the four subtypes of conduct disorder gave way to three other subtypes: (a) solitary aggressive type, (b) group type, and (c) undifferentiated type. For a child to qualify for the diagnosis of conduct disorder, he or she needed only to have manifested 3 of 13 types of undesirable behaviors.

Little in the definition of conduct disorder changed between DSM-III-R and DSM-IV. DSM-IV defines conduct disorder as “A repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated as manifested by the presence of three (or more) of 15 criteria in the past 12 months, with at least one criterion present in the past 6 months.” As can be seen in [Table 53.1](#), the 15 criteria include widely diverse kinds of behaviors, grouped into four very different categories from DSM-III-R, namely: (a) aggression to people and animals, (b) destruction of property, (c) deceitfulness or theft, and (d) serious violations of rules. The changes in categorization from DSM-III to DSM-III-R to DSM-IV reflect the difficulties framers of these documents had in getting a handle on the disorder. All 13 of the original behaviors listed in DSM-III-R reappear in DSM-IV, plus two additional behaviors, bullying or intimidating and staying out late in spite of parental prohibitions. Given the diversity of behaviors listed, a child who has shoplifted, lied, and stayed out late could receive the same diagnosis as a child who has robbed banks, pistol-whipped guards, and raped toddlers.

**Table 53.1. Diagnostic Criteria for Conduct Disorder (312.8)**

The DSM-IV distinguishes two kinds of conduct disorder that are based not on behaviors, but rather on age of onset. Childhood-onset type is defined by 3 or more of the 15 listed behaviors, at least 1 of which must have occurred before age 10 years. Adolescent-onset type is defined by an absence of any of these behaviors before age 10 years. This distinction is made because of the especially poor prognosis for children whose maladaptive behaviors start early in life because they tend to have more neuropsychiatric vulnerabilities, to require more psychiatric treatment later in life, and to act more aggressively ( [Lahey et al., 1999](#) ).

As with other diagnostic categories, diagnosticians are required by DSM-IV to specify the severity of the clinical picture by ratings of mild, moderate, or severe. Because all 15 of the behaviors listed are signs, not symptoms, the definition of conduct disorder does not distinguish among assaultive children who are paranoid and retaliate in response to imagined threats, those who fight because they are frequently the butt of teasing, those with episodic manic rages, or those with organic conditions that impair impulse control. Children with the disorder are described as callous, unempathic, and without remorse, terms more reflective of an examiner's attitude than his or her clinical judgment. It is therefore up to the user of DSM-IV to keep in mind that all 15 behaviors listed under conduct disorder, and combinations thereof, occur as part of numerous other diagnoses. The clinician must refrain from using the diagnostic category of conduct disorder in cookbook fashion, ticking off behavioral ingredients and measuring the length of time they have been heating up. Making a diagnosis is different from making a lamb stew.

**EPIDEMIOLOGIC AND SOCIOLOGIC PERSPECTIVES**

Because the criteria for making the diagnosis of conduct disorder vary from time to time and place to place, it is impossible to determine its prevalence. It is, however, one of the most frequently made diagnoses in the United States ( [Quaay, 1999](#) ), undoubtedly because of its broad definition. Estimates of prevalence have ranged from 1% to 10%, depending on the city, country, and period in which studies have been conducted. On the other hand, the occurrence of juvenile violence, commonly considered synonymous with conduct disorder, is easier to measure. Within societies, violence flourishes in urban areas ( [Elliott et al., 1989](#) ) and in the lowest socioeconomic sectors of cities ( [Blau and Blau, 1982](#); [Currie, 1998](#); [Shihedeh and Ousey, 1998](#) ). The association of poverty and violence is found in other cultures, as well as in the United States ( [Farrington, 1989](#); [Merton, 1963](#) ). It is not just poverty but, rather, *extreme* poverty, with which violence is associated ( [Krivo and Peterson, 1996](#) ). Disorganization, joblessness, single-parent homes, and what [Currie \(2000\)](#) has termed *brutalization* are more prevalent in conditions of extreme socioeconomic deprivation. Study after study confirms the finding that violent youths are disproportionately those who themselves have been victimized ( [Lewis et al., 1988, 1989](#); [Smith and Thornberry, 1995](#); [Widom, 1989](#) ). Other factors associated with the development of aggressive behaviors are broken homes ( [Farrington, 1998](#); [Henry et al., 1996](#) ), large family size ( [Capaldi and Patterson, 1996](#); [Farrington, 1998](#) ), and gang membership ( [Thornberry, 1998](#) ). Of note, studies indicate that violence often precedes membership in a gang ( [Thornberry, 1998](#) ) and therefore is not necessarily a result of membership.

[Farrington and Loeber \(2000\)](#) conclude that the most important variables contributing to ongoing aggressive adaptational styles are "individual (high impulsiveness and low intelligence, possibly linked to the executive functions of the brain); family (poor supervision, harsh discipline, child physical abuse, a violent parent, large family size, poverty, a broken family); peer delinquency; gang membership; urban residence; and living in a high crime neighborhood (characterized by gangs, guns, and drugs in the United States)." We would add that, in our clinical work, we have found that severe parental psychopathology also contributes greatly to the chaos and violence in the households of aggressive children and adolescents. Parental mental illness can be crucial to understanding a family's chaotic relationships, inability to provide adequate financial support, poor disciplinary practices, and violent, abusive behaviors.

**COURSE**

An abundance of data indicates that early severe behavioral problems often are followed by a variety of subsequent adaptational difficulties. [Bauermeister and colleagues \(1994\)](#), in their elegant literature review, concluded, "Preschool behavior problems are a strong risk factor for antisocial disruptive disorders at preadolescence." Several studies reported that aggressive behavior problems at 8 years of age were predictive of serious delinquency in adolescence ( [Lefkowitz et al., 1977](#); [West and Farrington, 1973](#) ). A study of youths from four U.S. communities documented a strong association between very early behavior problems and subsequent serious, aggressive behaviors and psychiatric problems ( [Lahey et al., 1999](#) ). As a result of these kinds of findings, DSM-IV distinguishes two types of conduct disorder, one, an aggressive type, starting in early childhood; the other, a nonaggressive type, starting in preadolescence or adolescence. Almost all studies, regardless of time, place, or sample, have found conduct disturbances to be far more common in boys than in girls.

It is important to keep in mind that, despite the aforementioned findings, most behaviorally disturbed children do *not* go on to become antisocial or criminal adults ( [Robins, 1966](#); [Rutter and Giller, 1984](#) ). Even among those children and adolescents whose behaviors bring them in conflict with the law, there is evidence that only a small proportion become recidivists ( [Tracy et al., 1990](#) ). However, this recidivistic group commits a disproportionate number of adult criminal acts.

Because of the diversity of the psychopathology underlying them, conduct disorders are followed by a wide range of psychiatric and adaptational problems other than antisocial or criminal behaviors ( [Rutter and Giller, 1984](#) ), including mood disorders, anxiety disorders, somatoform disorders, substance-related disorders, and psychoses ( [American Psychiatric Association, 1994](#); [Lewis et al., 1991, 1994](#); [Robins, 1966](#) ). [Hechtman and Offord \(1994\)](#) observed that, in addition to criminality and mental illness, early childhood disorders of conduct also are predictive of "widespread social malfunction, as seen in high rates of divorce and separation, poor work history, and unsatisfactory social relationships."

**CLINICAL DESCRIPTION**

Because disordered conduct can be a manifestation of such a wide variety of different neuropsychiatric conditions or can coexist with them, a useful way of approaching a child with behavioral problems is to try to identify first his or her intrinsic vulnerabilities to maladaptation. That is, are there any potentially remediable psychiatric, neurologic, medical, or cognitive problems that underlie and contribute to the child's undesirable behaviors? Second, what are the environmental stressors that influence the disruptive ways such vulnerabilities are expressed?

**Psychiatric Vulnerabilities**

Children with maladaptive, aggressive behaviors rarely appear to have serious neuropsychiatric problems. The key word in the previous sentence is *appear*. Unfortunately, their obnoxious behaviors overshadow underlying psychopathology and tend to deter clinicians from conducting the kinds of comprehensive evaluations necessary to uncover it. This section focuses on some of the most common psychiatric conditions underlying the behaviors associated with conduct disorder.

**ATTENTION DEFICIT HYPERACTIVITY DISORDER**

The diagnosis attention deficit hyperactivity disorder (ADHD) is characterized by impulsivity, short attention span, cognitive and social disinhibition, overactivity, social inappropriateness, poor judgment, and school problems, all of which are seen in children diagnosed with conduct disorder ( [Barkley, 1997](#) ). Evidence from neuroimaging studies suggests ADHD can be associated with a variety of different structural and functional brain abnormalities ( [Baving et al., 1999](#); [Mataro et al., 1997](#); [Rubia et al. 1999](#); [Zametkin et al., 1993](#) ). ADHD is one of the most common diagnoses of both violent and nonviolent delinquents, second only to that of



conduct disorder ([Biederman et al., 1991](#); [Offord et al., 1991](#)). However, the relationship between delinquency, aggression, and ADHD is not entirely clear. ADHD alone is not strongly predictive of a delinquent outcome. For example, longitudinal studies of boys with ADHD ([Lambert et al., 1987](#); [Mannuzza et al., 1989](#); [Weiss et al., 1971](#)) have shown that only 19% to 27% of hyperactive boys became antisocial in adolescence. When ADHD is coupled with other disorders, however, a poor outcome is more likely. Children with both ADHD and early conduct problems appear to be at greatest risk for later aggression and delinquency ([Dalteg and Levander, 1998](#); [Farrington et al., 1990](#); [Foley et al., 1996](#)).

One of the most interesting if misdiagnosed causes of episodic uncontrollable tantrums which can masquerade as conduct disorder/ADHD is the acute confusional state sometimes associated with migraine auras. These auras, indicators of brain dysfunctions deep in the brain and parts of the cortex, last minutes to hours and can occur before, during, after or in between migraine headaches. Children experiencing them may periodically go on rampages, destroying objects and attacking people who get in their way. These kinds of attacks may precede by years the development of headaches and can therefore, be extremely difficult to diagnose (see [chapter 29](#)).

The clinician must be cautious when considering a diagnosis of ADHD for behaviorally disordered or aggressive youngsters because inattention, overactivity, impulsivity, and poor judgment also are characteristic of other disorders [e.g., manic and hypomanic states ([Carlson, 1996](#)), dissociative disorders ([Lewis and Yeager, 1996](#))].

## MOOD DISORDERS

Depression and suicide attempts are well recognized concomitants of adolescent aggression and delinquency ([Loeber et al., 1991](#); [Zoccolillo, 1992](#)). More recently, however, child psychiatrists have begun to appreciate the relationship of mania to delinquent behaviors. In a study of 50 incarcerated youths, 20% were found to meet diagnostic criteria for mania ([Pliszka et al., 2000](#)). Mania in behaviorally disordered children looks very much like ADHD, oppositional defiant disorder, or conduct disorder, and does not have the "classic" presentation seen in adulthood ([Carlson, 1996](#)). In adolescence, the mood swings tend to be "mixed" or rapid cycling as opposed to the clearly defined episodes seen in adults with the disorder.

Whenever the clinician is confronted with a grandiose, boastful, belligerent, delinquent youngster, a bipolar diagnosis should be considered. Unfortunately, such youngsters alienate examiners and often are dismissed as merely narcissistic and sociopathic. Mania can manifest as vandalism sprees or sporadic episodes of robbery and burglary. One of our especially violent patients, when manic, shot out the street lights in his hometown. The disorder was not diagnosed until a second manic episode occurred during which a series of burglaries and a high-speed chase led to the shooting death of two officers.

## THOUGHT DISORDERS

Few seriously conduct-disordered or aggressive juveniles appear overtly psychotic. Nonetheless, their episodically violent and bizarre behaviors sometimes are manifestations of sporadic psychotic thinking and hallucinations. When not under stress, these children usually can maintain a normal facade. [Bender \(1959\)](#) made the observation that children who were psychotic in their early years appeared to be merely antisocial during adolescence. The most common psychotic symptom of aggressive children and adolescents is paranoid thinking ([Dodge et al., 1990](#); [Lewis et al., 1979b, 1989](#); [Myers et al., 1995](#); [Ulzen and Hamilton, 1998](#)). Unfortunately, their paranoia and grandiosity often are misconstrued as bravado or simply as the result of living in a tough neighborhood. Youngsters who view the world as a hostile, threatening place are primed to attack. Their grandiosity gives the appearance of remorselessness.

Our own studies suggest that the more violent or bizarre the youngster's acts, the more likely he or she has underlying psychotic symptomatology ([Lewis et al., 1979b, 1989](#)). Many aggressive juveniles who experience episodic auditory hallucinations try to hide these symptoms for fear of being labeled "crazy." We recently examined a 15-year-old boy who had gone on a shooting spree, killing and wounding several children. Periodically, since 12 years of age, he had heard voices in his head inciting him to violence. He had told no one about them.

## DISSOCIATIVE DISORDERS

It should come as no surprise that the scientific literature on delinquency and aggression fails utterly to recognize the existence of dissociative disorders, much less their contribution toward violent behavior. Our own early work is a case in point. We never considered the possibility that dissociative disorders, especially dissociative identity disorder, might account for particularly puzzling symptoms or bizarre behaviors in the violent youths we examined. Years ago, the fact that dissociation might have explained why some of the children we saw did not recall their acts never even occurred to us. However, as we learned, during dissociative episodes children may curse, steal others' belongings, set fires, even attack others, and subsequently deny their acts because they do not remember them. Many of the symptoms usually associated with complex partial seizures (e.g., lapses, staring spells, impaired memory, dreamlike states) also are characteristic of dissociation.

Children with severe dissociative disorders often hear voices in their heads ordering them to strike out at others or to kill themselves. Sometimes they are misdiagnosed schizophrenic. In fact, it has been reported that 19% of male patients with dissociative identity disorder are actually homicidal ([Loewenstein and Putnam, 1990](#)). Severe abuse and resultant dissociative disorders often are associated with extreme violence. In one study sample of severely abused, behaviorally disturbed children in residential treatment, dissociative symptoms abounded ([Trujillo et al., 1996](#)). However, because of their distractibility and episodic aggression, all of these children carried diagnoses of conduct disorder or ADHD. We reported the objective documentation of childhood dissociative symptoms and a history of extraordinary abuse in a series of 12 adult murderers with dissociative identity disorder ([Lewis et al., 1997](#)).

Among the many behaviors characteristic of the childhood externalizing disorders that also are typical of dissociation are episodic aggression, inappropriate sexual behaviors, mood lability, denial of behaviors, wandering away from home or school for hours or days, and apparent stealing (e.g., finding something in one's possession and denying any knowledge about how it got there). When the dissociative nature of these signs and symptoms goes unrecognized, children are dismissed as liars and thieves—in other words, as having a conduct disorder.

## SUBSTANCE ABUSE

Over half of violent crimes are committed when perpetrators are under the influence of alcohol ([Pihl and Peterson, 1993](#)). U.S. regional surveys have found that between 40% and 60% of male juvenile arrestees have drugs or alcohol in their systems at the time of detainment ([ADAM, 1999](#)). Surveys of high school students in urban areas reveal that drug and alcohol use is associated with increased risk of violent behavior, especially weapon carrying and fist fights ([Dukarm et al., 1996](#)). Similarly, violent adolescents are, reportedly, more likely than their less aggressive peers to use illicit substances ([DuRant et al., 1997](#)). Estimates of the prevalence of "multiproblem" youths in our society range from 4% to 20%—that is, youths who are violent, abuse substances, and have emotional problems ([Dryfoos, 1990](#); [Ellickson et al., 1997](#); [Elliot et al., 1989](#)).

The earlier and more serious the child's aggression, the greater the risk for subsequent substance abuse ([Loeber and Keenan, 1995](#)). Unfortunately, the causes underlying these aggressive behaviors rarely are determined before the onset of substance abuse. Looking backward, after an adolescent has committed aggressive acts and has started abusing alcohol or drugs, often all the clinician can discern in the distant past is a history of poor behavior, of conduct disorder.

Although the violence-promoting effects of most drugs seem to depend greatly on the psychological and physiologic condition of the user/abuser, and on environmental influences, there are certain substances that, in and of themselves, have reportedly engendered violence. These include crack cocaine ([Honer et al., 1987](#); [Taylor, 1990](#)), phencyclidine (PCP) (Fauman and [Fauman, 1982](#)), and the amphetamines ([King and Ellenwood, 1992](#)). We have evaluated several homicidal individuals, one a teenager, whose murderous acts took place while coming down from methamphetamine. The effects of benzodiazepines and barbiturates reportedly are dose dependent; low doses tend to decrease aggression, whereas moderate to high doses may increase it ([Cherek, 1990](#); [Taylor, 1990](#)). Marijuana, the most frequently used illicit substance among teenagers in the United States, usually causes relaxation and euphoria. In some individuals, however, it creates paranoia and panicky states that can lead to violence. It also may be laced with more dangerous substances (e.g., PCP) ([Hall and Solowij, 1998](#)).

The use of body-building substances also has been associated with violence. The anabolic steroids induce irritability, hostility, anger, and outright aggression ([Bahrke et al., 1990a, b](#); [Choi et al., 1990](#); [Parrott et al., 1994](#); [Uzych, 1992](#)). Some of the nonsteroidal substances consumed by body builders also can engender violence. We evaluated an otherwise nonviolent man who became homicidal while taking a mixture of ephedrine, caffeine, and chromium picolinate.

Substance abuse is used by different juveniles for different reasons, including attempts to self-medicate for depression, anxiety, panic episodes, psychosis,

dissociation, and hyperactivity. In fact, the drug of choice may provide a clue to the underlying disorder. It is therefore important to ask how the substance in question affects the youngster.

### Neurologic Vulnerabilities

As in the case of psychiatric vulnerabilities, neurologic impairment in conduct-disordered children rarely is obvious. In the past, neurologists tended to minimize the importance of "minor" head injuries (e.g., those that did not lead to prolonged periods of unconsciousness). We now know that even minor concussions from, for example, athletic injuries can have a cumulative effect, even when bleeding is not detectable on magnetic resonance imaging (MRI) ( [Pearl, 1998](#)). The temporal and frontal poles of the brain are especially vulnerable ( [Varney, 1999](#)). Recurrently violent children and adolescents have such horrendous medical histories ( [Lewis and Shanok, 1977](#); [Lewis et al., 1979a](#)) that it often is impossible to determine exactly when injury to the brain occurred. Even mild concussions may cause cognitive impairment ( [McCrea et al., 1997](#)), and children who have sustained one concussion are reportedly at higher risk for permanent damage from a second head injury than are their noninjured peers. Although the areas of damage cannot necessarily be localized, any injury to the brain may increase emotional lability, diminish judgment, and impair impulse control. Frontal lobe damage or dysfunction, in particular, has been associated with aggression ( [Raine et al., 1994, 1997b, 1998](#)). The frontal lobes contain the "executive" parts of the brain that are responsible for judgment, foresight, impulse control, and the appreciation of interpersonal cues. Damage to them impairs social functioning even when intellect remains intact. The frontal lobes also modulate the strong feelings emanating from the limbic system. Injury to the ventromedial parts of the frontal lobes affects autonomic responsiveness (e.g., blood pressure, heart rate, skin conductance). Studies of patients with damage to these areas revealed impaired physiologic responsiveness to emotionally charged materials ( [Damasio, 1998](#)).

Unfortunately, there is a paucity of data on orbitofrontal functioning in aggressive or delinquent children. Only one study has examined the effects of very early orbitofrontal damage on personality development and psychosocial adaptation in two children ( [Anderson et al., 1999](#)). Although both children reached developmental milestones at the usual times, both began to exhibit patterns of serious social maladaptation early on, including inability to follow rules or learn from punishment, lack of empathy toward others, lack of motivation and initiative, emotional lability, inability to develop friendships, and chronic lying, stealing, and physical fighting.

Few seriously conduct-disordered children or adolescents have epilepsy. However, many, perhaps by virtue of their adverse medical histories, have equivocal or diffusely abnormal electroencephalograms (EEGs). Psychomotor seizures (complex partial seizures), although uncommon in the conduct-disordered population, have been reported to be significantly more common in very violent delinquents than in the general population ( [Lewis et al., 1982](#)). Whether violent acts can occur during a seizure remains an area of heated debate. However, individuals with seizures often are more irritable interictally than persons without the condition.

### Intellectual and Cognitive Vulnerabilities

Most conduct-disordered children are not severely retarded. On the other hand, as a group, they tend to score in the low-normal or borderline ranges of intelligence as measured by standard tests ( [Hirschi and Hindelang, 1977](#); [Schonfeld et al., 1988](#); [Wilson and Herrnstein, 1986](#)). This association of behavior problems and intelligence quotient (IQ) has been documented to exist as early as 3 years of age, and hence is thought not to be the result of school failure ( [Richman et al., 1982](#)). Cognitive problems have been found to precede behavior problems ( [Schonfeld et al., 1988](#)). [Moffitt \(1990a\)](#) reported that aggressive delinquents whose behavior problems started early scored significantly lower on IQ tests than other samples of delinquent children. Because these intellectual deficits rarely are severe, behaviorally disordered children find themselves on the border of a diagnosis.

Learning disabilities, especially verbal learning deficits, are prevalent among behaviorally disordered and delinquent juveniles ( [Robbins et al., 1983](#)). Reading disabilities in particular have been associated with specific neurodevelopmental abnormalities in the language areas of the cerebral cortex ( [Galaburda et al., 1985](#); [Hynd et al., 1998](#)). Exactly how learning disabilities relate to behavior problems is unclear. We do know that verbal deficits limit a child's ability to express feelings in words rather than deeds. Furthermore, learning-disabled children experience more peer rejection and neglect than their normal schoolmates ( [Hazel and Schumaker, 1988](#)). It may be that learning disabilities are simply the most apparent sign of more pervasive neuropsychological impairment that, as a whole, diminishes judgment and increases impulsivity. Learning-disabled children whose difficulties include visual-spatial (right hemisphere) processing deficits frequently also have seriously impaired social skills ( [Reynolds, 2000](#); [Rourke and Furest, 1991](#)). These children tend to misread social cues, associated facial expression, body language, and tone of voice and, consequently, respond inappropriately.

The frontal lobe dysfunction characteristic of many conduct-disordered children impairs memory, abstract reasoning, and concentration, all of which are vital to the learning process ( [Moffitt and Lynam, 1994](#); [Skoff and Libon, 1987](#); [Wolff et al., 1982](#)). The cognitive deficits of behaviorally disordered children often appear clinically mild and such youngsters rarely score in the severely impaired range on testing. Hence, these children do not look "brain damaged." When such deficits in executive function are overlooked and untreated, children tend to be labeled simply as conduct disordered.

## ETIOLOGY AND PATHOGENESIS

For decades, the most influential theories regarding the development of antisocial behaviors were sociologic. Most influential was Merton's assertion that socioeconomically disadvantaged youngsters, unable to succeed through legitimate avenues, would, of necessity, turn to crime ( [Merton, 1938, 1957](#)). Others hypothesized that antisocial behaviors were not aberrant but, rather, were manifestations of a subculture of violence ( [Cohen, 1955, 1956](#)). However, more recently, even sociologists agree that socioeconomic disadvantage accounts only in part for antisocial behaviors.

### Possible Physiologic and Biochemical Contributions to Conduct Disorder

Over the past quarter century, investigators have struggled to find measurable physiologic characteristics with which to identify conduct-disordered and antisocial individuals. [Mednick \(1981\)](#) and [Hare \(1970\)](#) have hypothesized antisocial individuals suffer from an inherent autonomic hyporeactivity. They theorized that, as a consequence, delinquency-prone youngsters are less likely to inhibit their antisocial behaviors in response to aversive stimuli (i.e., punishment). [Porges and colleagues \(1996\)](#) reported an association between an infant's difficulties regulating the "vagal brake" (slowing of the heart rate) and subsequent behavioral problems at 3 years of age. More recently, [Damasio and colleagues \(1998\)](#) showed a relationship between damage to ventromedial parts of the frontal lobes and impaired autonomic responsiveness. This finding suggests that if autonomic hyporesponsivity exists in behaviorally disordered individuals, it may not be an inherited but, rather, an acquired condition. [Raine and colleagues \(2000\)](#) measured a variety of autonomic nervous system functions and found significant differences between a group of antisocial, aggressive individuals and comparison groups of substance abusers and healthy control subjects, and hypothesized a "significant brain basis" to antisocial personality disorder.

Studies of animals and humans suggest that such neurotransmitters as dopamine, norepinephrine, serotonin, and vasopressin influence aggressiveness ( [Ferris et al., 1998](#)). A strong association has been found between diminished amounts of serotonin in the central nervous system (CNS) and aggressive behaviors ( [Brown et al., 1979](#); [Coccaro et al., 1989](#); [Virkkunen et al., 1989](#)). Others are studying possible links between impaired metabolism of noradrenergic substances and aggressiveness ( [Pliszka et al., 1988](#); [Rogeness, 1994](#)). (For a more detailed discussion of the neurobiology of aggression, see [Chapter 29](#), Development of the Symptom of Violence).

The reader should bear in mind that the study of the neurophysiology of violence is in its infancy and that environment strongly influences the functioning of the CNS.

### Genetic Perspectives

Reports in the 1960s and 1970s suggested an association between certain chromosomal anomalies (47,XYY anomaly) and increased sociopathic tendencies ( [Casey et al., 1966](#); [Nielson, 1968](#)). However, few prisoners have the 47,XYY complement of chromosomes ( [Baker et al., 1970](#); [Jacobs et al., 1971](#)), and the overwhelming majority of people carrying the anomaly remain outside prisons and mental hospitals ( [Gerald, 1976](#)). Certain chromosomal abnormalities may impart a greater vulnerability to a variety of different problems in adaptation that manifest themselves as aggressiveness only in certain contexts ( [Owen, 1972](#)). There are studies that indicate that dysfunctional households may determine whether an individual with a particular genetic anomaly will manifest antisocial behaviors ( [Nielson, 1968](#)).

In another approach to issues of heredity, [Hutchings and Mednick \(1974\)](#) studied the records of adopted-away offspring of criminal fathers and found that their children were more likely to become antisocial than were the adopted-away children of noncriminal fathers. The children at greatest risk for antisocial behavior were those with criminal biological fathers and criminal adoptive fathers, a finding consistent with an interplay of genetic and environmental influences. Studies like these, relying as they do on registered parental criminality, must be interpreted with caution. They do not take into consideration the likelihood that parental criminality, especially in men, may simply be the most obvious manifestation of other, underlying neuropsychiatric disorders. There is, after all, strong evidence that the children



of parents who have a variety of different psychiatric disorders are more likely than the children of healthy parents to have behavioral problems ( [Moffitt, 1987](#); [Wilson and Herrnstein, 1986](#)). As [Walker et al. \(1989\)](#) have stressed, mentally ill parents often act in aggressive and neglectful ways and thereby contribute to maladaptation.

Regardless of etiology, the adaptational problems of males are especially likely to be manifested through aggressive acts compared with those of females. Unfortunately, disruptive males are more likely than females to be dismissed simply as delinquent or criminal and to be incarcerated, regardless of the neuropsychiatric underpinnings of their behaviors.

[Alsbrook and Pauls \(2000\)](#) observed, "Studying the genetics of behavior and psychiatric disorders is a particularly difficult endeavor since they are complex traits.... Genetic variables may be independent single genes (loci) or as is more likely to be the case, multiple loci functioning within the larger context of numerous environmental influences."

Researchers are only beginning to identify those genes that influence temperament and behavior ( [Buss and Plomin, 1984](#); [Gjone and Stevenson, 1997](#)). For example, genes have been identified that control the synthesis and degradation of particular neurotransmitters. [Brunner and colleagues \(1993\)](#) described a family in the Netherlands in which several of the male members exhibited aggressive, impulsive behaviors. Each of them was discovered to have a mutation in his monoamine oxidase-A (MAO-A)-regulating gene, which resulted in a deficiency of the enzyme. Similar findings have been reported for the gene regulating the production of catechol-O-methyltransferase ([Lachman et al., 1998](#); [Straus et al., 1997](#)).

Caution must be exercised in interpreting these kinds of genetic findings. The relationships of neurophysiologic predispositions to particular behaviors are complex. The transmission of behavioral traits from generation to generation depends on the interaction of numerous different heritable and environmental factors.

### Testosterone and Aggression

Although no abnormal constellation of chromosomes associated with violence has been identified, a normal constellation, XY, is strongly associated with it. Male aggressiveness is common to most animal species. There is reason to believe that male aggression is related to the masculinization of the brain *in utero* and is not simply a sociocultural phenomenon. Some studies of girls and boys born with adrenogenital syndrome have suggested that such children may be more energetic and physically active than their normal counterparts ( [Ehrhardt, 1975](#)). Other studies have called these findings into question ( [Hines, 1982](#)).

The relationship of serum testosterone levels to aggressive behavior is a subject of debate. [Persky and colleagues \(1971\)](#), in one of the first systematic studies of male aggression and testosterone, reported a positive correlation between self-assessed aggression and plasma levels of testosterone. Others have failed to replicate these findings ( [Meyer-Bahlburg et al., 1974](#); [Monti et al., 1977](#)). [Virkkunen et al. \(1994\)](#), in their study of adults, found elevated cerebrospinal fluid testosterone in impulsive, aggressive, antisocial, alcoholic men. [Mattsson and colleagues \(1980\)](#), in one of the few hormonal studies using juvenile delinquent subjects, found that incarcerated recidivist male subjects had slightly higher testosterone levels than did normal male adolescents. On the other hand, [Constantino and colleagues \(1993\)](#) found no significant differences in serum androgen levels between aggressive and nonaggressive prepubertal boys. Thus, the jury is still out on whether endogenous androgens distinguish more aggressive from less aggressive men.

### Abuse and Behavioral Problems

When all is said and done, the most important influences on violence are environmental or experiential. Although violence does not invariably beget violence, there is abundant evidence of the association of early maltreatment with the development of aggressive coping styles ( [Lewis et al., 1987, 1989](#); [Widom, 1989](#)). One of the most important characteristics of parents of seriously delinquent, violent juveniles is physical abusiveness toward their children and violence toward each other. The histories of severely behaviorally disturbed aggressive children again and again reveal a picture of physical or sexual abuse at the hands of adults, usually parents. This abuse usually is sufficient to leave evidence in terms of scars, but only occasionally is obvious enough to elicit protection of the child by the state. When abuse is serious enough to require hospital care, parents provide other explanations for children's injuries. The abuse remains hidden and nothing is done.

There are several ways to understand how abuse begets violence. First, parental violence becomes a model of behavior; second, it often results in CNS damage that contributes to a child's adaptational difficulties; and third, the stresses of maltreatment actually alter brain anatomy and physiology ( [Bremner et al., 1997](#)). People with posttraumatic stress disorder resulting from early sexual abuse reportedly have subtle signs of brain dysfunction and a plethora of neurodevelopmental problems ([Gurvits et al., 2000](#)). Finally, abuse engenders rage that usually is displaced from the abusing parent onto other figures in the child's environment, such as teachers and peers.

Our own work ([Lewis et al., 1979b, 1988, 1989, 1991, 1994](#)) and that of [Raine and colleagues \(1996, 1997a\)](#) documents some of the ways in which early deprivation and abuse, coupled with brain dysfunction, predispose children to early and ongoing violent, maladaptive behaviors (also see [Chapter 29](#)).

A word should be said about children who witness violence. Numerous studies have documented the cognitive, developmental, and psychiatric problems of children who witness family violence. These problems include not only anxiety, depression, and slow cognitive development ( [Edelson, 1999](#)), but aggression and antisocial behavior ([Henning et al., 1996](#); [Song et al., 1998](#); [Spaccarelli et al., 1995](#)).

### Summary of Etiologies

This review of the intrinsic and extrinsic contributors to disturbances of behavior illustrates that conduct disorder is not a single diagnostic entity. It is, rather, the final common pathway of the interaction of a variety of different kinds of psychobiological vulnerabilities with a variety of different kinds of environmental stressors. It is clear that children with disorders of conduct are on the border of numerous neuropsychiatric disorders and, in child psychiatry, the border of any condition is a precarious line on which to teeter.

## DIAGNOSTIC EVALUATION

Because the criteria for diagnosing conduct disorder are so broad, and so many different kinds of psychiatric disorders present as behavior problems, it is imperative that the clinician perform a detailed, comprehensive neuropsychiatric evaluation before dismissing a child as simply having a conduct disorder. Clinicians must therefore overcome negative feelings aroused by a child's obnoxious behaviors and embark on the evaluation with curiosity and an open mind.

A plethora of rating scales has emerged over the years, designed to enable the clinician to obtain information regarding behaviors from children, parents, and teachers. Among the most widely used are the Conners' Rating Scales ([Conners, 1997](#)) and the Child Behavior Checklist ([Achenbach, 1992](#)). Unfortunately, these scales focus almost exclusively on conduct, inattention, and hyperactivity; they do not encourage parents or teachers to share other kinds of invaluable observations relevant to diagnosis and treatment. Some of the semistructured interviews, such as the National Institute of Mental Health Diagnostic Interview Schedule for Children, Revised (NIMH-DISC-R) ([Schaffer et al., 1996](#)), Diagnostic Interview for Children and Adolescents (DICA) ([Welner et al., 1987](#)), and the Schedule for Affective Disorders and Schizophrenia, Childhood Version (K-SADS) ([Ambrosini and Dixon, 1996](#)), are of potentially greater clinical usefulness because the evaluator is obliged to explore areas other than just behavioral problems. Occasionally, the clinician uncovers a distinct diagnostic entity, such as a bipolar mood disorder or paranoid schizophrenia, masquerading as conduct disorder. More often the evaluation turns into a painstaking search for combinations of biopsychosocial vulnerabilities that affect behavior. Space precludes a detailed description of the diagnostic process; however, certain parts of the evaluation are highlighted.

### The Behaviors

First, the nature of the problematic behaviors must be explored in detail, including the circumstances in which they occur, the precipitants, and the amount of control the child seems to have over them. In the case of aggressive acts, it is essential to learn whether the child can tell in advance when the behavior will occur, the extent to which he or she can discontinue it voluntarily, the aftermath of the act (e.g., does the child feel shaky or dizzy or sleepy?), and the child's memory for the act. Many paranoid youngsters say that they can tell in advance when a fight will begin by the way the other person looks or stands. Children with complex partial seizures may experience auras before emotional outbursts and may feel confused, sick, or sleepy afterwards. After aggressive acts, paranoid or delusionally grandiose children often say they feel glad and that the victim "deserved it." Children with seizure disorders or dissociative disorders have trouble recalling what happened.

## The Medical History

One of the most important aspects of the evaluation is a detailed medical history, obtained from the child as well as the parent. Questions must be asked in ways that avoid “yes” or “no” responses, and answers must be pursued to their fullest. For example, after inquiring about the most serious accidents or injuries the child has experienced, the examiner, regardless of whether a negative response was given, must ask specifically about cars, bicycles, falls from high places, blows to the head, about dizziness, headaches, and blackouts. All positive responses should be followed up with such questions as, “And what *other* bicycle accidents have you had?” “And when else did you feel dizzy?” ([Hatzitaskos et al., 1994](#); [Stein et al., 1993](#)). We also have found the medical history to be a useful device for conducting parts of the mental status examination. Tough adolescents are unlikely to admit to hallucinations or delusions if asked about them directly. They would rather be thought bad than crazy. However, if these issues are integrated into the medical history (e.g., “How are your ears? Have you ever had an ear infection? Have your ears ever played tricks on you? Have you ever had the experience of thinking someone said something bad about you or your mom, and you turn around to fight, and the person really didn't say anything?”), the wary adolescent, who has experienced episodic hallucinations or paranoid misperceptions, may reveal them for the first time.

Many conduct-disordered youngsters carry evidence of their medical and family histories all over their faces, heads, arms, legs, and bodies in the form of scars. Part of the medical history is inquiry regarding the cause of every visible scar. Examiners should ask, “Are there any scars I can't see?” These kinds of questions often bring to light forgotten injuries and previously ignored evidence of child abuse. The child's or adolescent's body must be examined carefully in an appropriate setting. We recently examined a violent teenager and discovered a scar several inches in length running down his penis, into the scrotum.

## Inquiring About Sexual Abuse

Conduct-disordered children, especially those with sexually inappropriate or provocative behaviors, often have histories of having been sexually abused. The examiner must not only develop skill inquiring about sexual abuse, but must feel comfortable with the follow-up questions, “And who else bothered you? And who else? How about outside the family?” There is an art to enabling a child to reveal the unspeakable without posing unduly leading questions. We ask, “Who taught you about sex?” and “Have you ever had sex with someone much older? Much younger?”

## THE NEUROLOGIC EXAMINATION

Few children and adolescents who come in conflict with the law as a result of aggressive behaviors have obvious signs of CNS damage or dysfunction. On the other hand, as a result of the early, multiple insults to the CNS that are characteristic of the medical histories of violent children and adolescents ([Lewis and Shanok, 1977](#); [Lewis et al., 1979a](#)), they commonly have a myriad of subtle indicators of CNS dysfunction. Whether these vulnerabilities are recognized and addressed depends on the thoroughness of the neurologic investigation.

The way in which a referral is made affects the quality of the neurologic data obtained. It is therefore insufficient simply to refer a child to a clinic or to a particular neurologist for “a neurologic evaluation.” The clinician responsible for the overall assessment of the child or adolescent should already have taken a detailed medical and neurologic history, the findings from which should be presented as part of the referral to the neurologist (e.g., the child's failure to recall behaviors, history of head trauma, findings on neuropsychological testing). Without this kind of information, the neurologist will likely perform a “routine” examination and leave out assessments of subtle brain dysfunction (e.g., frontal lobe impairment, complex partial seizures).

Children with clear evidence of organic impairment or dysfunction on neuropsychological tests may have normal EEGs and MRI scans. On the other hand, EEG and MRI sometimes bring to light otherwise questionable brain damage.

## Psychological and Neuropsychological Testing

The usual tests of intelligence [e.g., the Wechsler Intelligence Scale for Children (WISC-III)] are helpful for documenting a child's level of understanding, but they do not tap frontal lobe or “executive functions” adequately. Neuropsychological assessment aims to tie observable abnormal behaviors to neurologic dysfunction or damage by parsing complex mental processes into their elemental cognitive components ([Moffitt, 1990b](#)). Neuropsychological examination of the aggressive child or adolescent should therefore attempt to identify the kinds of deficits that put the youngster at risk for responding maladaptively. It often can identify functional brain impairment not evident either on neurologic examination or neuroimaging. There is no one set of tests used routinely to assess neuropsychological functions. Selection should be based on the nature of the clinical questions to be addressed and, of course, reliability, validity, and the adequacy of norms for the patient's chronologic age and socioeconomic status.

The most commonly used personality assessment instruments for adolescents, namely, the Minnesota Multiphasic Personality Inventory—Adolescent version (MMPI-A) ([Butcher et al., 1992](#)) and the Millon Adolescent Clinical Inventory (MACI) ([Millon et al., 1993](#)), are more assessments of immediate psychopathology and emotional distress than of stable personality traits. Unfortunately, data gleaned from these instruments often are overinterpreted for purposes of predicting future delinquency and aggression and can be misused in juvenile justice and criminal justice settings. We have found frequently that adolescents with a multiplicity of symptoms who honestly subscribe to many different items risk being dismissed as fakers.

## Psychoeducational Assessment

Scholastic underachievement and school failure are common correlates of delinquency and aggression. Clinicians must not automatically ascribe academic problems simply to the juvenile's lack of motivation or bad attitude; instead, underlying cognitive, psychiatric, emotional, and environmental causes of academic underachievement must be investigated. School is perhaps the most potent source of social and psychological reinforcement for the older child and adolescent, and the inability to perform up to the expectations of parents, teachers, and peers has potentially life-long negative effects on self-esteem, social aptitude, and occupational stability. There is no one standard educational assessment battery. Suffice it to say that whatever tests are used must have appropriate norms and a high degree of validity and reliability. Findings from educational testing should be coordinated with those from neuropsychological testing.

## DIFFERENTIAL DIAGNOSIS

The diagnosis of conduct disorder usually just tells people what they already know: the child misbehaves. The differential diagnosis, however, is almost as broad as the entire field of child and adolescent psychiatry. Learning disorders, attention deficit hyperactivity disorder, mild mental retardation, seizure disorders, schizophrenia, mood disorders, dissociative disorders, even dissociative identity disorder (multiple personality disorder), all may manifest themselves as disorders of conduct. Most often, the conduct-disordered child or adolescent is found to have a variety of different kinds of psychiatric, neurologic, cognitive, and environmental vulnerabilities that, together, contribute to maladaptation. Each must therefore be identified and addressed. When these specific vulnerabilities are addressed, the child's conduct almost invariably improves.

Conduct disorder is much like fever; it can have many different causes, and it resolves only when those causes are identified and treated specifically. Whenever possible, a diagnosis reflective of underlying psychiatric or neurologic impairment should be used, rather than conduct disorder. However, just as, occasionally, in the absence of adequate information, the clinician must make the descriptive diagnosis of fever of unknown origin, so from time to time he or she may be forced to settle for the purely descriptive designation *conduct disorder*, meaning “behavior problem of unknown origin.” Often, conduct disorder proves to be an interim diagnosis on the way to a better understanding of the child's psychopathology and to a more therapeutically useful conceptualization of the clinical picture.

## TREATMENT

Many different treatment modalities for conduct-disordered children and adolescents have been tried, including psychotherapy and community-based programs ([Kazdin, 2000](#)). The success of most of these diverse modalities, unfortunately, remains untested. According to Kazdin, at this time, only three modalities have been shown, through controlled clinical trials, replication, and follow-up evaluation, to be effective—that is, to be “evidence based.” These modalities are (a) parent management training (PMT), (b) cognitive problem-solving skills training (PSST), and (c) multisystemic therapy (MST). This does not mean that other approaches may not be equally as good or superior. It means only that the research demonstrating success has not yet been done. Hence, this section focuses on the evidence-based modalities.



## Parent Management Training

Parent management training (PMT) focuses on teaching parents methods of interacting with their children to encourage appropriate behaviors and discourage antisocial, aggressive behaviors. It is based on the theory that many of the parents of behaviorally disturbed children unwittingly, through harsh punishments for infractions and failure to acknowledge good behavior, wind up reinforcing the very behaviors they wish to suppress ( [Dishion and Andrews, 1995](#)). Therapists work with the parents to help them use positive reinforcement for desirable behaviors, to use mild punishments for misbehavior, and to develop new ways of negotiating with their children ( [Patterson, 1982](#)). A literature review has singled out PMT as the only well established modality for the treatment of conduct disorder ( [Brestan and Eyberg, 1998](#)). In several studies, treatment gains were reportedly maintained from 1 to 3 years after treatment ( [Kazdin, 2000](#)).

A major drawback to successful outcome using PMT is the responsibility it places on parents to understand the basic principles underlying the program (e.g., the counterproductiveness of coercive disciplinary methods), to observe their children's behaviors systematically, to implement different ways of interacting and, of course, to attend training sessions. Unfortunately, the children and families in greatest need usually also are the most impaired and the least likely to follow through with a PMT program.

## Cognitive Problem-Solving Skills Training

Problem-solving skills training is a treatment modality that focuses on the child or adolescent ( [Spivak and Shure, 1974, 1978](#)). Based on the theory that conduct-disordered youngsters do not make proper use of their potential cognitive abilities, this modality tries to help children identify problems, recognize causation, appreciate consequences, consider alternative ways of handling difficult situations, and improve reality testing by cognitive means ( [Dodge and Frame, 1982](#); [Dodge and Tomlin, 1983](#); [Dodge et al., 1990](#)). Others have used PSST to enhance children's abilities to handle anger ( [Kendall and Braswell, 1982](#); [Lochman et al., 1991](#)). PSST attempts to help children think ahead, generate alternative solutions to problems previously dealt with impulsively, and to anticipate the potential consequences of behaviors. These are particularly difficult tasks for children with frontal lobe dysfunction. Older children seem to respond better than younger children to these kinds of modalities ( [Durlak et al., 1991](#)). It is not surprising that children with identified comorbid problems and those from chaotic homes respond less well to cognitive interventions than do more intact children from more stable households ( [Kazdin, 1995, 1997](#)).

## Multisystemic Therapy

Multisystemic therapy (MST) is based on the theory that a multiplicity of systems influence a child's adaptation, including family, peers, schools, and communities. Treatment focuses on the nature of these systems and how the child interacts with them. Treatment varies, depending on the child's and family's needs. It may therefore include PMT, PSST, marital counseling, or whatever other modalities are considered appropriate to identified problems. It is not surprising to learn that MST has been shown to be effective in several outcome studies, given that it recognizes the individual and environmental vulnerabilities of severely delinquent children and adolescents and attempts to address all of them. It is the antithesis of the "one size fits all" approach to treating conduct disorders. As such, it has been found superior to the usual services provided obstreperous or aggressive adolescents (e.g., probation, individual counseling) ( [Henggeler et al., 1998](#)). As [Kazdin \(2000\)](#) observes, MST poses replication difficulties because the precise techniques to use to address particular problem areas are not clearly specified. Nevertheless, he observes, "It may be the case that this model of treatment delivery is precisely what is needed for clinical problems that are multiply determined, protracted and recalcitrant to more abbreviated interventions" ( [Kazdin, 2000](#)).

## Pharmacotherapy

Because conduct disorder is not really a single entity, it is not surprising that no single medication or type of medication is especially useful. Such medications as stimulants, tricyclic antidepressants, lithium carbonate, MAO inhibitors, clonidine, antiepileptics, sedatives, and even beta blockers have been tried. Results, except in cases of the use of stimulants for clearly defined attentional problems, have been equivocal at best ( [Karper and Krystal, 1997](#); [Werry, 1994](#)). That is not to say that medication cannot be of enormous help. The choice of which medication or medications to use, however, must rest on the underlying symptoms to be addressed, and medication alone is rarely, if ever, sufficient. Used judiciously and on the basis of the specific clinical findings in each individual case, appropriate medication enhances the success of other treatment modalities.

The vulnerabilities of conduct-disordered children usually are chronic. Therefore, a successful treatment program must incorporate a plan for ongoing treatment and support systems. Medical, emotional, educational, and social supports often are required well beyond 16 years of age if severely behaviorally disordered children and adolescents are to adapt appropriately as adults.

## RESEARCH DIRECTIONS

It certainly is possible that neurochemical or physiologic factors related to conduct disorder will be discovered. However, the fact that so many different kinds of biopsychosocial problems are associated with behavior problems makes it unlikely that the search for a single biological or genetic factor peculiar to conduct disorder or its adult counterpart, antisocial personality, will be especially fruitful. It is likely, however, the development of laboratory tests for other kinds of psychiatric disorders such as schizophrenia, depression, and bipolar disorders will eventually help identify children whose antisocial behaviors obfuscate underlying, potentially more easily treatable mental illnesses. As neuroimaging studies of behaviorally disturbed children become safer and more feasible, it is possible that the diagnosis of particular frontal lobe syndromes will take the place of the diagnosis conduct disorder in many cases.

Recognition of the biopsychosocial interactions that contribute to deviant behavior raises important ethical and legal questions. Therefore, ethical issues regarding individual responsibility and the appropriate societal response to violent, antisocial behaviors deserve to be studied. The effect of incarceration on aggressiveness is a vital research question. Furthermore, the negative attitudes of clinicians toward behaviorally disordered, aggressive patients, and the ways these attitudes affect the diagnostic process, must become a focus of research. Aggressive patients, children and adults alike, rarely receive as careful, unbiased assessments as do patients with less frightening symptomatology. As we understand more and more about the psychobiological vulnerabilities of behaviorally disordered children, the global diagnosis conduct disorder will resolve into more discrete and treatable entities. It may even disappear.

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## 54 EATING AND GROWTH DISORDERS IN INFANTS AND CHILDREN

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Although eating disorders are frequently assumed to be synonymous with adolescent-onset anorexia and bulimia nervosa, a panoply of eating and growth disorders occurs earlier in life. This clinical myopia is reflected in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) ([American Psychiatric Association, 1994](#)), which recognizes only three eating disorders of early childhood: pica, rumination, and feeding disorder of infancy or early childhood. The lack of official diagnostic recognition of many eating disorders reflects their extraordinary complexity and resulting problems in nosologic definition. Eating and growth disorders are unique in medicine and psychiatry in their position at the dynamic interface of the somatopsychic boundary of developmental neuropsychiatric disorders, emotional/behavioral disturbances, and physical changes, which all interact in a complex fashion over time. Unfortunately, this complex interaction has not been recognized. For example, the two most common eating and growth disorders of infancy and childhood, failure to thrive (FTT) syndrome and obesity, are both currently categorized as medical conditions in DSM-IV and the 10th edition of the *International Classification of Diseases* ([World Health Organization, 1990](#)). The rationale for this approach is that both syndromes are defined by caloric nutritional status regardless of etiology. Therefore, some infants and children may have FTT or obesity but not actually have an eating disorder.

Until recently, understanding, and even categorization, of such disorders has been hampered by a reductionistic approach relying on deterministic, linear causality ([Woolston, 1988](#)) in which each disorder or developmental disturbance was viewed as having a single cause. This approach gradually has been replaced by the concept of a multifactorial, transactional model of development ([Sameroff and Chandler, 1975](#); [Sameroff and Fiese, 1989](#); [Woolston, 1989](#)). Despite this more sophisticated theoretical approach of developmental psychopathology, advancement in the understanding of early growth disorders has been hampered by a variety of controversies that actually arise from the complexity of the phenomena. Two such controversies are in phenomenology and etiology. The phenomenology of eating disorders has been plagued by a paucity of data and by persistent diagnostic confusion. For example, FTT and psychosocial dwarfism (PSD) frequently are used synonymously (e.g., [Green et al., 1984](#)); the distinctions among rumination, gastroesophageal reflux (GER), and psychophysiological vomiting rarely are delineated; and subtypes of such disorders as FTT ([Egan et al., 1980](#); [Woolston, 1983](#)), obesity ([Woolston, 1987](#)), and rumination ([Mayes et al., 1988](#)) have proliferated. Furthermore, controversy about etiology frequently has focused on a rigid dichotomy between an intrinsic, organic cause ("sick baby") versus an external, environmental disturbance ("bad mother") ([Woolston, 1991](#)). The counterproductive effect of such a dichotomous approach has been best demonstrated by the research in FTT syndrome, in which the organic/nonorganic differentiation has not held up to scrutiny ([Bell and Woolston, 1985](#); [Polan et al., 1991a](#)).

A fundamental obstacle to progress in the understanding and treatment of eating and growth disorders is lack of understanding of the basic mechanisms of hunger and satiety. The first major advance in this field occurred with the identification and coding of the ob gene, as well as its protein product, leptin ([Considine et al., 1995](#); [Green et al., 1995](#); [Zhang et al., 1994](#)). Experimental evidence suggests that leptin, produced in adipocytes, acts a hormonal feedback signal to regulate fat cell size



through hypothalamic mechanisms controlling food intake and metabolic rate ([Considine et al., 1996](#); [Lonnqvist et al., 1995](#); [Woods and Stock, 1996](#)). Leptin levels are highly correlated with body mass index in normal children, but not in children with eating disorders, resulting in a significantly elevated or reduced body mass index ([Argente et al., 1997](#)). These findings provide the first steps in elucidating the physiologic mechanisms of hunger and satiety.

## PICA

### Definition and Clinical Description

Pica would appear to be without the nosologic problems that beset other eating and growth disorders of infants and children. In fact, DSM-IV, which lists pica as the first feeding/eating disorder, defines it as the persistent eating of nonnutritive substances for at least 1 month in such a fashion that such eating is inappropriate to developmental level and is not part of culturally sanctioned practice. The two dependent clauses in this definition disqualify the two largest populations described in the extensive literature about pica: toddlers who ingest paint chips and young pregnant women who eat starch and clay ([Lacey, 1993](#)). This artificial clarity in definition attempts to solve the confusion that bedevils all aspects of the understanding of this disorder. Rather than being a narrowly homogeneous phenomenon, pica represents an eating disorder of enormously varied populations, including normally developing toddlers mouthing lead paint chips, rural pregnant women eating clay or starch, severely retarded adults eating feces, and normally developed adults chewing pencil erasers, fingernails, ice, and burnt matches. As if to create an aura of scientific understanding, pica as defined in literature before DSM-IV was subtyped according to the substance ingested. These terms include geophagia (eating clay), pagophagia (eating ice), plumbophagia (eating lead), amylophagia (eating starch), coprophagia (eating feces), cautoxyreophagia (eating burnt matches), trichophagia (eating hair), lithophagia (eating stones), and geomelophagia (eating raw potatoes). Aside from a review of Latin root words, such subtyping is most useful at documenting the extraordinary range of substances that people may ingest.

In many ways, the heterogeneity of pica precludes a coherent statement about age of onset, developmental outcome, etiology, or treatment. Because the pediatric literature has paid great attention to the phenomenology of lead poisoning, pica has become synonymous with children eating paint chips. For these children, pica has been reported to be more common in situations of relative environmental deprivation ([Madden et al., 1980](#)) or parental psychopathology. In this population, pica begins in the second and third year of life and may continue into childhood. In a second population, young pregnant women, the onset of pica begins with the first pregnancy in late adolescence or early adulthood and may continue intermittently for several decades. Pica in mentally retarded people begins in childhood and may diminish after middle age ([McAlpine and Singh, 1986](#)). In this population, the risk of pica appears to be related to the degree of retardation, so that the most severely retarded are most likely to have the disorder ([Danforth and Huber, 1982](#)). Pica in chronically anxious children or adults has never been studied or described in the literature. In this last group, a possible relationship between pica, trichotillomania, and other compulsive behavior disorders has never been investigated.

### Prevalence and Epidemiology

Incidence and prevalence figures for such a heterogeneous disorder are difficult to interpret. However, among specific at-risk populations, pica has been reported to be quite common. [Halstead \(1968\)](#) summarized findings from research studies that indicated that pica occurred in 25% to 33% of young children and 40% to 50% of poor, pregnant black women. The incidence of pica among institutionalized retarded adults has been reported to range from 9% ([McAlpine and Singh, 1986](#)) to 25% ([Danforth and Huber, 1982](#)).

Numerous studies have documented that the eating of specific substances such as clay and starch is strongly influenced by cultural and family factors ([Lacey, 1990](#)) because various social groups, especially poor and rural people, regard the eating of certain nonnutritive substances as acceptable. In addition, toddlers who are poorly supervised and understimulated are at high risk for ingesting inappropriate materials like paint chips.

### Biology and Pathogenesis

Except as mediated by mental retardation, there are no known genetic factors in the etiology of pica. Boredom, anxiety, and depression may exacerbate pica, but little has been reported about such associated psychopathology. Iron deficiency sometimes is associated with pica, but the direction of the causal association is unclear ([Vyas and Chandra, 1984](#)).

### Laboratory Studies and Differential Diagnosis

Although the extraordinary diversity of this disorder makes any useful classification schema unlikely, the remarkable prevalence and seriousness of its medical complications require a thoughtful, vigorous approach. These medical complications include heavy metal poisoning, mineral/vitamin deficiency, parasite ingestion, and intestinal obstruction. For toddlers and young children with a history of pica, a careful evaluation of the home environment and family functioning is required. In addition, these children should be evaluated for cognitive and psychiatric impairments. Pregnant women with pica presumably have more voluntary control over their eating. A careful assessment of their cultural and nutritional beliefs is essential, so that nutritional counseling can be helpful. Severely retarded people presenting with pica should be assessed for access to noxious materials and provided with sufficient supervision to prevent pica.

### Treatment, Natural History, and Outcome

Because of the extreme heterogeneity of the disorder, few comments are germane to the treatment and natural history for the wide spectrum of people who meet the criteria for pica. For toddlers and young children, as well as older mentally retarded individuals, proper supervision is the most important intervention. In addition, careful scrutiny of the environment to remove toxic substances is crucial. Older adolescents and young adults should be provided with psychoeducation and counseling about the medical risks associated with ingestion of nonnutritive substances.

## RUMINATION DISORDER

### Definition

Similar to pica, rumination disorder has the superficial appearance of a homogenous, unitary disorder. DSM-IV defines rumination as (a) repeated regurgitation and rechewing of food (in the absence of associated gastrointestinal illness) for a period of at least 1 month after a period of normal functioning and (b) not due to an associated gastrointestinal or other general medical condition (e.g., esophageal reflux).

### Prevalence and Epidemiology

Very little is known about the incidence and prevalence of rumination, although the most severely affected cases, which are the ones that have been reported, are rare. The sex ratio of the disorder also is unclear, although one case series ([Mayes et al., 1988](#)) reported a male predominance.

### Clinical Description

Rumination appears to require three factors: an impaired ability of the infant or retarded person to regulate his or her internal state of satisfaction, a physical propensity to regurgitate food, and a learned association that regurgitation helps relieve the internal state of dissatisfaction. The assessment of the child must include an evaluation of each of these factors, as well as the medical sequelae of rumination such as malnutrition and aspiration. Obviously, these medical sequelae will influence the vigor of the intervention, as well as serving as a baseline by which to measure the child's response to treatment.

### Biology and Pathogenesis

Unfortunately, the clinical reality of this phenomenon is more complex than portrayed by DSM-IV. Some infants and retarded adults may vomit the stomach contents rather than rechewing and reswallowing them. Although this phenomenon is called *psychophysiological* ([Ferholt and Provence, 1976](#)) or *operant* ([Johnston, 1993](#)) vomiting, its differentiation from rumination is problematic. Furthermore, the requirement that rumination not be associated with gastrointestinal disorders such as GER is overly restrictive because it implies that rumination has no underlying physiologic contributions. Indeed, the relationship, if any, between GER, rumination, and operant vomiting has not been investigated.

Because rumination is apparently a relatively rare disorder, virtually all of the information about the phenomenology must be derived from single case reports or small case series (e.g., [Mayes et al., 1988](#); [Sauvage et al., 1985](#); [Winton and Singh, 1983](#)) without comparison groups. In otherwise normally developing infants, the age of onset is in the first year of life, whereas in mentally retarded people, the age of onset can extend into adulthood ([Mayes et al., 1988](#)). Rumination has been reported to occur in nonretarded adults, many of whom have an additional comorbid eating disorder ([Eckern et al., 1999](#)). In some unknown proportion of cases, the disorder remits spontaneously, whereas in others, the course may be malignant, marked by aspiration, severe malnutrition, growth failure, developmental delay, and death. Some authors ([Mayes et al., 1988](#)) have proposed two diagnostic subgroups, psychogenic and self-stimulatory, to capture the difference in the course of retarded and nonretarded individuals. However, the relatively small sample size appears to make diagnostic subtyping premature.

Several authors have proposed that an adverse psychosocial environment is central to the development of rumination in otherwise normal infants (e.g., [Hollowell and Gardner, 1965](#); [Mayes et al., 1988](#); [Richmond et al., 1958](#)). The most common environmental factor cited in the genesis of rumination is an unsatisfactory mother–infant relationship that causes the infant to seek an internal source of gratification. This turning inward by the infant has been proposed to occur because the environment is more stimulating than the infant can tolerate or because the environment is not gratifying enough ([Richmond et al., 1958](#)), or because the environment is too stimulating with negative effects ([Ferholt and Provence, 1976](#); [Hollowell and Gardner, 1965](#)). Aside from these proposed contradictory environmental factors, no information has been reported about more general sociodemographic features. Similarly, the role of genetic factors is unknown.

Learning theorists have explained rumination in terms of the reinforcing response that it elicits ([Johnston, 1993](#)). These proposed feedback mechanisms include positive reinforcement when a desired event such as pleasure or attention follows rumination and negative reinforcement when an undesired event such as anxiety is reduced or removed. A more sophisticated theory involves combining the concept of positive and negative reinforcement by proposing a change in the valence of the behavioral consequences. For example, consequences that normally are behavior suppressing may acquire behavior-reinforcing characteristics if other, more usually positive, consequences are lacking ([Winton and Singh, 1983](#)). These concepts of the operant conditioning psychogenic factors in rumination are useful as the theoretical underpinnings of certain behavioral treatments of rumination.

The role of organic factors in rumination remains obscure, but some authors have argued that rumination is totally the result of physical disorders, including hiatal hernia and other esophageal abnormalities ([Herbst et al., 1971](#)). The relationship between the syndromes of rumination and GER is unknown, although high rates of association have been reported ([Shepherd et al., 1987](#)). GER, or chaliasia, is the syndrome of regurgitation of the stomach contents into the mouth and esophagus, apparently as a result of hypomotility of the gastric fundus and delayed gastric emptying rather than a weakened esophageal sphincter ([Papaila et al., 1989](#)). A possible but untested hypothesis is that GER is the physiologic substrate on which various psychosocial disruptions or deviant operant conditioning act so that rumination develops.

The assessment of the infant's capacity to regulate his or her internal state must include a general developmental assessment to evaluate for serious developmental delay, as well as hyperirritability states. In addition, the mother–infant relationship must be carefully examined for clues to stimuli that are noxious or disruptive to the infant.

### Laboratory Studies

The child's propensity to regurgitate can best be evaluated by procedures developed for GER, including esophageal pH monitoring, scintigraphic GER scan, endoscopy, and gastric emptying studies. In addition, various radiologic procedures that evaluate esophageal abnormalities should be considered.

### Treatment and Natural History

Similar to other eating disorders of early childhood, the extraordinary heterogeneity precludes specific treatments for all persons with this disorder. The learned aspect of rumination, especially in retarded people, may be crucial to modify. After careful evaluation of this factor, various behavioral interventions must be considered, especially if there are serious medical complications. These interventions include aversive techniques, in which a noxious stimulus is paired with rumination ([Glasscock et al., 1986](#)), and nonaversive techniques such as differential reinforcement of other incompatible responses.

## FAILURE TO THRIVE

### Definition

Failure to thrive is a disorder of infancy and early childhood characterized by a marked deceleration of weight gain and a slowing or disruption of acquisition of emotional and social developmental milestones. Deceleration of linear growth and head circumference growth are associated but not primary phenomena.

### Historical Note

The DSM-III ([American Psychiatric Association, 1980](#)) focused on the developmental delays presumably caused by psychosocial deprivation and virtually ignored the disordered growth and feeding. For this reason, DSM-III described FTT as reactive attachment disorder. DSM-IV ([American Psychiatric Association, 1994](#)) sought to redress this lack of recognition of the feeding disorder by new diagnosis of feeding disorder of infancy and early childhood. Although creating a new diagnostic category has the advantage of freeing it from a long history of misconception and controversy, it has the disadvantage of losing a long, rich history of research.

Despite a 45-year history of study, the understanding of FTT has been marked by confusion and controversy about such basic issues as the definition and the name of the disorder. The plethora of syndromic names provides a glimpse into the confused literature: hospitalism, anaclitic depression, institutionalism, environmental retardation, maternal deprivation syndrome, psychosocial deprivation dwarfism, deprivational dwarfism, deprivation syndrome, FTT, environmental FTT, and nonorganic FTT syndrome. This blizzard of interchangeable but nonsynonymous terms, which frequently represented the mistaken or oversimplified underlying conceptions of the investigators, has created a major obstacle to the course of research. These misconceptions arose out of the multifaceted nature of the syndrome. At different times, the three components of FTT, weight gain deceleration, linear growth delay, and developmental delays, were considered separately so that each was perceived as the central aspect, to the exclusion of the other parts. In fact, depending on the focus of the definition, FTT has been reported as consisting of only one component rather than a triad. For example, when the diagnosis of FTT is made on the basis of primary weight gain deceleration, developmental deficits are less evident ([Field, 1984](#)), and indeed, in one study using age-matched control subjects, there were no differences in development test scores between infants with FTT and normally growing infants ([Mitchell et al., 1980](#)). A second aspect of this confusion in the understanding of FTT has been the controversy over the relative contributions of emotional deprivation and of malnutrition. From the earliest observers to contemporary clinicians, the correlation between emotional misery and growth problems has been obvious. Perhaps too simplistically, some investigators argued that such disorders were directly caused by misery, mediated by some effect on the mind acting directly on the body, without requiring such external factors as altered caloric intake.

The starkest presentation of the argument has been the theoretical debate over the importance of love versus food in the etiology of FTT ([Widdowson, 1951](#)). Early clinical experience indicated that some infants with FTT who were given a normal caloric intake did not gain weight at their expected rate. These observations were used to bolster the argument that calories alone were not sufficient for weight gain. However, studies of malnourished children have demonstrated that they have supracaloric requirements before catch-up growth is possible ([Casey and Arnold, 1985](#)). In addition, [Whitten et al. \(1969\)](#) reported that even grossly understimulated infants with FTT gained weight rapidly if given enough food.

### Prevalence and Epidemiology

Failure to thrive is a common disorder, occurring at a rate of 1% to 5% of pediatric hospital admissions ([Berwick, 1980](#)). Surveys of low-income children in primary care suggest that nearly 10% show weight or length below the fifth percentile for age ([Koumjian and Marks, 1985](#)). Approximately 5% to 10% of all children 2 to 5 years of age in the United States exhibit poor growth that is unrelated to a medical disorder ([Drotar and Sturm, 1998](#)). This common condition is of great concern because FTT is associated with increased risk for lasting deficits in growth, cognition, and socioeconomic functioning (e.g., [Oates et al., 1985](#)). Despite the clinical importance of FTT in pediatric populations, the heterogeneity of etiology and phenomenology has impeded research efforts ([Benoit, 1993](#)).

### Clinical Description, Etiology, and Pathogenesis



The persistence of the debate over love versus food has several origins. First, FTT is a syndrome with physical (weight gain and growth deceleration) and behavioral/emotional (developmental delays) components. The overwhelming evidence indicates that inadequate caloric intake is the primary cause of growth deceleration, whereas emotional and socioeconomic deprivation is the primary cause of the developmental delays ( [Casey et al., 1984](#)). However, the depression-like symptoms associated with FTT probably reduce the infant's interest in feeding, rendering the infant harder to feed. Similarly, the significant malnutrition associated with FTT can produce a state of apathetic withdrawal. To complicate matters further, a small group of severely deprived and abused young children have a disruption of pituitary function even in the presence of adequate nutrition. Although these children represent a clearly defined and distinct diagnostic syndrome of PSD ( [Powell et al., 1967](#)), their condition is persistently confused with FTT.

Failure to thrive is a disorder with an onset in the first 3 years of life. Typically, infants who have onset of FTT before the end of 1 year of life are more likely to have been actively deprived of food or to have primary physiologic disorders that interfere with caloric intake. When the initial onset of FTT occurs in older infants and toddlers, there is more likely to be active interactional difficulties between the child and primary caregiver, which manifest as an eating disorder. Frequently, a young infant who presents with FTT responds rapidly to adequate feeding. However, the same social/familial conditions that are associated with such acute malnutrition also may be associated with chronic emotional and physical deprivation and poor infant-caregiver relationships. Therefore, in infants whose initial episode of FTT is rapidly ameliorated by refeeding, a second episode of FTT may develop that is characterized by a more chronic and internalized eating disorder. These toddlers and young children resist ingestion of adequate caloric intake and show secondary stunting of linear growth and head circumference as a result of chronic malnutrition.

The developmental outcome of children with FTT is remarkably heterogeneous, perhaps because of the heterogeneity of the syndrome. Significant variables that influence outcome include general factors such as socioeconomic status, maternal education, parental mental illness, and family social functioning. Because these factors are risk factors for both FTT and poor developmental outcome, they probably mediate their influence somewhat independently of FTT *per se*. Risk factors that are more directly linked to FTT include degree and chronicity of malnutrition, degree and chronicity of developmental delay, severity and duration of the dysfunction in infant-caretaker relationship, and severity of major medical disorder. Problems associated with FTT such as physical abuse, medical neglect, educational neglect, and social isolation interact with the general and specific risk factors to influence developmental outcome.

As indicated in the introduction, FTT is a common disorder, with an incidence ranging from 1% to 3% of inpatient pediatric admissions to 10% to 15% of some outpatient populations. Few data exist on historical trends for shifting prevalence of FTT. Sex ratio is reported to be approximately equal, although studies of older infants indicate a slight preponderance of boys.

Although FTT occurs in children of all social strata, it is more common in families where functioning is compromised by poverty, unemployment, social dislocation and isolation, and parental mental illness ( [Kessler and Dawson, 1999](#); [Fischaff et al., 1971](#)). As with any complex, multifactorial disorder, most infants develop relatively normally in conditions characterized by many of these risk factors, whereas a minority develop an eating/growth disorder such as FTT. On the other hand, FTT develops in some infants despite the apparent absence of any of these risk factors. Thus, social and family risk factors are just that: risk factors rather than specific etiologic agents.

Virtually nothing is known about what, if any, genetic factors exist in various forms of FTT. Obviously, there are important genetic contributions to various mental disorders that can influence caretaker adequacy.

Because the etiology of FTT is a multifactorial process, virtually any temperament or psychological factor that interferes with somatic homeostasis or attunement between infant and caretaker may well disrupt feeding and cause inadequate caloric intake ( [Wilson et al., 1986](#)). In young infants, the two most common such difficulties are marked irritability and apathy. Both of these states are themselves the result of multiple underlying factors, including basic psychophysiologic temperament, nutritional state, physical health, and affective interaction with the primary caregiver. This last factor has been the focus of considerable research as the major etiologic factor in the development of FTT. Investigators have postulated that any emotional or behavioral disturbance in the primary caretaker that causes her or him to be physically or emotionally absent will result in the state of apathetic depression in the infant. Alternatively, emotional disturbance in the caregiver that results in the infant's experiencing constant anger, rejection, irritability, or hatred will cause the infant to be irritable, difficult to soothe, or withdrawn.

### Laboratory Studies

Clinicians in the past sometimes have overemphasized the importance of the search for occult organic factors, as has been demonstrated by the low yield from exhaustive testing ( [Berwick et al., 1982](#); [Sills, 1978](#)). A major impetus for this excessive use of laboratory testing has been the misconception of the organic/nonorganic dichotomy. To be sure, the number of primary medical illnesses that can be associated with FTT is extensive. However, a thorough pediatric history, physical examination, and minimal screening laboratory tests usually identify physical illnesses that are contributing to FTT. These screening tests include complete blood count, lead level and free erythrocyte protoporphyrin, tuberculosis skin test (purified protein derivative), urinalysis and urine culture, and a sweat test in populations predisposed to cystic fibrosis ( [Frank and Zeisel, 1988](#)).

Some organic factors may be caused by the malnutrition associated with FTT as well as aggravating its course. Malnutrition suppresses immune functions so that children with FTT are at risk for chronic respiratory and gastrointestinal infections. The illnesses that these infections produce interfere with the infant's ability to ingest adequate calories, which exacerbates the infant's state of malnutrition. Similarly, infants with FTT are at risk for vitamin and mineral deficiencies, especially of calcium, iron, and zinc. These deficiencies result in blood dyscrasias and metabolic disturbances, which worsen the infant's clinical state. In addition, the mineral deficiencies and malnutrition increase the risk of lead toxicity because lead absorption is significantly increased in these deficiency states. Perhaps most important in evaluation of organic factors is the concept that illnesses that coexist with FTT belong in a continuum, from those that actually cause malnutrition (e.g., malabsorption) at one end to those that are caused by malnutrition (e.g., immunosuppression) ( [Woolston, 1985](#)) at the other.

### Treatment

As with other eating disorders that have associated malnutrition, ensuring adequate caloric intake is the most important intervention. Obviously, the clinician must identify the major reason for such inadequate caloric intake and remediate it. For infants who primarily are severely neglected, this simply means providing enough appropriate food. For older infants, toddlers, and children who have developed a more active pattern of food refusal, a more careful assessment of the triggers for food refusal must be performed to guide the intervention.

### Natural History and Outcome

The crucial axes on which to evaluate an infant with FTT include age of onset and duration of FTT, degree of malnutrition, degree of linear growth and head circumference stunting, presence of other physical illness, developmental delay, and level of family functioning.

Age of onset is important both in understanding the genesis of the FTT as well as in its prognosis. FTT with onset in the first 12 months of life in the absence of any concurrent medical illness is almost always a feeding disturbance resulting from either the infant's being deprived of adequate caloric intake or being so fussy and irritable that feeding is nearly impossible. In the former situation, the infant rapidly gains weight when offered enough food. Thus, if the factors that contributed to the inadequate caloric intake can be ameliorated, infants with this pattern of FTT have an excellent prognosis. Similarly, most infants who have severe problems with establishment of basic physiologic homeostasis develop more effective state regulation by the second and third year of life. However, the presence of other risk factors on the multiaxial approach to FTT puts such infants at high risk for poor developmental outcome. Some authors have been very specific in the linkage between developmental stage and etiology. For example, [Chatoor and colleagues \(1985\)](#) have proposed three distinct forms of FTT based on developmental stage of onset: (a) feeding disorder of homeostasis, in the newborn period; (b) feeding disorder of attachment, between 2 and 8 months of age, usually associated with maternal deprivation; and (c) feeding disorder of separation, between 6 months and 3 years of age, during the transition to self-feeding.

Calculation of degree of malnutrition is crucial in all aspects of the clinical management of FTT, including diagnosis, prognosis, and treatment. Frequently, the severity of the malnutrition is associated with the severity of the other risk factors that are influencing FTT. The degree of malnutrition affects the severity of medical complications associated with FTT, ranging from acute immunosuppression to permanent interference with brain growth. Calculation of degree of malnutrition is essential for the correct nutritional treatment required for compensatory catch-up growth.

The degree of height and head circumference growth stunting provides important data about the chronicity and severity of malnutrition ( [McLaren and Reed, 1972](#)). Such stunting usually is associated with a poorer growth prognosis because such growth arrest is both difficult to overcome, as well as strongly associated with

long-standing psychosocial deprivation. Stunting of head circumference growth is especially ominous because it reflects structural alterations in brain size. If nutritional interventions are delayed or inadequate, deficits in head circumference may be lifelong, even if weight and length deficits may be largely restored.

An important caveat in the calculation of growth stunting is the consideration of intrauterine growth retardation (IUGR) and prematurity. IUGR is defined as both weight and height less than the 10th percentile for gestational age (Frank and Zeisel, 1981). The growth prognosis for children with a history of IUGR varies with the nature of the prenatal insult. Infants with IUGR who are underweight compared with their length or head circumference have the best prognosis for later growth. Presumably such infants have been poorly nourished during the end of gestation and can regain adequate weight for height by appropriate caloric intake. In contrast, infants with IUGR whose weight and length are equally delayed frequently remain small despite intervention. These infants often have suffered a variety of systemic intrauterine pathologic events, including exposure to teratogens, infections, and chromosomal abnormalities ( [Frank and Zeisel, 1988](#)).

Infants who were born prematurely may appear to be growth delayed simply because they are being evaluated by chronologic age of birth rather than by gestational age. The age used to evaluate height and weight should be calculated by subtracting the number of weeks since birth ( [Frank and Zeisel, 1988](#)). Such corrections should be made for head circumference until 18 months after birth, for weight until 24 months, and for height until 40 months. Obviously, FTT may develop in children with IUGR and prematurity in addition to their preexisting conditions. In fact, because both conditions commonly are associated with such risk factors for FTT as maternal substance and alcohol abuse, family dysfunction, and maternal mental illness or poor competence, infants with prematurity or IUGR are at higher risk for development of FTT than are full-term, normally developed neonates. Thus, although the diagnosis of either of these conditions by no means precludes the diagnosis of FTT, prematurity and IUGR must be taken into account when assessing the infant's degree of growth delay.

The presence of other medical disorders associated with FTT is a complex phenomenon that covers the continuum from illnesses like malabsorption, which directly cause malnutrition, to recurring infections secondary to malnutrition-induced immunosuppression ( [Frank and Zeisel, 1988](#)). At one time, researchers and clinicians alike attempted rigidly to dichotomize children who were "only" malnourished (so-called nonorganic FTT) and children who had a presumably etiologic medical illness (organic FTT). This rigid dichotomy served to obscure both evaluation and treatment ( [Frank and Zeisel, 1988](#)). Rather than focusing only on a disorder that causes the malnutrition, the clinician must evaluate a child with FTT as being at high risk for chronic infectious illnesses, elevated blood levels of heavy metals, and mineral deficiencies.

As with the other factors in the multiaxial approach to diagnosis and assessment of FTT, developmental delay is an important, multiply determined, and transactional risk factor. Both malnutrition and emotional deprivation are independently associated with developmental delay ( [Frank and Zeisel, 1988](#)). Obviously, when they are combined, they are potent risk factors. In addition, developmental delays themselves may contribute to the infant's feeding problems.

Level of family functioning is a crucial variable in assessment and treatment of FTT. The clinician must come to understand which of a myriad of family dysfunctions is contributing to the infant's failure to ingest adequate caloric intake. Earlier researchers had wondered about a specific type of family and especially maternal problem. No such specific dysfunctions have been found for children with FTT as a group. However, many reports have described specific family-related problems that appear to be directly related to specific infants with FTT. Thus, the absence of a prototypical family dysfunction that is related to most cases of FTT should not discourage the clinician from the search for sources of family problems for a specific child with FTT. Such an exploration is crucial because the ongoing nutritional supplementation and emotional stimulation must be done in the context of the child's family. Frequently, the family problems that contributed to the infant's FTT serve as roadblocks to effective treatment.

## FEEDING DISORDER OF INFANCY OR EARLY CHILDHOOD

### Definition

The fourth edition of DSM addressed the complex and controversial issues associated with FTT syndrome by creating a new diagnostic category, feeding disorder in infancy or early childhood. DSM-IV defined this disorder as (a) a feeding disturbance as manifested by persistent failure to eat adequately with a significant failure to gain weight or significant loss of weight over at least 1 month; (b) not due to an associated gastrointestinal or other general medical condition (e.g., esophageal reflux); (c) not better accounted for by another mental disorder or lack of available food; and (d) having onset before age 6 years. Although this new diagnostic category has provided a rational basis for the development of a nosology for eating and growth disorders in early childhood, there are a number of problems with the current definition. First, DSM-IV did not quantify "significant" failure to gain weight. It is hoped that future iterations of DSM will provide some quantification of this in terms of percentage of ideal body weight or shift in percentile on weight and length growth curves. Second, DSM-IV ignored the proven lack of validity of the organic/nonorganic dichotomy implied by the criteria of the absence of a general medical condition ( [Bell and Woolston, 1985](#); [Polan et al., 1991a, b](#)). Perhaps most important, this new diagnostic category makes no provision for subtyping. The likelihood that a single general diagnosis will cover two developmental epochs is very small. As described in the section on FTT, [Chatoor and colleagues \(1998a, b\)](#) have proposed three distinct feeding disorders of infancy and early childhood that are well defined and have preliminary empirical support as valid and separate disorders within the broad diagnostic category.

## FUNCTIONAL DYSPHAGIA

### Definition

Functional dysphagia is an eating disorder characterized by a subjective experience of difficulty or discomfort associated with the act of swallowing that is not primarily due to an organic medical condition.

### Clinical Description

The two most commonly described variations are conversion dysphagia and posttraumatic dysphagia. ( [Culbert et al., 1996](#)). Functional dysphagia and its variants have a variety of alternate names, including *posttraumatic feeding disorder* ( [Chatoor et al., 1988](#)); *food aversion* ( [Siegel, 1982](#)); *food phobia* ( [Singer et al., 1992](#)); *feeding resistance* ( [Geerstma et al., 1985](#)); *food refusal* ( [Linscheid et al., 1987](#); [Ramsay and Zelazo, 1988](#)); *phagophobia* ( [Culbert et al., 1996](#)); and *globus hystericus* ( [Koon, 1983](#)). Although functional dysphagia can occur throughout the life cycle, its onset is most commonly in preschool- and school-aged children ( [Culbert et al., 1996](#)). Problems associated with functional dysphagia include total or partial food refusal, food selectivity, adipsia or polydipsia, rumination or vomiting, failure to gain weight or weight loss, and dehydration. Functional dysphagia frequently has a variety of initiators and maintainers. These include organic factors such as esophageal irritation or hyperactive gag reflex; psychosocial factors such as abuse related to feeding, dysfunctional family meal time culture, and social reinforcement; and emotional and behavioral factors such as actual or vicarious choking event, anxious/intense temperament, anxiety disorders, and distorted body image ( [Culbert et al., 1996](#)). Developmental outcome of functional dysphagia is completely unreported in the literature.

### Treatment

Treatment of functional dysphagia has included cognitive-behavioral therapy ( [Koon, 1983](#)), hypnotherapeutic therapy ( [Culbert et al., 1996](#)) as well a multimodal approach that integrates cognitive-behavioral, individual, family, and pharmacologic therapies ( [Atkins et al., 1994](#)).

## PSYCHOSOCIAL DWARFISM

### Definition

Psychosocial dwarfism (PSD), also called *derivational dwarfism* or *hyperphagic short stature*, is a syndrome of deceleration of linear growth combined with characteristic behavior disturbances (sleep disorder and bizarre eating habits), both of which are reversible by a change in the psychosocial environment ( [Gilmore and Skuse, 1999](#); [Green et al., 1984](#); [Powell et al., 1967](#); [Skuse et al., 1996](#)).

### Prevalence and Epidemiology

PSD apparently is a rare disorder, so virtually, all of the available literature consists of case reports or small case series. Incidence, prevalence, sex ratio, and sociodemographic features are unknown.



## Clinical Description

The deceleration of linear growth is remarkable in that it occurs in the absence of weight gain deceleration. In this way, PSD is quite distinct from growth stunting secondary to malnutrition (FTT), but rather resembles primary hypopituitarism (Blizzard, 1973; Powell et al., 1967). The most common, characteristic abnormal eating behaviors include polyphagia, gorging, vomiting, stealing and hoarding food, and eating from garbage pails and animal food dishes. Other behaviors reported include polydipsia, including drinking stagnant water, toilet bowl water, and dishwater. Various sleep disorders have been described, such as initial-onset insomnia and night wandering. These sleep disturbances may be related to growth delay because they interfere with the major nocturnal pulsatile release of growth hormone. Children with PSD may exhibit a variety of unusual patterns of relatedness and problems with behavior, including aggressiveness and impulsivity. Both specific language delays and delays in general intellectual development have been reported (Drash et al., 1986).

The age of onset is unclear but reportedly occurs between 18 and 48 months. Blizzard (1973) argued that the onset must occur after 24 months to distinguish it reliably from FTT.

## Etiology and Pathogenesis

The literature is virtually unanimous in describing parental psychopathology that results in maltreatment of the child (e.g., Powell et al., 1967), including overt abuse or neglect (Drash et al., 1968). Several reports have indicated that both the parental psychopathology and maltreatment of the child may be obscured in initial interviews. The parents have been reported to withhold clinical information and be uncooperative with treatment. The frequent association of parental psychopathology and the reversal of the associated features with change in environment appear to be quite convincing. However, because no studies have been reported that control for such basic variables as socioeconomic class, the validity of these findings should be viewed as preliminary.

## Laboratory Studies

Much of the research on PSD has focused on neuroendocrine abnormalities found in the syndrome in an attempt to unravel the relationship between growth rate and neuroendocrine changes. Unfortunately, these investigations have led to the discovery of no pathognomonic or consistently abnormal findings, with the possible exception of depressed somatomedin levels (Green et al., 1984). The abnormalities in hormonal levels and hypothalamic functioning have been found to normalize partially or completely after the subject's removal from the inimical environment. This normalization may occur in several weeks or require as long as 2 years, depending on the specific endocrine disturbance and the type of specific medical, hormonal, or psychiatric treatment (Green et al., 1984). In addition to this great variability in the normalization of endocrine abnormalities, none of the abnormal hormonal findings correlates specifically with growth failure. Growth failure has occurred with normal endocrine values, and catch-up growth has occurred with subnormal values (Green et al., 1984). The mechanisms causing the growth failure in PSD therefore are as yet unknown.

## Natural History and Outcome

Developmental outcome appears to be highly variable, although limited outcome data are available. Outcome appears to be highly contingent on the adequacy of the child's psychosocial environment. Hospitalization or removal to a less noxious home environment is presumed to be ameliorative because it is associated with reversal of some of the neuroendocrine, growth, and behavioral concomitants of the disorder (Powell et al., 1967). If the child is returned to the noxious home environment, the various gains that have been achieved may be arrested or reversed. Behavioral disturbance, developmental delays, and short stature are possible long-term sequelae. In addition, delayed puberty may occur (Howse et al., 1977).

The explanation for the rarity of PSD, even among children exposed to severe maltreatment, is a mystery. Possible factors may include genetic vulnerability to a disruption in the rate or rhythm of growth hormone production or some specific type of psychosocial deprivation that causes such neuroendocrine dysfunction. However, the unraveling of these mysteries awaits better designed studies that have larger sample sizes and matched control subjects.

## Treatment

The treatment and evaluation of PSD must include the three cardinal factors associated with the disorder: a reversible neuroendocrine and growth dysfunction, behavioral disturbances and developmental delays, and presumably a noxious psychosocial environment. The evaluation of the neuroendocrine and growth dysfunction is important in the differential diagnosis of other disorders that are associated with short stature as well as in monitoring the response to treatment. These other disorders involving short stature include primordial dwarfism, IUGR with persistent small size, hypopituitarism from a variety of etiologies, constitutional delayed growth, Turner's syndrome (XO chromosomal pattern), osteochondrodystrophies, and growth stunting secondary to chronic malnutrition with or without chronic disease. PSD is virtually unique in being reversible with a change in living situations and in the absence of any signs of physical illness except growth delay.

The behavioral disturbances and developmental delays found in PSD serve as important factors to help monitor response to treatment as well as to represent serious aspects of the disorder that must be addressed. A careful psychiatric evaluation of the child is required that assesses for the presence of bizarre and disruptive behaviors, as well as for problems in normal social relatedness. A developmental assessment must evaluate for both specific delays, especially in language, and general developmental delays. In older children, psychoeducational testing is helpful to assess specific intellectual, academic, and adaptive functioning. Psychotherapeutic and educational interventions should be guided by these findings.

The evaluation and intervention to ameliorate the psychosocial adversity is both the most important as well as most difficult task. According to all reports, caretakers of a child with PSD may be actively uncooperative in the assessment and treatment phases. Ideally, the caretakers can be engaged in the appropriate individual and family psychotherapies. In addition, they can use various social supports such as home aides and support groups. In the event of active treatment refusal, which may result in permanent damage to the child, the treaters must consider removal of the child from the home.

## CHILDHOOD OBESITY

### Definition

Obesity is defined as a state of excess adiposity, so that the body weight is greater than 120% of expected for age and height.

### Historical Note

Considerable attention in the literature has been focused on the eating disorders of early childhood that result in growth failure, such as FTT, PSD, and rumination. In contrast, the eating disorders that result in excessive weight gain have been virtually ignored. DSM-IV has reinforced this prejudice by refusing to classify any form of obesity as an eating disorder (American Psychiatric Association, 1994). Instead, it relegates obesity to Axis III as a physical disorder. In conjunction with this lack of interest in obesity in the psychiatric literature, there are many widely held misconceptions about its etiology, course, and even heterogeneity of subtypes. This state of clinical indifference about the fundamentals of obesity in general, and childhood obesity in particular, makes a scientific strategy for intervention difficult.

### Prevalence and Epidemiology

The incidence and prevalence of obesity in childhood is not nearly as well studied as obesity in adulthood. The few studies indicate that the prevalence rate of obesity is 5% to 10% of preschool-age children (Maloney and Klykylo, 1983). Occasionally, "epidemics" of infantile obesity have been reported with prevalence rates of 16.7% of infants younger than 12 months of age (Shukla et al., 1972). These epidemics appear to be caused by culturally determined misinformation or fads about infant feeding practices (Shukla et al., 1972).

Female obesity is nine times more common in working class families than in middle and upper social class families (Stunkard et al., 1972). The prevalence of obesity is linked to the socioeconomic status of the parents almost as strongly as it is to the subject's own social class (Goldblatt et al., 1965). This finding argues that socioeconomic status is linked to obesity in a causal rather than a simple associative manner, perhaps mediated through culturally determined eating habits and dietary misconceptions. A family-line analysis of obesity indicates that there is a strong correlation between the fatness of parents and their children. For example, by

17 years of age, the children of obese parents have three times the chance of being obese as the children of lean parents. If one sibling is fat, there is a 40% chance that a second sibling will be fat ([Garn and Clark, 1976](#)). If two siblings are fat, there is an 80% chance that the third sibling will be fat. Although these data seem to support a genetic basis for obesity, other nongenetic, but family-related, factors must be kept in mind. The same study that reported the sibling data indicates that if one spouse is fat, there is a 30% chance that the other spouse also will be fat ([Garn and Clark, 1976](#)). Obviously, this finding cannot be explained by genetic factors. Indeed, Birch and colleagues (e.g., [Birch and Fisher, 1998](#); [Johnson and Birch, 1994](#)) have demonstrated a significant psychosocially transmitted influence from parents to their children related to eating behaviors and obesity. However, well designed genetic studies involving monozygotic/dizygotic concordance ([Stunkard et al., 1986a](#)) and adoption samples ([Stunkard et al., 1986b](#)) support a strong genetic contribution to all forms of body habitus ranging from fatness to thinness.

Studies of overt psychopathology in obese adults have been as contradictory as studies of other aspects of obesity. [Silverstone \(1969\)](#) attempted to reconcile these discrepant reports by differentiating between late-onset adult obesity secondary to a gradual accumulation of fat and early-onset adult obesity characterized by a sudden increase in fatness that was the result of anxiety-driven overeating. Indeed, psychogenic obesity of infancy and early childhood has been reported to occur only in the context of a disorganized family in which the child's needs are poorly perceived and even more poorly differentiated ([Kahn, 1973](#); [Woolston, 1987](#); [Woolston and Forsyth, 1989](#)).

### Clinical Description

The first step in the elucidation of any new field of study is an operational definition that is phenomenologically reliable and valid. In the study of obesity, there needs to be an easy, accurate, reliable method of defining the clinical condition. Because obesity denotes being excessively fat, the operational definition must differentiate the condition of having excessive adipose tissue for chronologic age from simply being heavy for chronologic age as a result of large muscle mass or skeletal frame. Triceps skinfold thickness ([Garn and Clark, 1976](#)) and an obesity index using weight gain, suprailiac skin fold, and waist circumference ([Crawford et al., 1974](#)) are two well standardized measurements that appear to satisfy the requirements for a useful, operational definition of obesity. A simpler, if slightly less valid, measure of obesity is defined by exceeding 120% of ideal body weight for height for a given age and sex. Specifically, ideal body weight is calculated by dividing actual weight by the expected weight for a given age and sex and height percentile.

One of the most obvious explanations for the contradictory results of various studies in regard to developmental course and etiology is that obesity in childhood is a heterogeneous syndrome. Many authors (e.g., [Maloney and Klyklo, 1983](#); [Stunkard, 1980](#)) have identified multiple factors that contribute to the development of obesity, including emotional, socioeconomic, genetic, developmental, and neurologic.

Although preliminary attempts have been made to subdivide forms of juvenile-onset obesity into phenomenologically homogeneous groupings ([Woolston, 1988](#); [Woolston and Forsyth, 1989](#)), such attempts probably are premature. Many of the same issues related to diagnostic classification of FTT apply to obesity. Fundamentally, all forms of obesity have the same organic basis: caloric intake that exceeds caloric expenditure. Like FTT, there are a multitude of primary medical illnesses that are associated with obesity. However, because it is the excess caloric intake that actually mediates the obesity, there frequently are psychosocial/emotional factors that complicate all forms of obesity, including those associated with developmental syndromes. The database of obesity of early childhood is too small to permit construction of clinically useful categorization. Instead, like FTT, a multiaxial list of major risk factors is more helpful. These should include age of onset and duration, degree of obesity, family history of obesity, presence of a medical illness or syndrome associated with obesity, and type of family functioning.

In a similar fashion to FTT, obesity that is associated with an acute onset in the first 12 months has an excellent prognosis because the treatment is simply a matter of reducing the infant's caloric intake to the recommended level for height and age. However, this benign condition can become much more problematic if there are other risk factors such as severe family dysfunction, a strong family history for obesity, or a medical syndrome associated with obesity. The longer the obesity persists beyond infancy and the later is its onset in childhood, the more persistent it is likely to be.

Obesity in infancy and childhood is a heterogeneous syndrome that has been relatively neglected in the eating disorder literature. The development of obesity is influenced by the interplay of social, familial, emotional, and physical factors. Analogous to FTT, obesity has been subtyped according to the presence or absence of physical disorders that are associated with obesity. Much less attention has been given to the different roles of genetics, cultural eating habits, and psychogenic factors.

### Natural History and Course

The study of the natural history of obesity in infancy and childhood is in its beginning stages. Data about the typical course of this disorder are contradictory. The most widely held belief is that obesity of early onset is a chronic and steadily progressive disorder with very few remissions ([Charney et al., 1976](#); [Eid, 1970](#)). However, more recent works ([Poskitt, 1980](#); [Shapiro et al., 1984](#)) have reported that obesity in infancy is a poorer predictor of later childhood obesity than was believed previously.

This rather poor correlation between obesity in infancy and obesity in later childhood calls into question the notion of relentlessly progressive obesity that is triggered by fat cell proliferation in infancy. Rather than there being a critical phase in infancy for fat cell proliferation, it is more likely that the degree and duration of obesity are the major determinants of total adipose cell number in humans.

### Laboratory Studies and Differential Diagnosis

In the differential diagnosis of infantile or childhood-onset obesity, clinicians frequently search for the discovery of discrete genetic, endocrinologic, or neurologic syndromes associated with obesity, including Prader-Willi, Klinefelter, Fröhlich, Laurence-Moon-Biedl, Kleine-Levin, and Mauriac syndromes.

Although these syndromes associated with obesity frequently are searched for as an etiology for obesity in childhood, they are quite rare. In addition, a clinical rule of thumb distinguishes between obesity associated with a developmental syndrome and that associated with otherwise normal development. Children with a developmental syndrome usually are below the 25th percentile in height and have delayed bone age, whereas children with normal development are above the 50th percentile in height and have an advanced bone age.

### Natural History and Outcome

The degree of obesity, expressed either as a percentile of ideal body weight for height and age or as a Z score, predicts prognosis in that the degree of obesity may reflect the seriousness of the underlying risk factors. More important, the degree of obesity helps guide the clinician regarding the likelihood of serious medical sequelae of obesity such as slipped femoral head epiphysis, sleep apnea, pickwickian syndrome, hypercholesterolemia, and diabetes mellitus. In addition, the degree of obesity must guide the nutritional intervention. Caloric restriction below the recommended amounts for height, age, and sex rarely is indicated. Instead, the primary goal is to arrest continued weight gain until the child's height and weight are proportional.

### Treatment

Preliminary data indicate that children with a positive family history for being overweight as their only risk factor may have a relatively good response to brief, family-based behavioral intervention. Using a prospective, randomized, controlled experimental design, [Epstein et al. \(1990\)](#) reported that an 8-week behavioral intervention program for both the obese child and his or her parents significantly reduced the obesity in the child at the end of the 10-year follow-up. Unfortunately, this study has not been replicated in the absence of other risk factors. Children with this pattern of obesity rarely become morbidly obese, and so rarely develop the major medical complications of severe obesity. As indicated earlier, children with obesity as part of a primary developmental syndrome usually are below the 25th percentile in height and frequently are cognitively impaired.

Level of family functioning may be crucial in both the genesis of obesity as well as its treatment. Severely dysfunctional families provide the child with an environment of emotional neglect, a sense of abandonment, and no limit setting around the use of food. A small number of children who grow up in such environments acquire a pattern of enormous caloric intake and a rapid onset of severe obesity, usually beginning between 3 and 5 years of age. These children are at high risk for poor developmental outcome because the constellation of risk factors conspire to produce a powerfully negative synergistic spiral. These children usually have low self-esteem, which is exacerbated by their obesity. They are likely to suffer the most serious medical complication of obesity. Most important, they are at high risk because the only effective intervention for treatment of obesity is mediated through the family system. This intervention includes the orchestration of appropriate



medical and mental health services, effective limit setting, and the development of more positive self-esteem and emotional security. Unfortunately, the very risk factor that contributed so heavily to the genesis of the disorder, family dysfunction, makes intervention difficult ( [Woolston and Forsyth, 1989](#)).

## **BENIGN NUTRITIONAL DWARFING**

### **Definition**

Benign nutritional dwarfing (BND) is a disorder of school-age children and young adolescents in which maladaptive eating patterns unaccompanied by psychiatric or medical disorders result in a marked slowing of weight gain for at least 1 year, followed by a deceleration of linear growth.

### **Prevalence and Epidemiology**

Because of the relatively recent reporting of BND, satisfactory data on its incidence and prevalence do not exist. In most reports of BND, there is a slight predominance of boys over girls.

### **Clinical Description**

Delayed puberty may be associated with this disorder. Because the subnormal weight gain in such children is accompanied by a proportionate decline in growth velocity, body weight-for-height deficits frequently are not evident. Therefore, children with BND do not present with emaciation, as is commonly observed in cases of severe malnutrition. Although dwarfing secondary to chronic malnutrition in childhood is as old as humankind, BND is a relatively newly described disorder ( [Apley et al., 1971](#); [Davis et al., 1978](#); [Lifshitz et al., 1971](#); [Pugliese et al., 1983](#); [Sanberg et al., 1991](#)). Although various authors report various different motivations for the inadequate caloric intake, all reports have been consistent in describing an absence of weight loss (as opposed to a slowing of weight gain) and an absence of body image distortion. One well controlled study found virtually no differences in various measures of psychological functioning between children with BND and matched children with constitutional growth delay or familial short stature ( [Sanberg et al., 1991](#)).

Typically, the age of onset of BND is middle childhood to early adolescence (8 to 14 years), although some authors have included children as young as 2 years ( [Davis et al., 1978](#)) and as old as 15 years of age ( [Lifshitz et al., 1971](#)). Although the consequences of chronic insufficient caloric intake can include delayed puberty and permanent short stature, children with BND frequently respond to relatively straightforward nutritional counseling with an increase in caloric intake sufficient to reinstate normal growth. There are no reports of associated development or psychiatric disorders either during the course of BND or as its sequelae.

### **Etiology and Pathogenesis**

Benign nutritional dwarfing is characterized by the absence of social or familial dysfunction that typifies other eating and growth disorders. There are no reports that indicate any particular parental psychopathology or genetic factors. Some reports have described special nutritional concerns ( [Pugliese et al., 1983](#)) and chronic nonspecific feeding disturbances ( [Davis et al., 1978](#)).

The only psychogenic factors reported to be related to BND are inaccurate nutritional beliefs about weight gain or special dietary needs.

As indicated earlier, inadequate caloric intake to maintain normal weight gain is the only known organic factor. Various mineral and vitamin deficiencies may complicate the inadequate caloric intake ( [Lifshitz et al., 1971](#)).

### **Differential Diagnosis and Treatment**

Benign nutritional dwarfing is characterized by chronic inadequate caloric intake as a result of mistaken nutritional beliefs. Although a variety of medical and psychiatric disorders may result in chronic malnutrition, children with BND have a normal physical and psychiatric examination except for a history of weight velocity deceleration, followed by growth velocity deceleration. A careful history of growth and of dietary beliefs and eating habits is crucial to establish the diagnosis and guide the intervention. The treatment of BND should focus on nutritional counseling of the child and parents. Follow-up visits should be scheduled to ascertain weight gain.

## **PREPUBERTAL ANOREXIA NERVOSA**

### **Definition**

Prepubertal anorexia nervosa has all of the essential features of typical adolescent-onset anorexia, including intense fear of becoming fat, disturbance of body image, significant and defined weight loss, and a refusal to maintain a minimum normal body weight. The only modifications of the standard diagnostic criteria are those required by the physical characteristics of prepubertal children. Obviously, because these children are prepubertal, amenorrhea is not a meaningful criterion.

### **Historical Note**

Although prepubertal anorexia nervosa was reported in the same year ( [Collins, 1894](#)) that adolescent-onset anorexia nervosa was first described, substantial case reports were not recorded until nearly 70 years later. Indeed, full recognition of the special characteristics of prepubertal anorexia nervosa awaited reports in the 1980s to establish this as a relatively rare but bona fide form of anorexia nervosa ( [Bostic et al., 1997](#); [Growers et al., 1991](#); [Irwin, 1984](#); [Jacobs and Isaacs, 1986](#); [Lask and Bryant-Waugh, 1992](#); [Russell, 1985](#)).

### **Prevalence and Epidemiology**

Although anorexia nervosa is a relatively rare disorder, prepubertal anorexia is considerably rarer. The sex ratio and sociodemographics appear to be similar to those in the more typical anorexia nervosa. Current published reports indicate that the same types of social and family factors are implicated in the genesis of prepubertal anorexia nervosa as with more typical anorexia nervosa. These factors include middle or upper middle class and an enmeshed, intrusive family communication style. Like social and family factors, genetic factors may play an important role in anorexia nervosa regardless of the age of onset ( [Garfinkel and Garner, 1982](#); [Morgan and Russell, 1975](#)).

### **Clinical Description**

Because prepubertal children have a smaller percentage of body fat than adolescents, a smaller percentage of weight loss has a greater physiologic impact. This relatively greater deleterious effect of weight loss sometimes is compounded by the fact that prepubertal children are more concrete in their thinking, so that they more frequently generalize their pathologic eating to include their fluid intake ( [Irwin, 1984](#)).

### **Etiology and Pathogenesis**

Although numerous theories exist about various psychogenic factors related to anorexia nervosa, all remain relatively speculative. Children with prepubertal-onset anorexia express fewer concerns about sexual maturity ( [Jacobs and Isaacs, 1986](#)) than those with later-onset anorexia. In addition, prepubertal children with anorexia nervosa have less cognitive ability to understand various concepts of nutritional counseling. Some of the emotional and social goals in the treatment of adolescent anorexia related to differentiation from the family may be inappropriate for a prepubertal child.

As with other forms of anorexia nervosa, there are no known organic factors related to etiology. Acute malnutrition has a variety of important consequences for neuroendocrine functioning and growth.

## Natural History

Because relatively few cases of prepubertal anorexia nervosa have been reported, conclusions about any aspect of natural history remain tentative. However, existing reports support the notion that prepubertal anorexia carries all of the possible negative sequelae of adolescent anorexia nervosa. In addition, the experience of an episode of acute malnutrition severe enough to cause significant weight loss in a prepubertal child will likely have a deleterious effect on growth ( [Russell, 1985](#)).

## Treatment

The prepubertal age of onset in this typically adolescent and adult disorder is the main stumbling block to accurate diagnosis and treatment. In general, clinicians who typically treat anorexia nervosa have little knowledge about the special cognitive, emotional, or physical needs of prepubertal children. Analogously, clinicians who treat prepubertal children have little experience in the diagnosis or treatment of anorexia nervosa. Once these obstacles have been overcome, the diagnosis and treatment are at once as straightforward and as difficult as in more typical anorexia.

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### CASE ILLUSTRATION CHRONIC FAILURE TO THRIVE WITH SECONDARY DWARFING

George M. was a 5-year, 8-month-old child who was admitted to a children's psychiatric inpatient service because of his protective service case worker's "concern about his not picking up weight" and because of evidence of depression as indicated by statements such as "I want to be nothing. If I don't eat, I will be nothing."

George had been diagnosed as having failure to thrive (FTT) at age 3 years, 2 months when he was admitted to a pediatric unit of a community hospital for evaluation of his poor weight gain and linear growth. The workup for an organic etiology of his FTT was negative except that he was noted to consume inadequate calories. His mother was charged with medical neglect, and he was committed to the state, which placed him in a series of foster homes with continued visitation with his mother. Finally, at age 4 years, he was placed in a permanent foster home, which allowed ongoing visitation with his mother. His growth failure continued, so that he was evaluated on an outpatient basis at a children's hospital. Again, the pediatric workup was negative for an organic etiology except for his inadequate caloric intake. The evaluators were concerned by the unresponsive and apathetic styles of interaction with his biological mother and recommended termination of parental rights. However, George's biological mother appeared to improve her parenting style after she was informed of the intention to terminate her parental rights. Because of his apparent improvement, the protective service agency dropped the termination petition.

In the several months before admission, the protective service agency had attempted to reunite George with both his biological mother and father. However, this proved difficult, especially because George's parents had separated as a result of his father's alcohol abuse and violent temper. George was observed to have a picky and poor appetite in general but especially during and after visits with his parents. At these times, he ate very little and vomited what he did eat.

On admission, George presented as a small, thin, 5-year, 8-month-old boy with a flat affect and a depressed mood. He engaged easily with the interviewer and occasionally his affect brightened somewhat. He was unable to describe any wishes because he felt wishes were pointless and they would never come true. He had a realistic assessment of his body image, stating he was too skinny and needed to gain weight. George was quite articulate about his concerns about his future placement because he knew that his mother and his foster parents were vying for his custody.

George's developmental history was characterized by a full-term gestation, normal vaginal delivery, and normal early developmental milestones. By age 4 years, he was noted to have mild delays in language and in fine, gross, and visual motor skills.

His admission physical examination was remarkable only for his small stature, with a weight of 13.7 kg and height of 102.9 cm. These measurements represent a height age of 3 years, 2 months, a weight age of 2 years, 8 months, and a percentage of ideal body weight of 85%. His admission laboratory evaluations, including complete blood count, sedimentation rate, electrolytes, thyroid functions, urinalysis, somatomedin-C, head computed tomography scan, and bone age, were all within normal limits.

During the course of his 3-month hospitalization, George went through three phases in his process of developing greater trust and reliance in close attachments: first was avoidance and rejection of caregivers, second was testing the stability of these relationships, and third was his realization that he was indeed cared for. Despite ongoing visitation by George's biological mother, she continued her highly ambivalent and highly volatile relationship with George. Although she was sometimes warm and affectionate, at other times she was harsh and overtly rejecting. She persistently maintained that, "George needed to learn that you can't always be the center of attention." She interpreted his slow and picky eating as a personal attack on her parenting skills. Her overall personality structure was so fragile that the stress of a difficult visit with George and her taxi not arriving led her to experience a transient episode of a gross thought process disorder.

The treatment team and protective service agency decided that George could not return to live with his mother. Before discharge, a legal risk adoptive mother was identified and introduced to George. By the time of discharge, he had gained only 1 kg but had progressed considerably in his self-esteem and ability to verbalize his feelings. For the next 1½ years, George continued to improve emotionally and developmentally but remained a picky eater. However, within several months of termination of his mother's parental rights and finalization of his adoption, he began to eat with gusto. His rate of weight gain increased, so that he moved from below the 5th percentile to above the 25th. Several months later, his linear growth followed suit.

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# 55 ANOREXIA NERVOSA AND BULIMIA NERVOSA

Katherine A. Halmi, M.D.

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## DEFINITION

Anorexia nervosa and bulimia nervosa are the two major eating disorders. They are complex syndromes with considerable psychiatric and medical comorbidities. Current diagnostic criteria for anorexia nervosa and bulimia nervosa from the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) (American Psychiatric Association, 1994) are shown in [Table 55.1](#) and [Table 55.2](#). For those patients who have serious problems with eating behavior but who do not fall into the diagnostic categories of anorexia nervosa or bulimia nervosa, the DSM-IV has designated a category of Eating Disorder Not Otherwise Specified (EDNOS). Most cases of eating disorders have an onset during adolescence.

- A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).
- B. Intense fear of gaining weight or becoming fat, even though underweight.
- C. Disturbance in the way in which one's body weight or shape is experienced; undue influence of body weight or shape on self-evaluation; or denial of the seriousness of the current low body weight.
- D. In postmenarcheal females, amenorrhea (i.e., the absence of at least three consecutive menstrual cycles).

From American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. Washington, DC: American Psychiatric Association, 1994.

**Table 55.1. Diagnostic Criteria for Anorexia Nervosa**

- A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
  2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).
- B. Recurrent inappropriate compensatory behavior to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise.
- C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months.
- D. Self-evaluation is unduly influenced by body shape and weight.
- E. The disturbance does not occur exclusively during episodes of anorexia nervosa.

From American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. Washington, DC: American Psychiatric Association, 1994.

**Table 55.2. Diagnostic Criteria for Bulimia Nervosa**

There are four major criteria that define anorexia nervosa. The first criterion is a guideline for defining weight loss because there is no specific amount of weight loss associated with the other symptoms that constitute anorexia nervosa. Therefore, an adult is considered "underweight" if he or she weighs less than 85% of a weight that is considered normal for that person's age and height. For children up to the age of 18 years, pediatric growth charts should be used. Some children may not have weight loss but still weigh less than the expected weight because they have failed to make weight gains during a growth in height. The second criterion, intense fear of gaining weight, is present even during emaciated states. Anorectic patients often deny this fear because they are resistant to treatment, and hence, their fear of gaining weight often must be inferred from reports of their behavior that reveal rigorous attempts to prevent weight gain, such as severe food restriction and exercising. The third criterion, pertaining to body image disturbance, has evolved into a more complex concept. The significance of body weight and shape are greatly distorted in these individuals. Some feel globally overweight, and others realize they are thin but feel certain parts of their body, especially the abdomen and thighs, are too fat. The distorted significance of body weight and shape is related to a feeling of being very ineffective. Losing weight and being thin is one area in which these individuals can be effective and in control. The latter undoubtedly influences their denial of the serious medical complications of their malnourished state.

The fourth criterion for diagnosis of anorexia nervosa in the DSM-IV is amenorrhea. In some adolescents who have never menstruated, the amenorrhea is primary and menarche is delayed by the anorexia nervosa. Amenorrhea can appear before noticeable weight loss has occurred ([Halmi and Falk, 1981](#)). Because it is difficult to obtain an accurate history of menses and because of the great variation associated with weight loss in menses, some academicians are advocating abolishing this criterion.

There are two subtypes of anorexia nervosa: the restricting type and the binge-purge type. Studies have consistently demonstrated that impulsive behaviors, including stealing, drug abuse, suicide attempts, self-mutilations, and promiscuity, are more prevalent in anorectic-bulimics compared with anorectic restrictors. The anorectic-binge-purge type also has a higher prevalence of premorbid obesity, familial obesity, debilitating personality traits, and specific medical complications compared with the anorectic restrictors ([Eckert et al., 1987](#); [Halmi and Falk, 1982](#); [Strober et al., 1982](#)).

The criteria for bulimia nervosa are more arbitrary and less specific than the criteria for anorexia nervosa. There is no consensus on what constitutes a binge and how frequently bingeing must occur to warrant a diagnosis of this disorder. In the first criterion for bulimia nervosa in DSM-IV, binge eating is defined as eating more food than most people eat in similar circumstances and in a similar period. The sense of losing control is a significant subjective aspect that needs to be present. The second criterion, which is the recurrent use of inappropriate compensatory behaviors to avoid weight gain, usually means self-induced vomiting. However, bulimic patients often use cathartics for weight control and have an eating pattern of alternate binges and long fasting periods. The third criterion designed to address

chronicity and frequency is not based on specific research but rather clinicians' consensus about obvious impairment of function. The fourth criterion acknowledges that patients with bulimia nervosa also are concerned about their body shape and weight and tend to place excessive estimation of their worth in terms of appearance. The fifth criterion for bulimia nervosa differentiates the latter from the binge-purge subtype of anorexia nervosa. The diagnosis of bulimia nervosa also is subtyped into a purging type for those who regularly engage in self-induced vomiting or use of laxatives or diuretics and a nonpurging type for those who use strict dieting, fasting, or rigorous exercise but do not engage in purging behaviors. Patients with bulimia nervosa who do not purge tend to have less body image disturbance and less anxiety concerning eating compared with those who purge ([Davis et al., 1997](#)).

Binge eating disorder is listed in the EDNOS category of the DSM-IV for eating disorders. People with this disorder lack the compensatory weight control behaviors and the exaggerated concern with weight and shape ([Devlin et al., 1992](#)). Field trials are being conducted to provide evidence as to whether binge eating disorder should be a specific diagnostic category.

An EDNOS diagnosis also is given to persons who vomit after eating small amounts of food but who maintain a weight within a normal range and menstruate.

## HISTORICAL CONTEXT

Disturbances of eating behavior are described in the Middle Ages. Well documented case reports of anorexia nervosa are found in the literature describing early Christian saints. The severe starving behavior and bingeing episodes of Saint Catherine of Sienna are recorded in monastery documents, along with the kind of reed she used to induce vomiting and the herbal cathartics she used for purging ([Bell, 1985](#)). Another example of this irreversible self-starvation in a fasting female saint is Princess Margaret of Hungary, who lived from 1240 to 1271 ([Halmi, 1994](#)). She was the daughter of a king and was raised in a Dominican convent, where she excelled in all of her studies and in the undesirable chores of the monastery. It is likely that the biological vulnerability factors are similar in those dieting for sainthood during the Middle Ages and those dieting for thinness (attractiveness) in the 20th century. In the 17th century, both [John Reynolds \(1669\)](#) and [Richard Morton \(1689\)](#) described cases of typical anorexia nervosa symptomatology and distinguished them from consumption. In the 19th century, [Marcé \(1860\)](#), [Gull \(1888\)](#), and [Laséque \(1873\)](#) described additional cases of anorexia nervosa and recommended treatment. In the 20th century, the first major publication on anorexia nervosa was a book by [Bliss and Branch \(1960\)](#) that presented endocrine studies as well as psychological descriptions of the disorder. A decade later, [Hilda Bruch \(1973\)](#) further articulated the psychology of anorexia nervosa in her phrases "the relentless pursuit of thinness" and "the paralyzing sense of ineffectiveness, which pervades all thinking and activities." In 1979, Russell identified bulimia nervosa as a separate entity from anorexia nervosa. Subsequently it became apparent that there were young women who had the full syndrome of bulimia nervosa without a history of anorexia nervosa.

## EPIDEMIOLOGY AND DEMOGRAPHIC CHARACTERISTICS

Most studies of the incidence and prevalence of eating disorders have been conducted on restricted populations in very restricted areas of their countries. Thus, our knowledge about the incidence and prevalence of anorexia nervosa and bulimia nervosa in various countries is most likely inaccurate. Some figures, however, may be a close approximation to reality. An incidence study conducted in northeastern Scotland showed that between 1965 and 1991, there was almost a sixfold increase in the incidence of anorexia nervosa, from three cases per 100,000 population to 17 per 100,000 ([Hoek, 1993](#)). In a large representative sample of the Dutch population in Holland, [Hoek \(1991\)](#) reported the incidence of bulimia nervosa as 9.9 per 100,000 per year during the period 1985 to 1986 and 11.4 per 100,000 during the period 1986 to 1989. In Rochester, Minnesota, a study covering a 50-year span found an overall adjusted incidence for women of 14.6 per 100,000 per year; for men, the corresponding figure was 1.8 per 100,000 ([Lucas et al., 1991](#)). This study showed no change in the rates for women 20 years of age and older. Among 10- through 19-year-old girls, the incidence rates increased substantially from 1950 to 1984. In summary, when estimates are based on the population at large, the incidence rate of anorexia nervosa in industrialized countries is estimated at 8.1 per 100,000 per year.

[Soundy et al. \(1995\)](#) found that the community-based incidences of bulimia nervosa rose sharply from 1980 to 1983 and then remained relatively constant through 1990. The incidence rates in Rochester, Minnesota, during that decade were 26.5 per 100,000 population for women and girls and 0.8 per 100,000 for men and boys. The mean age for women is 23 years. Among 15- through 24-year-old adolescent girls and young women, it had become at least twice as common as anorexia nervosa.

Prevalence studies of eating disorders are more numerous and easier to conduct. The average prevalence of anorexia nervosa in England, Sweden, and Scotland using strict diagnostic criteria was 0.28% of young women ([Hoek, 1993](#)).

Over 50 prevalence studies of bulimia nervosa conducted between 1981 and 1989 gave a fairly consistent prevalence rate of 1% for bulimia nervosa in adolescent and young adult women ([Fairburn and Beglin, 1990](#)).

The male-to-female ratio for eating disorders in clinical samples lies consistently between 1:10 and 1:20 ([Hoek, 1991](#)). The onset of anorexia nervosa usually is between the ages of 10 and 30 years, with 85% of all anorectic patients developing the illness between the ages of 13 and 20 years ([Halmi, 1974](#)). In one large sample study, a bimodal distribution of age onset was found, with peaks at 14.5 and 18 years ([Halmi et al., 1979](#)). The stress of dieting may be greater at these times, during mid-puberty and at 18 years of age, when adolescents are preparing to leave home for a job or attend college. Because attractiveness is equated with better acceptance, young women may be more concerned about their appearance when they are preparing to leave the safe and dependent home environment.

## CLINICAL DESCRIPTION

Two hallmark characteristics of patients with anorexia nervosa are denial of the seriousness of their illness and resistance to treatment, both of which make obtaining an accurate history and producing an effective treatment result a challenge. Anorectic people demonstrate their intense fear of gaining weight by their intense preoccupation with thoughts of food and irrational worries about fatness. They frequently look in mirrors to make sure they are thin and incessantly express concern about their appearance. They take a great deal of time cutting up food into small pieces and rearranging food on their plates in order to eat less. An overwhelming feeling of inadequacy and ineffectiveness is a core symptom of all anorectic patients. Their success at losing weight is an impressive accomplishment and boosts their self-confidence. Obsessive-compulsive behaviors often develop or become worse as their anorexia nervosa becomes more severe. Obsessions with cleanliness and an increase in cleaning activities and compulsive studying commonly are observed. Perfectionist traits are common in the patient with the restricting type of anorexia nervosa.

Many adolescent anorectic patients have delayed psychosocial development, and adults often have a markedly decreased interest in sex with the onset of anorexia nervosa.

There are important physiologic differences between the two subtypes of anorectic patients. Most of the physiologic and metabolic changes in anorexia nervosa are secondary to the starvation state or purging behavior. These changes revert to normal with nutritional rehabilitation and the cessation of purging behavior.

In the patients with anorexia nervosa who engage in self-induced vomiting or abuse laxatives and diuretics, hypokalemic alkalosis may develop ([Table 55.3](#)). This electrolyte disturbance is associated with physical symptoms of weakness, lethargy, and, at times, cardiac arrhythmias. The latter condition may result in sudden cardiac arrest, a cause of death in patients who purge. Mild elevation of serum liver enzymes may occur both in the emaciated anorectic phase and during refeeding. This reflects some fatty degeneration of the liver. Elevated serum cholesterol levels tend to occur more frequently in younger patients and return to normal with weight gain. Other common laboratory findings in emaciated patients with anorexia nervosa are listed in [Table 55.4](#).

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1. Dental enamel erosion and caries
  2. Perioral dermatitis
  3. Periodontitis
  4. Subconjunctival hemorrhage
  5. Esophageal or gastric rupture
  6. Metabolic alkalosis with hypokalemia
  7. Cardiac arrhythmias
  8. Cardiomyopathy and cardiac failure secondary to ipecac abuse
  9. Renal failure
  10. Seizures
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**Table 55.3. Complications of Binging and Purging Behavior**

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1. Hematologic
Anemia
Leukopenia with relative lymphocytosis
2. Serum and plasma
Hypercarotemia
Hypoproteinemia
Hypercholesterolemia
3. Endocrine
Decreased estrogens
Decreased testosterone (in males)
Immature secretion pattern of luteinizing hormone
Decreased or blunted luteinizing hormone-releasing hormone
Decreased thyrotropin
Increased corticotropin-releasing hormone
Increased fasting and impaired growth hormone secretion responses
Blunted diurnal cortisol levels
Uncoupled vasopressin secretion from osmotic challenge
Low basal metabolic rate
Reduced bone density

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**Table 55.4. Common Laboratory Findings in Emaciated Anorexia Nervosa**

Laboratory findings present with binging and purging behavior are listed in [Table 55.5](#).

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Hypokalemia
Hypochloremic alkalosis
Elevated serum amylase
Electrocardiogram—QT interval and T-wave changes
Photon absorptiometry—reduced bone density

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**Table 55.5. Common Laboratory Findings with Binging and Purging Behavior**

Patients with bulimia nervosa should not be below 15% of the normal weight range. If they are, in most circumstances the correct diagnosis will be anorexia nervosa, binge-purge subtype. Patients with bulimia nervosa can be overweight. The sense of losing control of eating is a significant subjective aspect that occurs during binge eating. Abdominal pain or discomfort, self-induced vomiting, sleep, or social interruption usually terminate the bulimic episode, which is followed by feelings of guilt, depression, or self-disgust. Bulimic patients have a fear of not being able to stop eating voluntarily. Thus, ironically, they may fast for long periods, lose control because of severe hunger, and then binge eat. Hence, they completely forego a normal eating pattern and establish a routine of alternate binges and fasts. The food consumed during a binge usually has a high caloric content and a texture that facilitates rapid eating. Frequent weight fluctuations occur in bulimia nervosa but without the severity of weight loss present in anorexia nervosa. Most bulimic patients have difficulty feeling satiety at the end of a normal meal. They usually prefer to eat alone and at their homes. Approximately one-fourth to one-third of these patients have had a previous history of anorexia nervosa.

Most patients with bulimia nervosa have depressive signs and symptoms. They have problems with interpersonal relationships, self-concept, and impulsive behaviors and also show high levels of anxiety and compulsivity. Alcohol abuse and other drug dependencies are not uncommon in this disorder. Bulimic patients abuse amphetamines to reduce their appetite and lose weight. As is present in the patient with the binge-purge type of anorexia, patients with bulimia nervosa can have severe erosion of the enamel of their teeth, pathologic pulp exposures, loss of integrity of dental arches, diminished masticatory ability, and an obvious unaesthetic appearance of their teeth.

Parotid gland enlargement is associated with elevated serum amylase in bulimic patients who binge and vomit. Other complications of binging and purging behavior are listed in [Table 55.3](#). Severe abdominal pain in bulimic patients should alert the physician to a diagnosis of gastric dilatation and a need for nasal gastric suction, radiography, and surgical consultation.

Cardiac failure may be caused by a cardiomyopathy from ipecac abuse. This is a medical emergency that usually results in death. Symptoms of pericardial pain, dyspnea, and generalized muscle weakness associated with hypotension, tachycardia, and electrocardiographic abnormalities should alert the clinician to possible ipecac intoxication.

## ETIOLOGY AND PATHOGENESIS

The development of anorexia nervosa and bulimia nervosa is best conceptualized within the framework of a multidimensional model, which states that these disorders begin with dieting behavior. Antecedent conditions such as sociocultural influences, family environment, and psychological or personality characteristics and biological vulnerabilities affect the dieting behavior to produce the full-blown disorders of anorexia nervosa and bulimia nervosa. As fasting behavior, weight loss, and binge-purge behaviors continue, significant psychological and physiologic changes occur. Some of these changes are strong secondary reinforcers that allow the process of fasting, weight loss, and binge-purge behavior to continue.

Secondary psychological reinforcement occurs when the young women initially receive compliments for their weight loss and later realize this is one area of their life in which they can be extremely effective and in control. Patients who binge soon achieve a relief of anxiety during their binge eating even though this is followed by unpleasant feelings of guilt and depression. The physiologic reinforcements are less precisely defined. For example, with a period of only 8 hours of fasting, there is an increased secretion of corticotropin-releasing hormone (CRH), which is a potent anorectic agent. This may be effective in assisting some anorectic patients to continue their decreased calorie intake. Exercising causes a release of norepinephrine and endogenous opioids, which may also reinforce a feeling of exhilaration.

### Social and Cultural Influences

Anorexia nervosa and bulimia nervosa seem to be predominantly a “Western” disorder in that they are largely associated with the effects of industrialization and the resulting affluence. The Japanese health care system has been facing increasing numbers of patients with anorexia nervosa since World War II and the greater influence of Western values ([Suematsu et al., 1985](#)). Transcultural studies show that anorexia nervosa is rare in non-Western, poorly industrialized countries ([Lee et al., 1989](#)). When non-Western people are exposed to Western ideals of thinness, they are significantly affected by the exposure. For example, [Fichter et al. \(1988\)](#) found the prevalence of anorexia nervosa in Greek girls living in Germany and exposed to Western ideals of thinness was twice that of Greek girls who remained in Greece and were not exposed to Western values of body image. There have been suggestions that cultural differences in dietary habits, patterns of parent-child interactions, value orientation, and family structure may reveal more about the societal impact on the development of eating disorders ([Pate et al., 1992](#)).

Feminist theories emphasize that women are indoctrinated into a belief system that overvalues feminine beauty and thinness in particular. Women cannot achieve satisfactory self-esteem without attaining ideals that are impossible to fulfill. Eating disorders then become the adaptive response to the stress of demands that

women conform to an impossible and oppressive social expectation.

## Stressful Life Events

Studies investigating the role of sexual abuse as a risk factor in the initiation of an eating disorder have produced contradictory results ( [Connors and Morce, 1993](#)). Overall, approximately 30% of eating-disordered patients have been sexually abused in childhood, and this figure is comparable with rates found in the normal population. Studies have shown that abuse in multiple forms increases the likelihood of lifetime comorbid Axis I disorders and personality pathology among bulimic patients ( [Rorty et al., 1994](#)). It probably is most reasonable to suggest that for some patients there may be a direct link between sexual trauma and eating disorders but that, in general, sexual abuse may best be considered a risk factor for development of a wide range of psychological and psychiatric problems.

In the pre-morbidly vulnerable individual, normative developmental events such as the onset of puberty, leaving home, beginning school, and the start of new relationship have been precipitating events for the onset of an eating disorder ( [Cooper, 1995](#)). Adverse life events such as the death of a close relative, the breakup of a relationship, or an illness also have been related as precipitating eating disorders.

## Biological Factors

### *Genetic Factors*

Genetic vulnerability may be direct through inheritance of genes that are directly associated with the development of anorexia nervosa or bulimia nervosa. An indirect genetic predisposition, on the other hand, could become manifest under adverse conditions such as inappropriate dieting or emotional stress. These predispositions could manifest as a particular personality type, a susceptibility to psychiatric instability (especially affective or anxiety disorders), or a hypothalamic dysfunction. Six controlled studies have found a familial aggregation of anorexia nervosa and bulimia nervosa in the first-degree relatives of anorexia nervosa and bulimia nervosa probands ( [Kaye et al., 2000](#)). In a series of 67 twin probands, [Treasure and Holland \(1989\)](#) found that the concordance for a restricting anorexia nervosa was markedly higher for monozygotic (66%) than for dizygotic twins (0%). In a large, population-based twin registry study, [Kendler et al. \(1991\)](#) showed that concordance for bulimia nervosa was significantly higher in monozygotic than in dizygotic twin pairs. In the first large, uncontrolled study of siblings of anorectic patients, [Theander \(1970\)](#) found that in a sample of 94 anorectic patients, there was a 6.6% risk for female siblings to be diagnosed with anorexia nervosa.

### *Neuroendocrine Factors*

Under stresses such as severe dieting, vulnerability to destabilization of some of the endocrine and metabolic mechanisms affecting eating behavior may influence the development of a full-blown eating disorder. Most of the neuroendocrine changes in the eating disorders are directly related to dieting behaviors, weight loss, and reduced caloric intake. These changes revert to normal with resumption of normal eating behavior and nutritional rehabilitation. For example, increased CRH secretion occurs in underweight anorectic patients and in normal-weight, dieting individuals, but returns to normal with weight restoration. However, CRH is a potent anorectic hormone and may have a role in maintaining anorectic behaviors and initiating a relapse.

It is well established that pituitary cells producing luteinizing hormone are understimulated in patients with anorexia nervosa because of hyposecretion of gonadotropin-releasing hormone (GnRH) by the hypothalamus. A dysfunction may be present in the neurotransmitter systems that influence GnRH release and secretion. The considerable difference in patterns of severe dieting and fasting in patients with bulimia nervosa probably explains the variability in the function of the hypothalamic-pituitary-ovarian axis as measured in various studies of bulimic patients. Some anorectic women remain amenorrheic for a long time (several years) after weight restoration. Studies have shown these women to be more psychologically disturbed compared with those who have a rapid resumption of menses ( [Halmi and Falk, 1983](#)).

The neurotransmitter serotonin is inhibitory to feeding, exploratory, and sexual behaviors, all of which are inhibited in anorexia nervosa. Studies of serotonin receptor polymorphisms have been contradictory. Both serotonin agonists and antagonists have been found useful as adjunct treatments of anorexia nervosa. Cyproheptadine, a serotonin antagonist, was shown to facilitate weight gain in anorectic restrictor patients. This drug most likely acts on hypothalamic appetite centers to decrease satiety and, hence, increase food intake. Clomipramine and fluoxetine (selective serotonin reuptake inhibitors) have been useful in preventing weight relapse in anorexia nervosa and may specifically target the characteristic obsessive-compulsive behaviors associated with food and weight control. Decreased serotonergic turnover in the cerebrospinal fluid (CSF) has been associated with impulsive, suicidal, and aggressive behavior. Animal studies have shown that impaired serotonergic function leads to overeating and obesity. The bingeing and purging behaviors of patients with bulimia nervosa are suggestive of impulse control and satiety regulation problems. Several studies have reported impaired serotonergic function in bulimic patients. Patients with bulimia nervosa have lower CSF 5-hydroxyindoleacetic acid levels compared with control subjects, and blunted prolactin responses to pharmacologic challenge with meta-chlorophenylpiperazine (m-CPP), and to the serotonin agonist fenfluramine ( [Jimmerson et al., 1997](#); [Levitan et al., 1997](#)).

The abnormal pleasure-reward response to food seen in both anorectic and bulimic patients could be related to a dysfunction of the dopaminergic system and its role in self-stimulating or addictive behaviors. This area needs further study. The neurotransmitters norepinephrine, dopamine, and serotonin all interact and affect secretions of neuropeptides that in turn can have appetite-stimulating or satiety-producing effects. At present, there is no definitive evidence that any of the neuropeptides actually cause the initiation of an eating disorder ( [Halmi, 1995](#)).

Serum levels of leptin, the product of an obesity gene, were shown to correlate significantly with body mass index and the amount of adipose tissue in patients with anorexia nervosa. Thus, emaciated anorectic patients have extremely low levels of leptin, which increase as the patients gain weight ( [Eckert et al., 1998](#)). It is unlikely that leptin is a causal agent for producing eating disorders. [Crisp \(1984\)](#) espoused the theory that anorexia nervosa reflects an attempt to cope with maturational problems through the mechanism of avoidance of biological maturity. He found that girls who have an early menarche and who are mildly overweight are at greater risk for development of eating disorders.

## Psychological Factors

Assessing psychological factors as risk factors in the development of eating disorders is problematic because any such study must be retrospective. In a study of recovered patients with anorexia nervosa, [Casper \(1990\)](#) and [Strober \(1980\)](#) found that compared with control subjects and with their sisters, anorectic patients with long-term recovery had greater obsessive-compulsive personality disorder traits. More recently, a multinational collaborative study showed perfectionism and obsessive-compulsive characteristics to be robust traits in patients with anorexia nervosa ( [Halmi, 1999](#)). Restricting anorectic patients tend to receive Cluster C (anxious-fearful) diagnoses such as avoidant or dependent personality disorder. By contrast, anorectic bulimic patients have an equal likelihood of receiving a Cluster B (dramatic-erratic personality diagnoses such as borderline personality disorder) or Cluster C diagnosis. There appears to be no fundamental personality trait that serves as the major risk factor for the development of bulimia nervosa.

## Family Factors

Because family interactions are studied during or after the development of an eating disorder, it is impossible to determine specific interactions as risk factors. There also is the problem of designing valid instruments with which to assess family interactions. In bulimic families, studies have uncovered deficits such as lack of parental affection, negative, hostile and disengaged interactions in the family, parental impulsivity, and family alcoholism and obesity ( [Strober and Humphrey, 1987](#)). Patients with anorexia nervosa perceive their families as stable, nonconflictive, cohesive, and adequately nurturing ( [Vandereycken et al., 1989](#)). In contrast, bulimic patients rate their families as conflictive, badly organized, noncohesive, and lacking in nurturance ( [Wonderlich et al., 1990](#)). Undoubtedly, family factors have a role in the development, maintenance, and relapse of eating disorders, but what percentage of the "variance" can be attributed to family factors is unknown.

## DIFFERENTIAL DIAGNOSIS

In making a diagnosis of anorexia nervosa, it is important to be certain the patient has no medical illness that can account for weight loss. Occasionally, a patient may have both anorexia nervosa and a medical illness contributing to weight loss. In this situation, the diagnosis of anorexia nervosa is made by the positive criteria for the disorder, and both the underlying medical condition and the anorexia nervosa are diagnosed and treated as such. Weight loss frequently occurs in depressive disorders. However, in the latter, the patient usually has a decreased appetite, whereas an anorectic patient denies the existence of an appetite. The hyperactivity seen in anorexia nervosa differs from the agitated activity seen in depressive disorders in that the anorectic's activity is planned and ritualistic, such as exercising programs of jogging and cycling. The preoccupation with the calorie content of food, collecting recipes, and preparing meals for others is typical of the anorectic



patient but not present in those with a depressive disorder. The latter do not have a fear of becoming fat or disturbance in their body image, as is characteristic in anorexia nervosa.

Delusions about food in schizophrenia rarely are concerned with the calorie content of food. A fear of becoming obese and hyperactivity also is uncommon in the schizophrenic but is typical of the anorectic patient.

Chronic medical illnesses frequently associated with weight loss are Crohn disease, hyperthyroidism, Addison disease, and diabetes mellitus. Overeating episodes may occur in the Klüver-Bucy syndrome, which consist of visual agnosia, compulsive licking and biting, inability to ignore any stimulus, and hypersexuality. Another uncommon syndrome associated with hyperphagia is the Kleine-Levin syndrome, which is characterized by periodic hypersomnia lasting for several weeks.

## TREATMENT

### Anorexia Nervosa

There are fewer than 10 randomized, controlled treatment studies in anorexia nervosa. This is most likely because these patients are resistant to and uninterested in treatment, as well as being prone to the development of serious medical complications, which requires withdrawal from research treatment protocols. Open studies have indicated that a multidimensional treatment approach that includes medical management, psychoeducation, and individual therapy using both cognitive and behavioral principles is the most effective treatment. Controlled studies have shown that children younger than 18 years of age do better if they have family therapy or counseling. Nutritional counseling and pharmacologic intervention also can be useful components in the treatment plan.

The severity of illness determines the intensity of treatment required for patients with anorexia nervosa. Treatment levels can range from a medical intensive care unit, to a specialized eating disorder inpatient unit, to a general psychiatric hospital unit, to a day program, to outpatient care. Outpatient therapy as an initial approach has the best chance of success in anorectic patients who have had the illness for less than 6 months, are not bingeing and vomiting, and have parents who are likely to cooperate and effectively participate in family therapy.

Cognitive and behavior therapy principles can be applied in both inpatient and outpatient settings. Although there have been controlled inpatient studies of the effectiveness of behavior therapy for inducing weight gain ([Wullemier et al., 1995](#)), there have been no satisfactory controlled studies of any other type of individual psychotherapy in the treatment of anorexia nervosa. An in-depth discussion of cognitive-behavioral therapy (CBT) for anorexia nervosa can be found in articles by [Garner and Bemis \(1982\)](#) and [Kleifield et al. \(1996\)](#). Alliance building with the patient is essential because patients with anorexia nervosa find it extremely difficult to participate honestly in any therapeutic relationship. At best, they are ambivalent about any treatment attempt or therapeutic relationship that may change their behavior. It is important to foster the patient's cooperation in the treatment program, and this can be done by conveying respect for the patient's dependence on anorexia as a means of coping. Weight restoration and nutritional rehabilitation are extremely important because the state of being underweight is associated with depression, difficulty concentrating, irritability, and preoccupation with food. Also, cognitive impairment is present during this state of calorie restriction. To accomplish necessary changes with psychotherapy, the patient must be in a condition in which she can actually concentrate on the issues of psychotherapy. Monitoring is an important part of CBT. In an outpatient setting, the patient is taught to record not only her food intake but stressful circumstances during the day and her emotional responses to them. Cognitive restructuring is a method in which patients are taught to identify their disturbing cognitions and challenge their core beliefs. In this process, they become aware of specific negative thoughts and present arguments and evidence both to support and to cast doubt on their validity. From this, they try to form a reasoned conclusion based on the evidence. Problem solving is another method whereby patients learn to reason through difficult food-related or interpersonal situations. Adolescents from age 12 years on are fully capable of participating in this type of therapy.

A family analysis should be done on all anorectic patients who are living with their families. From this analysis, a clinical judgment should be made as to what type of family therapy or counseling is clinically advisable. There will be some cases in which family therapy is not possible, and in those instances family relationships can be addressed in individual therapy. In other situations, brief counseling sessions with the immediate family members may be the best way of dealing with family issues. A controlled family therapy study ([Russell et al., 1987](#)) has shown that anorectic patients younger than 18 years of age benefited from this, whereas patients older than age 18 years did worse in family therapy compared with the control therapy. There are no controlled studies of the combination of individual and family therapy. In actual practice, many clinicians provide individual therapy and some sort of family counseling in managing anorexia nervosa.

Medications can be useful adjuncts in the treatment of anorexia nervosa. There have been many large-sample, open studies in Europe concerning the use of chlorpromazine in the treatment of anorexia nervosa. Unfortunately, there are no controlled, double-blind studies to prove definitely the efficacy of this drug for inducing weight gain. However, this medication on an observational basis is especially effective in severely obsessive-compulsive and overwhelmed anorectic patients in that it helps reduce their preoccupations and makes them more amenable to therapy. It may be necessary to start at a low dose of 3 mg three times a day and gradually increase the dosage. There have been several double-blind studies to show that cyproheptadine, especially in high doses up to 24 mg/day, is effective in facilitating weight gain and reducing depressive symptomatology. This drug has the advantage of very few side effects, which makes it attractive for use in emaciated anorectic patients. Unlike the tricyclic antidepressants, it does not reduce blood pressure and increase heart rate. There is some indication that fluoxetine may be effective in preventing relapse in weight-restored patients with anorexia nervosa ([Kaye et al., 1991](#)).

Anorexia nervosa is a complex disorder that responds best to a multifaceted treatment approach that includes medical rehabilitation with weight restoration, individual cognitive psychotherapy, and family therapy or counseling for patients younger than 18 years of age. At times, specific medication such as chlorpromazine, fluoxetine, or cyproheptadine can be useful as adjuncts in the treatment of anorexia nervosa.

### Bulimia Nervosa

Cognitive-behavioral therapy (CBT) is the first-line treatment for bulimia nervosa. It has been found to be the most effective treatment in over 35 controlled studies. In these studies, the treatment program usually lasted 16 to 20 weeks, after which 40% to 50% of the patients were abstinent from both bingeing and purging. A reduction of bingeing and purging occurred in 70% to 95% of patients. Another 30% of those who did not show improvement immediately after treatment did show improvement to full recovery 1 year after treatment. In bulimia nervosa, CBT interrupts the self-maintaining cycle of bingeing and purging and alters the person's dysfunctional cognitions and beliefs about food, weight, body image, and overall self-concept. For therapists to be effective in using CBT for the treatment of bulimia nervosa, they need to be trained from the specific manuals available for this purpose. [Wilson and Fairburn \(1993\)](#) have written an excellent review of these studies.

Although there are no family studies of bulimia nervosa, it is common practice for most clinicians to include the family either in counseling sessions or a form of family therapy in the program of those patients younger than 18 years of age.

Over a dozen double-blind, placebo-controlled trials of antidepressants, including desipramine, imipramine, amitriptyline, nortriptyline, phenelzine, and fluoxetine, have been conducted in normal-weight outpatients with bulimia nervosa. The dosage of antidepressant medication used was similar to that used for the treatment of depression. In all trials, antidepressants were significantly more effective than placebo in reducing binge eating. These medications also improved mood and reduced eating disorder symptoms such as preoccupation with shape and weight. However, the abstinence rate from bingeing and purging was only 22% on the average. [Mitchell and DeZwaan \(1993\)](#) provide an excellent review of these studies. There is some indication that combining antidepressant medication with CBT is helpful in some individuals ([Agras et al., 1992](#)).

Although group therapy has not been studied in a controlled manner, in practice, many clinicians are conducting group CBT for patients with bulimia nervosa. This seems to be especially effective and popular with college students and young adults. Furthermore, it is a cost-effective treatment modality for both patients and therapist.

Adolescents from age 12 years on are fully capable in participating in CBT therapy for bulimia. This treatment has been widely investigated and consistently shown effective in reducing binge eating and purging, improving mood and self-esteem, improving interpersonal functioning, and reducing concerns about shape and weight. These results usually are well maintained. Interpersonal psychotherapy also shows promise as an alternative treatment but requires further investigation. When CBT is not available or possible, most clinicians opt to use a serotonin reuptake inhibitor in treating bulimic patients because this class of drugs has fewer side effects than the tricyclic depressants.

## OUTCOME AND COURSE OF ILLNESS

Long-term outcome studies have been conducted only on patients who have presented for treatment, and therefore, a generalization cannot be made about the course of all patients with eating disorder. Long-term follow-up research (10 years or longer for all patients) indicates that most patients with anorexia nervosa have ongoing problems with the illness (Eckert et al., 1995; Hsu and Crisp, 1979; Theander, 1985). Approximately one-fourth recover, one-fourth stay chronically ill with no improvement, and one-half have partial improvement. Mortality rates at 10 years after presentation for treatment are 6.6%, and at 30 years, 18% to 20%. Most follow-up studies for bulimia nervosa have had a 6-month to 2-year posttreatment follow-up. A review of 88 articles on the course and treatment follow-up studies on bulimic patients (Keel and Mitchell, 1997) summarize mortality rates between 0% and 3%. For those patients who were followed for between 5 and 10 years, approximately 50% fully recovered, whereas 20% continued to meet criteria for bulimia nervosa. Relapse poses a serious threat for bulimics, with approximately one-third of recovered bulimics relapsing within 4 years after treatment. Approximately 20% of bulimic patients seem to sustain an unremitting bulimic illness.

For anorexia nervosa, predictors of outcome have varied from study to study. Most studies, however, have shown that age of onset between 12 and 18 years portends a better outcome than onset before age 12 and after 18 years. Repeated hospitalizations, purging behavior, long duration of illness, and very low weight at presentation for treatment also are predictors of worse outcome in most studies (Eckert et al., 1995). For bulimia nervosa, larger patient samples and longer-term follow-ups are needed to obtain more accurate predictors of outcome in this disorder. A review of research studies on bulimia nervosa (Keel and Mitchell, 1997) concluded that personality disorders marked by problems with impulse control were associated with a worse prognosis in these patients.

## RESEARCH DIRECTIONS

Since the advent of new and efficient techniques in genetic research, the search for the genetic underpinnings of eating disorders has undergone rapid development. Several multinational collaborative studies are under way using a variety of methodologies. The search is on for identification of genetic markers, clusters of genes associated with anorexia and bulimia nervosa, and receptor polymorphisms of specific neurotransmitters in these disorders. Although it will be many years before any clinically meaningful application can be derived from these studies, such research nonetheless is an important and essential undertaking in the next decade.

Improving treatment strategies is another extremely important area for further research. Strategies for motivating eating-disordered patients are necessary to keep them in treatment. Treatment studies conducted over the course of 1 year invariably show high dropout rates, with less than half of the patients achieving a complete cure. Thus, more immediately effective treatment strategies need to be developed, as well as effective means of giving those patients who need more time to deal with a greater number of complex problems the motivation to stay in treatment. Valid assessments for evaluating family interaction need to be developed to ascertain better the family's effect on the course of eating disorders. Cost efficacy studies are necessary to present evidence for adequate insurance coverage for the treatment of eating disorders.

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### CASE ILLUSTRATION

Alice is a 17-year-old, single Caucasian girl who has just completed 4 months in an eating disorder specialty unit for the treatment of anorexia nervosa and obsessive-compulsive disorder (OCD). She arrived weighing 30 kg (66 lbs.) and was 162 cm (5ft4in) tall. She met DSM-IV criteria for anorexia nervosa and OCD, as well as meeting criteria for an Axis II obsessive-compulsive personality disorder.

Her parents describe Alice at 4 years of age as having to do everything perfectly and having rituals around cleaning things thoroughly and putting her toys in their proper places. These behaviors continued throughout grade school. She was always extremely competitive and obtained straight A's. When she was 12 years of age and in sixth grade, she began engaging in peculiar food handling, such as eating food slowly and reducing her total calorie intake. The following summer, she was hospitalized because of weight loss and was given the diagnosis of anorexia nervosa. Her obsessions and compulsions became more severe as her anorexia nervosa progressed. She developed rituals around cleaning, straightening and arranging objects, counting, and exercising. She felt and continues to feel that she must constantly be moving and accomplishing things because she fears becoming "lazy." Alice often was unable to leave her room in the morning for hours because she had to touch objects and perform a number of rituals. This caused her frequently to be late for school. The following year, her weight dropped to 50 lbs. and she was again hospitalized. This was beginning of a series of repeated hospitalizations and weight losses up to the time of the current hospitalization.

Alice denies any bingeing, purging, or drug or alcohol use. She also denies using cigarettes or caffeine. She only restricts her intake and exercises vigorously. She is very distressed about her obsessions and compulsions, but not about her eating behavior. She has primary amenorrhea and has never shown signs of depression. In her past hospitalizations, she was placed on both fluphenazine and clomipramine, but she discontinued medication shortly after she was placed in an outpatient program.

Alice began gymnastics when she was 8 years of age and continued until the onset of her anorexia nervosa. She has never dated or had a boyfriend, and has never been sexually active. She has a very antagonistic and angry relationship with her father, from whom she has always wanted attention and approval and claims she has never received it. She is the youngest of three children. Her two older sisters have done well academically and professionally.

Alice was placed on a liquid diet when she was admitted to a specialized eating disorder program. She had great difficulty adhering to unit rules and procedures and would drink her formula only out of fear of receiving a nasogastric tube if she did not gain weight. She was constantly in motion and became very upset and angry when confronted by staff. Although she was exceedingly harsh, critical, and self-punishing, she was on the other hand very sensitive to criticism about herself. During her fourth month of hospitalization, she was able successfully to maintain her weight within a normal range. Her confidence was greatly improved with her ability to eat three meals and maintain her weight. She actually enjoyed eating because she was allowed to be a vegetarian. She was more sociable, interacting with peers and vocal in community meetings and groups. Her rituals had greatly decreased and she actually was able to get through most of the day without any rituals. However, evenings were still difficult for her because at this time she had preoccupations about "being lazy."

During her fourth month of hospitalization, when she had reached her target weight, Alice was placed on clomipramine 225 mg/day. This medication was helpful in reducing her obsessions and compulsions. Throughout her hospitalization, Alice was treated with CBT and family counseling. She was discharged to a specialized eating disorder outpatient program with cognitive psychotherapy, family counseling, and group therapy.

### Comment

Alice had perfectionistic traits, a feature of obsessive-compulsive personality disorder, as a young child. There is growing evidence that a perfectionistic personality trait is a risk factor for development of anorexia nervosa. Alice was obsessed by her counting and checking obsessions and compulsions, but she was not distressed by her eating disorder rituals and preoccupations. This is a distinguishing factor between the egodystonic obsessions and compulsions of the obsessive-compulsive patient and the egosyntonic preoccupations and rituals associated with an eating disorder. Both of these phenomena had specific expressions in Alice's life, as well as an interaction. Alice's obsessions and compulsions became worse as her anorexia nervosa became more severe. She was managed effectively with cognitive-behavioral therapy techniques, a unit milieu program in a specialized eating disorder unit, and family counseling. The nutritional rehabilitation and medical management was part of the unit milieu program. When Alice reached a normal weight range and still showed bothersome obsessions and compulsions, she was then treated with clomipramine. Alice was able to complete her high school education and graduated as valedictorian.

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# 56 MODERN APPROACHES TO ENURESIS AND ENCOPRESIS

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## ENURESIS

### Definition and Historical Note

Enuresis is subclassified into two subtypes, primary and secondary. *Primary enuresis* encompasses children who have never achieved continence, whereas the term *secondary enuresis* refers to those children who maintain continence for at least 1 year, only to lose it at some point after that. The term itself is derived from the Greek *enourein*, “to void urine” and has come to imply nocturnal events, although that connotation is not inherent in the derivation of the word itself.

There is a rich literature concerning enuresis and its treatment over the centuries. In retrospect, many of these treatment approaches now appear to have been quite sadistic. This history has been summarized in an excellent review by [Glicklich \(1951\)](#), which covers material dating back to the Papyrus Ebers of 1550 B.C.

There has been substantial progress in the treatment of enuresis. This therapeutic progress has contributed to a greater understanding of the fundamental pathophysiologic processes that contribute to enuresis. These advances are reviewed in this chapter.

### Prevalence and Epidemiology

Statistics concerning the prevalence of enuresis also must take into account the severity of the disorder. For example, in the Isle of Wight Study, Rutter and colleagues found that 15.2% of boys were wet less often than once a week, whereas only 6.7% wet at least once a week. The corresponding figures for girls were 12.2% and 3.3%. By age 14 years, only 1.9% of boys were wet less often than once a week, and 1.1% were wetting at least once a week, with the corresponding figures for girls being 1.2% and 0.5% ([Rutter et al., 1973](#)). Longitudinal data from the Isle of Wight Study have illustrated that wetting develops in many children between the ages of 5 and 7 years. Enuresis also was found in greater frequency in children who were undergoing psychosocial stress and in those living in socially disadvantaged circumstances ([Rutter, 1989](#)).

A Scandinavian study of 3,206 7-year-old children found an overall prevalence of 9.8%; 6.4% of this group was accounted for by children with night wetting, 1.8% by day wetters, and 1.6% by those with mixed day and night wetting. This study also showed a strong genetic influence in that the risk of a child's having enuresis was 7.1 times greater if the father manifested enuresis after 4 years of age and 5.2 times greater if the mother did ([Jarvelin et al., 1988](#)).

An 8-year longitudinal study in New Zealand found a prevalence of 7.4% for nocturnal enuresis in 8 year olds. This figure was accounted for by 3.3% with primary enuresis and 4.1% with secondary enuresis ([Fergusson et al., 1986](#)).

More recent studies have found remarkably similar results. In a group of 392 7-year-old children from the west coast of Sweden, [Wille \(1994c\)](#) reported a prevalence of 7.3% for monosymptomatic primary enuresis. A questionnaire study involving a large cohort of Australian children in the 5- to 12-year-old range reported an overall incidence of 5.1% for nocturnal enuresis of least weekly frequency and 1.4% for daytime wetting of similar frequency ([Bower et al., 1996](#)).

### Clinical Description

As noted, the term *enuresis* itself denotes only the voiding of urine. Over the years, the term has acquired both a pathologic and a nocturnal connotation. Daytime wetting is correctly referred to as *diurnal enuresis*, whereas nighttime wetting is referred to as *nocturnal enuresis*.

The fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) ([American Psychiatric Association, 1994](#)) defines functional enuresis as “repeated voiding of urine during the day or at night into bed or clothes.” DSM-IV goes on to note that “to qualify for a diagnosis of enuresis, the voiding of urine must occur at least twice per week for at least three months or else must cause clinically significant distress or impairment in social, academic (occupational) or other important areas of functioning.” The child must also have reached an age at which continence could definitively be expected. DSM-IV uses a chronologic age of 5 years as a cutoff or a mental age of 5 years for those children with developmental delays. DSM-IV also stipulates that the wetting not be the result of “the direct physiological effects of a substance (e.g., diuretics) or a general medical condition (e.g., diabetes, spina bifida, a seizure disorder).” Three subtypes of enuresis are defined: nocturnal only (nighttime wetting), diurnal only (daytime wetting), and nocturnal and diurnal (mixed day and night wetting). A distinction also is made between “primary” and “secondary” enuresis. Primary enuresis refers to those children who have never achieved urinary continence, whereas secondary enuresis refers to those children who have achieved continence and then lost it. The period of continence necessary to differentiate between primary and secondary enuresis had variously been thought to be 6 months to 1 year. DSM-IV does not put forward a precise period of time for the distinction, but instead makes reference to “a secondary type in which the disturbance develops after a period of established urinary continence.” A child is not considered to have primary functional enuresis until 5 years of age. Secondary enuresis can begin at any time, once the criterion of initial continence has been fulfilled, but the usual onset is between 5 and 7 years of age ([Rutter, 1989](#)).

### Etiology and Pathogenesis

The physiologic manifestations of this disorder have led to a wide range of etiologic theories. A primary focus of these studies has naturally been the anatomy of the bladder and urinary tract. [Shaffer et al. \(1984\)](#) elegantly combined an investigation of bladder anatomy and physiology with the covariable of behavioral disturbance. It might intuitively be expected that children with dysfunctional or abnormal bladders would be those without a concomitant behavioral disorder to explain their enuresis and that those whose enuresis could be explained on the basis of psychopathology would have normal bladders. The results were counterintuitive in that those children who were behaviorally disturbed also had significantly lower functional bladder volumes and more developmental delays. Thus, although not providing a



parsimonious explanation to differentiate the etiology of enuretic events between psychiatrically disturbed and nondisturbed children with enuresis, the study did lend further support to a theory of general developmental delay, which would explain both the enuresis and the high frequency of behavioral disturbance. In a more recent study, which investigated bladder capacity in children with primary nocturnal enuresis, former enuretic patients, and control subjects, also failed to find any significant difference in bladder capacity between the groups ([Wille, 1994a](#)). It also has been demonstrated that fluid loading can produce enuretic events in children who do not have a history of enuresis ([Kirk et al., 1996](#); [Rasmussen et al., 1997](#)). Children with enuresis were found to have developmental delays twice as often as those without in a large longitudinal population study ([Essen and Peckham, 1976](#)). A study that compared 35 otherwise healthy children with enuresis with a control group found that the bone age of the children with enuresis displayed a significant lag behind chronologic age, leading the authors to speculate about delayed maturation of central nervous system regulatory functions ([Mimouni et al., 1985](#)).

There is an obvious relationship between enuresis and bladder infection ([Hansson, 1992](#)); thus, an infection of the urogenital tract should be ruled out before a diagnosis of functional enuresis is made. This is especially important for girls, who are more prone to urinary tract infections ([Hjalmas, 1992](#)). The possibility of urinary tract obstruction as a widespread cause of enuresis has been reported ([Mahony, 1971](#)) but has been criticized because such a hypothesis can lead to unnecessary surgery ([Smith, 1969](#)). After extensively reviewing the literature on this subject, [Shaffer \(1985\)](#) concluded, "There is no evidence that urethral dilatation or bladder neck repair are effective treatment for enuresis." The only exception to this would be if there were very specific pathophysiologic findings.

More recent investigations into the role of urodynamic abnormalities in the pathogenesis of primary nocturnal urinalysis support Schaffer's conclusion ([McDermott and Merrick, 1994](#)). In a large study, [Kawauchi et al. \(1996a\)](#) found an incidence of urologic abnormalities of 1.8% on intravenous pyelography (n = 940), 7.1% on voiding cystourethrography (n = 695), 11.5% on cystometry (n = 487); and no abnormalities on renal ultrasonography (n = 58). Of those who did manifest reflux on voiding cystourethrography, the degree of reflux was assessed as mild in 92.1%.

The nature of the enuretic phenomenon has naturally led to speculation concerning a psychodynamic etiology. These hypotheses have in general evolved from case reports or have been derived from theoretical considerations. There has been one rigorous attempt to define the generalizations derived from the literature regarding enuresis and encopresis and then to determine with what frequency these generalizations were borne out by an analysis of the clinical material. This elegant study by [Achenbach and Lewis \(1971\)](#) revealed "only two of the twenty-four generalizations derived from the literature regarding encopresis and enuresis received support at the conventional level (probability = 0.05) of statistical significance."

Epidemiologic studies have, however, shown a correlation between psychological disturbance and enuresis, which is more pronounced in older children ([Rutter, 1989](#)). This observation then raises the question of the nature of the relationship: Is it a causal, incidental, or secondary relationship? The aforementioned link between enuresis and developmental delays, which also are linked to psychopathology, would suggest that there is a common underlying maturational factor that predisposes vulnerable children to manifest both behavioral disturbances and enuresis. In further support of this hypothesis are the observations that the nature of the behavioral disturbance in children with enuresis is nonspecific ([Mikkelsen et al., 1980](#)) and that no physiologic marker can be found that reliably differentiates psychologically disturbed from nondisturbed children with enuresis ([Mikkelsen and Rapoport, 1980](#); [Shaffer et al., 1984](#)). When children with enuresis have been compared with other children attending a child psychiatric clinic, the only discernible differences were an increased frequency of developmental delays and a lesser frequency of eating disorders and specific neurotic patterns ([Steinhausen and Gobel, 1989](#)). [Biederman et al. \(1995\)](#) have evaluated the possible linkage between enuresis and attention deficit hyperactivity disorder (ADHD). Their findings indicated that enuresis did not increase the risk for psychopathology in children with or without ADHD but was associated with increased risk for learning disability in normal control children, but not in those with ADHD. The association with behavioral disturbance has been reported as being greater for secondary enuresis ([Feehan et al., 1990](#); [McGee et al., 1984](#); [von Gontard et al., 1999b](#)) and with enuresis persisting into adolescence ([Fergusson and Horwood, 1994](#)). One study that specifically evaluated risk factors for development of secondary enuresis found that delayed attainment of initial nocturnal continence and exposure to four or more stressful life events in a year were significantly related to the development of secondary enuresis ([Fergusson et al., 1990](#)). Similar results with regard to the relationship between psychosocial stress and secondary enuresis have been reported by [von Gontard et al. \(1997\)](#). However, at least one large study in the Netherlands found no difference in psychopathology between children with primary and secondary enuresis ([Hirasing et al., 1997](#)).

The occurrence of the enuretic episodes during sleep naturally led to a series of studies investigating the relationship between sleep states and the occurrence of enuretic events. The earliest of these studies suggested that the enuretic events occurred in "deep sleep" and led to a theory that enuretic events were dream equivalents ([Pierce et al., 1961](#)). This theory was subsequently supplanted by Broughton's ([Broughton, 1968](#); [Gastaut and Broughton, 1964](#)) view that enuresis was a disorder of arousal. This research suggested that enuretic episodes were preceded by arousal signals and originated in delta sleep. A further elaboration of this theory held that psychiatrically disturbed children with enuresis received normal arousal signals but did not respond to them, whereas those without psychiatric disturbance did not generate arousal signals ([Ritvo et al., 1969](#)). The largest and most convincing sleep studies indicate that enuretic episodes occur in each sleep stage in proportion to the time spent in that stage, when time of night also is considered ([Kales et al., 1977](#); [Mikkelsen et al., 1980](#); [Robert et al., 1993](#)). There have been three studies that suggested that children with primary enuresis may be more difficult to arouse from sleep than control subjects, although the methodology is somewhat subjective with regard to defining arousability ([Neveus et al., 1999a](#); [Wille, 1994c](#); [Wolfish, 1999](#)). Recent research in this area has focused on combining sleep studies with cystometry ([Norgaard et al., 1989](#)). This work may lead to the identification of subtypes of children with enuresis ([Imada et al., 1998](#); [Watanabe and Azuma, 1989](#); [Watanabe et al., 1994](#)).

The development of desmopressin acetate (DDAVP) as a treatment for enuresis (see later) has led to the observation that some children with enuresis do not have the ability to concentrate the urine they produce during the night and reduce urine volume ([Miller et al., 1992](#)). In a further investigation of this hypothesis, [Rittig et al. \(1991\)](#) compared the circadian variation of plasma atrial natriuretic peptide (ANP) with the clearance of creatinine and the excretion of sodium and potassium. Subjects in the study consisted of 15 children with nocturnal enuresis and 11 control subjects matched for age, sex, and weight. The children with enuresis did not differ from control subjects with regard to ANP, but during the first hours of sleep, they did manifest significantly more polyuria, natriuresis, and kaliuresis despite normal levels of ANP. The authors concluded that children with enuresis display abnormal diurnal rhythmicity in the urinary excretion of potassium and sodium that is not correlated with plasma levels of ANP. They speculated that the abnormalities in sodium and potassium may be related to abnormal tubular handling. This hypothesis has been further supported by subsequent research ([Natochin and Kuznetsova, 1999](#); [Vurgun et al., 1997](#)). [Steffens et al. \(1993\)](#) used a radioimmunoassay to evaluate the circadian rhythmicity of plasma arginine vasopressin (AVP) in 55 children with enuresis and 15 control subjects. The AVP levels were measured under conditions of controlled water intake three times per day for 72 hours. Only 14 of the 55 children with enuresis had a significant decrease in AVP compared with control subjects. Nine of these 14 AVP-deficient children subsequently were found to be totally dry with DDAVP treatment.

The circadian rhythmicity of AVP has continued to be a focus of investigation because it theoretically could explain both the pathophysiology of enuresis and its response to DDAVP. Accordingly, researchers have been particularly interested in any differences that could be detected between DDAVP responders and nonresponders. One study has reported significant differences in morning values of AVP between normal control subjects (n = 7) and children whose enuresis responded to DDAVP (n = 6), as well as between the responders (n = 6) and nonresponders to DDAVP (n = 5). Thus, the morning AVP levels were able to differentiate the children with enuresis from the control subjects, and the responders from the nonresponders ([Medel et al., 1998](#)). However, further complicating this line of research has been the finding that AVP is secreted in a "pulsatile pattern," which dictates frequent sampling of plasma levels to be meaningful ([Wood et al., 1994](#)). Studies using frequent measurements of AVP have produced mixed results. Two studies that used more frequent AVP measurements ([Lackgren et al., 1997](#); [Wood et al., 1994](#)) found no differences between responders and nonresponders to DDAVP. [Aikawa et al. \(1998, 1999\)](#) addressed this question in a series of studies that measured AVP secretion on an hourly basis for 24 hours. The first set of these studies looked at AVP secretion profiles in children with enuresis (n = 9) and control subjects (n = 8). The results did establish that the plasma AVP level was significantly lower in the children with enuresis in the 11 P.M. to 4 A.M. time period. They then looked at the secretion dynamics in two phenomenologic subgroups of children with enuresis: those with low urinary osmotic pressure and large nocturnal urine output, as opposed to a group with normal urinary osmotic pressure and small nocturnal urine output. The results showed that the mean nocturnal AVP levels were significantly lower in the first (large nocturnal output) group and that treatment with DDAVP did produce a significant increase in AVP for this group as a whole, but not for each child.

Another area of research has been the role of urine osmolality in the production of nocturnal enuresis. Three separate studies that looked at first morning urinary specific gravity in preschool children have suggested that children who wet the bed tend to have lower mean urinary specific gravity than those who do not, but the findings do not reach statistical significance ([Kawauchi, 1996b](#); [Mevorach et al., 1995](#); [Salita et al., 1998](#)).

The effect of fluid restriction on AVP levels and urine osmolality also has been investigated. These studies indicate that AVP levels are increased in both control subjects and in children with enuresis in response to fluid restriction, and that the degree of AVP secretion is related to plasma osmolality ([Eggert et al., 1999](#)). When DDAVP responders and nonresponders are compared with control subjects, all three groups manifest an increase in AVP, but the DDAVP responders showed a lower increase than the other groups ([Hunsballe et al., 1999](#)). Studies involving adolescents and adults with refractory enuresis also have suggested that the primary pathophysiologic mechanism may be an abnormal tubular processing of sodium related to a relative insensitivity to AVP ([Hunsballe et al., 1998](#); [Robertson et al., 1999](#)) that is corrected to some degree by DDAVP. Similar research in children led [Eggert and Kuhn \(1995\)](#) to hypothesize that the primary difference between



children with enuresis and control subjects may be at the receptor level.

Although this line of research has primarily involved DDAVP, it also has led to a reexamination of the therapeutic effect exerted by imipramine. [Hunsballe et al. \(1997\)](#) reported a decrease in urine output and reduced osmolal clearance induced by imipramine that, in part, was contributed to by a lower excretion of sodium and potassium.

One of the newest areas of research has been the exploration of genetic linkages. It has long been known that enuresis tends to run in families and that a positive family history can be related to positive treatment outcome ([Hogg and Husman, 1993](#)). In general, the new genetic studies involve large numbers of families with multigenerational transmission of primary nocturnal enuresis. The chromosomes that have been identified to date include 13q, 12q, 8, and 22 ([Arnell et al., 1997](#); [Eiberg et al., 1995](#); [von Gontard et al., 1999a](#)). In some families, an autosomal dominant mode of transmission with penetrance above 90% has been identified.

### Laboratory Studies

The fact that urinary tract infections can precipitate enuretic events in children means that a urinalysis should be performed to rule out this readily treatable cause of enuresis.

The use of more invasive and painful studies remains controversial. Although it is certainly possible that altered bladder physiology may lead to primary enuresis, the yield from these studies does not appear to be of sufficient magnitude to warrant subjecting all children with enuresis to them. A thorough review of this subject has been performed by [Cohen \(1975\)](#), who found the incidence of obstructive lesions in children with enuresis to be 3.7% in a primary care pediatric setting. Accordingly, he suggested that “contrast studies are indicated only when there is significant evidence of anatomical or functional pathology by history or exam.” Subsequent studies have supported this general position ([Blickman and Schimmelpenninck-Scheiffers, 1984](#); [Kawauchi et al., 1996a](#); [McDermott and Merrick, 1994](#)), while suggesting that those children with daytime wetting and overt symptoms of voiding disturbance are more apt to have urinary tract abnormalities than those who wet solely at night ([Jarvelin et al., 1990](#)).

### Differential Diagnosis

The differential diagnosis includes the possibility of urinary tract infection and altered bladder physiology. There are scattered case reports of enuresis being secondary to other primary medical problems, such as hyperthyroidism ([Stoffer, 1988](#)), constipation ([Oregon et al., 1986](#)), and central hormonal abnormalities ([Kikuchi et al., 1989](#)). Although such reports are infrequent, the clinician should do a thorough physical examination and consider the possibility of underlying organic illness. Psychological testing in conjunction with structured interviews may provide further insight into the coexistence of psychopathology. However, the studies reviewed previously suggest that any coexisting psychological disorder should be viewed as an accompanying finding rather than as a causal effect.

The distinction between primary and secondary enuresis can be made by history.

### Treatment

Although psychotherapy may be helpful for managing the behavioral disorders that accompany enuresis, it appears to have little effect on primary enuresis itself, with studies showing a success rate of 20%, which may largely be accounted for by spontaneous remission ([Cohen, 1975](#)). Psychotherapy may be more useful for those children with secondary enuresis, especially those whose episodes begin after a traumatic event or parental divorce.

It has been shown that having nocturnal enuresis has a negative impact on self-esteem, which can be normalized by effective treatment ([Hagglof et al., 1998](#); [Moffatt et al., 1987](#)).

The two primary means of treating children with enuresis fall into the categories of behavioral methods and psychopharmacologic methods.

Behavioral treatment should be attempted first because it usually is more innocuous than pharmacologic intervention. The underlying assumption of the behavioral strategy is that it is helping children with enuresis and their families master an affliction rather than tacitly implying that the children are either consciously or unconsciously causing the wetting themselves. One unfortunate consequence of various reward–punishment strategies is that they can subtly imply to children and their families that the disorder is quasi-volitional. The bell and pad method of conditioning is a reasonable first approach. A review of this treatment modality indicated that it was first reported in 1904 and has been in routine use since the 1930s ([Rappaport, 1997](#)). In reviewing the results of several studies involving over 1,000 children, [Werry \(1966\)](#) found a success rate of 75%. Recent studies are consistent with this ([Berg et al., 1982](#); [Devlin and O’Cathain, 1990](#)). There appear to be two subgroups of responders: those who sleep through the night after treatment without wetting, and those who wake up spontaneously to go to the bathroom ([Bonde et al., 1994](#)). Psychiatric disorder in the child and family stress appear to be negative prognostic factors when predicating outcome with this modality ([Devlin and O’Cathain, 1990](#)). An investigation into the effectiveness of a simple bell and pad method as opposed to two variations that had more behavioral embellishments found no statistical difference between the modalities. However, one variation that included reward contingencies tended to have a slightly lower rate of relapse than the bell and pad alone ([Kaplan et al., 1989](#)). As noted, whenever reward–punishment contingencies are considered, it is extremely important to ensure that one is not communicating to the parents and child that the disorder is quasi-volitional. Although their frequency has decreased with improved bell and pad technology, reports of “buzzer ulcers” do continue to appear, and parents should be alerted to this ([Diez and Berger, 1988](#)).

An attempt has been made to investigate the relationship between bladder capacity and response to behavioral treatment. A study involving 50 children who were wet at least 2 nights a week found that children with small pretreatment maximal functional bladder capacities did better with the bell and pad method in conjunction with retention–control training, whereas the children with larger bladder capacities responded to the bell and pad method alone. However, this was a qualitative difference in response, because 92.5% of the 40 children who completed the study met the outcome criteria of 14 consecutive dry nights, regardless of which group they were in ([Geffken et al., 1986](#)). A similar study that examined the impact of bladder capacity on response to the bell and pad system found no association with outcome ([Berg et al., 1982](#)). Both of these investigations indicated that behavioral disturbance was related to failure to respond to conditioning techniques ([Berg et al., 1982](#); [Geffken et al., 1986](#)).

Bladder capacity also has been investigated with regard to changes occurring during treatment. [Oredsson and Jorgensen \(1998\)](#) measured bladder capacity in 18 children with severe nocturnal enuresis before beginning a 6-week period of treatment with the bell and pad and again after treatment. Ten of the 18 children ceased wetting, but overall there was a significant increase in bladder capacity for the entire group that did not correlate with outcome.

Behavioral treatment continues to evolve. In a study involving 125 children, an attempt was made to replace the bell and pad mechanism with a simple alarm clock that was either set to go off at a time when the bladder might be expected to be reaching maximal capacity (group I) or after 2 to 3 hours of sleep (group II). The results were comparable with previously published figures for the bell and pad, with success noted in 77.1% of group I and 61.8% of group II, and respective 6-month relapse rates of 24.1% and 14.7% ([El-Anany et al., 1999](#)).

Another innovation involves replacing the pad that signals the enuretic event with a small ultrasonic monitor mounted to an elastic abdominal belt that signals the alarm when bladder capacity is reaching a predetermined threshold ([Petrican and Sawan, 1998](#)). Results of a clinical trial of this methodology were comparable with those obtained with the traditional bell and pad technique, and increases in nighttime bladder capacity also were noted ([Pretlow, 1999](#)).

An approach using bladder biofeedback has been developed for children with enuresis who are refractory to other forms of treatment, have small bladder capacities, and have evidence of an unstable detrusor. Specifically, the authors noted that of 24 children who fit these criteria, 17 experienced complete remission (two of these later relapsed), six experienced a decrease, and in one, there was no change ([Hoekx et al., 1998](#)).

As noted earlier, the presence of behavioral or family functioning problems can have a negative impact on the outcome of behavioral treatment. A successful intervention in refractory children with severe wetting who have these issues is to combine traditional alarm therapy with treatment with DDAVP ([Bradbury, 1997](#); [Bradbury and Meadow, 1995](#)).

The Australian psychiatrist, [MacLean \(1960\)](#), first described the efficacy of imipramine for nocturnal enuresis in 1960. Since then, there have been over 40 double-blind studies confirming the efficacy of imipramine for nocturnal enuresis, whereas results with other classes of psychotropic agents have been in general equivocal or negative ([Blackwell and Currah, 1973](#)). Lack of response to imipramine often can be traced to the reluctance of primary care physicians to exceed



dosages of 25 to 50 mg. Nevertheless, it is reasonable to begin at a dose of 25 mg and to titrate up slowly because some children respond to the lower dosages. Allowing 4 to 7 days between dosage increments makes it possible to detect these low-dose responders. Most children respond in the 75- to 125-mg range. The upper range of dosage is determined by the child's weight, with the standard upper limit being 5 mg/kg. A baseline electrocardiogram should be obtained before instituting treatment with imipramine, and monitoring is advised above 3.5 mg/kg ( [Mikkelsen and Rapoport, 1980](#)). A case of sudden death has been reported in a girl receiving 14.7 mg/kg for school phobia ( [Robertson and Barker, 1976](#)).

The relatively high rate of spontaneous remission in enuresis militates against keeping children on medication for long periods. A practical approach is to taper slowly and discontinue the imipramine every 3 months. If wetting resumes as the dosage is tapered or after it is discontinued, then the dosage can simply be titrated back up to the effective dose for another 3-month period. It has been the author's impression that more children do not experience a reactivation of the enuresis after a 3-month period of imipramine treatment than can be accounted for by spontaneous remission alone, but this has not been statistically proven.

There have been tragic reports of children who reasoned that if three pills would stop the wetting for a night, then taking the whole bottle should stop it permanently. Thus, it is important to warn parents about the magical thinking of children in this regard and the importance of controlling the medication. Younger siblings also are at risk of overdosing with the medication if it is not controlled. In cases of mild to moderate overdose, supportive measures, including the symptomatic management of seizures and cardiac arrhythmias, may be sufficient. Physostigmine also may be useful in more severe overdoses ( [Greene, 1981](#)).

There have been five studies investigating the relationship between blood level of imipramine and clinical response. One study found no correlation between improvement in enuresis and the blood level of imipramine either alone or in conjunction with its metabolites ( [Devane et al., 1984](#)). However, three studies have now demonstrated a significant correlation between the diminution of enuretic events and the steady-state concentrations of imipramine plus its metabolite desipramine ( [de Gatta et al., 1984, 1990](#); [Jorgensen et al., 1980](#); [Rapoport et al., 1980](#)). One of these studies found an optimal effect when the combined steady-state imipramine plus desipramine concentrations were above 60 mg/L ( [Jorgensen et al., 1980](#)), and another reported favorable outcomes when steady-state combined levels were greater than 80 ng/mL ( [de Gatta et al., 1984](#)). The most recent study in this line of investigation ( [Fritz et al., 1994](#)) evaluated the blood level–efficacy equation in 18 children who, after baseline and placebo, received increasing dosages of imipramine at 2-week intervals. The specific dosages used were 1, 1.5, 2, and 2.5 mg/kg. They found that efficacy was “moderately but significantly” related to increasing dose. However, there was tremendous (700%) variation in serum levels between the individual children at every dosage level. There is a good correlation between side effects and blood level, especially dry mouth ( [Rapoport et al., 1980](#)). This may prove clinically useful in monitoring children who are extremely phobic about having their blood drawn.

As might be expected, the advent of treatment with DDAVP has led to a marked decline in new research concerning imipramine.

One multicenter, randomized, double-blind, placebo-controlled study did compare imipramine with the tetracyclic antidepressant mianserin, which does not have significant anticholinergic effects ( [Smellie et al., 1996](#)). The authors reported that imipramine was statistically significantly superior to both mianserin and placebo, leading them to conclude that imipramine's efficacy was not related to its antidepressant effect. As noted previously, one study has suggested that imipramine may exert a nocturnal antidiuretic effect mediated by effects at the renal tubular level ( [Hunsballe et al., 1997](#)).

Despite the efficacy of behavioral interventions, survey studies tend to indicate that in clinical practice medication is more apt to be used than behavioral interventions ( [Devlin, 1991](#)). A large population-based study found that only 38% of children with enuresis had seen a physician. Over one-third of this physician-treated group had been prescribed some form of pharmacotherapy, and only 3% had been advised to use the bell and pad conditioning technique. This study also revealed that over half of the children were psychologically distressed by the enuresis, and two-thirds of the parents expressed concern ( [Foxman et al., 1986](#)). This may be changing, because a more recent study reported 80% of physicians recommended the bell and pad ( [Vogel et al., 1996](#)). Throughout the literature there also are reports of novel treatment approaches such as acupuncture ( [Capozza et al., 1991](#); [Minni et al., 1990](#); [Roje-Starcevic, 1990](#)), a prostaglandin synthesis inhibitor ( [Metin and Aykol, 1992](#)), an anticholinergic calcium antagonist ( [Elmer et al., 1991](#)), the oral synthetic androgen mesterolone ( [el-Sadr et al., 1990](#)), and hypnosis ( [Banerjee et al., 1993](#)). Oxybutynin hydrochloride has been reported as effective for children who are refractory to imipramine and also have inadequate bladder storage function ( [Kosar et al., 1999](#)). Although many of these studies are controlled, the use of these approaches in enuresis still should be considered experimental.

The newest research into pharmacotherapy for enuresis involves the use of DDAVP. [Moffatt et al. \(1993\)](#) reviewed all of the then-existing controlled studies concerning the use of DDAVP for enuresis. In the process, they located 18 randomized, controlled trials (11 crossover and seven parallel), which included a total of 689 subjects, most of whom had been refractory to prior treatment. The decreased frequency of enuretic events in the study ranged from 10% to 91%. In general, wetting resumes once the medication is discontinued. Those studies that reported long-term follow-up indicated that 5.7% remained dry after stopping the medication. The most common side effects were nasal stuffiness, headache, epistaxis, and mild abdominal pain. Positive prognostic factors appear to be fewer initial (pretreatment) wet nights and age greater than 9 years. [Hogg and Husmann \(1993\)](#) and [Terho \(1991\)](#) particularly looked at the efficacy of DDAVP for children who had been refractory to conditioning treatment and imipramine, using a randomized, double-blind, placebo-controlled crossover study. Of the 52 children studied (age range, 5 to 13 years), 53% had a complete cessation of wetting, 19% were partial responders, and 28% had no or minimal response. The dosages used ranged from 20 to 40 µg (intranasal) and response did not persist after termination of treatment. In a 5-year retrospective review of 59 children, [Key et al. \(1992\)](#) suggested that lower doses may be just as effective. In their series, 5 µg at bedtime was the initial starting dose, and 81% improved on less than 10 µg. A single-blind study did reveal a dose–response phenomenon ( [Fjellestad-Paulsen et al., 1987](#)).

A study investigating the differential response of children with enuresis to DDAVP and the bell and pad method of conditioning found that 70% improved with the DDAVP and 86% improved with the alarm method, yielding no significant differences ( [Wille, 1986](#)). A similar experiment that compared the therapeutic benefits of DDAVP in combination with the bell and pad to placebo found that the combination of DDAVP and the alarm resulted in significantly more dry nights ( [Sukhai et al., 1989](#)). A follow-up study of children receiving treatment for an average of 13 months with DDAVP revealed no hormonal or hematologic side effects, and it was concluded that treatment of this duration was safe ( [Rew and Rundle, 1989](#)). However, there have been reports of hyponatremia ( [Kallio et al., 1993](#)) and hyponatremic seizures ( [Beach et al., 1992](#); [Schwab et al., 1996](#); [Yaouyanc et al., 1992](#)) with intranasal use of DDAVP. A case report and literature review documented 14 cases in the English language literature with symptomatic hyponatremia involving seizures or mental status changes ( [Bernstein and Williford, 1997](#)). A similar review noted that excess fluid intake was identified in 6 of 11 case reports, leading the authors to recommend that patients receiving DDAVP for nocturnal enuresis should not ingest more than 8 ounces of fluid on the nights when DDAVP is administered ( [Robson et al., 1996](#)).

Sufficient time has now elapsed since the institution of the widespread use of DDAVP to permit more follow-up studies. A large, multicenter Swedish study used a 4-week observation phase, a 6-week dose titration period (20 to 40 µg DDAVP), and a 1-year long-term treatment period into which a treatment-free week was introduced every 3 months to assess for remission. Subjects were 399 children with primary nocturnal enuresis aged 6 to 12 years. Sixty-one percent (245) experienced a 50% or greater reduction in wet nights during the dose titration phase and then entered the long-term phase. The average number of wet nights decreased from 5.3 during the observation phase to 0.8 during the last 3-month interval. Within 6 months of treatment initiation, 77 children became dry. There were significantly more responders in the older age groups. Overall, long-term treatment at these dosages was found to be safe ( [Hjalmas et al., 1998](#); [Tullus et al., 1999](#)).

Another strategy has been rapidly to titrate the dosage of DDAVP (maximum, 50 µg) until dryness is achieved, maintain this dosage for at least 4 to 6 weeks, then decrease the daily dosage by 10 µg every 4 dry weeks. This strategy resulted in 71% achieving complete dryness with no relapses. A further 7% achieved dryness after relapses, 7% showed partial improvement, and 15% showed little or no response. The mean dose was 20 µg, the mean duration of treatment was 28 weeks, and median follow-up was 18 months ( [Riccabona et al., 1998](#)).

The formulation of oral DDAVP has made it significantly easier to administer. In an early open 6-week trial involving 33 children with primary nocturnal enuresis, five children responded to 200 µg/day and 17 to 400 µg/day, whereas seven showed no response, and four dropped out. A subsequent 2-week treatment with 40 µg of the nasal spray showed similar efficacy and was able to increase the number of dry nights in two of the nonresponders ( [Matthiesen et al., 1994](#)). A multicenter, randomized study compared 200- and 400-µg doses of the oral preparation, as well as a 20-µg dose of the spray. No significant differences were found between any of the treatment conditions. However, there tended to be fewer wet nights when the children who initially received 200 µg of the oral preparation were increased to 400 µg ( [Janknegt et al., 1997](#)).

The dose response of oral DDAVP was explored in a randomized, placebo-controlled study that used 200-, 400-, and 600-µg daily doses. The 400- and 600-µg doses were significantly more effective than placebo, and there also was a significant linear trend for decreases in wet nights with increasing dosage ( [Skoog et al., 1997](#)).

An early oral DDAVP study with adolescents ( [Stenberg and Lackgren, 1994](#)) also has produced long-term follow-up results ( [Lackgren et al., 1998](#)). The initial study included two 12-week treatment periods with most of the patients receiving 400 µg/day. The initial studies showed that oral DDAVP was significantly more effective than placebo. The long-term, 7-year follow-up indicated that the “cure rate” at both the 2-year and 7-year follow-ups was greater than would be expected by data on

the rate of spontaneous remission.

As indicated earlier, in the section on Pathophysiology, the success of treatment with DDAVP has led to an increase in research regarding the etiology of enuresis. Related to this are studies that try to elucidate factors related to successful treatment with DDAVP. A consistent finding in this regard is older age, larger bladder capacity, and fewer pretreatment numbers of wet nights ([Butler et al., 1998](#); [Eller et al., 1997, 1998](#); [Folwell et al., 1997](#); [Neveus et al., 1999b](#); [Rushton et al., 1996](#)).

Urine osmolality parameters also have been extensively investigated as potential predictors of response to DDAVP. [Rushton et al. \(1995\)](#) found that DDAVP treatment is associated with a significant increase in nocturnal urine osmolality, as well as nocturnal diurnal osmolality ratios. Although responders tended to have higher urine osmolality than nonresponders, the differences did not reach statistical significance and the authors concluded that it therefore was not a reliable predictor of response. Similar results have been reported by other investigators ([Eller et al., 1997](#); [Folwell et al., 1997](#); [Neveus et al., 1999b](#)).

### Outcome and Follow-up

The natural history of primary enuresis must be taken into account in any treatment plan, whether it is primarily behavioral or pharmacologic. There is a high rate of spontaneous remission between the ages of 5 and 7 and again after age 12 years. Accordingly, the clinician might want to wait until after age 7 years before instituting pharmacologic treatment, unless other factors indicate otherwise. Similarly, the strong possibility of spontaneous remission should be considered in any positive treatment response after 10 or 11 years of age.

Pharmacologic studies have indicated that treatment with imipramine can result in three subtypes of response. There are true responders who have a sustained response, and there are also true nonresponders. There also is a surprisingly large group of transient responders. These children have an initial response to imipramine and then lose it over 2 to 3 weeks. When the dosage is increased by another 25 mg, they again respond for another 2 to 3 weeks. Eventually, the dosage required to maintain a response becomes prohibitive. Although long-term treatment with imipramine is not an option for these children, imipramine still can be used for brief, socially important periods, such as camp, because the initial response can be recaptured after a medication-free period ([Rapoport et al., 1980](#)).

There has been one large follow-up study that compared observation, imipramine, DDAVP, and the alarm system. Patients were weaned from therapy after 6 months. Continence was assessed at the 3-, 6-, 9-, and 12-month points of the protocol, so that the 12-month assessment would be 6 months after treatment ceased. Among the observation group (n = 50), only 6% were continent at 6 months and 16% at 12 months. Of the imipramine group (n = 44), 36% were continent at 6 months while still on medication, but this decreased to 16% at the 12-month assessment. The corresponding figures for DDAVP (n = 88) were 68% continent at 6 months, but only 10% at 12 months. The alarm system showed the best long-term effects, with 63% continent at 6 months, and 56% at 12 months ([Monda and Husman, 1995](#)).

### Areas for Future Research

As indicated by the new research reviewed previously, there has been a dramatic increase in research into the etiology and treatment of enuresis. Much of this has been stimulated by the recognition that DDAVP is an effective treatment for the disorder. Despite this new research, a definitive explanation that would link DDAVP efficacy with the pathophysiology of the disorder has not yet been proven, and there is no consistently reliable way to differentiate DDAVP responders from nonresponders, either retrospectively or prospectively.

This and other findings related to bladder physiology and anatomy suggest that we might ultimately be able to differentiate distinct phenomenologic subgroups that relate to treatment outcome.

The long-standing observation that enuresis has a hereditary basis has been advanced by the elucidation of genetic linkages in some large, multigenerational pedigrees. This research will likely continue to advance and may ultimately provide important information on clinical subtypes.

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#### CASE ILLUSTRATION

John is an 8-year-old boy with primary enuresis. He wets the bed an average of 3 to 4 nights per week. At age 5 years, his pediatrician found no psychological explanation for the enuresis. The family elected to wait on treatment for a few years to see if John would spontaneously remit. The family handled the wetting in a matter-of-fact manner, with John changing the bed each morning. However, the wetting was now beginning to become a social problem for John because it interfered with his plans to sleep overnight with friends and was an embarrassment at summer camp. Accordingly, the family sought treatment, and their pediatrician recommended the bell and pad method of conditioning. John did not rapidly respond to the bell and pad, and it was quite disruptive for the family because they lived in a small house and John shared a room with his younger brother. At this point, the pediatrician decided that medication might be warranted, and he referred the family to a child psychiatrist with whom he was familiar. After receiving medical clearance from the pediatrician and discussing side effects with the family, the child psychiatrist prescribed oral DDAVP 200 µg/day. John did not respond to this dosage but did respond rapidly at a dosage of 400 µg/day. The child psychiatrist would taper and discontinue the DDAVP every 3 months. During the first such tapering process, John developed breakthrough wetting at 200 µg, and the dosage was returned to 400 µg with good results. Three months later, the DDAVP was again tapered. On this occasion, there was no resumption of wetting as the DDAVP was decreased, and it appeared that John's enuresis had now spontaneously resolved.

#### Comment

*Was John's remission spontaneous or due to the DDAVP treatment?* There is no firm indication that a period of treatment with DDAVP leads to cessation of enuresis after the medication is removed, and relapse usually occurs on removal. Thus, John most likely had a spontaneous remission. However, there is one study that suggests that the remission rate after discontinuation of successful treatment with DDAVP is greater than could be anticipated from the spontaneous remission rate.

*If John had not responded to DDAVP, what would have been the next step?* The family could have been asked to try the bell and pad method for a longer time. Alternatively, the child psychiatrist could discuss with the pediatrician a possible trial of imipramine, which has success rates comparable with those of DDAVP.

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## ENCOPRESIS

### Definition

Encopresis is defined by DSM-IV ([American Psychiatric Association, 1994](#)) as the "repeated passage of feces into inappropriate places." The manual goes on to note that the soiling must occur at least once a month for at least 3 months and that the mental or chronological age of the child must be at least 4 years. Physical disorders must, of course, be ruled out. If there has been a period of fecal continence preceding the recurrence of soiling, it is classified as secondary encopresis.

The DSM-IV also denotes two subtypes of encopresis, which it labels as "with constipation and overflow incontinence" and "without constipation and overflow incontinence." The former category roughly corresponds to what has been referred to in the literature as *retentive encopresis*, whereas the latter corresponds to what has been known as *nonretentive encopresis*.

### Prevalence and Epidemiology

A study involving 8,863 children found a frequency of 1.5% among children between 7 and 8 years of age, with a male:female ratio of over 3:1 ([Bellman, 1966](#)). In the Isle of Wight Study, Rutter and colleagues found that 1.3% of boys between the ages of 10 to 12 years soiled at least once a month, with the corresponding figure for girls being 0.3%. That study also found a significant relationship between enuresis and encopresis ([Rutter et al., 1981](#)).

### Clinical Description

Encopresis has been classified in different ways. As noted, DSM-IV makes a distinction between primary and secondary encopresis and has added subtypes that denote the distinction between retentive and nonretentive encopresis. Retentive encopresis is characterized by a cycle of several days of retention, a painful expulsion, and another period of retention. While the fecal mass is growing, there may be leakage around the mass. The category of nonretentive encopresis applies to those children who simply do not control the expulsion of feces on either a psychological, physiologic, or combined basis.

[Hersov \(1985\)](#) has proposed three categories: (a) children who have adequate bowel control and volitionally deposit feces in inappropriate places; (b) children who either are unaware that they are soiling or are aware but unable to control the process; and (c) situations where the soiling is due to excessive fluid, which may be caused by diarrhea, anxiety, or the retentive overflow process described previously. The last mechanism is responsible for approximately 75% of this category.



## Etiology and Pathogenesis

There have been extensive investigations into the physiologic basis for encopresis. [Loening-Baucke \(1987\)](#) found that 56% of children with retentive encopresis were unable to defecate rectal balloons, and most of these children had abnormal contractions of the external anal sphincter. This study also had prognostic significance in that only 14% of those who were unable to defecate the rectal balloons had responded to treatment after 1 year, whereas 64% of those who could defecate the balloons recovered after 1 year. Similarly, only 13% of patients who were unable to relax the anal sphincter at initial evaluation were improved 1 year later, whereas the corresponding figure for those who could relax the sphincter was 70%. Interestingly, none of the patients who presented with an abdominal fecal mass at the time of the initial evaluation showed improvement 1 year later, regardless of ability to defecate the rectal balloons. Constipated children subsequently were compared with control subjects on a wide range of physiologic measures during the act of bearing down ([Loening-Baucke and Cruikshank, 1986](#)). These studies revealed that the act of bearing down led to decreased anal sphincter activity in 100% of control children, 58% of constipated children who were able to defecate a rectal balloon, and 7% of those constipated children who were unable to defecate the balloon. The latter group was significantly less likely to respond to conventional laxative treatment, and the authors concluded that the increased external sphincter activity could relate to their chronic fecal retention and encopresis. A companion study ([Loening-Baucke et al., 1987](#)) investigated the social competence and behavioral profiles of 38 children with encopresis and correlated these variables with the physiologic variables of anorectal manometric and electromyographic evaluations as they related to treatment outcome. The study found that social competence and behavioral rating scores were not significantly different between those boys who were or were not able to defecate the balloons. The behavioral problem ratings also were similar in both physiologic subgroups of girls. The social competence score of the girls who could not defecate the balloons was lower than that of those who could. The follow-up data indicated that the behavioral and social competence scores did not correlate with successful outcome at 6-month and 1-year follow-ups, but there was a significant negative correlation between positive outcome in the inability to defecate the balloon and the inability to relax the sphincter. Thus, the physiologic variables were predictive of outcome, and the psychological variables were not. A subsequent study by the same group with a similar design continued to demonstrate some predictive value of the balloon test, in that children with functional constipation and encopresis who were able to defecate the balloon were twice as likely to have recovered at 12-month follow-up. However, the author concluded that even though these results were statistically significant, the calculation of predictive value indicated that the defecation test could not, in and of itself, reliably predict recovery ([Loening-Baucke, 1996](#)).

A similar study at a different center concluded that a significant number of boys with encopresis have abnormalities of anorectal expulsion dynamics, but the researchers could not find abnormalities of anorectal sensory or motor function ([Wald et al., 1986](#)). [Sentovich et al. \(1998\)](#) specifically investigated pudendal nerve terminal motor latency in 23 children with encopresis, compared with 23 control subjects, and could find no significant difference. However, anal electromyography did indicate nonrelaxation of the external anal sphincter in 75% of the encopretic children, as opposed to 13% of the control subjects ( $p < 0.001$ ), as well as lower pressures at rest and with squeezing ( $p < 0.01$ ). Complementary abnormalities of anal sphincter function have been reported by others ([Catto-Smith et al., 1998](#); [Sutphen et al., 1997](#)).

Another approach has been to investigate potential involvement of hormones that affect gastrointestinal motility. [Stern et al. \(1995\)](#) measured plasma levels of gastrin, pancreatic polypeptide, cholecystokinin, motilin, thyroxin, estrogen, and insulin at several intervals after the administration of a standardized meal to 10 children with encopresis and the same number of matched control subjects. The authors reported significant differences for postprandial levels of pancreatic polypeptide, which peaked earlier and remained higher in children with encopresis, as well as a lower motilin response. However, the authors could not entirely rule out that their findings were not the result of chronic constipation, rather than the cause ([Stern et al., 1995](#)).

The dramatic nature of the soiling incident has naturally led to psychodynamic speculation. [Bemporad and Hallowell \(1987\)](#) have identified a "small number" of children with intractable encopresis for which they have proposed the term *chronic neurotic encopresis*. The characteristics of these children were (a) history of neurologic delay, (b) early or harsh bowel training, and (c) a distant father and a neurotic mother.

Environmental factors have been noted for some time and include the observations of [Freud and Burlingham \(1943\)](#), who noted a high frequency of soiling and wetting in children separated from their parents during World War II.

At least two studies revealed no correlation between social class and soiling ([Rutter et al., 1981](#); [Stein and Susser, 1967](#)). [Foreman and Thambirajah \(1996\)](#) specifically looked at associated psychopathology in boys with primary encopresis, compared with those with secondary encopresis. They found that the children with primary encopresis were more likely to have experienced developmental delays and to have associated enuresis, whereas those with secondary encopresis had experienced more psychosocial stressors and had higher rates of associated conduct disorder.

## Laboratory Studies

The physiologic studies described previously must be considered research investigations and not the representation of a usual and customary workup. However, they do suggest that a more detailed physiologic investigation than usually is done may be warranted. Usually, once the more obvious physiologic problems, such as Hirschsprung's disease, are ruled out, the problem is considered to be psychogenic.

The plain abdominal roentgenogram reveals evidence of fecal retention. In general, a positive rectal examination is sufficient to determine fecal retention, but a negative rectal examination does not rule it out, and in those cases, the abdominal roentgenogram can be helpful in establishing the diagnosis ([Rockney et al., 1995](#)).

One of the most important investigations may well be a thorough history that documents the frequency, nature, and circumstances of the soiling events in great detail. This history should be elicited both from the parents and from the child.

Psychological testing and evaluation are important in providing a thorough picture of the child, but it remains difficult to know if concomitant psychological problems are associated, causal, or secondary.

## Differential Diagnosis

The differential diagnosis of encopresis must take into account that the soiling can be either a symptom of another problem or the primary problem itself. For example, historically encopresis and enuresis have been reported to occur under stress in normal children and to remit when the stressor is removed. Similarly, in children who are retarded or significantly developmentally delayed, the encopresis may be only one expression of the primary problem. Children who are impulsive and hyperactive may have occasional episodes of encopresis simply because they do not attend to the stimuli until it is too late. Thus, the symptom of encopresis must be viewed in the context of the child's larger psychological and environmental profile. Strictly medical causes, such as Hirschsprung disease, stenosis of the rectum or anus, smooth muscle disease, and endocrine abnormalities, also should be ruled out.

## Treatment

The most widely accepted first line of treatment is one that encompasses educational, psychological, and behavioral approaches. As outlined by [Levine \(1982\)](#), this approach entails an initial meeting that is designed to educate both the parents and child about bowel function and to diffuse the psychological tension that may have developed in the family around the encopresis. This educational and psychological intervention is then followed by an initial bowel catharsis, after which the child receives daily doses of laxatives or mineral oil. There also is a behavioral component to the treatment, which consists of daily timed intervals on the toilet with rewards for success. A 78% success rate has been reported for this approach, without symptom substitution ([Levine and Bakow, 1976](#); [Levine et al., 1980](#)).

[Stark et al. \(1997\)](#) have replicated earlier work reporting the efficacy of group treatment with an educational and behavioral focus in conjunction with medical management for children who had not responded to medical management alone.

The adjunctive use of oral laxatives and conditional rectal cathartics also has been investigated ([Sprague-McRae et al., 1993](#)). Specifically, the authors compared the results obtained with children who were all treated with a high-fiber diet, initial bowel evacuation, behavior modification program, and random assignment to either oral laxatives ( $n = 24$ ) or conditioning rectal cathartics ( $n = 37$ ). Only 61 of 136 patients evaluated completed treatment, and thus there was a high dropout rate. No significant outcome difference was found between the two groups, and 87% continued in remission at 6- to 12-month follow-up. [Nolan et al. \(1991\)](#) used a random allocation design to compare combined treatment with laxatives and behavior modification ( $n = 83$ ) to behavior modification alone ( $n = 86$ ). At 12-month follow-up, 51% of the combined therapy group had at least one 4-week period without an encopretic episode, compared with 36% of the behavior modification group. After the authors excluded children with poor compliance, there was no statistical difference between groups, although the authors maintained that from a clinical perspective,

use of laxatives combined with behavior modification was superior to behavior modification alone.

[Loening-Baucke \(1990\)](#) has expanded on the pathophysiologic studies described earlier by exploring the utility of biofeedback training in children with abnormal defecation dynamics. Specifically, patients (ages 5 to 16 years) were randomly assigned to traditional medical treatment alone (n = 19) or conventional treatment plus up to six biofeedback sessions. Eighty-six percent of the biofeedback group had learned normal defecation dynamics at the conclusion of biofeedback treatment. At 7-month follow-up, 77% of the biofeedback group had normal defecation dynamics, as opposed to only 13% of the conventionally treated ( $p < 0.01$ ). The improvement in defecation dynamics was correlated with clinical improvement at 12 months (16% with conventional treatment and 50% with biofeedback;  $p < 0.05$ ). Similar successful results were reported in a European study ([Benninga et al., 1993](#)).

A longer follow-up study ( $4.1 \pm 1.5$  years) compared the long-term outcome in 129 children with constipation and encopresis, as well as abnormal defecation dynamics, who were treated with conventional treatment, with 63 children who received additional biofeedback training that was directed toward normalizing the defecation dynamics. The results indicated that both groups showed similar rates of improvement (86% of the conventionally treated and 87% of the biofeedback group). At long-term follow-up, complete recovery was documented in 62% of the conventionally treated, 50% of those who had achieved success with the biofeedback treatment, and 23% of those who had not responded to biofeedback treatment. Length of time at follow-up was significantly related to recovery for the group as a whole ( $p < 0.01$ ), suggesting that the natural history of the disorder is to move toward continence. The author concluded that biofeedback treatment cannot be demonstrated to be statistically superior to conventional treatment ([Loening-Baucke, 1995](#)).

A subsequent study by another group with a somewhat similar design reached the same conclusion ([Nolan et al., 1998](#)). However, research into biofeedback treatment continues using a newly developed portable apparatus ([Griffiths et al., 1999](#)), and as an adjunctive treatment in combination with other behavioral strategies and laxative therapy ([Cox et al., 1998](#)).

More intensive psychotherapeutic intervention may be required for those children with intractable encopresis who appear to fit the psychological profile described by [Bemporad and Hallowell \(1987\)](#), where it seems clear that interpersonal–psychodynamic factors are perpetuating the problem.

Pharmacologic treatment with imipramine also has been reported as useful for encopresis. There have been 15 reported cases of children with encopresis responding to imipramine, which have been described in six papers ([Abrahams, 1963](#); [Connell, 1972](#); [Gavanski, 1971](#); [Geormaneanu and Voiculescu, 1980](#); [Siomopoulos, 1976](#); [White, 1977](#)). All but three of the reported subjects are male. In general, the therapeutic effect occurred within a few days to 2 weeks. The doses of imipramine reported are relatively low, in the 25- to 75-mg range. There also is a similar positive case report involving amitriptyline treatment of a 6-year-old ([Dosssetor et al., 1998](#)). There is one double-blind study demonstrating the effectiveness of the prokinetic agent cisapride (Propulsid) for encopresis related to constipation (Nurko et al., 2000). However, this agent has been removed from the market in the U.S. by the F.D.A., due to serious side effects.

### Outcome and Follow-up Data

The 78% success rate described by Levine suggests that most children will respond to a relatively innocuous approach that involves educational, behavioral, and physiologic components, as do the follow-up data of [Loening-Baucke \(1995\)](#). In general, the longer-term follow-up studies consistently indicate that the passage of time is an important contributor to remission of the disorder ([Loening-Baucke, 1995](#); [Rockney et al., 1996](#)). The epidemiologic data also indicate that the effects of maturation will provide a significant number of spontaneous remissions from year to year. The evaluation of any long-term intervention such as psychotherapy should take this factor into account. All but a few children will have either responded to treatment or spontaneously remitted by age 16 years, and persistence beyond that age is quite unusual ([Rex et al., 1992](#)).

### Areas for Future Research

This review suggests that encopresis is an excellent paradigm for assessing the relative impacts of biological, psychological, and social factors. For example, do the physiologic findings described previously represent a constitutional vulnerability, or are they the result of the effects of chronic constipation on the bowel? The symptom of encopresis in its various presentations can be a fruitful area of research for those interested in elucidating the interrelation between mind, body, and culture in children.

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#### CASE ILLUSTRATION

Peter is a 10-year-old boy with a 2-year history of encopresis occurring at a frequency of once to twice per week. The feces are formed, and the episodes usually occur at home after school. Frequently he hides the soiled underclothes, only to have his parents find them around the house a few days later.

Peter's biological parents divorced 3 years ago. His father was awarded custody of Peter and his younger brother and sister. His father remarried 2 years ago. Peter and his stepmother have been at odds since she moved in.

Treatment with the conventional catharsis and bowel-training regimen was not successful. The family sought treatment with a child psychiatrist. It was apparent that there was considerable tension between Peter's father and stepmother. His father tended to be removed and distant, which greatly angered his stepmother. The couple tended to blame all of their problems on Peter's encopresis. Peter was treated with once-weekly individual and family psychotherapy. As an adjunctive measure, imipramine was prescribed for the encopresis because it was thought that removing the symptom would help to clarify the other significant family issues. Baseline data on the frequency of encopresis were collected for 1 month. After this, imipramine was begun at a dose of 25 mg and titrated up to a dose of 75 mg. Imipramine was effective, and the frequency of encopresis declined from five times per week to two times per week. However, approximately 6 weeks later, the frequency of soiling began to escalate back to its pretreatment levels. In the family sessions, it emerged that Peter's stepmother had become very inconsistent in giving him the imipramine. An attempt was made to have his father administer the medication, but he refused.

Even though the encopresis did recur, the response to medication coupled with the parents' apparent sabotaging of the treatment led to a fuller clarification of the family dynamics. The stepmother became more forceful in expressing her hatred of Peter and eventually took the position that either Peter would leave or she would. The father capitulated on this demand, and Peter went to live with his paternal uncle's family. One month after the move, the encopresis completely remitted. Because the family lived nearby, Peter was able to maintain close contact with his father and siblings.

#### Comment

Given the relatively high success rate with the bowel training approach, why did Peter not respond? The literature clearly shows that children who have more psychological disturbance or are from more troubled families are less apt to respond to this intervention.

What can be inferred from Peter's positive response to imipramine? This would suggest that imipramine's efficacy in encopresis could be a nonpsychotropic effect. Alternatively, it could be argued that Peter was concomitantly depressed and that the therapeutic effect on the encopresis was secondary to an antidepressant effect. It also is quite likely that the response was a placebo effect because there have not been large, placebo-controlled, double-blind studies of imipramine's efficacy for encopresis.

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## 57 FIRESETTING

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Firesetting behavior in youngsters is the result of a complex interaction of individual, social, and environmental factors. There is a range of firesetting activities representing increasingly severe levels of psychopathology. Although current epidemiologic information is somewhat thin, there are studies suggesting that youngsters 18 years of age and younger represent a significant proportion of individuals involved in firesetting and arson-related behaviors. Several theoretical perspectives present hypotheses regarding the causative factors of firesetting. Research supporting these theories suggests that there is a common group of clinical features describing pathologic firesetting. Swift diagnosis of firesetting behavior can result in successful intervention and remediation. There are many clinical reports suggesting the availability of a wide variety of therapeutic methods that are effective in abating pathologic firesetting behavior in youngsters.

### DEFINITION OF TERMS

The development of fire behavior in youngsters can be viewed as following a naturally occurring sequence of psychosocial phases ( [Gaynor and Hatcher, 1987](#); [Grolnick et al., 1990](#); [Kafry et al., 1980](#)). There are at least three sequential phases of fire behavior: fire interest, firestarting, and firesetting. These categories of fire behavior represent increasing levels of involvement with fire. By experiencing each of these sequential phases, most children learn age-appropriate, fire-safe behaviors. However, for a percentage of youngsters, the influence of risk factors, such as deficits in emotional functioning, difficulties in the family environment, or frequent stressful life events, affect their development and produce such behaviors as unsupervised firestarting, repeated, intentional firesetting, and arson.

Fire interest is experienced by most children 3 to 5 years of age ( [Gaynor and Hatcher, 1987](#); [Hanson et al., 1995](#); [Jackson et al., 1987](#)). There are several ways in which youngsters express their interest in fire. For example, they may ask questions such as, "How hot is fire?" and "What makes fire burn?" Or they may incorporate fire into their play by wearing fire hats, playing with toy fire trucks, and cooking food on their toy stoves. Fire interest represents one of the many curiosities youngsters have about the physical properties of their natural environment.

Firestarting occurs when children experiment with matches or other ignition sources. This type of behavior emerges in children between the ages of 5 and 9 years, and it is observed predominantly in young boys ( [Gaynor and Hatcher, 1987](#); [Showers and Pickrell, 1987](#)). When this experimentation takes place under controlled and supervised conditions, the result is the development of competent, fire-safe behaviors. For example, if youngsters request and are given permission to light the candles on their birthday cake, they are learning the conditions under which it is safe to strike a match and light candles. Children can be given the appropriate responsibility for fire at specific ages, although this responsibility should not be given prematurely ( [Grolnick et al., 1990](#)). Teaching children the importance of fire in their environment and helping them to gain mastery and control over it are benefits of participating in supervised fire-related activities.

Unfortunately, a greater majority of youngsters participate in unsupervised firestarting activities. Studies estimate that over 60% of children interested in fire engage in at least one unsupervised firestarting incident ( [Cole et al., 1986](#); [Gaynor and Hatcher, 1987](#)). In addition, many youngsters will not admit to having participated in these firestarting activities unless specifically questioned about them ( [Kafry et al., 1980](#)). Most unsupervised firestarting is a single-episode event primarily motivated by curiosity. The resulting fires are either accidental or unintentional, and if a fire becomes out of control, youngsters make serious attempts either to extinguish the fire themselves or go for help ( [Cole et al., 1986](#); [Gaynor and Hatcher, 1987](#); [Kafry et al., 1980](#)). Although unsupervised firestarting incidents are unplanned and occur only once or twice for most youngsters, it is estimated that although the probability is fairly low that first-time firestarts will result in a significant fire, this probability increases significantly for subsequent firestarting activities ( [Lewis and Yarnell, 1951](#)). Hence, although unsupervised firestarts can be the result of innocent curiosity or accidental behavior, the risks of starting a significant fire and the resulting consequences exceed the benefits of the learning that may result from the youngster's experimentation.

By 10 years of age, most children have learned the rules of fire safety and prevention and are capable of engaging in age-appropriate firesetting activities such as helping to light the family barbecue or building a campfire ( [Cole et al., 1986](#); [Gaynor and Hatcher, 1987](#)). If adequate fire-related experiential and educational efforts have occurred at home and in school, youngsters will have achieved a sense of competence and mastery over a powerful and fascinating aspect of their physical environment. However, for a number of children, what begins as one or two unsupervised firestarting incidents leads to repeated, intentional firesetting behavior. Hypothesized explanations for the development of pathologic firesetting follow in a subsequent section. [Table 57.1](#) presents a description of the factors distinguishing unsupervised firestarting from pathologic firesetting.

Factor	Firestarting	Firesetting
History	Single episode	Recurrent
Method	Unplanned	Planned
Motiv	Accidental	Intentional
Ignition	Available	Acquired
Target	Nonspecific	Specific
Behavior	Extinguish fire	Run away

**Table 57.1. Factors Distinguishing Firestarting and Pathologic Firesetting**

Pathologic firesetting is characterized by a history of multiple ignitions that take place over at least a 6-month period ( [Gaynor and Hatcher, 1987](#)). The nature and extent of these ignitions may vary, ranging from parents finding burned candles concealed in their youngsters' rooms to fires requiring fire department suppression. The ignitions usually are planned as opposed to being a spur-of-the-moment, impulsive activity ( [Gaynor and Hatcher, 1987](#); [Icove and Estep, 1987](#)). Ignition sources, such as matches and lighters, often are searched for, acquired, and concealed until they are needed ( [Benians, 1981](#); [Gaynor and Hatcher, 1987](#); [Icove and Estep, 1987](#); [Kolko and Kazdin, 1986](#)). The firesetting activity usually takes place in a concealed or isolated area in or near the home, where there is little possibility of immediate detection by an adult or authority figure ( [Icove and Estep, 1987](#)). When a fire is set, there often is an attempt to gather flammable and combustible materials, such as old newspapers and rags, or chemicals, such as paints and alcohol, to use as aids in spreading the fire ( [Gaynor and Hatcher, 1987](#); [Icove and Estep, 1987](#)). These fires can be motivated by a variety of reasons, including revenge and anger, attention, malicious mischief, watching the fire burn, and, in a small number of adolescent cases, profit ( [Benians, 1981](#); [Gaynor and Hatcher, 1987](#)). The target of the firestart often has significant meaning for the firesetter because of the emotional importance of the fire ( [Canter and Fritzon, 1998](#); [Gaynor and Hatcher, 1987](#)). For example, if a youngster has set fire to the cushions of his mother's favorite chair, then it is likely that one of the primary motivations for the firesetting lies in the nature of the relationship between the mother and her child. After the fire, youngsters usually do not voluntarily admit to their involvement in the ignition ( [Gaynor and Hatcher, 1987](#)). If the fire is out of control, rather than calling for help, they

are likely to run away to a safe spot, sometimes to watch the fire burn and await for the arrival of the fire department ( [Gaynor and Hatcher, 1987](#); [Icove and Estep, 1987](#)). Although individual case reports reveal variations in ignition patterns, these are the predominant characteristics that represent pathologic firesetting behavior.

## EPIDEMIOLOGY

Information on the prevalence of firesetting behavior in youngsters is derived from four general areas of study. The first is the annual fire cause and origin rates in the United States. There are two primary resources for this annual data—the U.S. Fire Administration's National Incidence Reporting System and the National Fire Protection Association's United States Arson Trends and Patterns. In 1997, children started 65,060 fires, causing 284 deaths, 2,158 injuries, and \$283.3 million in direct property damage ( [Hall, 2000](#)). Children 5 years of age and younger were responsible for three-fourths of the deaths caused by child-set fires ( [Hall, 2000](#)). Most often these victims were the younger siblings or playmates of the children who started the fires. Although firesetting is the second leading cause of death (second to automobile accidents) for children and adolescents ages 6 to 14 years, it is the number one cause of death (accounting for 33% of the total) for preschoolers ( [Hall, 2000](#)). Hence, very young children are not only most likely to start fires, but they also are most likely to be their victims.

The second source of information relates to the prevalence of unsupervised firestarting and firesetting in populations of school-age children. One study interviewing kindergarten, second, and fourth grade children and their parents (n = 139) from three randomly selected schools reveals that 60% of the children admitted to engaging in unsupervised firestarting and that 77% of them reported to be present when their friends participated in the same behavior ( [Gaynor and Hatcher, 1987](#)). Of the unsupervised firestarting group (n = 83), 37% of the children admitted to one incident, 40% said they started fires two times, and 23% disclosed they had participated in unsupervised firestarting more than twice ( [Gaynor and Hatcher, 1987](#)). Of the group reporting to firestart more than once (n = 52), 55% of the children admitted to burning purposely items such as grass, leaves, rugs, clothes, and paper products ( [Gaynor and Hatcher, 1987](#)). Of these children, 23 were boys, and 15 were girls; boys were more likely to burn items and to set fires outside the home, whereas girls were more likely to do it indoors. None of these fires required firefighting suppression ( [Gaynor and Hatcher, 1987](#)). In addition, 47% of the parents reported that they had engaged in unsupervised firestarting during their childhood ( [Gaynor and Hatcher, 1987](#)). Another study surveying school-age youngsters in grades one through eight (n = 77) indicated that 75% of their sample reported knowing someone who had firestarted with matches or lighters, 58% of the youngsters had actually witnessed unsupervised firestarting, and 38% admitted to engaging in at least one unsupervised firestart ( [Cole et al., 1986](#)). Of this sample, 3% of the firestarting incidents resulted in fires requiring fire department intervention. Hence, these studies offer preliminary findings suggesting that most school children may be exposed to or participate in unsupervised firestarting activities, with fortunately few of these incidents resulting in major fires.

The third type of epidemiologic data examines the prevalence of firesetting behavior among psychiatric populations. An early study indicated that 2% of the evaluations in an outpatient child psychiatry clinic of a large voluntary hospital presented with firesetting as the primary complaint, and 3% of the evaluations conducted in a suburban private practice were firesetting cases ( [Vandersall and Weiner, 1970](#)). Two studies confirm these estimates. One study reports that between 1973 and 1981, 2.45% of their outpatient clinic population (104 of 4,242 children) presented with definite or marked firesetting behavior ( [Jacobson, 1985a](#)). This study also characterized firesetting as a subgroup of the conduct disorder diagnosis. Firesetters differed significantly from a matched group of youngsters with conduct disorder in that they demonstrated more antisocial, aggressive, and destructive behaviors. A second study shows that 2.1% of 100 consecutive referrals to an adolescent forensic mental health service presented with significant firesetting behavior ( [Jasper et al., 1998](#)). When fire behavior in psychiatric populations is examined, a slightly different picture emerges. [Kolko and Kazdin \(1988\)](#) delineated five categories of fire behavior—match play, participation in at least one unsupervised firestart, setting fire more than twice, setting a fire causing serious damage, and setting a fire requiring firefighter suppression—occurring in psychiatric outpatient and inpatient populations. Their results indicate that the prevalence rates for match play and participation in at least one unsupervised firestart were 24% and 19%, respectively, for outpatients and 52% and 35%, respectively, for inpatients ( [Kolko and Kazdin, 1988](#)). Although these outpatient rates are somewhat lower than previously cited rates in a sample of school children (60% for match play and 37% for firestarting) ( [Kafry et al., 1980](#)), the rates for inpatients are more closely aligned ( [Kafry et al., 1980](#); [Kolko and Kazdin, 1988](#)). However, a further comparison of the fire involvement rates from these two studies indicates that more youngsters in the outpatient and inpatient groups were involved in recurrent firesetting (outpatients, 11/21; inpatients, 18/25) than those children sampled from the school population (19/83) ( [Kafry et al., 1980](#); [Kolko and Kazdin, 1988](#)). In addition, whereas none of the firestarts in the school sample resulted in significant fires, 13 of 22 fires started by outpatients resulted in serious damage, and 9 of 24 fires started by inpatients resulted in serious damage ( [Kafry et al., 1980](#); [Kolko and Kazdin, 1988](#)). Therefore, these studies suggest that although the proportion of psychiatric cases presenting with the primary complaint of firesetting remains in range of 2% to 3%, the frequencies with which outpatient and inpatient populations engage in recurrent fire behaviors, some of which lead to serious and damaging fires, may be remarkably higher.

The final type of epidemiologic information on firesetting comes from the arson arrest records of juveniles. Youngsters starting fires that warrant firefighting suppression and that result in significant property damage, loss, or personal injury are at risk for being investigated by law enforcement for the crime of arson. If probable cause can be demonstrated, that is, if existing evidence indicates that firestarting is the result of a malicious and willful intent to destroy by fire, and youngsters have reached the age of accountability, then they can be arrested for arson. During the last two decades, juveniles have accounted for 38% to 56% of all arson arrestees ( [Federal Bureau of Investigation, 1979–1999](#)). In 1998, arson had the highest juvenile arrest rate (52%) of all violent and property crimes indexed by the [Federal Bureau of Investigation \(1999\)](#). Demographic information indicates that most (85%) of these arrested juveniles are white and male ( [Federal Bureau of Investigation, 1979–1999](#)). In addition, most of these youngsters are between the ages of 15 and 18 years (in 1995, 63% were older than 15 years of age) ( [Federal Bureau of Investigation, 1979–1999](#); [Hall, 2000](#)). Hence, youngsters involved in firesetting not only pose significant problems for the mental health community, they represent a major burden for the law enforcement and juvenile justice systems.

## CLINICAL DESCRIPTION

Information on the clinical features of firesetting youngsters primarily comes from descriptive studies, with a smaller proportion of data emerging from empirical investigations that meet rigorous scientific standards of acceptability. Therefore, the essential clinical features reportedly associated with firesetting must be viewed as representing trends rather than empirically verified patterns of behavior.

There is a growing amount of clinical speculation suggesting motivational subtypes to describe firesetting behavior ( [Canter and Fritzon, 1998](#); [Gaynor, 2000](#); [Grolnick et al., 1990](#); [Kocsis et al., 1998](#)). Although these motivational subtypes are intuitively useful, they offer a relatively singular explanation of the development of firesetting behavior in children and they lack empirical investigation and support. They are mentioned because they provide a useful starting point to understanding firesetting behavior. The four motivational subtypes are curiosity—fires set as a result of experimentation or accident; troubled—fires set as an expression of psychological distress; delinquent—intentionally set fires for the purpose of control, destruction, or harm; and pathologic—fires set because of severe mental disorder. Although these subtypes present a conceptual framework for describing four general motives for firesetting behavior, these motives have not yet been empirically validated, nor have they been linked to the clinical features that describe different types of children and adolescents who are firesetters.

It has been proposed that the clinical features of firesetting can be organized into three major categories: individual characteristics, social circumstances, and environmental conditions ( [Fineman, 1980, 1995](#); [Gaynor, 2000](#); [Gaynor and Hatcher, 1987](#)). Each of these categories comprises specific risk factors that predict the essential features of firesetting youngsters. In addition, there is a body of research supporting the notion that firesetting youngsters can be divided into two major age groups, younger children averaging 8 years and adolescents 13 years of age and older ( [Jacobson, 1985a, 1985b](#)). It is hypothesized that the bimodal nature of this age distribution distinguishes two different classes of firesetters, a younger group whose clinical features and motives for firesetting differ significantly from those of the older group ( [Jacobson, 1985a, 1985b](#); [Kolko, 1988](#)). Given these general assumptions, [Table 57.2](#) and [Table 57.3](#) outline the essential clinical features describing these two age groups of firesetting youngsters.

Feature	Description
<b>Individual characteristics</b>	
Demographic	Residence in young (age range 10 to 15) or older (age range 16 to 18) areas; living in a high-risk area; low socioeconomic background
Intelligence	Normal to above average intelligence; high IQ; high IQ; high IQ
Spontaneity	A greater number of reported fires; more frequent and varied types of fires; more frequent and varied types of fires
Proximity	Overwhelming feelings of anger and aggression; contact with or proximity to highly flammable materials
Motivation	General interest in fire; curiosity; desire to test limits; desire to cause damage; desire to cause damage; desire to cause damage
<b>Social circumstances</b>	
Family	Engagement in fire with or without other children; living in a high-risk area; living in a high-risk area; living in a high-risk area
Peer	Socially isolated; contact with or proximity to highly flammable materials; contact with or proximity to highly flammable materials
School	Peer pressure; performance; evidence of learning disabilities; academic achievement; academic achievement
<b>Environmental conditions</b>	
Availability of materials	Specific materials used for fire; availability of materials
Accompanying behavior	The act of firesetting represents the emotional release of anger, rage, and aggression
Consequences	Reactivity to both immediate positive reinforcing properties of attention and effect with the parents; negative outcomes if property is lost and punishment



**Table 57.2. Clinical Features of Pathologic Firesetters, Children 12 Years of Age and Younger**

Feature	Description
<b>Individual characteristics</b>	
Demographics	The majority are white boys. They all seem of average intelligence from middle socioeconomic backgrounds.
Intelligence	Normal levels of intelligence, but long histories of academic failure.
Appearance	A higher than average number of accidents starting in plastic models, higher levels of animal abuse, repetitive enuresis, general conduct and conduct problems.
Enuresis	There is a lack of emotional depth and restricted capacity of expression. Firesetting appears to be a coping strategy and aggressive behavior of defiance and defiance of the fire with a common aim: to seek a difficult separating episode of full fire burning.
Behavior	Violent, impulsive, and defiant are typical behaviors coupled with higher than average levels of fire setting. A specific strategy of early withdrawal of contact observed from the moment of fire ignition and dependence on others to extinguish the fire.
<b>Social environment</b>	
Home	Single-parent homes where the father is absent or the mother is over-protective. There is a high degree of marital discord, severe discipline and superior, poor communication, parental alcoholism and maternal depression. There is a history of the water patterns of early firesetting (see text).
Peer	Isolation and withdrawal from the company of other children and non-firesetting peers.
School	Characterized by poor academic achievement, failure to advance in school grades and conduct and behavioral problems with frequent absences and suspensions.
<b>Developmental conditions</b>	
Attachment issues	The same group supports and encourages firesetting, which often is the result of enuresis or incontinence before setting, with the encouragement of others to set a fire.
Accompanying behaviors	Other antisocial and disruptive behaviors such as alcohol consumption, self-harm, and violence towards others at the same time as or before firesetting in the company of other children or in the home.
Consequences	The immediate, reinforcing properties of firesetting include the resulting attention from family, friends, and the fire department. There is a tendency for a continuation of a cycle of the negative outcomes of property loss and destruction.

**Table 57.3. Clinical Features of Pathologic Firesetters, Adolescents 13 Years of Age and Older**

### Firesetting Children

Demographic information indicates that young firesetting children averaging 8 years of age are predominantly boys coming from a mixed socioeconomic background (Kolko et al., 1985; Showers and Pickrell, 1987). Most studies suggest that these youngsters are of average intelligence (Kolko et al., 1985; Ritvo et al., 1982). However, there are widespread reports indicating that firesetting children typically do not perform well in elementary school, and this finding may be related to a handful of studies suggesting that these youngsters have a higher-than-average incidence of learning disabilities (Kuhnley et al., 1982; Vandersall and Weiner, 1970).

There are scattered studies in the literature pointing to certain types of experiences that occur in the lives of these children that may have a relationship to their firesetting behavior. For example, one study suggests that firesetting children between the ages of 6 and 10 years have a greater number of chronic physical illnesses, including allergies and respiratory problems, than their nonfiresetting counterparts (Siegelman and Folkman, 1971). No attempts have been made to replicate these findings, although there is a hypothesized relationship between chronic illness and the development of violent behavior in children (Group for the Advancement of Psychiatry, 1999). There is a continuing controversy regarding the relationship between enuresis, firesetting, and delinquent behavior, with some studies suggesting that childhood enuresis and firesetting predict later arson arrests and convictions (Lewis and Yarnell, 1951; Repo et al., 1997), whereas other studies indicate no such relationship (Heller et al., 1984; Jacobson, 1985a; Showers and Pickrell, 1987). Also, some studies show a significant relationship between abuse, neglect, and firesetting behavior in children (Jasper et al., 1998; Wooden and Berkey, 1984; Yeo, 1998). The specific nature of the relationship between experienced abuse, neglect, and firesetting behavior remains to be explained both clinically as well as empirically, although one hypothesis suggests that firesetting is an inappropriate strategy for coping with an adverse environment or a method for sending a "smoke signal" to indicate psychological distress (Gaynor and Hatcher, 1987; Yeo, 1998).

The emotional style of these children has been described by clinical studies as one in which there appears to be marked difficulty in modulating feelings of anger and revenge (Bumpass et al., 1983, 1985; Sakheim and Osborn, 1999; Sakheim et al., 1991). It has been suggested that not only are these children unable to recognize or understand these types of feelings when they occur, but they do not have the experience nor skill to express them in socially acceptable ways (Bumpass et al., 1983, 1985). Consequently, they engage in firesetting as a means of expressing accumulated feelings of displaced anger, revenge, and aggression (Bumpass et al., 1983, 1985). This is partially substantiated by clinical work suggesting that the targets of young children's firestarts often are the objects and possessions of those to whom their anger is directed (Gaynor and Hatcher, 1987). For example, there have been clinical reports of youngsters who admit to setting their mother's bed on fire because they were angry with her (Minuchin, 1974). The primary justification of this emotional style comes only from clinical observations, however, and further empirical work needs to be pursued to verify these findings.

The behavior pattern of firesetting children has been characterized as overactive, impulsive, and mischievous (Babinski et al., 1999; Jacobson, 1985a; Sakheim and Osborn, 1999; Sakheim et al., 1991). Although the early documentation of the interrelationship of the behavioral triad of enuresis, cruelty to animals, and firesetting has failed to hold up under more recent scrutiny (Fine and Louie, 1979; Heller et al., 1984; Jacobson, 1985a; Showers and Pickrell, 1987), other recent work suggests there may be a relationship between animal abuse and property offenses, including firesetting (Arluke et al., 1999; Sakheim et al., 1991). Clinical reports indicate young firesetters have difficulty controlling their tempers and have histories of destroying their toys and other personal objects in fits of anger and rage (Gaynor and Hatcher, 1987). This behavioral pattern is fairly consistent with the previously described emotional style of accumulated anger and aggression typically being discharged in a socially inappropriate and unacceptable manner. Clearly, there remains a great deal of work to be done at arriving at a detailed description of the linkages between the emotional and behavioral styles of firesetting children.

There is a growing amount of literature indicating that young firesetting children come either from single-parent homes or from families where one of the two parents, typically the father, is absent for long periods (Fine and Louie, 1979; Gruber et al., 1981; Stewart and Culver, 1982; Vandersall and Weiner, 1970). Studies suggest that, when the family is intact, there appears to be a higher-than-average amount of marital discord (Kazdin and Kolko, 1986; Kolko and Kazdin, 1990). Patterns of family interaction for firesetting children have been characterized by a greater use of overly harsh methods of discipline, including corporal punishment, and an unaffectionate, distant, negative, and conflicted environment (Jacobson, 1985a; Siegelman and Folkman, 1971; Vandersall and Weiner, 1970; Yeo, 1998). In addition, there have been reports that families of firesetting children tend to exhibit more verbally and physically aggressive patterns of family interaction than families of nonfiresetting children (Patterson, 1975; Patterson et al., 1998). Finally, there are a number of studies suggesting that parents of firesetting children have significant psychiatric histories, including one or more family members carrying a diagnosis of antisocial personality, alcoholism, or depression (Group for the Advancement of Psychiatry, 1999; Gruber et al., 1981; Kazdin and Kolko, 1986; Stewart and Culver, 1982). Although much of this information on family patterns comes from clinical impressions, it represents a strong starting point for further empirical work.

There is somewhat limited information on the behavior of firesetting children in social situations. Preliminary work suggests that these children have a great deal of trouble establishing and sustaining meaningful interpersonal relationships (Heath et al., 1985; Vandersall and Weiner, 1970). In addition, studies have shown that when firesetting youngsters find themselves in social situations, they exhibit poor judgment and are unable to plan and anticipate outcomes (Bullis et al., 1998; Sakheim and Osborn, 1994; Sakheim and Osborn, 1999; Sakheim et al., 1991). The degree to which these youngsters feel detached, alone, and isolated may have an effect on their need to gain attention and recognition through firesetting. This is somewhat verified by the findings that young children typically firestart alone rather than in the company of friends (Benians, 1981). If youngsters gain recognition from their peers as a result of their firesetting, then their involvement with their peer group not only reinforces their firesetting behavior, but increases the likelihood that it will recur (Fineman, 1995; Patterson et al., 1998). These observations regarding their behavior in social relationships are speculative, and until more systematic work is undertaken, they are presented only as possible clues for further investigation.

There is evidence indicating that elementary school children with histories of firesetting experience significant academic and behavior problems in school (Bullis et al., 1998; Gruber et al., 1981; Kuhnley et al., 1982; Showers and Pickrell, 1987; Vandersall and Weiner, 1970). The higher proportion of learning disabilities experienced by firesetting children may account partially for this poor academic showing (Showers and Pickrell, 1987). In addition, because some firesetting children carry a diagnosis of hyperactivity, which sometimes is accompanied by the feature of shortened attention spans, they may have difficulty concentrating on and completing their assigned work (Gruber et al., 1981; Loeber et al., 1998). As a result of their academic failures, these children may feel frustrated and angry and they may act out their feelings, thereby becoming conduct and behavior problems in school. Hence, these studies suggest that, for a variety of reasons, firesetting children are not likely to have successful and rewarding experiences in school.

It has been hypothesized that there are immediate environmental conditions that set the stage for firesetting behavior in young children and reinforce the behavior once it has occurred. It has been suggested that there are antecedent stressors that take place in the lives of these youngsters and that trigger particular emotional reactions (Fineman, 1980, 1995; Gaynor and Hatcher, 1987). Specifically, these antecedent stressors trigger accumulated feelings of anger and revenge experienced by these children. The act of firesetting is motivated by the emotional release of displaced anger and aggression or by feelings of revenge. Firesetting holds both the

positively reinforcing properties of attention and effect, as well as the negative outcomes of property loss, injury, and punishment. Although intuitively interesting, these assumptions have yet to be examined in any systematic fashion, either through clinical observation or empirical investigation.

The following case example is presented for the purpose of illustrating the clinical features of children exhibiting pathologic firesetting behavior.

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#### CASE ILLUSTRATION

Frank is a 10-year-old who lives with his mother and his 11-year-old sister in a lower-middle income urban area. Frank's mother and father have been divorced for 8 years. Frank's mother had been living with a man who, she says, was very generous to her family. He liked to spend time with her children, but he could be extremely rigid and overly punitive, particularly when Frank did something that displeased him. Recently, the mother separated from him because he physically abused her. Frank had witnessed this abuse several times and also may have been a victim. However, when asked, both mother and Frank deny that any of the physical abuse was directed at Frank.

During the time of the separation, Frank's mother began to notice some behavior problems in her son. His ability to concentrate dramatically decreased, and reports came from school that he was involved in fights with his peers. At home, Frank had frequent temper outbursts during which he would become so angry that he would destroy his toys. Also, Frank was verbally aggressive and belligerent toward his sister. He was being generally disruptive both at school and at home. His mother reported that this was unusual behavior for Frank and that she was very concerned for his well-being.

Frank's mother noted that he had been curious about fire since the age of 3 years, when she found him playing with matches in their kitchen. At that time, she taught him how to strike a match correctly. Together they would practice striking matches, blowing them out, and throwing them in the fireplace. Frank was told never to play with matches. He also was encouraged to come to his mother if he ever felt the urge to strike a match, and they would do it together. Despite these rules about firestarting, during the past few weeks, Frank's mother found some scrapes of burned newspaper in their fireplace. One afternoon she arrived home early from work in time to see Frank rolling up pages of newspaper, lighting them, and throwing them into the fireplace. She became angry, describing the possible consequences of what could happen if the flaming newspaper accidentally caught the house on fire.

Frank's most recent fire episode involved igniting his model airplane on the kitchen stove and running with it to the living room fireplace. Unfortunately, he did not reach the fireplace in time, and the burning airplane fell on the carpet, starting a small fire. Frightened and confused, Frank ran out of the house screaming for help. Luckily, his mother was nearby and heard his screams. She and Frank ran back to the house and together they were able to extinguish the fire.

#### Comment

Frank's firesetting episodes are typical of this age group in that his unsupervised firestarts occur alone and in his home. Despite his mother's attempts to be firm and constructive in teaching him fire safety rules, he continues to engage in repeated and intentional firesetting behavior. The question becomes, Why has the frequency of his firestarting increased and intensified at this time? There may be several reasons for Frank's apparent preoccupation with firesetting. First, there could be a relationship between his fire behavior and the discord and separation in his family. Children with family histories of divorce, neglect, or physical or sexual abuse may firestart to express their distress. Frank's firestarting may be his way of expressing his inner turmoil about his family situation. Second, he could be testing the limits of his mother's authority. Third, firesetting may be Frank's way of asking for more attention from his mother. Finally, Frank's fire behavior may represent a "cry for help." Frank may be experiencing general psychological distress, and he may not have the capability of understanding or expressing his pain and conflict. Firesetting becomes his method for calling attention to his emotional difficulties and his way of asking for help.

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### Firesetting Adolescents

There are far fewer systematic studies focused on describing the clinical characteristics of firesetting adolescents than there are studies describing firesetting children. Demographic information indicates that it is 10 times more common for adolescent boys than girls to firestart ([Heath et al., 1985](#)). Most of these young boys are white and typically fall into the age range of 15 to 18 years ([Federal Bureau of Investigation, 1979–1999](#); [Hall, 2000](#)). There is some work indicating that these youngsters are of average intelligence; however, they have long histories of academic and behavior difficulties in school ([Bullis et al., 1998](#); [Kolko et al., 1985](#); [Kuhnley et al., 1982](#); [Ritvo et al., 1982](#)).

There are some preliminary studies suggesting that firesetting adolescents are more likely to have certain types of experiences and conflicts than their nonfiresetting counterparts. For example, one study suggests that adolescents with histories of firesetting have a higher than average number of accidents resulting in physical injuries ([Jackson et al., 1987](#)). A plausible explanation of this finding is that adolescent firesetters are greater risk takers and that risk-taking behavior more frequently results in accidents. Also, a series of studies shows that firesetting adolescents may experience a higher level of sexual arousal and fantasy excitement than nonfiresetters ([Freud, 1932](#); [Lewis and Yarnell, 1951](#); [Sakheim and Osborn, 1994](#); [Sakheim et al., 1991](#); [Yarnell, 1940](#)). There also is some work reporting that firesetting adolescents experienced more sexual conflicts and gender conflicts ([Bumpass et al., 1985](#); [Sakheim and Osborn, 1986](#); [Sakheim and Osborn, 1994](#); [Sakheim and Osborn, 1999](#); [Sakheim et al., 1985](#); [Sakheim et al., 1991](#)). One study indicates that adolescent firesetters are more frequently involved in sexual misbehavior ([Jacobson, 1985a](#)). This information, however, emerges from a limited number of investigations that rely heavily on clinical impressions rather than on controlled empirical observation.

There is very little work examining the emotional style of adolescent firesetters. Clinical studies characterize them as angry, aggressive youngsters who have little regard for social rules and norms ([Gaynor and Hatcher, 1987](#)). They may feel excitement and defiance before the actual firestart. Once the fire is set, they often do not feel guilt or remorse for their actions ([Gaynor and Karchmer, 1988](#)). These youngsters are unable to experience a depth of emotional feeling and their resulting behavior reflects a restricted capacity of expression. This pattern of emotional functioning is similar to that found in antisocial and delinquent personality patterns (American Psychiatric Association, 1994; [Sakheim and Osborn, 1994](#); [Sakheim and Osborn, 1999](#); [Sakheim et al., 1991](#)).

The behavior of firesetting adolescents is characterized as restless, impulsive, mischievous, and defiant ([Gaynor and Hatcher, 1987](#); [Repo and Virkkunen, 1997](#); [Wooden and Berkey, 1984](#)). In addition, there is some work indicating that these adolescents demonstrate higher levels of risk-taking behavior ([Kafry et al., 1980](#)). These somewhat preliminary findings suggest that, if these youngsters are in a situation where ignition sources or fire materials are readily available, they will be less likely to control their impulse or urge to firestart and more likely to follow through with their aggressive and defiant firesetting behavior.

There is emerging work investigating the relationship between the presence of mental disorder, psychiatric diagnosis, and firesetting. Several studies indicate a relationship between the early childhood onset of conduct disorder and the development of antisocial behavior in adolescents ([Lahey et al., 1999](#); [Repo and Virkkunen, 1997](#); [Werry, 1997](#)). Studies of adolescents involved in recurrent firesetting resulting in arrest reveal that criminal recidivism was significantly related to alcohol dependence for boys, and self-mutilation and suicide attempts for girls ([Bernburg and Thorlindsson, 1999](#); [Coid et al., 1999a](#); [Coid et al., 1999b](#); [Repo et al., 1997](#)). In addition, it appears as if adolescent firesetters who carry a psychiatric diagnosis are more likely to recommit arson crimes than those firesetting adolescents who are not diagnosed with a mental disorder ([Barnett et al., 1997](#)).

There is clinical and empirical agreement on the relationship between the quality of family life and the prediction of firesetting behavior in adolescents ([Repo, 1998](#); [Sakheim and Osborn, 1999](#); [Scholte, 1999](#)). Clinical reports suggest that the characteristics distinguishing the families of firesetting children also may describe the families of adolescent firesetters ([Gaynor and Hatcher, 1987](#)). That is, firesetting adolescents come from single-parent households in which patterns of inconsistent supervision and discipline are typical ([Gaynor and Karchmer, 1988](#)). Increased parental control and decreased parental supervision appear to be related to delinquent firesetting behavior in adolescents ([Scholte, 1999](#)). There are some studies suggesting that firesetting adolescents are likely to have histories of physical abuse and other violent patterns of interaction in their family ([Jasper et al., 1998](#); [Jayaprakash et al., 1984](#); [Ritvo et al., 1982](#); [Showers and Pickrell, 1987](#)). Dissertation work demonstrated that families of adolescent firesetters showed significantly more pathologic functioning in the areas of problem solving, communication, responsiveness, and involvement than did families of nonproblem adolescents ([Reis, 1993](#)). Finally, there is a strong indication that parental psychopathology is related to firesetting behavior and arson arrest ([Group for the Advancement of Psychiatry, 1999](#); [Scholte, 1999](#)). Specifically, evidence suggests that paternal alcoholism and maternal psychosis increase the risk of adolescent firesetter recidivism ([Repo, 1998](#)). There is a great need for an increase in work to explain how these specific family factors contribute to the development of adolescent firesetting.

Just as with the behavior of normal adolescents, there is work indicating that peer group participation greatly influences the behavior of firesetting adolescents ([Bernburg and Thorlindsson, 1999](#); [Gaynor and Hatcher, 1987](#)). Current studies confirm the effect of a negative peer group on the development of delinquent behavior ([Dahlberg, 1998](#); [Rapp and Woodarski, 1997](#)). Most adolescents firestart with one or two friends ([Jackson et al., 1987](#)). Clinical cases indicate that the peer group supports, encourages, and condones firesetting, and to some extent provides a safe environment into which adolescents can retreat once they have intentionally set a destructive fire ([Gaynor J, unpublished reports from clinical case files, San Francisco, 2000](#)). Evidence suggests that continued affiliation with this type of peer group promotes the continuation of delinquent behavior during and beyond adolescence ([Fergusson and Lynskey, 1998](#)). Regardless of who leads the peer group, there may be an agreement among the members that firesetting is an acceptable behavior. In addition, clinical cases suggest that adolescents who firestart may believe that their behavior will gain them a certain degree of attention and recognition in the peer group ([Gaynor J, unpublished reports from clinical case files, San Francisco, 2000](#)). It is hypothesized that peer group support of firesetting behavior may be one of the most influential social factors in reinforcing the likelihood that the behavior will recur as long as the firestarting goes undetected and it results in no immediate nor long-term consequences.

Academic performance, behavior in school, and truancy are related to the development of delinquent behavior, including firesetting, in adolescents ([Kjelsberg and Dahl, 1999](#); [Lahey et al., 1999](#); [Loeber et al., 1999](#); [Su et al., 1998](#)). The academic achievement of firesetting adolescents is significantly below the average ([Kaufman et al., 1961](#); [Yarnell, 1940](#)). These youngsters have long histories of academic failures in school, and they may be one or more grades behind their class by the time they enter high school ([Kaufman et al., 1961](#)). Conduct reports also are less than favorable. One study indicates that firesetting adolescents often are disruptive in



class and they frequently engage in fights with their peers ([Wooden and Berkey, 1984](#)). Many of them either have been suspended several times or have been expelled from more than one school ([Kaufman et al., 1961](#)). In addition, statistical studies indicate that approximately 75% of middle and high school fires are caused by adolescents (National Fire Protection Association, 1978–1998). Hence, not only do these youngsters fail to achieve or adjust to their school environment, but there is evidence indicating that schools may be one of the primary targets of their firesetting.

There is some preliminary work suggesting that certain environmental conditions may be directly related to adolescent firesetting. Studies suggest that specific stressors may trigger firesetting in adolescents ([Fineman, 1980](#), [Fineman 1995](#)). Such stressors are likely to include frequent and sudden alterations in family relationships due to separation, divorce, or death ([Fineman, 1980](#), [Fineman, 1995](#); [Patterson et al., 1998](#); [Strachan, 1981](#); [Vandersall and Weiner, 1970](#)). In one study, parents of firesetting youngsters indicated more than twice the number of family disruptions than their nonfiresetting family counterparts ([Wooden and Berkey, 1984](#)). These parents also reported that their youngsters experienced a greater degree of stress in their lives 6 months before their firestart than at other times in their lives ([Wooden and Berkey, 1984](#)). Clinical reports suggest that, at least in some instances, immediately before firesetting, adolescents may engage in alcohol or drug consumption ([Gaynor J, unpublished reports from clinical case files, San Francisco, 2000](#); [Repo et al., 1997](#)). In addition, there are clinical cases in which firesetting also was accompanied by other delinquent acts, including petty theft and vandalism ([Gaynor J, unpublished reports from clinical case files, San Francisco, 2000](#)). It has been reported that, immediately after firesetting, adolescents may leave the fire scene and move to a safe spot to watch the fire burn. They are not likely to call for help to extinguish the fire. This is somewhat consistent with the notion that these youngsters also are unlikely to experience guilt or remorse once they have set the fire. Perhaps, if they did experience these feelings, they would take the appropriate actions to help suppress the fire. The attention and recognition that they may get from their peer group is likely to reinforce the firesetting behavior, especially if it goes undetected. It appears as if these adolescents experience relatively little fear of punishment for their delinquent activity, nor do they consider the potential negative outcomes of property loss or personal injury.

The following case describes one example of pathologic firesetting behavior in an adolescent.

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#### CASE ILLUSTRATION

Sixteen-year-old Carl lives with his father and younger brother in an upper-class urban neighborhood. His mother and father divorced when Carl was 12, and his mother recently remarried. Carl's father, a prominent attorney, leaves for work early, comes home late, and does a great deal of traveling. Although Carl's father is absent much of the time, a loving, elderly aunt, as well as a housekeeper, share the responsibility for the two boys. Carl's mother lives in another city a few hundred miles away and visits the two boys on an irregular basis.

Carl's father describes his son as "basically a good kid" but wishes he were more obedient and respectful. His mother says he is hard to talk to and never takes her seriously. Carl's aunt describes him as a charming boy who always seems to get into trouble. Although Carl is very bright, he has been expelled from several private schools because of his inability to follow the rules. These schools characterize him as the class bully and ringleader, and he always seems to be responsible for major class disruptions. Carl currently is attending a private day school, where his grades are average and his conduct marginally acceptable.

Carl has a history of antisocial and delinquent behavior, which began around puberty. Shortly after his mother and father separated, Carl began missing several days of school for unexplained reasons. He would leave home early in the morning and return by dinner, offering no explanation of where he had been or what he had been doing while not attending school. After talking with the school staff, it was decided to move Carl to another educational environment. Several months later, Carl's father received a late night telephone call from the local police reporting that they had caught Carl and a few friends slashing car tires at a nearby shopping mall. Carl's father was able to convince the police not to press charges. There have been two additional incidents of shoplifting, one from a local drugstore and one from a large sporting goods store. On both occasions, apologies were accepted, and no punishment or retribution was implemented. Carl has an apparent knack for getting into trouble and an ability to avoid experiencing the consequences of his behavior.

Carl's firestarting emerged at age 9 years when he was caught setting trash can fires at school. Both his father and the school authorities admonished him, and he was suspended from school for 2 days. The latest known firestart occurred 6 years later. Carl had been invited to spend the night at the house of his friend Kevin. Kevin's parents went out for the evening, and the two boys decided to drink the beer that they found in the refrigerator. Both of the boys together consumed about two six-packs of beer. They then left the house and rode their bikes down to the local park and recreation area. They thought it would be easy and fun to break into the building and steal the petty cash from the park director's desk. Once they had entered the building through an open window, they worked for several minutes to break into the desk where the petty cash was kept. Unsuccessful and frustrated by their attempt to obtain the money, they spotted a lighter on a nearby counter and ignited the papers in the trash can. They fled from the building on their bikes without attempting to extinguish the fire. On their way home, they heard the sound of fire engines and assumed that they were responding to the fire.

#### Comment

Carl has a long-standing history of aggressive and antisocial behavior. It appears as if he seizes every opportunity to get into trouble. Lacking the appropriate attention and supervision at home, he pursues recognition in socially unacceptable ways. Attempts by school authorities to control his disruptive behavior have been unsuccessful. In fact, there do not seem to be any people who are effective in guiding or shaping Carl's behavior. Carl apparently felt no guilt or remorse over having set the fire at the park and recreation building. In addition, neither he nor his friend made any attempt to extinguish the fire or call for help. Hence, together these boys successfully started a significant fire and escaped immediate detection. Unless they are caught, they are unlikely to experience any consequences of their firestarting behavior. Their perceived success in this episode may encourage them to pursue more illegal and dangerous firesetting activities in the future.

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## THEORY

There are several major theoretical frameworks that offer explanations specific to the development of firesetting behavior in youth. However, before they are presented, it is useful to explain firesetting behavior in the context of the development of aggressive and violent behavior in childhood and adolescence.

It is hypothesized that the development of aggressive and violent behavior is orderly rather than random and follows one or more behavioral pathways ([Loeber and Stouthamer-Loeber, 1998](#); [Nagin and Tremblay, 1999](#); [Vitaro et al., 1998](#)). Research supports a triple-pathway model to describe the development of aggressive and violent behavior in youth ([Loeber et al., 1993](#), [Loeber et al., 1997](#)). There is an overt or proactive pathway, that starts with minor aggression, and is followed by physical fighting, and then by personal violence such as assault, rape, and murder. The covert or reactive pathway is defined as a sequence of nonconfrontational behaviors such as frequent lying and shoplifting, and is followed by property damage such as vandalism, firesetting, and other moderate to serious forms of delinquency. The third pathway, unlike the previous two, exists only before 12 years of age and is an authority conflict pathway consisting of defiant, stubborn behavior that includes staying out late at night, running away, and truancy. Empirical work has validated this model and suggests that most aggressive behavior develops along either an overt or covert pathway, with the most severe forms of violence exhibiting both overt and covert behaviors ([Loeber et al., 1998](#); [Tolan and Gorman-Smith, 1998](#)).

Although this model explains the classification of firesetting as a covert aggressive behavior, it does not hypothesize the underlying causes that lead specifically to the development of firesetting behavior in youth. There are four theoretical frameworks—psychoanalytic, social learning, dynamic-behavioral, and functional analysis—that present hypotheses regarding the acquisition of firesetting behavior. Although they offer intuitively interesting explanations of firesetting, there is remarkably little empirical work to support or reject these theories. Therefore, the current value of these theories lies in their attempt to speculate on the psychological dynamics of firesetting.

Freud was the first to examine the psychological meaning of fire ([Freud, 1932](#)). His psychoanalytic theory hypothesized a relationship between sexual desires and fire. He wrote that fire symbolically represented the expression of libidinal and strong phallic-urethral drives. That is, men attempt to extinguish fires with their own urine, thereby symbolically engaging in a homosexual struggle with another phallus. Therefore, it is the relationship between sexual arousal and urination that is the underlying motive associated with the thrill of igniting and extinguishing fires. The act of firesetting represents a regression to the urethral-phallic phase of psychosexual development, and setting fire substitutes for forbidden masturbatory or sexual desires (Freud, 1932).

Given this hypothesized relationship between sexual arousal, urination, and firesetting, it is expected that firesetting youngsters might display one or more conflicts with respect to these areas of psychological functioning. There is some clinical, as well as empirical, work suggesting that various groups of firesetting youngsters have a higher-than-average incidence of sexual conflicts ([Sakheim and Osborn, 1986](#), [Sakheim and Osborn, 1994](#), [Sakheim and Osborn, 1999](#); [Sakheim et al., 1985](#), [Sakheim et al., 1991](#); [Yarnell, 1940](#)), abnormal sexual behavior ([Jacobson, 1985a](#)), and sexual dysfunction ([Stekel, 1924](#); [Yarnell, 1940](#)). However, there is substantial controversy over whether there is a significant relationship between enuresis, firesetting, and general patterns of delinquent behavior. Early research indicated that the behavioral triad of animal abuse, enuresis, and firesetting predicted aggressive and violent behavior in children and adolescents ([Hellman and Blackman, 1966](#)). Although later work does not confirm the predictive value of the behavioral triad, it does indicate a significant relationship between enuresis and firesetting ([Heller et al., 1984](#); [Prentky and Carter, 1984](#); [Repo et al., 1997](#)). More recent work also points to a significant correlation between animal abuse and property offenses, including firesetting ([Arluke et al., 1999](#); [Sakheim et al., 1991](#)). Hence, it may be that both enuresis and animal abuse are independently related to firesetting, and children involved in one or both of these behaviors also are more likely to be firesetters. To date, the major tenets of psychoanalytic theory explaining the specificity of the connection between sexual arousal, enuresis, and firesetting remain open until further work can verify the nature of the hypothesized relationships.

Social learning theory offers a significantly different explanation of the development of firesetting behavior. Whereas psychoanalytic theory emphasizes the relationship between instincts and firesetting behavior, social learning theory emphasizes the impact of the environment on shaping the development of firesetting behavior. Learning experiences and cues, personal repertoires, and parent and family influences and stressors are hypothesized to be related to firesetting ([Kolko and Kazdin, 1986](#); [Patterson et al., 1989](#)). Firesetting occurs because youngsters learn the behavior, that is, they may observe it, imitate it, model it, and perhaps even be rewarded for it. Firesetting is a form of learned aggression, which may first surface in the patterns of family interaction ([Kolko and Kazdin, 1990](#); [McKerracher](#)



and Dacre, 1966; Vreeland and Waller, 1979). Later firesetting activities may be reinforced by the peer group. Undetected firesetting episodes in which no significant behavioral or social consequences are experienced also reinforce the likelihood that firestarting will recur. Hence, using a developmental framework, initially firesetting behavior emerges and may be learned within the family system; however, it is reinforced later by the peer group, and it recurs because of the lack of socially applied sanctions prohibiting or punishing the behavior ( Patterson et al., 1989).

There is minimal work investigating the application of social learning theory to the development of firesetting behavior. One clinical study reported that fathers of adolescent firesetters all had significant fire-related employment (fireman, furnace stoker, automobile burner at a junkyard), with one youngster not only watching but participating with his father in the burning of automobiles ( Macht and Mack, 1968). There have been no empirical attempts to confirm or disclaim these clinical observations. Another clinical study reported that in a small percentage of firesetting cases (three of 27), parents admitted to using fire to punish their children, obviously conveying the message that fire is an acceptable form of retaliation ( Ritvo et al., 1982). Although these clinical studies represent thin evidence in support of a social learning theory explanation of firesetting behavior, there is a long history of empirical studies that have identified family variables as consistent covariates for early forms of antisocial behavior and for later delinquency ( Group for the Advancement of Psychiatry, 1999; Loeber and Dishion, 1983; Loeber and Stouthamer-Loeber, 1998; Loeber et al., 1998; Patterson et al., 1998). These covariates, such as harsh and inconsistent discipline, little positive parental involvement with the child, and poor monitoring and supervision, are strikingly similar to those variables characterizing the families of firesetting youngsters ( Kolko and Kazdin, 1990). Hence, what remains to be accomplished is the identification of the specific linkages between social learning theory and those family variables characteristic of firesetting youngsters.

Dynamic-behavioral theory is a broad-based conceptual framework designed specifically to explain firesetting behavior. This theory identifies a number of psychosocial determinants related to the development of firesetting behavior in youngsters. Although there have been various attempts to categorize these determinants (Fineman, 1980, Fineman, 1995; Gaynor and Hatcher, 1987; Heath et al., 1985), there is consensus on at least three major dimensions related to the development of firesetting behavior: (a) personality and individual characteristics, (b) family and social circumstances, and (c) immediate environmental conditions. Each of these three dimensions comprises specific variables. For example, personality and individual characteristics can include demographics, intelligence, emotional style, and experiential and behavioral variables. Social circumstances can include family dynamics, peer relationships, and school performance. Environmental conditions are those antecedent events that trigger firesetting and the consequences that may or may not be experienced as a result of the behavior. It is hypothesized that these variables can be further classified as either risk or protective factors. Risk factors are positively related to the emergence of firesetting behavior in children and adolescents and include the individual variables of chronic illness, neglect and abuse, sexual conflict, impulsivity, and psychiatric diagnosis; the social circumstances of family dysfunction, parental psychopathology, inadequate social skills, and school failure; and the environmental conditions of stressful events and alcohol and substance use and dependence. Much less is understood about those protective factors that discourage or prevent firesetting behavior, but certain social conditions such as intact family structures, prosocial peer groups, and supportive communities are thought to be related to the development of healthy, nonviolent, and well adjusted children and adolescents. The most useful feature of these proposed risk and protective factors is that they can be observed and measured to confirm or reject their hypothesized relationship to firesetting.

The major contribution of dynamic-behavioral theory is that research related to describing the psychosocial characteristics of firesetting youngsters can be organized and classified utilizing this conceptual framework. It presents the first predictive model of firesetting behavior by trying to define the domain of independent variables, such as personality and social and environmental factors, that will predict the occurrence of the dependent variable, firesetting behavior. Unlike psychoanalytic or social learning theory, it is broad-based in that it does not assume that one major factor, such as instincts or the social environment alone, predicts firesetting. Rather it presents a multivariate approach to predicting levels of firesetting behavior. As research emerges describing the various characteristics of firesetting youngsters, dynamic-behavioral theorists must empirically identify both the risk and protective factors that are significantly related to firesetting behavior. The result will be a quantifiable description of various types of youngsters involved in various levels of firesetting behavior.

There is one final theoretical framework that represents the latest attempt to explain firesetting behavior. Functional analysis offers a model for predicting the occurrence of arson and recidivistic fire behavior ( Jackson et al., 1987). The application of this framework is more useful in explaining the firesetting behavior of older children and adolescents, who are more likely to be involved in recurrent firestarting. This paradigm hypothesizes that certain psychosocial stimuli, in the context of major environmental conditions, predispose individuals toward firesetting, which in turn is either positively or negatively reinforced. The psychosocial stimuli and environmental conditions that influence firesetting are not unlike those identified by dynamic-behavioral theory. They include psychosocial disadvantage, dissatisfaction with life, ineffective social interaction, previous experience with fire, the occurrence of emotionally significant events, and opportunities to set fire. Firesetting is viewed as an attempt by firestarters to exert some type of change in their environment, where alternative behaviors motivated by the need to change have proven ineffective. Firesetters are characterized as basically ineffective individuals who have been unable to develop the behaviors necessary to express and satisfy their emotional and social needs. Consequently, they firestart as one way to assert a sense of mastery and control over their environment. This description of recurrent firesetting behavior and the general underlying assumptions is consistent with clinical case reports of adolescent firesetting in which social ineffectiveness coupled with the need to express aggressive behavior have been the primary motivating factors for firestarting ( Gaynor, 2000). Hence, a preliminary application of the tenets of functional analysis theory appears to be useful in understanding the clinical dynamics of recurrent firesetting.

## DIFFERENTIAL DIAGNOSIS

Current empirical work consistently reports that the most frequently occurring psychiatric diagnosis associated with pathologic firesetting is conduct disorder (Jacobson, 1985a; Repo, 1998; Sakheim and Osborn, 1999; Sakheim et al., 1985, Sakheim et al., 1991; Showers and Pickrell, 1987). The essential clinical features describing this disturbance, such as behavioral difficulties at home or at school, low self-esteem, low frustration tolerance resulting in irritability or temper outbursts, and poor academic achievement, closely match the observed behavior patterns of pathologic firesetters ( Gaynor and Hatcher, 1987). In addition, studies suggest that the occurrence of firesetting behavior is a significant predictor of the persistence of aggressive and antisocial behavior in youngsters diagnosed with conduct disorder (Jasper et al., 1998; Kazdin, 1987; Kelso and Stewart, 1986; Taylor et al., 1998). Therefore, the probability is high that firesetting youngsters diagnosed with conduct disorder will have poor outcomes and will continue to be involved in broader antisocial and delinquent behavior.

There is some evidence indicating that a conduct disorder diagnosis with aggressive features may be accompanied by additional psychopathology differentiating male and female firesetters (Coid et al., 1999a, Coid et al., 1999b; Jasper et al., 1998; Taylor et al., 1998). Male firesetters diagnosed with conduct disorder are more likely to have significant histories of alcohol dependence (Repo, 1998; Repo and Virkkunen, 1997; Repo et al., 1997). For female firesetters, a conduct disorder diagnosis often is accompanied by an underlying mood disorders with histories of self-mutilation and suicide attempts (Coid et al., 1999a, Coid et al., 1999b; Jasper et al., 1998). It also is more likely that firesetting behavior diagnosed in the context of an early onset of conduct disorder for both male and female firesetters leads to more persistent and recidivistic criminal behavior (Coid et al., 1999a, Coid et al., 1999b; Lahey et al., 1998; Repo, 1999; Repo and Virkkunen, 1997; Werry, 1997).

A handful of studies report that youngsters diagnosed with hyperactivity and attention deficit/hyperactivity disorder have histories of firesetting behavior as well as participation in other delinquent activities (Broling and Brotman, 1975; Gruber et al., 1981; Lahey et al., 1999; Vitelli, 1998). Conversely, it has been observed that a proportion of firesetting youngsters display general learning disabilities, which may be related to their below-average levels of achievement in school (Jasper et al., 1998; Wooden and Berkey, 1984). It is unclear from this research whether these learning disabilities are related to diagnosed hyperactivity and accompanying shortened attention span. Although preliminary information suggests a relationship between hyperactivity, attention deficit, firesetting, and conduct disorder, clearly more empirical investigation is necessary to determine the exact extent and nature of these connections.

There are several studies indicating a relationship between firesetting and severe mental disturbances such as schizophrenia, organic brain dysfunction, and mental retardation (Barnett et al., 1997; Jasper et al., 1998; Pontius, 1999; Taylor et al., 1998; Wooden and Berkey, 1984; Yarnell, 1940). Some of these studies suggest that firesetting occurs in psychotic youngsters in response to delusions involving themes of persecution, self-abuse, and control (Wooden and Berkey, 1984; Yarnell, 1940). In some cases of severe mental disturbance it is likely that the firestarting represents a reactive behavior, rather than a major or essential aspect of the disturbance. The exact role firesetting plays in various types of severe psychopathology is unknown and remains open to theoretical speculation and empirical evaluation.

Although pyromania, the only *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV) diagnosis related specifically to the behavior of firestarting, was initially reported in children in the classic treatise by Lewis and Yarnell (1951), subsequent empirical work shows that it is not used in diagnosing firesetting youngsters (Gaynor and Hatcher, 1987; Geller et al., 1997; Rider, 1980). The diagnosis of pyromania typically is reserved for adults (American Psychiatric Association, 1994). However, according to clinical and empirical observations, pyromania, described by the essential features of a recurrent failure to resist the impulse to set fires, an increased sense of tension before setting fires, and an experience of intense pleasure, gratification, or release at the time of ignition, occurs infrequently in adult populations and rarely is a psychiatric diagnosis attributed to adult firesetters (Barnett et al., 1997; Taylor et al., 1998; Zeegers, 1984). More than likely, adult firesetters, when detected, are legally apprehended and become the responsibility of the criminal justice system. Therefore, even if pyromania were an



appropriate diagnosis, most criminally incarcerated arsonists do not receive psychiatric evaluations and diagnostic classifications.

## TREATMENT

The goals of an effective treatment strategy are to abate firesetting behavior and sustain significant changes in the underlying psychopathology. Specific psychotherapies have been developed to work with youngsters presenting with firesetting as the primary behavior problem. Outpatient treatment is the method of choice; however, there are some inpatient settings that have programs designed exclusively for firesetting youngsters. Because theory and empirical evidence suggest that there is a complex set of variables associated with the development of firesetting behavior, there is a trend toward the application of multisystem or multicomponent therapies, as well as community-based interventions to the treatment of pathologic firesetting. Many of these therapeutic approaches are recently developed, and consequently, there is an absence of controlled empirical studies demonstrating their relative effectiveness. Nevertheless, preliminary clinical evaluations of many of these methods suggest that, at least in the short term, they are highly successful in eliminating firesetting behavior in youngsters. What is less clear is the relative impact these treatment strategies have on sustaining desirable changes in the underlying psychopathology.

Individual and family psychotherapy are the two predominant modalities used in the outpatient treatment of firesetting youngsters. The primary focus of individual psychotherapy is on the immediate elimination of firesetting behavior, with a secondary emphasis on adjusting or changing the underlying psychopathology ( [Gaynor and Hatcher, 1987](#)). Cognitive–emotion and behavior therapy are the two most highly developed outpatient treatment approaches. The major goals of cognitive–emotion psychotherapy are to teach youngsters how to recognize the urge to firestart, interrupt the behavior before it starts, and substitute socially appropriate types of behaviors to express their underlying emotions ( [Bumpass et al., 1983](#), [Bumpass et al., 1985](#)).

The primary therapeutic mechanism of the cognitive–emotion approach is the construction of a written graph by youngsters with help from their parents and the therapist. [Figure 57.1](#) represents an example of a graph constructed by a firesetting youngster. Both the feelings and specific events leading up to and following the most recent firestart are graphed. The *vertical axis* of the graph represents the time before, during, and after firesetting. The *horizontal axis* represents the intensity of the various emotions experienced during this time period. The firestarting episode is graphed in the center of the vertical axis. Youngsters are asked to list on the vertical axis, in order of occurrence, the significant events leading up to and following the firestart. For example, Johnny came home after school, and no one else was home. He wanted to play with some friends, but after calling around, he found that they were all busy. He turned on the television, but there was not anything that he wanted to watch. He decided to make a trip to the corner store to get some candy. He bought his favorite snack and picked up the free book of matches lying on the counter. On his way home he began striking matches and extinguishing them. He passed a vacant lot, which he entered. When no one was looking, he set a small plant on fire and ran home. When he got home, he turned on the television and watched cartoons for the rest of the afternoon. In the early evening, his grandmother arrived to make his dinner. After dinner, Johnny and some of his friends walked over to see the damage that occurred as a result of the fire at the vacant lot.



**Figure 57.1.** Graphing firesetting behavior.

Once these events are graphed, youngsters are asked to describe their feelings associated with this period. Each type of feeling, such as loneliness, anger, excitement, fear, and guilt, is graphed separately and represented by an individual line. Because specific feelings wax and wane over this period, their various intensity levels are represented by the relative amplitude of the “feeling” lines on the graph. For example, when Johnny arrived home after school to an empty house, initially he might have felt lonely. After calling around to his friends and finding them all busy, these feelings might have increased. Once he discovered that the television was not going to satisfy him, his feeling of loneliness again may have intensified. However, once he decided on a trip to the store, some of his lonely feelings subsided and were replaced by other types of emotions. Hence, the amplitude of the “loneliness” feeling line would gradually increase once Johnny arrived home from school; it stays elevated during his struggle to find some form of entertainment, and then it begins to decrease once his trip to the store was underway. In this way, one or more feelings and their relative patterns of experienced intensity are graphically represented in relation to the significant firestarting incident.

A typical or usual graph is expected for most youngsters involved in pathologic firesetting. A major assumption of this graphing technique is that although the specific events surrounding firestarting may vary, the particular pattern of feelings connected with firestarting episodes is expected to be somewhat similar ( [Bumpass et al., 1983](#), [Bumpass et al., 1985](#)). It is the role of the therapist, through the process of constructing the graph with the youngsters, to bring to their awareness the usual feeling states associated with the impulse of firesetting. A typical graph indicates that one or more significant events triggers a sequence of sad, lonely feelings. These feelings are replaced by intense, angry feelings, which in turn are controlled by a destructive urge that is significantly relieved by firestarting. A feeling of fear usually emerges before the firesetting and continues for a brief time after the fire is set. Feelings of guilt, if experienced, follow the firesetting. The therapist emphasizes the importance of the relationship between these experienced feelings and the desire to firestart. This pattern of feelings, the associated significant events, and firesetting are the focus of this particular method of cognitive–emotion psychotherapy.

The graphing technique is used as the starting point to help youngsters learn that they can prevent themselves from setting more fires. Once the graph is complete, usually by the second or third session, it is used as a teaching tool for the remainder of the psychotherapy. Youngsters are taught that the feelings they experience early in the typical pattern leading to firesetting, such as loneliness or sadness, are a signal that the impulse to firestart may be imminent. They are reminded that they do not always have to act on their feelings, especially if they firestart. They are told that they have a choice of what to do about their feelings. They are asked what constructive behaviors they can pursue when they are feeling lonely and sad. These alternative behaviors are listed on the graph. Youngsters are told that they probably will not set any more fires, but if they have the urge to firestart, they should pay attention to their feelings so that they can talk about them with their therapist. If they become overwhelmed with their feelings and want to set a fire, they are asked first to telephone and talk with their therapist. If subsequent firestarts occur, these firesetting episodes are graphed and become the focus for future sessions. If youngsters are successful in redirecting their urge to firestart, they are supported and praised by their therapist and their family. This form of cognitive–emotion therapy averages six to eight sessions and can be considered a form of brief psychotherapy.

A follow-up clinical assessment evaluating the effectiveness of this type of cognitive–emotion therapy indicated that 2 of 26 youngsters exhibited single episodes of firesetting behavior subsequent to the termination of their treatment ( [Bumpass et al., 1983](#), [Bumpass et al., 1985](#)). For six youngsters, additional psychotherapy was recommended once the graphing technique had been successful in abating their firesetting behavior. Unfortunately, these recommendations were not pursued; these youngsters became involved in other acting-out and antisocial behaviors, such as petty theft and vandalism, and were subsequently treated in inpatient and residential treatment facilities. Although there was no control group included in this follow-up evaluation, this preliminary assessment indicates that this method shows some promise in eliminating firestarting behavior. It remains unclear how successful this approach is in addressing the underlying psychopathology that frequently exists in the lives of firesetting youngsters and their families.

There are a number of behavior therapy approaches that have been reported as successful in abating firesetting behavior. The predominant behavior therapy methods used either alone or in combination are punishment, reinforcement, negative practice or satiation, and operantly structured fantasies. Two case studies report the successful application of various methods of punishment, including the use of threats, such as work penalties ( [Carstens, 1982](#)). Sometimes the use of punishment is coupled with positively reinforcing youngsters when they find and return to their parents conspicuously hidden empty matchbooks ( [Holland, 1969](#)). Negative practice procedures, coupled with the use of positive reinforcement to encourage more socially appropriate behaviors, also have been reported as successful in stopping severe and recurrent firestarting behavior ( [Koles and Jenson, 1985](#); [Kolko, 1983](#), [Kolko, 1988](#)). One behavior therapy case study avoided the application of punishment methods or negative fire experiences by using a positive reinforcement program coupled with the implementation of operantly structured

fantasies ([Stawar, 1976](#)).

All of the reported behavior therapy techniques—punishment, positive reinforcement programs, negative practice and satiation procedures, and operantly structured fantasies—are successful in stopping pathologic firesetting behavior in single-case applications. In addition, these methods are short-term interventions that can be used within an average of 6 to 12 sessions. However, questions remain regarding the effectiveness of the techniques with more than one or two cases. Also, although these methods appear to be successful in stopping the specific firesetting behavior, there is little evidence indicating that these specific behavioral techniques change or adjust accompanying psychopathology. Measuring the relative success of behavior therapy is a challenge for the future.

There are only a handful of cases reported in the literature using brief (three to six sessions) family psychotherapy to treat firesetting youngsters successfully ([Eisler, 1974](#); [Madanes, 1981](#); [Minuchin, 1974](#)). In two of the three cases, family members were taught how to ignite and extinguish matches safely in a controlled setting in the therapist's office ([Eisler, 1974](#); [Minuchin, 1974](#)). This controlled firestarting task was used as a vehicle to restructure the existing patterns of family communication and interaction. In particular, attention was focused on restoring the appropriate amount of parenting authority and reestablishing communication between youngsters and their parents regarding household rules designed for the safety and protection of the family. In the third case, the youngster's firesetting was viewed as the overt symptom of a dysfunctional family system (Madanes, 1981). Family therapy sessions were focused on a recognition of the underlying distress and helping family members to identify the changes that needed to happen to reshape the nature of their interactions. In all three cases, follow-up studies indicated the successful elimination of firesetting behavior and a higher level of satisfaction among family members regarding patterns of communication and interaction. Although these clinical reports are encouraging, the success of family psychotherapy in treating firesetting cases deserves more systematic and widespread application and evaluation.

Inpatient treatment programs for firesetting youngsters have been influenced by two major types of therapeutic philosophies ([Gaynor and Hatcher, 1987](#)). The first is a more traditional, psychodynamic approach where the treatment emphasis is on the nature of the therapeutic alliance formed between youngsters and program staff. Both individual and family psychotherapy are the techniques used and the treatment program is long term (ranging from 6 months to 2 years). The second theoretical approach is behavioral, where specific behaviors are identified for change and discrete interventions are designed to adjust these behaviors. The firesetting youngsters are the primary focus of the behavior therapy methods, with parents and family members included in the therapeutic endeavor once the firesetting behavior has been eliminated. Behavior therapy programs tend to be relatively short term (4 to 8 weeks) and currently are the most widely offered inpatient approach to the treatment of firesetting youngsters.

Studies indicate that several different types of behavior therapy methods have been used successfully in inpatient settings ([Cox-Jones et al., 1990](#); [DeSalvatore and Hornstein, 1991](#); [Koles and Jenson, 1985](#); [McGrath et al., 1979](#)). One inpatient program consists of three phases designed to work with both the child and the parents. During phase one, children participate in a series of behavioral exercises in which a choice must be made between toys and firestarting materials. These exercises are observed by therapists out of the view of the youngsters. If they choose matches and lighters as opposed to non-fire-related toys, the therapist intervenes and conducts a debriefing. The debriefing focuses on helping youngsters to realize the experienced emotions associated with choosing firestarting materials. The result of these exercises is that children are left with a mild aversion to firestarting materials. During the second phase, intensive family psychotherapy is used to adjust those environmental conditions in the family that are associated with the emergence of firesetting behavior. The third phase focuses on reentry into the family and community. Outpatient family psychotherapy is used, and an intensive effort is made to provide special community support services such as schooling and structured activities. At the end of the third phase, it is expected that youngsters will return to the family and social environment, not participate in firestarting activities, and function adequately in their interpersonal and social milieu. Follow-up contact shows no relapses in 100 cases treated in a 2-year period ([Birchill, 1984](#)).

One other inpatient approach is worthy of mention. It also involves the treatment of firesetting with a combination of behavior therapy techniques and fire safety education, with the goals of improving ego functioning while providing information directed at developing more adaptive and socially appropriate behaviors ([DeSalvatore and Hornstein, 1991](#)). The program takes place in a short-term diagnostic inpatient unit, and uses a milieu approach consisting of three phases—assessment, didactic, and practice. The fire safety materials use Smokey the Bear as the primary teaching theme. At 1-year follow-up, results indicated that 1 of 35 children was found to have set another fire ([DeSalvatore and Hornstein, 1991](#)). Although these behaviorally oriented inpatient programs show promise, they must be evaluated in the more rigorous context of controlled clinical trials.

The application of a multisystem approach to the treatment of firesetting is consistent with current theory and empirical support suggesting the linkage between a complex set of individual, social, and environmental factors and the development of pathologic firesetting behavior in youngsters. It has been proposed that the formulation of a treatment plan should address the elimination of firesetting behavior, improve skill deficits in social, emotional, or academic functioning, and improve the level of fire safety knowledge and appropriate fire survival skills ([Stadolnik, 2000](#)). There are several examples of multisystem treatment programs designed for firesetting juveniles ([Clare et al., 1992](#); [Cox-Jones et al., 1990](#); [Koles and Jenson, 1985](#); [Kolko, 1983](#); [Zingaro and Pittman-Wagers, 1992](#)). Although settings can vary from outpatient to inpatient, there is a consistent treatment approach involving the identification of the specific causative factors leading to the development of pathologic firesetting and the implementation of intervention focused on multiple targets of therapeutic change. For example, [Koles and Jenson \(1985\)](#) present the case of a young boy with a history of chronic firesetting related to poor impulse control and frequent temper outbursts, inadequate parental supervision and discipline, little or no interaction with peers, and below-average knowledge of fire survival and safety information. This case was successfully treated using the multisystem approach of overcorrection, relaxation training, and covert sensitization to establish cognitive control of impulse and temper, behavioral contracting to improve parental involvement, social skills training to facilitate interaction with peers, and fire safety education to increase survival and safety skills. Replication of these case studies and controlled clinical trials are critical to validating the effectiveness of the multisystem treatment of firesetting.

Community-based intervention programs for juvenile firesetters and their families reflect the multisystem approach to the treatment of firesetting behavior. Juvenile firesetter intervention programs (JFIPs) typically are coordinated by the fire service in their local community and supported by technical assistance from the U.S. Fire Administration and other national and statewide fire service organizations ([Gaynor, 2000](#)). The primary goal of JFIPs is to create a community network of treatment options for juvenile firesetters and their families. JFIPs allow at-risk youth and their families access to a coordinated network of human service agencies, including the fire service, law enforcement, school programs, mental health, social services, and juvenile justice. The centerpiece of a JFIP is the ability to identify, screen, and evaluate the likelihood or risk that youth will become involved in future firesetting incidents. Once firesetting risk is determined, juveniles and their families can participate in a number of intervention options, including fire survival and safety education, organized youth groups, school programs, mental health services, human resource programs, and the rehabilitation and correction services of juvenile justice. There have been at least two reported attempts to evaluate the effectiveness of community-based programs, and results indicate that even minimal intervention may have a beneficial effect in reducing firesetting behavior over a long period ([Adler et al., 1994](#); [Kolko, 1996](#)). Clearly, more systematic program monitoring and evaluation is necessary to determine the impact and value of community-based intervention programs.

Several outpatient, inpatient, multisystem, and community-based treatment methods report preliminary success in treating firesetting youngsters. The selection of the best intervention strategy depends on maximizing the fit between an assessment of the severity of the firesetting and associated clinical features and the philosophies, modalities, and targets of behavior change emphasized by the treatment approaches. If clinical assessments reveal youngsters who are a significant danger to themselves or others and there is evidence of severe psychopathology, then inpatient treatment must be strongly considered. If the need for inpatient treatment can be ruled out, then an outpatient therapeutic modality becomes a viable intervention option. In general, the cognitive-emotion approach uses the graphing technique to effect changes in emotional functioning, behavior therapy uses a variety of methods to extinguish firestarting and in some cases replace it with more socially acceptable behaviors, and family psychotherapy works within the family structure to adjust role relationships and patterns of communication and interaction. Because of the complex interaction of individual, social, and environmental risk factors related to the development of pathologic firesetting behavior, multisystem and community-based intervention programs have surfaced as viable treatment options for juvenile firesetters and their families. Until more rigorous standards of scientific evaluation can be applied to study the relative effectiveness of these therapeutic approaches, clinical evidence stands to support the success of these intervention strategies.

## RESEARCH DIRECTIONS

To date, there have been limited systematic attempts to mount an organized research effort to describe empirically the prevalence of firesetting in youngsters, to quantify the clinical features associated with firesetting, or to test the relative effectiveness of specific psychotherapies in treating the problem. Most work in the field consists of clinical studies, with little attention paid to rigorous standards of scientific evaluation. The state of the art with respect to research is, at best, qualitative. There is a desperate need for an organized effort to quantify empirically the clinical data from which emerges the bulk of our information on firesetting youngsters. The major priorities for future research include prospective epidemiologic studies focused on the prevalence of unsupervised firestarting behavior in normal populations of children, and an estimate of the proportion of these children who later become involved in pathologic firesetting; an empirical comparison of the clinical features of firesetting youngsters compared with those youngsters diagnosed with other specific delinquent or antisocial behaviors; and a randomized clinical trial comparing the relative effectiveness of two or more therapeutic approaches in not only eliminating firesetting behavior but in alleviating the associated psychopathology that



accompanies this destructive behavior. If these research goals are achieved, then significant steps will have been taken to prevent the occurrence of pathologic firesetting behavior in future generations of our youth.

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# 58 GENDER IDENTITY DISORDERS

Kenneth J. Zucker, Ph.D.

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## DEFINITION

At a nascent cognitive level, gender identity has been defined as a child's recognition that he or she is a member of one sex but not of the other sex. Four decades ago, [Stoller \(1964\)](#) coined the term *core gender identity* to refer to the development of a "fundamental sense of belonging to one sex," the "awareness" that one is a male or female. At an affective level, this sense of belonging is emotionally valued, so that a child experiences a sense of comfort or security from being a boy or girl. A child's gender identity is often closely tied to the adoption of culturally sanctioned behavioral markers of masculinity or femininity (*gender roles*).

What does one observe in children and adolescents who are said to have gender identity disorder (GID)? The most common feature is a strong identification with, and preference for, the gender role characteristics of the other sex. This can be inferred from various age-related behavioral manifestations of gender identification, such as toy interests, fantasy role and activity preferences, peer affiliation preferences, and personality traits. Cross-gender identification is also expressed through verbal statements that one is, or would like to be, a member of the other sex. Moreover, children with GID often have few positive things to say about their own sex and appear to experience a sense of *gender dysphoria*, or unease, about being of the sex that they are. By adolescence, when the clinical picture more closely resembles what one observes in adults with GID, the pervasive sense of gender dysphoria becomes even more difficult to ignore or dismiss.

## HISTORICAL NOTE

In the 1950s, two clinical developments led to an increased interest in the study of children with potential problems in their gender identity development. First, [Money and associates' \(1957\)](#) research on children with various forms of physical intersex conditions showed that key milestones in gender identity formation occurred sometime between 18 and 36 months of age, if not earlier. It was observed that, despite an ambiguous sexual biology, children with physical hermaphroditism could develop a stable gender identity if they were reared unambiguously as members of one sex or the other ([Zucker, 1999](#)). Second, retrospective clinical reports on the adult syndrome of GID, commonly known as *transsexualism* ([Benjamin, 1954](#)), led to the recognition that the behavioral markers of this condition were often expressed during the first few years of life.

Forty years ago, [Green and Money \(1960\)](#) reported on a series of five boys without detectable physical or biological abnormality who displayed what was described as "incongruous" gender role behavior, a pattern that was incorporated 20 years later into the *Diagnostic and Statistical Manual of Mental Disorders*, third edition (DSM-III) diagnosis gender identity disorder of childhood. Since this initial report, many studies of such children have been conducted, directed at, among other things, developing accurate diagnostic and assessment procedures, evaluating etiologic hypotheses, examining the effects of treatment, and tracking long-term development ([Green 1974, 1987; Zucker and Bradley, 1995; Zucker and Green, 1992](#)).

By the early 1980s, thinking about children with gender identity problems was being influenced by a third development. A series of studies on adult homosexuality, perhaps peaking with the volume by [Bell and colleagues \(1981\)](#), indicated that the presence of patterns of childhood cross-gender behavior was a strong developmental predictor of later homosexuality, which was later confirmed in a meta-analytic review by [Bailey and Zucker \(1995\)](#). Although efforts to treat homosexuality have declined substantially since 1973, when homosexuality per se was deleted from the DSM as a mental disorder, interest in the determinants of sexual orientation has continued. Indeed, in the 1990s, there was a renaissance in research on the origins of sexual orientation development ([Gladue, 1997](#)).

## EPIDEMIOLOGY

### Prevalence

No studies have formally assessed the prevalence of GID in children. It has been suggested, however, that conservative estimates of prevalence can be inferred from data regarding the prevalence of GID (transsexualism) in adults. Such data are based on the number of persons attending clinics that serve as gateways for surgical and hormonal sex reassignment. Because not all gender dysphoric adults make themselves known, this method may underestimate the prevalence of GID; in any case, the number of adult transsexuals is small—one recent estimate from the Netherlands suggests a prevalence of 1 in 11,000 men and 1 in 30,400 women ([Bakker et al., 1993](#)).

The prevalence of GID might also be derived from the literature on the epidemiology of homosexuality. Unfortunately, this literature presents two main problems. First, the true prevalence of exclusive, or near-exclusive, preferential homosexuality remains a source of debate ([Laumann et al., 1994; Wellings et al., 1994](#)); second, the retrospective literature on childhood cross-gender behavior in homosexual men and women often does not specify how to determine a cutoff score to dichotomize cases as cross-gendered versus not cross-gendered. In addition, cases classified as cross-gendered would not necessarily meet the complete diagnostic criteria for GID in the DSM-IV ([American Psychiatric Association, 2000; Friedman, 1988](#)).

More liberal estimates of prevalence can be judged from studies of children in whom specific cross-gender behaviors have been assessed. For example, the standardization study of the Child Behavior Checklist (CBCL) ([Achenbach and Edelbrock, 1981](#)), a widely used parent-report questionnaire of childhood behavioral psychopathology, included information on the percentage of mothers of both clinic-referred and nonreferred boys and girls who endorsed two items pertaining to cross-gender identification: "behaves like opposite sex" and "wishes to be of opposite sex." [Table 58.1](#) shows the percentage of mothers of nonreferred boys and girls, across the age range of 4 to 11 years, who endorsed these two items by giving ratings of either a 1 (somewhat or sometimes true) or a 2 (very true or often true) on a 0- to 2-point scale for frequency of occurrence. For both items, more mothers of girls gave ratings of either a 1 or a 2 than did mothers of boys; however, chi-square tests showed that the differences were significant only for the rating of a 1 for the item "behaves like the opposite sex."



Sex	Age Group (Years)									
	4	5	6	7	8	9	10	11	12	Total
Boys	41	40	33	40	23	20	40	00	00	23
	00	00	00	00	00	00	00	00	00	00
Girls	33	00	01	40	00	00	00	00	00	00
	41	00	00	00	00	00	00	00	00	00
Total	4	5	6	7	8	9	10	11	12	Total
	33	00	00	00	00	00	00	00	00	00
Total	00	00	00	00	00	00	00	00	00	00
	41	00	00	00	00	00	00	00	00	00
Total	00	00	00	00	00	00	00	00	00	00
	00	00	00	00	00	00	00	00	00	00

**Table 58.1. Percentage of Nonreferred Children Whose Mothers Endorsed Child Behavior Checklist Items Relevant to Cross-Gender Identification**

These findings suggest that there is a sex difference in the occurrence of mild displays of cross-gender behavior, but not with regard to more extreme cross-gender behavior. Indeed, in another analysis of these data, it was reported that none of these 398 boys were given ratings of a 2 on both items and only two of the 398 girls were rated in this way (Zucker and Bradley, 1995).

The main problem with such data is that they do not differentiate enduring patterns of cross-gender behavior from transient phenomena (Zucker, 1985). Accordingly, data of this kind would probably overestimate cases of GID, although the methods of data collection may be reasonable screening devices for more intensive evaluation (Lindsay, 1994; Pleak et al., 1989; Sandberg et al., 1993).

### Incidence

Has the incidence of GID changed over the past several decades? Unfortunately, the types of child epidemiologic data required to answer this question do not exist. Indirect sources of information are available, but they are difficult to employ. Consider, for example, the contemporary debate regarding the incidence of homosexual partner preference. Some authors maintain that the incidence is rather stable, whereas others hold that it has increased (Fay et al., 1989). If the former view is correct, then one might also expect a stable incidence of GID, of which some unspecified percentage of homosexual adults probably experienced in childhood (at least in muted form). If the latter view is correct, then one might expect to find a greater number of children experiencing symptoms of GID. Changes in patterns of child rearing with regard to gender role behavior are another indirect source of information on changes in the incidence of GID. Lothstein reported his clinical experience that mothers who had attempted to “masculinize” their daughters or “feminize” their sons in order to prepare them for “radically new social roles” did not produce “androgynous” children, as they had hoped, but rather children who evidenced a “stereotypical cross-gender role which was frightening to their parents” (Lothstein, 1983, p. 248). Whether parental efforts to shape children's gender role behavior in less conventional ways is inadvertently inducing GID has, in fact, not been well studied; therefore, no firm conclusions can be drawn as of yet.

### Sex Differences in Referral Rates

Consistently, it has been observed that boys are referred more often than girls for concerns regarding gender identity. This has been reflected in both research studies and case reports of treatment; for example, Zucker and colleagues (1997a) reported a referral ratio of 6.6:1 (N = 275) of boys to girls.

How might this disparity be best understood? One possibility is that the sex difference in referral rates reflects a true sex difference in prevalence. Another possibility is that social factors play a role in accounting for the disparity. For example, it is well established that parents, teachers, and peers are less tolerant of cross-gender behavior in boys than girls (Fagot, 1985), which might result in a sex-differential in clinical referral (Zucker and Bradley, 1995). Weisz and Weiss (1991) devised a “referability index” (RI) that reflected the frequency with which a child problem, adjusted for its prevalence in the general population, resulted in a clinic referral. All 118 items from the CBCL were analyzed in a comparison of clinic-referred and nonreferred children. Among parents in the United States, the 20 most referable problems (e.g., vandalism, poor schoolwork, attacks people) appeared to be relatively serious. In contrast, the 20 least referable problems (e.g., bragging, teases a lot, likes to be alone) appeared less so. Weiss (personal communication, March 4, 1992) indicated that, for boys, the CBCL item “wishes to be of opposite sex” had an RI of 91/118 (i.e., in the upper quartile) and “behaves like opposite sex” had an RI of 80/118. For girls, the RI was lower: 55/118 for “wishes to be of opposite sex” and 14/118 for “behaves like opposite sex.”

Weisz and Weiss' study (1991), along with studies from the normative literature, led to the prediction that referred girls would display more extreme cross-gender behavior than referred boys, which might account, therefore, at least in part, for the disparity by sex in referral rates. Zucker and colleagues (1997a) provided some data that supported this prediction, suggesting that girls may need to display more cross-gender behavior than boys before a referral is initiated. However, it is important to note that the sexes did not differ in the percentage who met the complete DSM criteria for GID; thus, there was no gross evidence for a sex difference in false positive referrals.

## CLINICAL DESCRIPTION

The initial behavioral signs of GID most typically appear during the toddler and preschool years (Green 1974, 1987), the years in which more conventional patterns of sex-typed behavior can also first be observed. In some cases, however, parents recall that such behaviors as cross-dressing began prior to the second birthday. The central clinical issue concerns the degree to which a pattern of behavioral signs is present, because this pattern is the basis for inference on the extent to which a child is cross-gender-identified.

In boys, the clinical picture, in its full form, includes at least eight characteristics: (a) an occasional or frequently stated desire to be a girl or an insistence that he is a girl; (b) verbal or behavioral expressions of “anatomic dysphoria” (e.g., saying that he does not like his penis and would prefer a vagina or vulva; urinating in the seated position to enhance the fantasy of having female genitalia); (c) frequent cross-dressing in girls' or women's clothing or use of other apparel (e.g., towels) to simulate feminine attire; (d) a preference for female roles in fantasy play and an avoidance of male roles in fantasy play; (e) a preference for stereotypical feminine toys and activities and an avoidance of stereotypical masculine toys and activities; (f) recurrent display of stereotypical feminine or effeminate mannerisms; (g) a preference for girls as playmates and an avoidance or dislike of boys as playmates; and (h) an avoidance of rough-and-tumble play and/or participation in group sports with males.

In girls, the clinical picture is similar. It includes: (a) an occasional or frequently stated desire to be a boy or an insistence that she is a boy; (b) verbal or behavioral expressions of anatomic dysphoria (e.g., stating a desire to have a penis; urinating in the standing position in order to enhance the fantasy of having male genitalia); (c) an intense aversion to wearing stereotypical feminine clothing and an insistence on wearing stereotypical masculine clothing; (d) a preference for male roles in fantasy play and an avoidance of female roles in fantasy play; (e) a preference for stereotypical masculine toys and activities and an avoidance of stereotypical feminine toys and activities; (f) recurrent display of stereotypical masculine mannerisms; (g) a preference for boys as playmates and an avoidance or dislike of girls as playmates; and (h) a strong interest in rough-and-tumble play and participation in group sports with boys.

During childhood, several developmental issues should be considered. One issue concerns the child's remarks about wanting to be of the other sex or insisting that he or she is a member of the other sex. Stoller (1968) argued that boys who literally believed that they were girls constituted an etiologically distinct subgroup within the universe of feminine boys and were most at risk for development as transsexuals. Subsequent clinical observations and empirical data suggest that Stoller's view is not entirely accurate. Relatively young boys who show multiple signs of cross-gender behavior, as was the case in Stoller's (1968) sample, are the ones most likely to misclassify themselves as girls (Zucker et al., 1993b, 1999b). It is not clear whether this subgroup is etiologically distinct. Developmentally, one would expect younger children to have more difficulty with regard to correct gender self-labeling, and this has been verified by empirical studies of normal children. Among children with GID, then, one would expect more difficulties with correct gender self-labeling to occur during the preschool years when the overall clinical picture is extreme and, perhaps, when there are rather severe problems in general psychosocial functioning. The most common clinical presentation is that of a child who knows what his or her sex is but desires to be the other sex (Zucker et al., 1993b).

A second developmental issue concerns variation in age-related markers of cross-gender identification. Some behaviors, such as cross-sex peer affiliation

preference, appear to remain stable throughout childhood; however, other behaviors, such as activity and role interests, may show important changes. Young boys, for example, who show a preoccupation with female dolls and role play as a mother are much less likely to play with female dolls (or, for that matter, with any dolls) or to role play female characters as they approach adolescence but may well continue to manifest feminine preoccupations, such as an intense interest in feminine fashion and idealization of female actresses or rock stars. The effeminate mannerisms of such boys may become more prominent. In addition, older children are less likely than younger children to verbalize the desire to change sex ([Zucker et al., 1984, 1998a](#)), although the persistence of gender dysphoria in these children may be expressed in other ways, such as devaluation of their own sex and idealization of the other sex.

A final developmental issue concerns the meaning of the desire to change sex during the childhood and adolescent years. That desire may be related to several factors in early childhood. For example, a child might reason that because he or she prefers cross-sex activities, it would make sense to become a person of the other sex. Or a child might believe that engaging in cross-sex activities actually changes his or her sex, a manifestation of the preoperational thinking that takes place prior to the achievement of *gender constancy*. Familial factors may also be important for young children who perceive that a parent would actually like them to be of the other sex (or some subtle variation of this communication). Thus, it is highly unlikely that young children conceptualize the wish to change sex in the same manner as adolescents.

## ETIOLOGY AND PATHOGENESIS

### Biological Mechanisms and Hypotheses

Children with GID invariably do not show signs of a gross physical intersex condition, which would rule out a marked prenatal hormonal anomaly ([Meyer-Bahlburg, 1994, 1998](#)). Thus, the search for biological influences on the development of GID must focus on factors that do not affect the configuration of the external genitalia.

#### ACTIVITY LEVEL

Activity level (AL) is a commonly accepted dimension of temperament, with some evidence for a genetic basis ([Saudino and Eaton, 1991](#); [Willerman, 1973](#)) and possibly prenatal hormonal influences ([Ehrhardt and Baker, 1974](#)). Regarding children with GID, AL as a predisposing factor is a promising possibility, because it shows a rather strong sex difference, with boys having a higher AL than girls ([Campbell and Eaton, 1999](#); [Eaton and Enns, 1986](#)). Rough-and-tumble (RT) play, another sex-dimorphic behavior, bears some similarity to AL, in that it is often characterized by high energy expenditure; however, a distinguishing feature of RT is that it is a social-interactive behavior involving such sequences as “play fighting” and “chasing.” Unlike AL, marked avoidance of RT is one of the defining features of GID for boys in the DSM-IV ([Green 1974, 1987](#)).

Using parent-report measures of AL, two studies found that boys with GID had a lower AL than control boys ([Bates et al., 1979](#); Zucker and Bradley, 1995). [Zucker and Bradley \(1995\)](#) also found that girls with GID had a higher AL than control girls; indeed, the girls with GID had a higher AL than the boys with GID, whereas for the controls, the typical sex difference was observed. It is possible, therefore, that a sex-atypical AL is a temperamental factor that predisposes to the development of GID. For example, a low-active boy with GID may find the typical play behavior of other boys to be incompatible with his own behavioral style ([Ruble and Martin, 1998](#)), which might make it difficult for him to integrate successfully into a male peer group.

Perhaps these within-sex variations in AL are related to variations in patterns of prenatal hormonal secretion and converge with recent studies in the experimental animal literature. For example, among female rhesus monkey offspring, it has been possible, by varying the timing of exogenous administration of hormones during the pregnancy, to alter the normal patterning of sex-dimorphic behavior, but to keep normal genital differentiation intact ([Goy et al., 1988](#)). This animal model—which shows a *dissociation* between sex-dimorphic behavioral differentiation and genital differentiation—has the most direct relevance for explaining the marked cross-gender behavior of children with GID.

#### BIRTHWEIGHT

On average, males weigh more than females at birth ([Arbuckle et al., 1993](#)). There are, of course, many factors that influence variations in birthweight (BW). One hypothesized factor is the sex difference in prenatal exposure to androgens. In one study, girls with congenital adrenal hyperplasia (CAH) had a higher mean BW than unaffected girls ([Qazi and Thompson, 1971](#)). In another study, genetic males with the complete form of the androgen insensitivity syndrome were comparable in BW to that of genetic females ([de Zegher et al., 1998](#)).

[Zucker and associates \(1999a\)](#) compared the BWs of boys with GID and clinical control boys and girls. The clinical controls showed the expected sex difference in BW, with an effect size of 0.29 (Cohen's  $d$ ). The boys with GID had a significantly lower BW than the clinical control boys ( $d = 0.18$ ), but did not differ significantly from the clinical control girls. Although it is not clear what factor or set of factors account for the proband-control difference in BW, the results are consistent with the possible role of prenatal hypoandrogenization among the GID probands.

#### HANDEDNESS

Slightly more males than females show a preference for using the left-hand in unimanual behavioral tasks, such as writing. There is no established consensus for understanding the basis of this sex difference. Genetic factors clearly play a role in determining hand preference. Another line of research implicates adverse prenatal and/or perinatal events that result in an elevation in left-handedness above the approximate gold standard of 10% in the general population.

[Zucker and coworkers \(2001\)](#) found that boys with GID ( $N = 205$ ) had a significantly elevated rate of left-handedness (19.5%) when compared to three separate quasi-epidemiologic samples of boys (11.8%, total  $N = 13,253$ ) and with a diagnostically heterogeneous sample of clinical control boys (8.3%,  $N = 205$ ). This finding parallels studies of adult males with GID, who also appear to have an elevated rate of left-handedness ([Herman-Jeglinska et al., 1997](#)), as well as studies of adult men with a homosexual sexual orientation ([Lalumière et al., 2000](#)). At present, the explanation for the elevation remains unclear, but candidate factors have centered on some type of perturbation in prenatal development that, in some way, affects sex-dimorphic behavioral differentiation.

#### SIBLING SEX RATIO AND BIRTH ORDER

Boys with GID have an excess of brothers to sisters (sibling sex ratio) and have a later birth order ([Blanchard et al., 1995](#); [Zucker et al., 1997b](#)). Some additional evidence shows that boys with GID are born later primarily in relation to the number of older brothers, but not sisters. In the Blanchard and colleagues study, clinical control boys showed no evidence for an altered sibling sex ratio or a late birth order. These findings mesh nicely with studies of adult males with GID and who have a homosexual sexual orientation, who also have an excess of brothers to sisters and a later birth order ([Blanchard and Sheridan, 1992](#); [Blanchard et al., 1996](#); [Green, 2000](#)). One study on birth order in girls with GID showed that they had an early birth order as compared to clinical control girls ([Zucker et al., 1998b](#)).

One biological explanation to account for the results in males pertains to maternal immune reactions during pregnancy. The male fetus is experienced by the mother as more “foreign” (antigenic) than the female fetus. Based on studies with lower animals, it has been suggested that one consequence of this is that the mother produces antibodies that have the consequence of demasculinizing or feminizing the male fetus, but no corresponding masculinizing or defeminizing of the female fetus ([Blanchard and Klassen, 1997](#); [Green, 2000](#)). This model would predict that males born later in a sibling might be more affected, since the mother's antigenicity increases with each successive male pregnancy, which is consistent with the empirical evidence on sibling sex ratio and birth order among GID probands. At present, however, this proposed mechanism has not been formally tested in humans.

#### PHYSICAL APPEARANCE

The influence of physical appearance, including attractiveness, on social perception and interaction has been widely studied by social psychologists. In a clinical study of several very feminine boys, [Stoller \(1975\)](#) made this serendipitous observation about their physical appearance: “We have noticed that they often have pretty faces, with fine hair, lovely complexions, graceful movements, and—especially—big, piercing, liquid eyes” (p. 43).

[Green \(1987](#); [Green et al., 1985](#); [Roberts et al., 1987](#)) systematically studied physical attractiveness in a sample of GID boys and a male control group. At the time of assessment, the parents were asked to describe the faces of their infant sons. Masked ratings of audiotaped interviews showed that the parents of the GID boys were more likely than the parents of the controls to describe their sons during infancy as “beautiful” and “feminine.” The parents of the GID boys were also more likely to



recall that strangers commented, "He would make a beautiful girl." Retrospective distortion, however, could have operated in this situation. [Zucker and associates \(1993c\)](#) provided some data that were consistent with these parental recollections. University students, masked to group status, rated the attractiveness of boys with GID and clinical control boys from photographs taken at the time of clinical assessment. Boys with GID were judged to be significantly more attractive, beautiful, cute, handsome, and pretty than were the control subjects. In another study, girls with GID were judged to be significantly less attractive, beautiful, cute, and pretty than were clinical and normal control girls ([Fridell et al., 1996](#)). In a third study, boys with GID were judged to be significantly less "all-boy," masculine, and rugged than control boys, whereas girls with GID were judged to be significantly more masculine, rugged, and tomboyish in appearance than control girls ([McDermid et al., 1998](#)).

It should be noted that attractiveness need not be conceptualized as a fixed biophysical trait—social shaping of physical appearance is clearly possible. Clinical observations suggest that some parents subtly alter the physical appearance of children with GID so as to induce a "feminine" look in boys and a "masculine" look in girls. Moreover, some of the children themselves insist on altering their physical appearance. For example, some girls with GID insist on cutting their hair short and wearing clothing that will enable them to pass successfully as boys. Physical appearance, then, may be a predisposing factor in the development of GID, may serve to perpetuate the disorder, or may simply be one of the disorder's clinical signs.

### Psychosocial Mechanisms and Hypotheses

Psychosocial factors, to truly merit causal status, must be shown to influence the emergence of marked cross-gender behavior in the first few years of life. Otherwise, such factors would be better conceptualized as perpetuating rather than predisposing.

#### SEX ASSIGNMENT AT BIRTH

Because most children with GID do not have a co-occurring physical intersex condition, sex assignment at birth is invariably in accordance with the external markers of biological sex. In some physical intersex conditions, sex assignment is delayed and, on occasion, changed from the initial sex assignment. It has been argued that prolonged delay or uncertainty about the child's "true" sex can contribute to gender identity conflict in affected individuals ([Meyer-Bahlburg et al., 1996](#); [Money et al., 1957](#)). This does not, however, appear to be the situation for children with GID.

#### PRENATAL GENDER PREFERENCE

It is common for parents to express a prenatal gender preference. Other things being equal, parents will have a child of the nonpreferred sex about 50% of the time. Are parents of children with GID more likely than control parents to report having had a desire for a child of the opposite sex? The simple answer appears to be no, at least with regard to the mothers of boys with GID ([Zucker et al., 1994](#)). We did find, however, that the maternal wish for a girl was significantly associated with the sex composition and birth order of the sibship. Among the GID boys with only older brothers, the percentage of mothers who recalled a desire for a daughter was significantly higher than among the probands with other sibship combinations; however, the same pattern was observed in a control group ([Zucker et al., 1994](#)).

#### SOCIAL REINFORCEMENT OF CROSS-GENDER BEHAVIOR

Understanding the role of parent socialization in the genesis and/or perpetuation of GID (e.g., via reinforcement principles or modeling) has been influenced by the normative developmental literature on sex-dimorphic sex-typed behavior ([Ruble and Martin, 1998](#)). It has also been influenced by the seminal observations of [Money and colleagues \(1957\)](#) that the rearing environment was the predominant determinant of gender identity in children with physical intersex conditions.

It should be recognized that some critics are quite skeptical of the role of parent socialization in inducing sex differences in sex-typed behavior among ordinary children ([Lytton and Romney, 1991](#)) or within-sex variations. In recent years, the importance of the rearing environment has also been questioned within the literature on physical intersex conditions ([Zucker, 1999](#)). This literature has recently been discussed in relation to the long-term gender identity outcome of a normal boy, whose penis was accidentally ablated during a routine circumcision at 7 months, and subsequently reassigned as a girl around the age of 2. The patient apparently differentiated and maintained a female gender identity through at least age 9 years ([Money, 1975](#)). Longer-term follow-up, however, revealed that the patient reverted to living as a male in early adolescence ([Diamond and Sigmundson, 1997](#)), which has been interpreted by some to indicate a much stronger role for biological rather than psychosocial influences on gender identity differentiation. However, critics have pointed out that there are alternative interpretations to this case ([Meyer-Bahlburg, 1999](#)). Moreover, in another case of ablatio penis, in which a gender-reassignment to female occurred at 7 months, the patient's gender identity was judged to be unequivocally female at age 26 ([Bradley et al., 1998](#)). The literature on GID must be appraised against this backdrop of competing views on the role of socialization.

Clinicians of diverse theoretical persuasions have consistently reported that the parental response to early cross-gender behavior in children with GID is typically neutral (tolerance) or even encouraging ([Zucker and Bradley, 1995](#)). Regarding boys with GID, Green assessed parental recall of such responses taken from clinical and structured interviews at the time of assessment and concluded that "what comes closest so far to being a *necessary* variable is that, as any feminine behavior begins to emerge, there is *no* discouragement of that behavior by the child's principal caretaker" ([Green, 1974](#), p. 238, italics in original; [Green 1987](#); [Roberts et al., 1987](#)). In a structured interview study, [Mitchell \(1991\)](#) found that mothers of GID boys were more likely to tolerate or encourage feminine behaviors and less likely to encourage masculine behaviors than were the mothers of both clinical and normal control boys. The following vignette with parents of a 4-year-old boy is illustrative:

- Interviewer: What is the first memory that you have of Eric's interest in "girls' things?"  
(I):  
Mother (M): Well, when he was about 2, he started to wear my shoes. He would wear them every day.  
I: What did you think about that?  
M: I thought it was cute, the way he was clumping around in them.  
I: What about you (to father)?  
Father (F): I wasn't around much, working and all, so I didn't see much of it. She (his wife) told me about it, and I said that I didn't like it, but what can you do?  
I: What happened after that?  
M: Well, then he got interested in my dresses (laughs), and he looked so cute, running around in the heels and my long dress. We even took pictures of him like that.  
F: Yeah, right.  
I: Anything else?  
M: When I took him to the toddler group, he went immediately over to the girls and he got into the Barbie dolls. He seemed obsessed with their hair and we wound up buying him three or four of them. His favorite one is the Barbie with roller-blades. I asked his doctor about it when he was 3, and the doctor said that it was a phase and that he would grow out of it.  
F: Yeah, and then he was pretending that he was a girl all the time and said that he would grow up and be a mommy. And then he said that he wanted to cut off his penis.  
I: What happened when he said that?  
M: Well, that's why we're here. I'm getting worried that he won't grow out of it ... it's getting worse.  
I: Before he said that he wanted to cut off his penis, how do you think you have reacted to his interest in the girls' things over the past couple of years?  
M: Well, I've pretty well let him do what he wants, and, anyways, the doctor told me not to worry. I thought that if he played with the Barbies, maybe he'd be a good father, but he doesn't want to be the father; he's only pretending to be me.  
F: Yeah, I'm getting more worried myself, but my wife told me not to be so worried, and, well, you know, she's kind of the boss when it comes to raising the kids.

Of course, the limitations of this kind of interview data need to be recognized. Nonetheless, one aspect of these data deserves special comment. As noted, clinicians of diverse theoretical persuasions have observed the apparent tolerance, or even encouragement, of feminine behavior shown by parents of boys with GID. However, the fact that these parents have sought out a clinical assessment usually means that they are now concerned about their child's gender identity development ([Zucker, 2000](#)). From the standpoint of attribution theory ([Weiner, 1993](#)), one might predict that parents would minimize their encouragement or tolerance of cross-gender behavior, since it has such an obvious bearing on "causality." Yet a majority of the parents whom we have assessed do not recall systematic efforts to limit or redirect

their child's cross-gender behavior, particularly during the initial period of symptom onset and for various periods of time thereafter.

The reasons why parents might tolerate, if not encourage, early cross-gender behaviors appear to be quite diverse, suggesting that the antecedents to this "end state" are multiple in origin. As noted earlier, for example, some parents report being influenced by ideas regarding non-sexist child rearing. In other parents, the antecedents seem to be rooted in pervasive conflict that revolves around gender issues. For example, a small subgroup of mothers (about 10%) of boys with GID appear to experience something akin to what we have termed *pathologic gender mourning* (Zucker, 1996). During the pregnancy, there is a strong desire for a girl [in all of the cases, the mother had already borne at least one other son, but no daughter—except in three instances in which the daughter was given up for adoption (one case) or had died in infancy (two cases)]. After the birth of the "nonpreferred" son, this wish seems to color strongly the mother's perception and relationship with her newborn, and there are strong signs of ambivalence about his gender status. Zucker (1996) identified at least 10 possible signs of pathologic gender mourning, including severe postpartum depression related to the birth of a son, recurrent night dreams about being pregnant with a girl, delayed naming, and active cross-dressing of the boy (Zucker and Bradley 1995, 2000; Zucker et al., 1993a). The most common psychological trait associated with the strong wish for a daughter appears to be the need to nurture and be nurtured by a female child, which often reflects compensatory needs originating in childhood (Gibson, 1998).

#### PARENT-CHILD RELATIONSHIPS

Apart from attempts to examine the specific interactions that occur during the initial display of sex-dimorphic behavior, attempts have been made to examine the general quality of the relationships between gender-disturbed children and their parents.

In the case of boys, Stoller (1968, 1975, 1985) observed clinically an overly close relationship between mother and son and a distant, peripheral father-son relationship. He presented his hypothesis simply: "The more mother and the less father, the more femininity" (Stoller, 1985, p. 25). Stoller (1985) argued that GID in boys is a kind of "developmental arrest ... in which an excessively close and gratifying mother-infant symbiosis, undisturbed by father's presence, prevents a boy from adequately separating himself psychically from his mother's female body and feminine behavior" (p. 25). In several respects, Stoller's notions point to the crucial role of preoedipal events in the development of GID, a view that is shared by many other contemporary psychoanalytic clinicians and theorists.

Green (1987; Green et al., 1985; Roberts et al., 1987) had the parents of feminine boys and control boys rate the amount of "shared time" with their sons during the first 5 years of life. Contrary to prediction, the mothers of feminine boys recalled spending less time with their sons compared with the amount of time the mothers of control subjects remembered spending. Consistent with prediction, the fathers of feminine boys recalled spending less time with their sons from the second to fifth year compared with the amount of time the fathers of control subjects remembered and the father's shared time with masculine male siblings. Thus, this method of assessing parent-son relationships confirmed the paternal side of the equation but yielded results opposite from prediction for the maternal side. Thus, at least with regard to the mother, the quality of the mother-child interaction may have been more important than the sheer amount of time that mother and child spent together.

Despite Green's (1974, 1987) finding regarding shared time, there is little question that boys with GID feel closer to their mothers than to their fathers. In part, this may be a function of a boy's perception of similarity to the mother, but it also evolves from the day-to-day subtleties of parent-child interaction.

Unfortunately, no systematic studies on parent-child relationships have been conducted for girls with GID. Preliminary clinical studies suggest that for such girls the mother-daughter relationship is often impaired, leading to what might be described as a "disidentification" from the mother (Green, 1974; Stoller, 1975; Zucker and Bradley, 1995). During the early years of these girls, a variety of factors appear to impair the development of a close mother-daughter relationship; as a result, there is a devaluing of femininity and an overvaluing of masculinity, outcomes that the parents seem to encourage. These preliminary clinical studies suggest, therefore, that the quality of parent-child relationships in gender-disturbed boys is quite different from that in gender-disturbed girls.

#### GENERAL PSYCHOPATHOLOGY

The role of maternal psychopathology in the genesis and perpetuation of GID has received a great deal of clinical and theoretical attention but, unfortunately, only limited empirical evaluation. At the outset, it should be noted that the available empirical studies have been delimited to the mothers of boys with GID—comparable studies are not available regarding the mothers of girls with GID (Zucker and Bradley, 1995).

Marantz and Coates (1991) found that the mothers of boys with GID showed more signs of psychopathology than did the mothers of demographically matched normal boys, including more pathologic ratings on the Diagnostic Interview for Borderlines and more symptoms of depression on the Beck Depression Inventory.

Over the past several years, the author has collected systematic data on maternal psychopathology and marital discord, some of which was reported by Mitchell (1991) and Zucker and Bradley (1995). To date, the data show that, on average, mothers of boys with GID have levels of emotional distress and psychiatric impairment comparable to that of clinical control mothers, but higher than that of normal control mothers. There was not, however, a between-group difference on a measure of marital discord.

On one measure—the Symptom Checklist 90-Revised—mothers of GID boys had higher scores on most of the subscales and the composites than did the mothers of the normal control boys, whereas the scores of the clinical control mothers fell in between the two other groups. The GID mothers had peak scores on the obsessive-compulsive, depression, and hostility subscales. On another measure, the Diagnostic Interview Schedule (DIS), 30% of the mothers (total  $N = 140$ ) had two DIS diagnoses, and 24% had three or more DIS diagnoses. The most common diagnoses were major depressive episode (39.6%) and recurrent major depression (32.1%). Overall, the rate of psychiatric impairment appears to be higher than our available data on mothers of both the clinical control and normal boys. Because more control group mother data are required, these results should, of course, be viewed cautiously. Nonetheless, it is apparent by reference to epidemiologic data that, on average, mothers of GID boys have a history of elevated psychiatric disorder.

The emerging data on emotional distress and psychiatric impairment in the mothers of boys with GID indicate that it is more common than in the mothers of normal control boys and at least comparable to the mothers of clinical control boys. Still we are left with the problem of specificity (Garber and Hollon, 1991), in that these maternal characteristics are not unique to the mothers of GID boys, but common to the mothers of clinic-referred boys in general. Accordingly, maternal emotional distress/impairment functions, at best, only as a nonspecific risk factor in the development of GID. If the mother's emotional state truly is involved in the genesis of GID, then there should be evidence of psychiatric impairment prior to and during the emergence of the child's symptoms. The data are suggestive that this is the case, and that the presence of emotional difficulties in the mothers is not simply a reaction to having a child with GID (Zucker and Bradley 1995).

Coates and her colleagues have argued that the presence of psychopathology renders the mothers emotionally unavailable, which results in anxiety and insecurity in the son, and that it is this state of affairs that is partly responsible for symptom onset. Indeed, Coates and Person (1985) advanced a very specific hypothesis, namely that the erratic and uneven emotional availability of the mothers activated separation anxiety in the boys, which, in turn, activated the symptoms of GID: "In imitating 'Mommy' [the boy] confuse[s] 'being Mommy' with 'having Mommy.' [Cross-gender behavior] appears to allay, in part, the anxiety generated by the loss of the mother" (Coates and Person, 1985, p. 708). Indeed, boys with GID appear to have high rates of separation anxiety traits, as judged by maternal report on a structured interview schedule (Zucker et al., 1996) and their own responses on the Separation Anxiety Test (Birkenfeld-Adams, 1999).

The possible role of separation anxiety in the genesis of GID raises more general questions about the quality of the mother-son relationship. Over the past few years, the author's group has studied the quality of attachment to the mother among young boys and girls with GID (ages 3 to 6). Among a sample of 22 boys, Birkenfeld-Adams (1999) found that the majority (73%) were classified as insecurely attached, a rate comparable to that of an internal clinical control group and of other studies of clinical populations (Greenberg et al., 1991).

Because insecure attachments and separation anxiety are likely nonspecific risk factors (i.e., many boys who have these qualities do not have GID), the crucial question that remains is why only a small minority of boys develop the "fantasy solution" of wanting to be a girl. Various predisposing factors have been implicated, including temperamental characteristics of the child, the premorbid relationship with the mother, the position of the father in the family system, that the family psychopathology must occur during the putative sensitive period for gender identity formation (Money et al., 1957), and so on. At present, however, the question of specificity remains unanswered in any satisfactory manner.

One possible mediating variable might be the importance of the child's gender to the mother or her attitudes toward men and masculinity in general. In this regard, pathologic gender mourning, as discussed earlier, may be a potential prototype. As noted, pathologic gender mourning appears to be part of the family history in only a small minority of cases; thus, other pathways are required to account for the role of maternal impairment in the genesis of GID.



It appears therefore that there are diverse pathways that lead to how parents respond to the child's early cross-gender behavior (either by encouraging or tolerating it). Thus, from a clinical and therapeutic point of view, it is important to identify the putative motivations with regard to the selective reinforcement of sex-typed behaviors.

The role of paternal influences in the genesis and perpetuation of GID has also received a great deal of clinical and theoretical attention but, again, only limited empirical evaluation, which has been delimited to the fathers of boys with GID.

One account implicates the father's role by virtue of his absence from the family matrix. Across 10 samples of boys with GID, the rate of father absence (e.g., owing to separation or divorce) was 34.5% ([Zucker and Bradley, 1995](#)). It is unlikely, however, that this rate would differ significantly from the rate found in clinical populations in general, if not the general population. [Green \(1987\)](#) found that paternal separations occurred earlier in the families of GID boys than normal control boys; therefore, it is possible that timing is an additional variable to consider. [Green \(1987\)](#) also found that the fathers of GID boys (both father-present and father-absent) recalled spending less time with their sons than did the fathers of control boys during the second year of life, years 3 to 5, and at the time of assessment. The inclusion of a clinical control group would be helpful in gauging the specificity of this finding.

Unfortunately, there is little in the way of systematic research on paternal psychopathology. [Wolfe \(1990\)](#) conducted a small but detailed study of 12 fathers, predominantly of an upper middle-class background, of boys with GID. On the Structured Clinical Interview for DSM-III, all of the fathers received an Axis I diagnosis for either a current or past disorder (most frequently substance abuse and depression) and eight also received at least one Axis II diagnosis. Unpublished DIS data from the author's clinic on 90 fathers indicate that alcohol abuse (22.2%) has been the most common diagnosis. This percentage may underestimate the prevalence of alcohol abuse in fathers of GID boys because we were not able to interview a substantial number of fathers who were no longer part of the family matrix. On the other hand, it should be noted that about half the fathers who were interviewed did not meet criteria for any DIS diagnosis. Whatever the exact patterning of paternal emotional functioning proves to be, the same issues of interpretation discussed earlier regarding mothers apply.

## LABORATORY STUDIES

### Biological Assessment

There are no known biomedical tests that can identify children with GID. As noted, various parameters of biological sex, such as the sex chromosomes and the appearance of the external genitalia, are invariably normal ([Green, 1976](#)). As noted in the section on differential diagnosis, children with certain physical intersex conditions may be at some risk for gender dysphoria. For these youngsters, pediatric endocrinologic and urologic or gynecologic evaluations are warranted, because certain biomedical treatments may be required; however, it is rare that such conditions have not already been identified prior to psychiatric evaluations.

### Psychological Assessment

A number of parent-report and behavioral measures can be used to assess sex-typed behavior in children with GID ([Green, 1974](#); [Zucker, 1992](#); [Zucker and Bradley, 1995](#)). From a diagnostic standpoint, it should be recognized that no one test is a replacement for a diagnosis that is established by a clinical interview that covers the behavioral signs for GID. Nevertheless, these measures have strong discriminant validity and constitute one strong line of evidence that GID is, in fact, a distinct syndrome. [Green \(1987\)](#), for example, found that a discriminant function analysis of parent interview data required 6 of 16 sex-typed behaviors to classify correctly all boys as members of either the group of boys with GID or the control group of boys. As reviewed elsewhere, data from psychological tests, in general, show a consistent pattern in that the percentage of false negatives appears to be higher than the percentage of false positives ([Zucker, 1992](#); [Zucker and Bradley, 1995](#)).

## DIFFERENTIAL DIAGNOSIS

### Childhood Diagnostic Issues

During childhood, several differential diagnostic issues should be considered in relation to GID. There is a type of cross-dressing in boys that appears to be qualitatively different from the type of cross-dressing that characterizes GID. In the latter, cross-dressing typically involves outerwear, such as dresses, shoes, and jewelry, that helps enhance the fantasy of being like the other sex. In the former, cross-dressing involves the use of undergarments (e.g., panties and nylons). Clinical data show that other signs of cross-gender identification do not accompany such cross-dressing; in fact, the appearance and behavior of boys who engage in it are conventionally masculine. Clinical experience suggests that this type of cross-dressing has some sort of self-soothing function. Many male adolescents and adults who display transvestic fetishism recall such cross-dressing during childhood ([Zucker and Blanchard, 1997](#)). It should be noted, however, that prospective study has not verified the assumption that this behavior pattern is contiguous with later transvestism.

When all the clinical signs of GID are present, it is not difficult to make the diagnosis. But the clinician who accepts the notion that there is a spectrum of cross-gender identification must be prepared to identify what Meyer-Bahlburg described as the "zone of transition between clinically significant cross-gender behavior and mere statistical deviations from the gender norm" ([Meyer-Bahlburg, 1985](#), p. 682). Clinical experience suggests that boys who fall into this ambiguous zone do poorly in male peer groups, avoid rough-and-tumble play, are disinclined toward athletics and other conventionally masculine activities, and feel somewhat uncomfortable about being male; however, these boys do not wish to be girls and do not show an intense preoccupation with femininity. [Friedman \(1988\)](#) coined the term *juvenile unmasculinity* to describe such boys, who, he argued, suffer from a "persistent, profound feeling of masculine inadequacy which leads to negative valuing of the self" (p. 199). It is not clear whether this behavior pattern actually constitutes a distinct syndrome or is simply a mild form of GID; in any case, the residual diagnosis gender identity disorder not otherwise specified (GIDNOS) could be employed in such cases.

In girls, the primary differential diagnostic issue concerns the distinction between girls with GID and tomboys. The study of a community sample of tomboys by [Green and colleagues \(1982\)](#) showed that these girls shared a number of the cross-gender traits observed in clinic-referred gender-disturbed girls ([Zucker and Bradley, 1995](#)). In part, the DSM-III-R criteria for GID in girls were modified in the hope of better differentiating these two groups of girls ([Zucker, 1982](#)). At least three characteristics may be most useful in making the differential diagnosis: (a) By definition, girls with GID indicate an intense unhappiness with their status as females, whereas this should not be the case for tomboys; (b) girls with GID display an intense aversion to the wearing of culturally defined feminine clothing under any circumstances, whereas tomboys do not manifest this reaction, although they may prefer to wear casual clothing, such as jeans; and (c) girls with GID, unlike tomboys, manifest a verbalized or acted-out discomfort with sexual anatomy.

The final differential diagnostic issue concerns children with physical intersex conditions. In DSM-III, an intersex condition was an exclusion criterion for the adult diagnosis of transsexualism, but this criterion was eliminated in DSM-III-R. The DSM-III-R did not formally address the issue of whether children who display significant cross-gender identification and have an intersex condition should be given a diagnosis such as GID. Consider, for example, girls with CAH, in which there is excessive prenatal exposure to androgens. On average, these girls display more masculine behavior than do nonaffected girls ([Berenbaum and Hines, 1992](#)). A small percentage of these girls also appear to be gender dysphoric ([Zucker, 1999](#)). The phenomenology in these cases appears similar to that observed in physically normal girls with GID, but the etiology may well be different ([Meyer-Bahlburg, 1994](#)). Thus, there may be a risk in applying the same diagnosis as that used with physically normal children. On the other hand, the risk is reduced if it is recognized that a particular diagnosis does not dictate identical treatment across cases. The DSM-IV Subcommittee on Gender Identity Disorders debated these issues ([Bradley et al., 1991](#); [Meyer-Bahlburg, 1994](#)). The final decision was to not diagnose GID in children or adults with a co-occurring physical intersex condition.

### Adolescent Diagnostic Issues

During adolescence, the clinician is likely to encounter at least four types of psychosexual problems ([Sullivan et al., 1995](#); [Zucker and Bradley, 1995](#)). Children with an unresolved GID are at risk for seeking hormonal and surgical solutions to their distress. The DSM-IV criteria for GID for adolescents or adults emphasize the persistence of cross-gender identification and discomfort with one's assigned sex, including the desire for hormonal or surgical sex-reassignment. Diagnostically, it is important to assess the strength and persistence of the desire to change sex, because clinical management decisions are influenced by the relative intractability of the condition. The residual diagnosis GIDNOS can be used for clinically gender dysphoric individuals who do not meet the complete diagnostic criteria for GID.

A second type of psychosexual problem occurs among adolescents who have had a history of GID or a variation of it. These adolescents continue to show signs of cross-gender identification but do not acknowledge a homosexual orientation or profess a desire to change sex. They are often referred because of continued social ostracism. The degree of felt distress varies with regard to the continued cross-gender identification. It is unlikely that any formal DSM-IV diagnosis would apply in

such cases, although the residual diagnosis GIDNOS could be employed to indicate that the adolescent continues to struggle with gender identity issues.

A third type of psychosexual problem characterizes adolescents who have been referred by either themselves or by significant others because of homosexual behavior or orientation. Some of these youngsters have a history of GID or a variation of it. The reason for referral varies, but from a differential diagnostic standpoint it is important to rule out continuing problems that center on gender identity. For those youngsters who are distressed about their sexual orientation, the diagnosis of sexual disorder NOS can be given.

The last type of psychosexual problem concerns adolescent boys who cross-dress, in part, for the purpose of sexual arousal. The extent of the cross-dressing varies. In its full form, the diagnosis of transvestic fetishism can be applied. These boys appear to have a nascent heterosexual orientation and appear unremarkably masculine in their demeanor. A history of GID is not part of the clinical picture, but some of these boys think about sex-reassignment surgery and are at risk for transsexualism. In DSM-IV, adolescents who meet the criteria for GID with this type of developmental history typically have a heterosexual sexual orientation.

## TREATMENT

Several therapeutic approaches have been employed to treat children with GID, including behavior therapy, psychotherapy, family therapy, parental counseling, group therapy, and eclectic combinations of these strategies ([Green 1974, 1987](#); [Zucker 1985, 1990](#); [Zucker and Green, 1989](#)). As reviewed elsewhere ([Zucker, 1985](#); [Zucker and Green, 1989](#)), all of these strategies appear to have clinical utility; unfortunately, formal comparative studies have not been conducted; therefore, the most efficacious types of treatment remain unclear. (For specific references to the case report literature, see [Zucker, 2001](#); [Zucker and Bradley, 1995](#).)

Three general comments about treatment of GID will be made. First, clinical experience suggests that intervention can more readily reduce gender identity conflict during childhood than adolescence. The prognosis for reducing severe gender dysphoria after puberty is rather poor. Accordingly, the earlier treatment begins the better. Second, the importance of working with the parents of children with GID has been much discussed in the literature. When there is a great deal of marital discord and parental psychopathology, treatment of these problems greatly facilitates more specific work on gender identity issues. Management of the child's gender behavior in his or her daily environment requires that the parents have clear goals and a forum in which to discuss difficulties. Because parental dynamics and ambivalence about treatment may contribute to the perpetuation of GID ([Newman, 1976](#)), it is important for the therapist to have an appropriate relationship with the parents in order to address and work through these issues. Last, the therapist needs to consider closely the goals of treatment ([Coates and Zucker, 1988](#); [Green 1974, 1987](#); [Zucker 1985, 1990, 2001](#); [Zucker and Green, 1989](#)). In part, this issue is conceptualized within the therapist's theoretical framework, but is also a function of the parents' concern and, to some extent, of the child's concerns. Two short-term goals have been discussed in the clinical literature: The reduction or elimination of social ostracism and conflict and the alleviation of underlying or associated psychopathology. Longer-term goals have focused on the prevention of postpubertal gender dysphoria and homosexuality. Little disagreement about the advisability of preventing gender dysphoria in adolescence or adulthood has been expressed in the clinical literature. Contemporary and secular-minded clinicians are, however, sensitive to the importance of helping people integrate a homosexual sexual orientation into their sense of identity ([Friedman, 1988](#); [Green, 1987](#)). Not surprisingly, however, most parents of children with GID probably prefer the development of a heterosexual orientation. It is, therefore, important that clinicians point out to such parents that, as of yet, there is no strong evidence that treatment affects later sexual orientation. The author, as well as other experienced clinicians in the field, has preferred to emphasize the merit of reducing childhood gender identity conflict per se and, when it is present, associated behavioral problems, and to orient the parents of children with GID to the short-term goals of intervention.

## OUTCOME AND FOLLOW-UP DATA

[Green \(1987\)](#) has conducted the most comprehensive long-term follow-up study of boys with GID. An original sample of 66 feminine and 56 control boys was assessed initially at a mean age of 7.1 years (range, 4 to 12), and about two-thirds of the boys in each of these groups were reevaluated at a follow-up mean age of 18.9 years (range, 14 to 24). A semistructured interview was employed to assess sexual orientation in fantasy and behavior, using Kinsey scale criteria ([Green, 1987](#)). All 35 control boys were heterosexual in fantasy at follow-up; of the 25 control boys who had had overt sexual experiences, one was classified as bisexual, and the remainder were classified as heterosexual. In contrast, 75% (33 of 44) of the GID boys were classified as either bisexual or homosexual in fantasy; of those who had had overt sexual experiences, 80% (24 of 30) were classified as either bisexual or homosexual. The remaining boys were classified as heterosexual. One of the GID boys, who was erotically attracted to males, was seriously entertaining the notion of sex-reassignment surgery.

[Green's \(1987\)](#) controlled follow-up study has shown that the most common long-term outcome of GID is homosexuality, a finding that has been reported in other studies, albeit without comparison groups ([Zucker, 1985](#)). As discussed in more detail elsewhere ([Green, 1987](#); [Zucker 1985, 1990](#)), these data converge neatly with the retrospective studies of adult male homosexuals that have evaluated the presence of childhood cross-gender behavior ([Bailey and Zucker, 1995](#)).

Adults with GID, particularly those with a homosexual orientation (i.e., erotic attraction to persons of the same biological sex), invariably recall a childhood cross-gender history. Prospective studies of children with GID, mainly boys, have found that only a very small proportion persist in their wish to change sex after puberty, although the percentage does appear considerably higher than what one would expect based on general population rates for transsexualism. Why is this? As noted elsewhere ([Zucker, 1985](#)), there are two main possibilities. One possibility is that the low base rate of adult GID, even within the population of children with GID, would require a large sample size to identify the few cases of gender dysphoria; the other possibility is that the assessment process and therapeutic intervention (when it occurs) alter the natural history of cross-gender identification. It should be noted that these two explanations are not mutually exclusive.

Clearly, childhood cross-gender identification is a behavioral marker for either later homosexuality or unresolved adult GID. Can more fine-grained predictions be made (e.g., predicting later homosexuality versus GID; homosexuality versus heterosexuality)? Clinical experience has suggested that children who do not move away from extreme cross-gender identification as they enter adolescence may be at greater risk for later gender dysphoria. [Zucker and associates \(1986\)](#) found that the persistence of gender dysphoria, including the desire to change sex, was considerably higher among patients who were first assessed during adolescence and followed-up later, compared with patients who were first assessed during childhood and followed-up later. This suggests that the clinical situation at or near the transition from childhood to adolescence may be crucial in differentiating a gender dysphoric outcome from other outcomes.

[Green \(1987](#); [Green et al., 1987](#)) compared a number of childhood variables in the feminine boys who were subsequently classified as bisexual or homosexual with the feminine boys who were subsequently classified as heterosexual. Although some feminine behaviors distinguished the two subgroups, a composite childhood femininity score only approached conventional levels of significance and only for the rating of sexual orientation in fantasy, not behavior. The lack of a stronger correlation is somewhat surprising because one might have expected that the degree of cross-gender identification would be related to long-term outcome. However, Green did find that the continuation of certain feminine behaviors throughout childhood was associated with later homosexuality. Thus, it may be that the persistence of the feminine behavior is more important than its extent during the early childhood years.

Less father-son shared time in the first 2 years of life was also associated with later homosexuality, but the amount of mother-son shared time was not. Involvement in treatment also did not distinguish the two subgroups. It should be noted, however, that Green's study was not formally designed to assess the effects of therapy. Cases were not randomly assigned to receive or not receive treatment, and the therapy that occurred was quite variable with regard to length, type, experience of the therapist, and so on.

## RESEARCH DIRECTIONS

Over the past 40 years, considerable progress has been made in demarcating the phenomenology of GID. Assessment procedures for a proper diagnostic work up are now widely available ([Zucker, 1992](#)). The strong connection between GID and later homosexuality has been verified by prospective study. Treatment appears to be capable of interrupting the natural progression toward adult GID, but it is far less clear whether treatment can (or should) influence later sexual orientation.

The biology of gender identity, gender role, and sexual orientation remains an area of intense inquiry. Continued study of the effects of prenatal sex hormones on postnatal sex-dimorphic behavior should help clarify the boundaries of their predisposing influence ([Collaer and Hines, 1995](#)). Research over the past decade has begun to identify some characteristics of children with GID, particularly boys, which may well have a specific biological basis. Corresponding studies of girls have been fewer in number, largely because of problems in sample size that limit statistical power. In many respects, it has been easier to rule out candidate biological explanations, such as the influence of gross anomalies in prenatal hormonal exposure, than it has been to identify the relevant biological mechanisms that are involved in affecting sex-dimorphic behavioral differentiation, but not sex-dimorphic genital differentiation. However, the identification of new potential biological markers may open avenues for further empiric inquiry.

Data on psychosocial influences have been able to rule out some hypothesized pathways and have lent some support for others. Social reinforcement of



cross-gender behavior when it first appears in the toddler and preschool years appears to be the most common psychosocial influence on the disorder's consolidation. The role of family influences, including parental psychiatric impairment and emotional distress, appears to be an important area for further empiric inquiry.

The relation between GID and sexual orientation calls for a better understanding of the development of eroticism in its own right, including its development during the years prior to puberty (Bem, 1996, 2000a, 2000b; Green, 1987; Herdt and McClintock, 2000). How do children eroticize stimuli? How is meaning given to the state of sexual arousal? How fixed or flexible is sexual orientation? Over the past few decades, and even in the short time since previous editions of this volume (Lewis, 1991, 1996), important advances have occurred in our understanding of psychosexual differentiation and its disorders. In the third millennium, there will be more to come.

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# 59 TIC DISORDERS

James F. Leckman, Bradley S. Peterson, and Donald J. Cohen

- Definitions
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- Clinical Descriptions
  - Transient Tic Disorder
  - Chronic Motor or Vocal Tic Disorder
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Tic disorders are transient or chronic conditions associated with difficulties in self-esteem, family life, social acceptance, or school or job performance that are directly related to the presence of motor and/or phonic tics. Individuals with tic disorders may manifest a broad array of behavioral difficulties, including uninhibited speech or conduct, impulsivity, distractibility, motoric hyperactivity, and obsessive-compulsive symptoms. Some of these associated “non-tic” symptoms may be a major source of impairment.

Historically, tic disorders have been the province of both neurologists and psychiatrists. They are remarkable disorders that provide a glimpse into some of the processes that govern the interface between mind and body. The psychological dimension is offered not only by the associated behavioral difficulties but also by the “choice” and timing of the tic behaviors themselves.

Although tic symptoms have been reported since antiquity, systematic study dates only from the 19th century, with the reports of [Itard \(1825\)](#) and [Gilles de la Tourette \(1885\)](#). Gilles de la Tourette, in his classic study of 1885, described nine cases of tic disorder characterized by motor “incoordinations” or tics, and “inarticulate shouts accompanied by articulated words with echolalia and coprolalia.” This report also hinted at the hereditary nature of the syndrome and the association between tic disorders and obsessive-compulsive symptoms.

The current classification of tic disorders in *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV) includes Tourette's disorder, chronic motor or vocal tic disorder, transient tic disorder, and tic disorder not otherwise specified ( [American Psychiatric Association, 1994](#)). The diagnostic criteria for these conditions are presented in [Table 59.1](#). These criteria focus on the phenomenology and natural history of the disorder and are readily applied in clinical settings. They also serve as the basis for the diagnostic descriptions contained in the [World Health Organization's International Classification of Diseases \(ICD-10\) \(1988\)](#).

Table 59.1. DSM-IV Tic Disorder Classification

## DEFINITIONS

A tic is a sudden, repetitive movement, gesture, or utterance that typically mimics some aspect of normal behavior. Usually of brief duration, individual tics rarely last more than a second. They tend to occur in bouts and, at times, have a paroxysmal character. Individual tics can occur singly or together in an orchestrated sequence. Tics vary in their frequency and forcefulness. Many tics can be temporarily suppressed. Young children experience their tics as being involuntary. Older children and adults, however, often describe their tics as being associated with unwanted somatosensory “urges” that are momentarily relieved with the performance of the tic ([Bliss, 1980](#); [Leckman et al., 1993](#)). Motor tics vary from simple, abrupt movements, such as eye blinking, head jerks, or shoulder shrugs, to more complex, purposeful-appearing behaviors, such as facial expressions or gestures of the arms or head. In extreme cases, these movements can be obscene (copropraxia) or self-injurious (e.g., hitting or biting). Phonic or vocal tics can range from simple throat-clearing sounds to more complex vocalizations and speech. In severe cases, coprolalia (obscene speech) is present.

## PREVALENCE AND EPIDEMIOLOGY

Tic behaviors are commonplace among children. Community surveys indicate that 1% to 13% of boys and 1% to 11% of girls manifest “frequent tics, twitches, mannerisms, or habit spasms” ([Zohar et al., 1998](#)). The instability of these estimates owes in part to the timing of the interviews in relation to the waxing and waning course of tic symptoms, wording of items on symptom inventories, identity of the informant, and demographic characteristics of the sample studied. Children between the ages of 7 and 11 years appear to have the highest rates. Although boys are more commonly affected with tic behaviors than are girls, the male:female ratio in most community surveys is less than 2:1. Urban living may be associated with elevated rates ([Zahner et al., 1988](#)). Although race and socioeconomic status have not been shown to influence the point prevalence of tics, some authors have suggested that tic symptoms may be more common among white and Asian racial groups.

Much less is known concerning the prevalence of tic disorders per se. Once thought to be rare, current estimates from registers of clinically diagnosed subjects suggest that 1% to 2% of the general population may be affected. Unfortunately, these estimates are preliminary, because only a few rigorous population-based studies have been performed. For example, [Apter and colleagues \(1993\)](#) screened 28,037 16- to 17-year-old individuals prior to their induction into the Israeli army, and reported a point prevalence of 4.3 per 10,000 for Tourette's disorder. Although the rate of any tic disorder was not reported, their data suggest that this figure



would be approximately an order of magnitude higher—in the range of 47 per 10,000. Using a similar design, [Costello and associates \(1996\)](#) examined 4,500 children 9, 11, and 13 years of age in rural North Carolina. The children were directly examined, as was one parent; both were asked about symptoms during the previous 3-month period. Altogether, 10/10,000 children met criteria for Tourette's syndrome, 13/10,000 for boys and 7/10,000 for girls. These figures are consistent with the view that children are more likely to be identified as having a tic disorder than adults and that males are more commonly affected than females (with male:female ratios of 9:1 and 3:1 for children and adults, respectively) (Burd et al., [1986a](#), [1986b](#)).

The true prevalence rate for Tourette's disorder, however, may be substantially higher based on studies that have used longitudinal classroom monitoring techniques. For example, [Mason and coworkers \(1998\)](#) completed a study of all 13- to 14-year old pupils in a mainstream secondary school in the United Kingdom. Students were investigated using a two-stage procedure employing standardized questionnaires completed by parents, teachers, and pupils. Personnel trained to identify tics carried out classroom observations. Those pupils identified as having tics underwent a semistructured interview to determine whether they had Tourette's syndrome (TS). Five subjects were identified as having TS, yielding a prevalence estimate of 299 per 10,000 pupils in this age group. If confirmed, these results suggest that TS in the community as a whole is more common and milder than earlier population-based studies suggest.

## CLINICAL DESCRIPTIONS

Relatively few longitudinal studies of tic disorders have been performed with the exception of TS ( [Bruun, 1988](#); [Goetz et al., 1992](#); [Leckman et al., 1998b](#); [Shapiro et al., 1988](#)). Thus, most of the information provided in the following is based on cross-sectional studies and anecdotal clinical experience.

### Transient Tic Disorder

Almost invariably a disorder of childhood, transient tic disorder is usually characterized by one or more simple motor tics that wax and wane in severity over weeks to months. The anatomic distribution of these tics is usually confined to the head, neck, or upper extremities. More rarely transient phonic tics, in the absence of motor tics, can occur. The age of onset is typically 3 to 8 years. Boys are at greater risk. The initial presentation may be unnoticed. If medical consultation is sought, family practitioners, pediatricians, allergists, and ophthalmologists are typically the first to see the child. Missed diagnoses are common, particularly because the symptoms may have completely disappeared by the time of the consultation. As prescribed by the prevailing diagnostic criteria, the subsequent natural history of this condition is limited to fewer than 12 consecutive months of active symptomatology. As such, this is often a retrospective diagnosis because the clinician is unable to know with certainty which children will show progression of their symptoms and which children will display a self-limiting course.

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#### CASE ILLUSTRATION

Todd is a 6-year-old boy with a history of intense eye blinking of 2 weeks duration. The symptom was noticeable to others and interfered with his ability to maintain visual attention. The symptoms began toward the end of a family vacation, shortly before Todd was to enter first grade. By the time a visit to the family pediatrician was arranged, the symptoms had diminished considerably. In light of a positive family history of tic disorder in the father and an older sibling, the pediatrician tempered his reassurance but indicated to the family that the condition might well be transient in nature. The family was counseled to avoid central nervous system (CNS) stimulants, decongestants, and other sympathomimetics.

#### Comment

Although the value of avoiding CNS stimulants has not been convincingly demonstrated in prospective studies, such recommendations are commonplace and may have merit in light of the available anecdotal literature. Recently, systematic studies of the use of stimulants in children with Tourette's disorder have rekindled this controversy ([Sverd et al., 1999](#)) and have shown that short-term treatment with stimulants is not usually associated with increased tic symptoms. Definitive longer-term studies have not been reported.

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### Chronic Motor or Vocal Tic Disorder

This chronic condition can be observed among children and adults. As with other tic disorders, a waxing and waning course and a broad range of symptom severity characterize this condition. Chronic simple and complex motor tics are the most common manifestations. A majority of tics involve the head, neck, and upper extremities. Although some children may display other developmental difficulties, such as attention deficit hyperactivity disorder (ADHD), the disorder is not incompatible with an otherwise normal course of development. This condition also can appear as a residual state, particularly in adulthood. In such instances, a predictable repertoire of tic symptoms may only be seen during periods of heightened stress or fatigue.

Chronic vocal tic disorder, by all accounts, is a rare condition. Some authors exclude "chronic cough of adolescence" from this category ( [Shapiro et al., 1988](#)).

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#### CASE ILLUSTRATION

Richard is 16-year-old sophomore in high school. His coach observed chronic motor tics during baseball practice. These included a mouth stretching movement and a head tilt that occurred together at times. The coach was impressed by how little awareness Richard seemed to have concerning these movements despite their disfiguring character and the occasional teasing they prompted by his teammates. When approached, Richard's mother indicated that tic symptoms had been present off and on since he was 7 years old and that the family had noticed that they had been more frequent as he approached the end of the school term. She mentioned that they were in regular contact with a clinic and that, thus far, no medications had been used to control the symptoms.

#### Comment

A conservative approach (as illustrated in this vignette) is often indicated given the potential unwanted physical and psychological effects of many of the medications used to treat tic disorders.

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### Tourette's Syndrome (Chronic Motor and Phonic Tic Disorder)

The most severe tic disorder is best known by the eponym, "Gilles de la Tourette's syndrome." Typically, the disorder begins in early childhood with transient bouts of simple motor tics such as eye blinking or head jerks. These tics initially may come and go, but eventually they become persistent and begin to have adverse effects on the child and his or her family. The repertoire of motor tics can be vast, incorporating virtually any voluntary movement by any portion of the body. Although some authors have drawn attention to a "rostral-caudal" progression of motor tics (head, neck, shoulders, arms, torso), this course is not predictable. As the syndrome develops, complex motor tics may appear. Often they have a "camouflaged" appearance (e.g., brushing hair away from the face with an arm) and can only be distinguished as tics by their repetitive character. Rarely, complex motor tics can be self-injurious and further complicate management (e.g., punching one side of the face or biting a wrist). Single tics may occur in isolation, or there may be well-orchestrated combinations of motor tics that involve multiple muscle groups.

On average, phonic tics begin 1 to 2 years after the onset of motor symptoms and are usually simple in character (e.g., throat clearing, grunting, squeaks). More complex vocal symptoms, such as echolalia, palilalia, and coprolalia occur in a minority of cases. Other complex phonic symptoms include dramatic and abrupt changes in rhythm, rate, and volume.

Motor and phonic tics tend to occur in bouts. Their frequency ranges from nonstop bursts that are virtually uncountable to rare events that occur only a few times a week. The timing of tics appears to have a fractal character in which bouts of tics occur in bouts and bouts-of-bouts of tics occur in bouts ( [Peterson and Leckman, 1998](#)). This pattern of self-similarity over differing time scales is characteristic of fractals and suggests that the same multiplicative processes underlying the occurrence of bouts of tics may govern the basic neurobiological systems that generate tics at shorter time intervals (milliseconds), as well as the waxing and waning course over longer time intervals (months).

The forcefulness of motor tics and the volume of phonic tics can also vary tremendously, from behaviors that are not noticeable (a slight shrug or a hushed guttural noise) to strenuous displays (arm thrusts or loud barking) that are frightening and exhausting.

Consistent with available epidemiologic data, tic disorders tend to improve in late adolescence and early adulthood ( [Leckman et al., 1998b](#)). In many instances, the phonic symptoms become increasingly rare or may disappear altogether, and the motor tics may be reduced in number and frequency. Complete remission of both motor and phonic symptoms has also been reported ([Bruun, 1988](#); [Shapiro et al., 1988](#)). In contrast, adulthood is also the period when the most severe and debilitating forms of tic disorder can be seen. In addition to the tic behaviors, associated behavioral and emotional problems frequently complicate Tourette's

syndrome. These difficulties range from impulsive, “disinhibited,” and immature behavior to compulsive touching or sniffing. There is no clear dividing line between these abrupt and disruptive behaviors and complex tics on the one hand and comorbid conditions of ADHD and obsessive-compulsive disorder (OCD) on the other.

Although most children with TS are loving and affectionate, maintaining age-appropriate social skills appears to be a particularly difficult area for many patients with TS ([Bawden et al., 1998](#); [Dykens et al., 1998](#); [Stokes et al., 1991](#)). Whether this is caused by the stigmatizing effects of the tics, the patients' own uneasiness, or some more fundamental difficulty linked to the neurobiology of this disorder is unknown. Coexisting ADHD, if present, can aggravate these difficulties ([Bawden et al., 1998](#); [Dykens et al., 1998](#); [Spencer et al., 1998](#)). Similarly, children with autism and other pervasive developmental disorders are at higher risk for developing TS ([Baron-Cohen et al., 1999](#)).

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#### CASE ILLUSTRATION

Arthur is an 11-year-old prepubertal boy who was referred for psychiatric evaluation and possible placement in a residential treatment facility. His past psychiatric history included symptoms suggestive of ADHD from an early age and the presence of motor and phonic tics that had increased steadily over the past 2 years. The immediate reason for referral was the emergence of loud coprolalic utterances of “fuc...” that would interrupt class and reverberate through the house. His parents reported that, initially, both his teachers and parents saw his tic symptoms as intentional and deliberate. He was teased by his peers and regularly provoked the anger of his parents. Six months earlier, during the course of an educational evaluation, the school psychologist suggested that a medical consultation was needed because of probable attention deficit disorder and possible TS. Shortly thereafter, a pediatric neurologist saw him, diagnosed TS, and placed him on 2 mg/day of haloperidol. Within a week, the tics had diminished considerably; however, he continued to do poorly in school and began to put on weight. Over the Christmas vacation, the tics again worsened despite increasing the haloperidol to 6 mg/day. On return to school, his tic symptoms were severe, and he was unable to concentrate on his schoolwork. His parents became increasingly alarmed at his dazed appearance and weight gain and abruptly took him off the haloperidol. Initially, he appeared to do better, but within 2 weeks, the situation had completely deteriorated, with a marked exacerbation of his motor and phonic tics. A day before the evaluation, the parents had been told that Arthur could no longer remain in school.

#### Comment

The waxing and waning character of tic symptoms frequently continues despite the use of medications. “Chasing” the symptoms with increases in medication often is not an effective strategy. Reassurance and educational interventions with both the family and school often are sufficient to help a child over a period of tic exacerbation. Exacerbations of tics some weeks after neuroleptic withdrawal are frequently observed and are usually self-limiting over a period of 4 to 8 weeks.

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## NEUROPSYCHOLOGICAL FINDINGS

Although motor and phonic tics constitute the core elements of the diagnostic criteria for TS, perceptual and cognitive difficulties are also common ([Walkup et al., 1998](#)). Neuropsychological studies of TS have focused on a broad array of functions. The most consistently observed deficits occur on tasks requiring the accurate copy of geometric designs, that is, “visual-motor integration” or “visual-graphic” ability. (See [Schultz et al., 1998](#), for a review.) There was no evidence to suggest that comorbid ADHD or depressive symptomatology could account for the observed group differences. Even after controlling statistically for visual-perceptual skill, intelligence, and fine motor control, children with TS continued to perform somewhat worse than controls on the visual-motor tasks, suggesting that the integration of visual inputs and organized motor output is a specific area of weakness in individuals with TS. However, these findings reflect a mild to moderate deficit with the performance distributions overlapping greatly across TS and control subjects, and with most of the patients performing within the normal range.

## ETIOLOGY AND PATHOGENESIS

### Genetic Factors

Twin and family studies provide evidence that genetic factors are involved in the vertical transmission within families of a vulnerability to TS and related disorders ([Pauls and Leckman, 1986](#)). The concordance rate for TS among monozygotic twin pairs is greater than 50%, whereas the concordance of dizygotic twin pairs approaches 10% ([Hyde et al., 1992](#); [Price et al., 1986](#)). If cotwins with chronic motor tic disorder are included, these concordance figures approach 90% for monozygotic and 30% for dizygotic twin pairs. Differences in the concordance of monozygotic and dizygotic twin pairs indicate that genetic factors play an important role in the etiology of TS and related conditions. These figures also suggest that nongenetic factors are critical in determining the nature and severity of the clinical syndrome.

Other studies indicate that first-degree family members of TS probands are at substantially higher risk for developing TS, chronic motor tic disorder, and OCD than are unrelated individuals ([Pauls et al., 1991](#); [Walkup et al., 1996](#)). For example, in one study ([Pauls et al., 1991](#)), the risk to male first-degree family members approximates 50% (18% TS, 31% chronic motor tics, and 7% OCD), whereas the overall risk to females is less (5% TS, 9% chronic motor tics, and 17% OCD).

The pattern of vertical transmission among family members has led several groups of investigators to test whether or not mathematical models of specific genetic hypotheses could account for these data. The bulk of this work favors models of autosomal-dominant transmission ([Pauls and Leckman, 1986](#)). These studies have also led directly to the identification of large multigenerational families with the disorder, to facilitate genetic linkage studies ([Kurlan et al., 1986](#)). Thus far, traditional linkage studies have not been successful in determining the chromosomal location of the putative TS gene or genes ([Pakstis et al., 1991](#)).

More recently, nonparametric approaches using families in which two or more siblings are affected have been undertaken ([The Tourette Syndrome International Consortium for Genetics, 1999](#)). This sib-pair approach is suited for diseases with a complex mode of inheritance and has been used successfully in studies of other complex disorders, such as diabetes mellitus and essential hypertension. In this study, two areas are suggestive of linkage to TS, one on chromosome 4q and another on chromosome 8p. Currently, this international consortium of researchers is completing high-density maps of these genomic regions in an effort to refine and extend their preliminary results. In addition, a new sample of sibling pairs is being ascertained to determine if these preliminary linkage results can be replicated.

It is also noteworthy that none of the chromosomal regions (e.g., 3 [3p21.3], 8 [8q21.4], 9 [9pter], and 18 [18q22.3]) in which cytogenetic abnormalities have been found to cosegregate with TS showed any convincing evidence for linkage in the sib-pair study; however, it is still possible that rare susceptibility genes may be found in one or more of these regions using molecular cytogenetic techniques.

### Neuroanatomic Substrates

The basal ganglia and related structures in the midbrain and cortex are likely to serve as the neuroanatomic substrates for TS and related disorders ([Leckman et al., 1997](#); [Peterson et al., 1998a, 1998b, 2001](#)). This speculation is based on an emerging appreciation of the functional interrelationships among these structures and their regulation of cognitive and sensorimotor activities, the occurrence of stereotypies, and the formation of habits. Preliminary neuropathological and neuroimaging studies, and a large body of neurochemical and neuropharmacologic data have also directly implicated the basal ganglia and related cortical structures in the pathobiology of TS.

The basal ganglia have long been recognized as providing a critical way station in cortico-striato-thalamo-cortical (CSTC) circuits involved with motor control and sensorimotor integration ([Marsden, 1982](#)). The identification of interrelated circuits that link thalamic sites with areas in the prefrontal and limbic cortex ([Alexander and DeLong, 1985a, 1985b](#)) has intensified speculation concerning the role of these structures in the pathobiology of TS ([Leckman et al., 1991b, 1997](#); [Leckman and Riddle, 2000](#)) and OCD ([Rapoport, 1989](#)).

Animal studies have indicated that the balance of activity of medium spiny neurons located in the striosomes versus the matrix of the striatum may crucially determine an individual's vulnerability to a range of sudden, repetitive tic-like head and paw movements, as well as sniffing ([Canales and Graybiel, 2000](#)). Neurons located in the striosomes receive afferents from prefrontal and limbic regions. Tic-like movements and vocalizations emerge when these striosomal cells are differentially activated.

Habits involve the automatic and coordinated activity of thousands of medium spiny neurons ([Graybiel, 1998](#)). If habits are coordinated ensembles of action, then conceptually tics may be conceptualized as prewired bits of behavior (motor or vocal) that are available to be assembled into habits ([Leckman et al., 2000](#)). Like habits, tic sequences often arise in response to a heightened and selective sensitivity to particular environmental cues from within the body or from the outside world. In the case of TS these perceptual cues include faint premonitory feelings or urges that are relieved with the performance of tics, and a need to perform tics or compulsions until they are felt to be “just right.”

Few neuropathologic studies have been performed. [Balthasar \(1956\)](#) reported hypoplasia of the corpus striatum (caudate and putamen), with a loss of the interneuronal neuropile. These findings, however, were based on a single case and were not consistent with an earlier report that found no distinctive histological pattern ([Dewulf and van Bogaert, 1941](#)). The case reported by Dewulf and van Bogaert was atypical with regard to age of onset (18 years) and the unremitting



progress of this individual's neurologic symptoms. [Laplaine and coworkers \(1981\)](#) described two intriguing case reports of individuals with basal ganglia lesions that were associated with tics, obsessive-compulsive symptoms, and deficits in motivation.

Neuroimaging studies have provided limited support for the involvement of the basal ganglia and related cortical structures in TS ([Braun et al., 1993](#); [Peterson et al., 1993](#); [Singer et al., 1993](#)). More recent large-scale morphometric studies have identified cortical changes that are age- and sex-dependent ([Peterson et al., 2001](#)). Similarly, recent functional magnetic resonance imaging (fMRI) studies present a more complex picture in which altered patterns of activation and/or deactivation in the prefrontal cortex likely influence voluntary tic suppression or active ticking ([Peterson et al., 1998b](#)). If confirmed, these findings are consistent with the view that prefrontal cortex-basal ganglia circuits participate in the shaping of the motor output of the basal ganglia (Leckman and Riddle, in press). A similar picture has emerged in the study of individuals with OCD using functional neuroimaging techniques ([Baxter et al., 1992](#)).

Recent studies using transcranial magnetic stimulation have reported altered patterns of cortical inhibition/excitation in TS patients. Specifically, following focal transcranial magnetic stimulation to the primary motor cortex, [Ziemann and colleagues \(1997\)](#) have documented that the cortical silent period was shortened and the intracortical inhibition reduced in TS patients. This suggests either disinhibited thalamic input or impaired intracortical inhibition directly at the level of the motor cortex.

In summary, converging lines of evidence for pathology in CSTC circuitry in TS comes from a number of preliminary neuroimaging, neuropathologic, and neurophysiologic studies. Thus far, these findings generally suggest subtle developmental differences.

### Neurochemical Systems and Neuropharmacologic Data

The functional status of a number of neurotransmitter and neuromodulator systems has been evaluated in TS. The most compelling evidence has focused attention on nigrostriatal and nigrocortical dopaminergic projections; serotonergic projections from the dorsal raphe to the substantia nigra, globus pallidus, and striatum; and noradrenergic projections from the locus ceruleus ([Leckman et al., 1997](#)).

Based largely on parallels between the tics, vocalizations, and obsessive-compulsive behaviors seen in some patients with encephalitis lethargica, [Devinsky \(1983\)](#) has suggested that TS is the result of altered dopaminergic function in the midbrain. The substantia nigra and the ventral tegmental area (VTA) are adjacent midbrain sites that contain large numbers of dopaminergic neurons. The dopamine cell bodies in the substantia nigra give rise to the ascending nigrostriatal pathway. The dopamine neurons of the VTA send ascending projections to various cortical regions, including the anterior cingulate gyrus and other limbic structures. The dopamine neurons in the substantia nigra and VTA also give rise to descending pathways projecting to the dorsal pontine tegmentum in the region of the noradrenergic locus ceruleus.

Other data implicating central dopaminergic mechanisms include clinical trials in which haloperidol and other neuroleptics that preferentially block dopaminergic D2 receptors have been found to be effective in the partial suppression of tics in a majority of TS patients ([Shapiro and Shapiro, 1988](#)). [Friedhoff \(1986\)](#) has suggested that the relationship between D2 and D1 dopamine systems may be of interest in TS in that a blockade of the D2 receptors by relatively selective agents such as haloperidol and pimozide disinhibits the D1 system.

Tic suppression has also been reported following administration of  $\alpha$ -methyl-para-tyrosine and following trials of tetrabenazine ([Shapiro and Shapiro, 1988](#)). In contrast, Tourette's-like syndromes have appeared following withdrawal of neuroleptics, and tics are often exacerbated following exposure to agents that increase central dopaminergic activity, such as L-Dopa and central nervous system (CNS) stimulants, including cocaine.

These pharmacologic data, taken together with reports of altered baseline and post-probenecid mean cerebrospinal fluid (CSF) levels of homovanillic acid (HVA) (a major metabolite of brain dopamine) in some TS patients compared to available contrast groups ([Butler et al., 1979](#); [Cohen et al., 1978](#); [Singer et al., 1982](#)), have led several groups of investigators to suggest that TS is a disorder in which postsynaptic dopaminergic D2 receptors are hypersensitive. Although preliminary positron emission tomography studies (PET) of brain dopamine receptors do not support the view that there are increased numbers of D2 receptors in all TS patients ([Brooks et al., 1992](#); [Singer et al., 1991](#)), [Wolf and colleagues \(1996\)](#) did find that differences in D2 dopamine receptor binding/affinity in the striatum were correlated with the degree of difference in observed tic severity among monozygotic twins discordant for TS. If confirmed, this finding suggests that certain environmental factors, as yet unknown, may influence tic severity via the density of D2 dopamine receptors.

Preliminary postmortem studies have suggested that there are an increased number of dopamine transporter sites in the basal ganglia of patients with TS ([Singer et al., 1991](#)). This work has been extended to *in vivo* single photon emission computerized tomography (SPECT) studies of dopamine transporter sites ([Malison et al., 1995](#)), which confirm the earlier postmortem data in a small number of subjects. Additional imaging and neuropathological studies are needed to address fully the potential abnormalities of receptor number, affinity, and distribution. If confirmed, the increased number of dopamine transporter sites in the striatum may reflect a dopaminergic hyperinnervation of the striatum in some patients. Given the role of dopaminergic projections in mediating the output of the basal ganglia ([Aosaki et al., 1994](#)) and the importance of the balance of D1 versus D2 dopamine receptor stimulation ([Filion and Tremblay, 1991](#); [Filion et al., 1991](#)), it is possible that the severity of tics may, in part, be mediated either by an increased density of D2 versus D1 receptors and/or by dopaminergic hyperinnervation of the striatum.

In summary, although the evidence that dopaminergic pathways are intimately involved in the pathobiology of TS is compelling, the exact nature of the abnormality remains to be elucidated. Multiple mechanisms may be involved and patients appear to differ with regard to the nature of the dopaminergic alterations observed ([Leckman et al., 1997](#)).

Although serotonergic mechanisms have been repeatedly invoked as potentially playing an important role in the pathophysiology of TS, there is very little hard evidence to support this contention. Serotonergic inputs from the dorsal raphe to both the substantia nigra and striatum have been identified and partially characterized, but there are no direct neuropathologic data implicating them in TS. Medications that act by increasing or decreasing serotonergic activity do not consistently alter tic symptoms. Studies of CSF 5-hydroxyindoleacetic acid (5-HIAA), the principal central metabolite of serotonin, have reported a range of low and normal levels in TS patients compared to contrast groups ([Butler et al., 1979](#); [Cohen et al., 1978](#); [Singer et al., 1982](#)). Despite this lack of evidence, serotonergic hypotheses continue to be attractive. Circumstantial evidence includes their neuroanatomic projections and their likely involvement in the pathophysiology of OCD.

### Stress-Related Neuroendocrine Factors

Tic disorders have long been identified as "stress-sensitive" conditions ([Chappell et al., 1994](#); [Jagger et al., 1982](#); [Shapiro and Shapiro, 1988](#); [Silva et al., 1995](#)). Typically, symptom exacerbations follow in the wake of stressful life events. As noted by Shapiro, these events need not be adverse in character to exacerbate tics ([Shapiro and Shapiro, 1988](#)). Clinical experience suggests that, in some unfortunate instances, a vicious cycle can be initiated in which tic symptoms are misunderstood by the family and teachers, leading to active attempts to suppress the symptoms by punishment and humiliation. These efforts can lead to a further exacerbation of symptoms and a further increase in stress in the child's interpersonal environment. Unchecked, this vicious cycle can lead to the most severe manifestations of TS and dysthymia, as well as maladaptive character traits. Although psychoanalytic and dynamic formulations of TS have been largely discredited ([Mahler and Rangell, 1943](#)), the intimate association of the content and timing of tic behaviors with dynamically important events in the lives of children make it difficult to overlook their contribution to the intramorbidity course of these disorders ([Cohen and Leckman, 1994](#)).

The neurobiologic substrates for these effects may involve the central noradrenergic pathways and their role as a regulator of the hypothalamic-pituitary-adrenal (HPA) axis. For example, a series of adult TS patients were found to have elevated levels of CSF norepinephrine ([Leckman et al., 1995](#)) and high levels of urinary norepinephrine in response to the stress associated with a lumbar puncture ([Chappell et al., 1994b](#)). These elevated levels of CSF norepinephrine may also contribute to the elevations in CSF corticotropin releasing factor and increased plasma levels of adrenocorticotrophic hormone seen in some TS patients ([Chappell et al., 1996](#)).

The neurobiological interface between a heightened stress response and the appearance or exacerbation of tics is not well understood but may relate to the emotionally laden inputs from the amygdala, the orbital frontal cortex, and other limbic structures to the striosomal medium spiny neurons ([Leckman and Riddle, 2000](#)). Activation of these afferents would then lead to a differential activation of these neurons and the emergence of tics (stereotypies).

In addition to the intramorbidity effects of stress and anxiety, which have been well characterized, some authors have suggested that premorbid stress may play an important role as a sensitizing agent in the pathogenesis of TS among vulnerable individuals ([Leckman et al., 1984](#)).

## Perinatal Factors

The search for nongenetic factors that mediate the expression of a genetic vulnerability to TS and related disorders has also focused on the role of adverse perinatal events (Leckman and Peterson, 1993). This interest dates from the report of Pasamanick and Kawi (1956), who found that mothers of children with tics were 1.5 times more likely to have experienced a complication during pregnancy than mothers of children without tics. Other investigators have reported that, among monozygotic twins discordant for TS, the index twin with TS uniformly had a lower birth weight than the unaffected cotwin (Hyde et al., 1992; Leckman et al., 1987). Severe maternal life stress during pregnancy and severe nausea and vomiting during the first trimester also have been implicated as potential risk factors in the development of tic disorders (Leckman et al., 1990). Other studies have reported that low birth weight children with parenchymal lesions involving the basal ganglia are at an increased risk for developing a tic disorder (Whitaker et al., 1997). In addition to disrupting the functional integrity of the cortical-subcortical pathways, some of these factors may have an enduring effect on the status of the developing dopaminergic or noradrenergic pathways (Friedhoff, 1986) or stress responsivity. For example, animal studies have shown that severe maternal stress toward the end of gestation can lead to enduring changes in stress responsivity of the HPA axis in the adult offspring as well as long-lasting changes in the dopamine sensitivity (Henry et al., 1994, 1995)—changes similar to those seen in some TS patients (Chappell et al., 1994b).

## Sex-Specific Neuroendocrine Factors

The increased prevalence of TS and other tic disorders among males may be related to exposure of the developing male CNS to elevated levels of dihydrotestosterone and/or other gender-related factors (Leckman et al., 1993; Peterson et al., 1992). This hypothesis is based on a series of interrelated observations. First, although TS is more prevalent among males, it does not appear to be genetically transmitted as a sex-linked trait (Pauls and Leckman, 1986); therefore, the observed sex differences are unlikely to result from a genetic cause. Second, the onset of tics is typically well before puberty, so that activation of the hypothalamic-pituitary-gonadal axis during puberty is not crucial for the development of tics. Earlier hormonal effects during gestation (Sikich and Todd, 1988) or during adrenarche therefore may be more important (Peterson et al., 1992). Third, high levels of exogenous androgens can induce a marked and persistent tic exacerbation (Leckman and Scahill, 1990). Fourth, some, but not all, Tourette's disorder patients respond to compounds that block androgenic receptors (Peterson et al., 1998b). In summary, there is strong evidence to support a role for sex-specific hormonal factors in the pathogenesis and intramorbidity course of TS; however, the precise mechanisms and timing of these events remain to be elucidated.

## Postinfectious Autoimmune Mechanisms

It is well known that group A hemolytic streptococci (GABHS) can trigger immune-mediated disease in genetically predisposed individuals (Bisno, 1991). Speculation concerning a postinfectious etiology for tic disorder symptoms dates from the late 1800s (Kushner, 1999). Acute rheumatic fever (RF) is a delayed manifestation of GABHS, occurring approximately 3 weeks following an inadequately treated upper respiratory tract infection. Inflammatory lesions involving the joints (polymigratory arthritis), heart (rheumatic carditis), and/or central nervous system (Sydenham's chorea [SC]) characterize rheumatic fever. SC, TS, OCD, and ADHD likely share common anatomic targets—the basal ganglia of the brain and related cortical and thalamic sites (Husby et al., 1976). For example, SC patients often display motor and vocal tics, obsessive-compulsive, and ADHD symptoms (Mercadante et al., 1997; Swedo et al., 1989).

It has been suggested that pediatric autoimmune neuropsychiatric disorder associated with streptococcal infection (PANDAS) represents a distinct clinical entity (Swedo et al., 1998). The most compelling evidence that acute exacerbations of TS and OCD can be triggered by GABHS comes from two independent reports demonstrating that the vast majority of patients with TS or OCD have elevated expression of a stable B-cell marker (Murphy et al., 1997; Swedo et al., 1997). The D8/17 marker identifies close to 100% of RF patients (with or without SC) but is present at low levels of expression in healthy control populations. The identity of the D8/17 epitope is not yet known, but several non-B-cell types can express it (Kemeny et al., 1994). Additional evidence comes from Swedo and colleagues (1998), who reported that in children who met PANDAS criteria, GABHS infection was likely to have preceded neuropsychiatric symptom onset for 44% of the children, whereas pharyngitis (no culture obtained) preceded onset for another 28% of the children. There were 144 episodes of symptom exacerbation among these 50 children. Cognitive deficits, oppositional behaviors, and motor hyperactivity were reported to be “particularly common” in addition to tic and obsessive-compulsive symptoms during periods of exacerbation. Thirty-one percent of these exacerbations were associated with documented GABHS infection, and 42% with symptoms of pharyngitis or upper respiratory infection (no throat culture obtained). These results are intriguing, although preliminary. The magnitude of some of the reported associations may have been biased upward because individuals were selected for having a recent or recurrent history of GABHS infections.

In summary, a substantial body of circumstantial evidence exists that links postinfectious autoimmune phenomena with TS; however, these data are not compelling with regard to specific immunologic mechanisms (Singer et al., 1998), nor do they establish where these immune changes occur in the sequence of causal events. These potentially important findings require replication in independent samples and warrant more intensive investigation.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of simple motor tics includes a variety of hyperkinetic movements: myoclonus, tremors, chorea, athetosis, dystonias, akathic movements, paroxysmal dyskinesias, ballistic movements, and hyperexplexia (Towbin et al., 1998). These movements may be associated with genetic conditions, such as Huntington's chorea or Wilson's disease; structural lesions, as in hemiballismus (associated with lesions to the contralateral subthalamic nucleus); postinfectious autoimmune processes, as in SC (Swedo et al., 1989); idiopathic functional instability of neuronal circuits, as in myoclonic epilepsy; and pharmacologic treatments, such as acute akathisia and dystonias that are associated with the use of neuroleptic agents. Differentiation between these conditions and tic disorders is usually accomplished on clinical grounds and is based on the presentation of the disorder and its natural history. For example, although aspects of tics, such as their abruptness, paroxysmal timing, or suppressible nature, may be similar to symptoms seen in other conditions, it is rare for all of these features to be combined in the absence of a bona fide tic disorder. Occasionally, diagnostic tests are needed to exclude alternative diagnoses.

Complex motor tics can be confused with other complex repetitive behaviors, such as stereotypies or compulsive rituals. Differentiation among these behaviors may be difficult, particularly among retarded individuals with limited verbal skills. In other settings, where these symptoms are closely intertwined, as in individuals with both TS and OCD, efforts to distinguish between complex motor tics and compulsive behaviors may be futile.

Involuntary vocal utterances, in the absence of a tic disorder, are uncommon neurologic signs. Huntington's disease may be associated with sniffing and brief sounds. Involuntary moaning can be heard in Parkinson's disease, particularly as a result of L-Dopa toxicity. Complex phonic tics, characterized by articulate speech, can be distinguished from other conditions, including voluntary coprolalia. Phonic tics can play an important role in differential diagnosis because of their rarity in other syndromes.

Anamnesis, family history, observation, and neurologic examination are usually sufficient to establish the diagnosis of a tic disorder. There are no confirmatory diagnostic tests. Neuroimaging studies, electroencephalogram-based studies, and laboratory tests are usually noncontributory, except in atypical cases.

Inventories such as the Yale Child Study Center Tourette's Syndrome/Obsessive-Compulsive Disorder Specialty Clinic Questionnaires (Appendices I and II, Leckman and Cohen, 1998), completed by the family prior to their initial consultation, can be valuable ancillary tools to gain a long-term perspective of the child's developmental course and the natural history of the tic disorder. In addition, several valid and reliable clinical rating instruments have been developed to inventory and quantify recent tic symptoms, including the Yale Global Tic Severity Scale (Leckman et al., 1989), the Shapiro Tourette Syndrome Severity Scale (Shapiro et al., 1988), and the Hopkins Motor and Vocal Tic Scale (Walkup et al., 1992). Some clinicians also make regular use of standardized videotape protocols to assess current tic severity (Chappell et al., 1994a; Shapiro et al., 1988; Tanner et al., 1982).

## ASSESSMENT

Table 59.2 presents an outline of clinically relevant areas that should be assessed in patients presenting with a tic disorder. Once the diagnosis has been established, care should be taken to focus on the overall course of an individual's development, not simply on his or her symptoms. This may be a particular problem in the case of TS, where the symptoms can be dramatic and the temptation to organize all of an individual's behavioral and emotional difficulties under a single all-encompassing rubric is relatively great.



**Table 59.2. Clinical Evaluation of Tic Disorders and Closely Related Conditions**

The principal goal of an initial assessment is to determine the individual's overall level of adaptive functioning and identify areas of impairment and distress. Close attention to the strengths and weaknesses of the individual and his or her family is crucial. Relevant aspects include the presence of comorbid mental, behavioral, developmental, or physical disorders; family history of psychiatric and/or neurologic disease; relationships with family and peers; school and/or occupational performance; and the history of important life events.

Medication history is important, particularly if the disorder is long-standing or medications have been prescribed for physical disorders. It may be necessary to evaluate the adequacy of prior trials with pharmacologic agents used to treat tic disorders.

## TREATMENT

Tic disorders are frequently chronic, if not lifelong, conditions. Continuity of care is desirable and should be considered before embarking on a course of treatment. Major approaches include education, supportive interventions, and treatment with neuropsychopharmacologic agents. Typically, patients with chronic motor tics alone do not require drug treatment. Psychoanalytically oriented psychotherapy can be a useful adjunct in selected cases to help address the adverse effects of chronic illness on self esteem, social acceptance, coping skills, and character formation. Behavioral and dietary treatments have not yet provided consistent positive effects.

### Educational and Supportive Interventions

Although the efficacy of educational and supportive interventions has not been rigorously assessed, they can have profoundly positive effects by reshaping negative expectations (Cohen et al., 1988). This is particularly true when the family and others have misconstrued the tic behaviors as voluntary and intentionally provocative. Families also find descriptions of the natural history comforting, in that the disorders tend not to be relentlessly progressive and usually improve during adulthood. This information often contradicts the impressions gained from the available lay literature on TS, which typically focuses on the most extreme cases. In addition, the characterization of TS as a neuropsychiatric disorder determined by a neurobiological vulnerability and influenced by psychosocial factors can help families fend off the stigmatizing effects of a purely mental disorder. For children, contact with their teachers can be enormously valuable. By educating the educators, clinicians can make significant progress toward securing for the child a positive and supportive environment in the classroom. Self-help organizations such as the Tourette Syndrome Association can also be of assistance in providing audiovisual aides as well as peer support and counseling.

### Pharmacotherapy of Tics

The decision to begin medication is based on the level of symptoms and the clinical presentation of the individual case. Many cases of TS can be successfully managed without resorting to the use of medication. When patients present with coexisting ADHD, OCD, depression, or bipolar illness, it is often better to treat these "comorbid" conditions first, as successful treatment of these disorders often will diminish tic severity.

A variety of therapeutic agents are now available to treat tics (Chappell et al., 1997; Kurlan 1997; Leckman et al., 2000). Each medication should be selected on the basis of expected efficacy and potential side effects. Dopamine D2 receptor antagonists remain the most predictably effective tic-suppressing agents in the short-term. Documentation of haloperidol's effectiveness in the early 1960s was landmark in the history of TS, as it called into question the prevailing view that tics were psychogenic in nature (Kushner, 1999). The most widely used D2 receptor antagonists are haloperidol, pimozide, fluphenazine, and tiapride (not presently available in the United States). Favorable data from double-blind clinical trials are available for each of these agents (Borison et al., 1982; Eggers et al., 1988; Sallee et al., 1997; Shapiro et al., 1989; Tourette Syndrome Study Group 1999). The US Food and Drug Administration has approved TS as an indication for haloperidol and pimozide use. Long-term with these agents experience has been less favorable, and the "reflexive" use of these agents should be avoided (Kurlan, 1997; Silva et al., 1996). Typically, treatment is initiated with a low dose (0.25 mg of haloperidol or 1 mg of pimozide) given before sleep. Further increments (0.5 mg of haloperidol or 1 mg of pimozide) may be added at seven to 14 day intervals if the tic behaviors remain severe. In most instances, 0.5 to 6.0 mg per day of haloperidol or 1.0 to 10.0 mg per day of pimozide administered over a period of 4 to 8 weeks are sufficient to achieve adequate control of tic symptoms. Common potential side effects include tardive dyskinesia, acute dystonic reactions, sedation, depression, school or social phobias, and weight gain. In many instances by starting at low doses and adjusting the dosage upward slowly clinicians can avoid these side effects. The goal should be to use as little of these medications as possible to render the tics "tolerable." Efforts to stop the tics completely often risk overmedication.

More recently, "atypical" neuroleptics, including risperidone, ziprasidone, and olanzapine have been used to treat tic symptoms. These agents have potent 5-HT<sub>2</sub> blocking effects, as well as more modest blocking effects on dopamine D<sub>2</sub>. Initial favorable double-blind clinical trials have now been reported for both risperidone and ziprasidone, although ziprasidone is not currently available in the United States (Bruggeman et al., 2001; Sallee et al., 2000). Risperidone use often causes weight gain and complaints of sedation. Ziprasidone appears to have a more favorable side effect profile, but more data are needed before a judgment can be made regarding its efficacy and safety.

Clonidine is a potent  $\alpha_2$ -receptor agonist that is thought to reduce central noradrenergic activity. Although initial open studies were favorable, subsequent double-blind clinical trials have had mixed results (Goetz et al., 1987; Leckman et al., 1991a; Tourette's syndrome study groups, 2002). Clinical trials indicate that subjects can expect on average a 25% to 35% reduction in their symptoms over an 8- to 12-week period. Motor tics may show greater improvement than phonic symptoms. The usual starting dose is 0.05 mg given either on awakening or at bedtime, if the individual's sleep-wake cycle is disturbed. Further 0.05 mg increments at 3- to 4-hour intervals are added weekly until a dosage of 5 mg/kg is reached or the total daily dose exceeds 0.25 mg. Although clonidine is clearly less effective than haloperidol and pimozide for immediate tic suppression, it is considerably safer. The principal side effect associated with its use is sedation, which occurs in 10% to 20% of subjects and usually abates with continued use. Other side effects include dry mouth, transient hypotension, and rare episodes of worsening behavior. Clonidine should be tapered and not withdrawn abruptly, to reduce the likelihood of symptom or blood pressure rebound (Leckman et al., 1998b). Initial results from a double-blind clinical trial with the closely related compound, guanfacine, are also promising (Scahill et al., 2001).

Many other medications have been used in the treatment of tics; however, with the exception of flutamide, an androgen receptor antagonist, and pergolide, a dopaminergic agonist, none have been evaluated in double-blind randomized clinical trials (Gilbert et al., 2000; Peterson et al., 1998c). Promising agents include agents that deplete presynaptic dopamine concentrations (tetrabenazine) (Carpenter et al., 1999). Cholinergic agents, including nicotine formulated as a patch and used in combination with a neuroleptic, have also been recommended (Sanberg et al., 1997; Silver et al., 2000). Building on its success in treating dystonia, reports have appeared concerning the benefits of botulinum toxin injections that temporarily weaken muscles associated with severe motor or vocal tics (Jankovic, 1994). Finally, GABAergic agents, particularly clonazepam, have also been widely used.

### Special Populations: PANDAS

Postinfectious autoimmune mechanisms are likely to contribute to the pathogenesis of 10% to 20% of TS cases (Swedo et al., 1998). Preliminary evidence suggests that individuals with abrupt onset and coexisting ADHD and/or OCD may be at greater risk (Peterson et al., 2000). As in SC, the precise mechanisms are in doubt. Clarification of these mechanisms is likely to lead to improved treatment and possibly preventive interventions. Although initial results from immunomodulatory treatments, including plasma exchange, are promising, caution is warranted (Perlmutter et al., 1999). It is also premature to recommend prophylactic antibiotic

treatment ([Garvey et al., 1999](#); [Shulman, 1999](#)).

## FUTURE DIRECTIONS

A deepening appreciation of the clinical phenomenology, recent progress in neuroanatomy, systems neuroscience, functional *in vivo* neuroimaging, and genetics has set the stage for a major advance in our understanding of TS. The evidence supporting a genetic basis for TS is stronger than any common neuropsychiatric disorder other than Huntington's disease. The age of onset, stress sensitivity, and sex distribution are specific clues that TS syndrome. Success will lead to the targeting of specific brain circuits for more intensive study. Diagnostic, prognostic, and treatment advances also can be expected as the neural circuits involved are identified and as techniques are developed to modify their activity ([Vandewalle et al., 1999](#)).

Given this potential, TS can be considered a model disorder to study the dynamic interplay of psychological and neurobiological systems during development. It is likely that the research paradigms utilized in these studies and many of the empirical findings resulting from them will be relevant to other disorders of childhood onset, especially OCD and ADHD, and will enhance our understanding of normal development.

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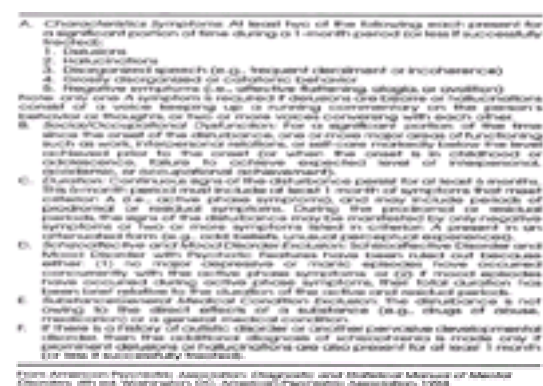
# 60 CHILDHOOD SCHIZOPHRENIA

Fred R. Volkmar, M.D. and Katherine D. Tsatsanis, Ph.D.

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## DEFINITION

The definition of schizophrenic disorders in childhood has been the topic of considerable debate. There is ample evidence from clinical, neuropsychological, brain imaging, and neurobiological studies to suggest that the patterns of abnormality are similar in both childhood and adult schizophrenia ( [Frazier et al., 1997](#); [Jacobsen et al., 1996b](#); [Zahn et al., 1997](#)). However, controversies have surrounded the stringency of diagnostic criteria and assumptions about the nature of the disorder. *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV) ([American Psychiatric Association, 1994](#)) criteria for the condition are presented in [Table 60.1](#). In light of the available evidence supporting a fundamental continuity among childhood, adolescent, and adult schizophrenia, the criteria apply to all age groups.



**Table 60.1. DSM-IV Diagnostic Criteria: Schizophrenia**

Relative to the DSM-III-R ([American Psychiatric Association, 1987](#)) criteria for schizophrenia have been simplified and the disorder is defined on the basis of characteristic psychotic symptoms, deficits in adaptive functioning, and duration of at least 6 months. The criteria for the characteristic psychotic symptoms have been streamlined with examples moved out of criteria into text; these characteristic psychotic symptoms include delusions, hallucinations, disorganized speech, grossly disorganized or catatonic behavior, and negative symptoms such as flattened affect. As indicated in Criterion B, some consideration of age should be made relative to social and occupational dysfunction. Signs of disturbance must be present for at least 6 months with at least 1 month during which active symptoms are present. The disorder cannot be diagnosed in the presence of schizoaffective or mood disorder with psychotic features, nor can it result from the direct effects of substance abuse or a general medical condition.

Unfortunately, the attempt to produce a more concise and efficient criterion set may complicate the task of diagnosis of childhood schizophrenia; certain symptoms such as disorganized speech and behavior are relatively common in nonpsychotic children, and the lowered threshold for Criterion A may similarly produce overdiagnosis ([Werry, 1996](#)). On the other hand, particularly in adolescents who develop schizophrenia and where there is a history of substance abuse, Criterion E may tend to produce underdiagnosis of the condition. The reworking of the criterion related to comorbid diagnosis of schizophrenia and autism (Criterion F) reflects the finding that there is no more than a chance association of these conditions ( [Volkmar and Cohen, 1991](#)). Given the lack of familiarity of many clinicians with schizophrenia in childhood, the condition may be misdiagnosed in both directions; that is, both false-positive and -negative diagnoses are common ( [McKenna et al., 1994](#)). The DSM-IV text indicates differences in symptomatology in children and addresses some aspects of differential diagnosis relevant to children. Review of both text and criteria is indicated relative to a diagnosis of schizophrenia in children and adolescents. The approach to diagnosis of the disorder in *International Classification of Diseases* (ICD)-10 is roughly similar ( [World Health Organization, 1990](#)), although that definition is even less developmentally oriented than DSM-IV ([Werry, 1996](#)).

Although DSM-IV and its predecessor, DSM-III-R, in some respects represent substantial advances in the classification of schizophrenic disorders, certain problems with respect to diagnosis of the disorder in childhood remain. Problems arise both in applying concepts such as psychosis to children and in the specification of criteria applicable to disordered children.

The concept of psychosis, as applied to children, is problematic in several respects. It is clear from the work of [Piaget \(1954\)](#) and others that children's conceptions of reality change over the course of normal development and that fully adult conceptualizations of reality are achieved only in adolescence. Many children have normatively appropriate beliefs in fantasy figures, which would not of themselves suggest psychosis. Finally, it is important to note that comorbid associations with conditions such as mental retardation or other developmental disorders might pose additional problems in assessing psychotic thinking in children because many of these severe disorders have important deleterious effects on development. Issues of the definition of psychosis or thought disorder become extremely complex in relationship, for example, to children who do not talk and are likely never to do so ( [Caplan, 1994](#)). These observations suggest the importance of incorporating a truly developmental point of view in conceptualizing the nature of psychosis in childhood.

Changes in diagnostic practice and the relative infrequency of the disorder have complicated the development of better definitions of childhood schizophrenia. The relatively few studies evaluating DSM-III criteria ( [American Psychiatric Association, 1980](#)) for schizophrenia in childhood have provided mixed and somewhat conflicting results.

In DSM-III, the term childhood schizophrenia was discarded as a separate diagnostic concept, whereas the validity of infantile autism as a diagnostic concept was recognized; this major change in classification reflected the consensus of most investigators that the term childhood schizophrenia had been used too broadly ( [Rutter, 1972](#)). [Cantor and coworkers \(1982\)](#) noted problems with this approach because several subjects they believed exhibited schizophrenia had evidenced signs of the



disturbance relatively early in life; important developmental correlates of psychotic symptoms were noted. This report suggested that it is more difficult to apply DSM-III criteria for schizophrenia to younger children. In a study comparing schizophrenic and autistic children, [Green and associates \(1984\)](#) suggested that DSM-III criteria for schizophrenia could be meaningfully applied to younger children. In a review of 228 children with childhood “psychosis” using DSM-III criteria, [Volkmar and associates \(1988\)](#) noted difficulties in applying DSM-III criteria for schizophrenia, particularly in relation to documenting a period of deterioration associated with the illness or, in younger children, in relation to the stringency of criteria for hallucinations and delusions. At present, the extent to which adult criteria for schizophrenia can be applied to children remains unclear. Some children clearly do meet such criteria, but the classification of children—particularly younger or developmentally delayed individuals—with schizophrenic-like disorders remains problematic. It is hoped that these issues will be addressed in future editions of DSM.

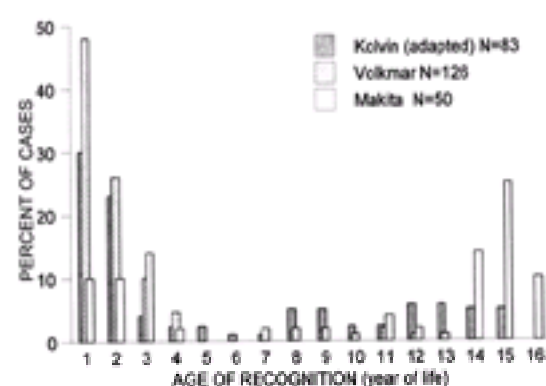
## HISTORICAL NOTE

There have been marked changes in the way childhood schizophrenia is both conceptualized and diagnosed. These changes reflect both the recognition of similarities to adolescent and adult forms of schizophrenia and the difficulties inherent in simplistic extensions to children of concepts derived largely from work with adults. In the 19th century, considerable interest centered on the role of experience versus endowment; by the mid-19th century, Maudsley proposed an extension of concepts of “insanity” to childhood. By the late 19th century, Kraepelin had proposed the term dementia praecox to describe a severe disorder with an onset in late adolescence or early adulthood and with characteristic features. This concept, now termed schizophrenia, proved particularly influential. [Kraepelin \(1919\)](#) noted that some cases appeared to have an onset in childhood; [De Sanctis \(1906\)](#) proposed the term dementia praecocissima for children who developed the disorder in childhood. Although [Potter \(1933\)](#) attempted to define childhood schizophrenia on the basis of specific features (e.g., loss of interest in the environment, disturbances in thinking or affect, or changes in behavior), the term became synonymous with childhood psychosis despite the fact that the disorder in children appeared less common than in adults and was more difficult to characterize.

It is important to note that disagreements regarding the nature of the adult schizophrenic syndrome resulted in rather different diagnostic approaches to the disorder. Different clinical features, such as a deteriorating course, disturbances in thinking or specific pathognomonic symptoms were variously emphasized ([American Psychiatric Association, 1987](#)). These controversies were also reflected in the apparent relative overdiagnosis of schizophrenia in adults in the United States compared with that in the United Kingdom ([Cooper et al., 1972](#)). Similarly, some investigators' approaches to childhood schizophrenia have been narrower and more conservative ([American Psychiatric Association, 1987](#); [Potter, 1933](#); [Rutter, 1972](#)), whereas others, particularly those of [Bender and associates \(1947\)](#), have argued for a very broad and inclusive definition. Early attempts to propose definitions (e.g., by the British Working Party on Childhood Psychosis) ([Creak, 1963](#)) were overinclusive, and schizophrenia was broadly defined so as to include all “psychotic” children. Although the narrow view currently predominates, it should be noted that this is a relatively recent historical phenomenon and that, for example, until DSM-III ([American Psychiatric Association, 1980](#)), childhood schizophrenia was the only “official” diagnostic term available to describe all psychotic children.

More importantly, the modern controversy over the relationship of childhood psychosis to adult schizophrenia can be traced to [Kanner's \(1943\)](#) description of the syndrome of early infantile autism. Kanner's initial report of autism noted various ways in which his proposed disorder appeared to differ from schizophrenia; at the same time, his use of the term autism suggested some point of phenomenologic similarity. The continuity or discontinuity of autism with schizophrenia became the topic of considerable debate. In retrospect, it appears that a central aspect of this controversy related to an assumption of continuity based on severity; that is, because autism is a severe psychiatric disorder with a very early onset, must it not be the case that it represents the earliest manifestation of a similarly severe psychiatric disorder in adults?

By the 1970s, various lines of evidence began to suggest that infantile autism and childhood schizophrenia differed in important ways. The work of [Kolvin \(1971\)](#) and [Rutter \(1972\)](#) suggested that differentiations could be made within the broad group of “psychotic” children on the basis of various features (e.g., age of onset, clinical characteristics, family history, and evidence of central nervous system [CNS] dysfunction). In particular, it was apparent that almost all cases of childhood psychosis fell into one of two groups on the basis of age at onset of the disorder, and this is true for series of cases collected at different centers in various countries ([Fig. 60.1](#)). The early-onset group exhibited difficulties in the first years of life more consistent with Kanner's description of autism, whereas the late-onset group exhibited the delusions, hallucinations, and clinical features more commonly associated with schizophrenia ([Kolvin, 1971](#)). It was also clear that family members of the late-onset cases were more likely to exhibit histories of schizophrenia. Thus, a major difference between autism and childhood schizophrenia is the early-onset of the former (always by, and usually well before, 36 months) and the comparatively much later onset (usually after 7 years and more commonly after puberty) of the latter. Together these various lines of evidence convinced most investigators that autism and schizophrenia were unrelated; this was reflected in the 1980 edition of DSM ([American Psychiatric Association, 1980](#)). It should be noted that, although most investigators, including the authors of this chapter, have adopted this view, a small number continue to advocate the earlier, broader diagnostic conceptualizations ([Cantor, 1988](#)).



**Figure 60.1.** Reported age of onset of childhood psychosis. (From Volkmar FR, Cohen DJ, Hoshino Y, et al.: Phenomenology and classification of the childhood psychoses. *Psychol Med* 18:191–201, 1988.)

Recently, the distinction between autism and schizophrenia in childhood has been extended to a consideration of the clinical spectrum of these disorders. Specifically, there have been attempts to identify subgroups of children within the broader spectrum who do not meet DSM criteria but who do show some features of the disorders. Rapoport and colleagues have been examining children with treatment refractory childhood-onset schizophrenia as part of a National Institute of Mental Health (NIMH) study since 1990. Of the children and adolescents referred with a diagnosis of childhood-onset schizophrenia, about 20% of the cases screened met DSM-III-R criteria; of those excluded at screening, 30% presented with complex developmental disorders and brief psychotic symptoms ([Jacobsen and Rapoport, 1998](#); [Kumra et al., 1998c](#)). A subgroup of children was proposed, labeled multidimensionally impaired (MDI), that shared the following features: (a) poor ability to distinguish fantasy from reality; (b) nearly daily periods of emotional lability disproportionate to precipitants; (c) impaired interpersonal skills despite desire to initiate social interactions; (d) cognitive deficits, indicated by multiple deficits in information processing; and (e) absence of formal thought disorder. This group was predominantly male, and further characterized by marked comorbid attentional and impulse control difficulties. A similar subgroup within the larger pervasive developmental disorder not otherwise specified (PDD-NOS) category has also been described ([Cohen et al., 1994](#)). Termed multiplex developmental disorder (MDD), this subtype is defined by difficulties in social interaction, deficits in affect regulation, and vulnerability of thought process. The two subtypes reflect the varied emphasis of features closer to childhood schizophrenia or PDD. A further distinction may lie in family history; [Kumra and colleagues \(1998c\)](#) found that family members of children with MDI were affected with schizophrenia spectrum disorders but not autism or mental retardation. More careful research is needed, and it should be noted that none of these terms is as yet broadly accepted, but represents current attempts to understand the broader phenotype of these disorders.

## PREVALENCE AND EPIDEMIOLOGY

Given the marked changes in the diagnostic concept over the past decade, it is not surprising that information on the prevalence and epidemiology of the disorder, as strictly defined, is quite limited. Few studies conducted prior to 1980 are readily interpretable, and major epidemiologic studies have yet to be conducted. Accordingly, information is limited to estimates provided from case series and comparisons of the frequency of schizophrenia in relation to diagnoses of autism. Available research indicates that the disorder is probably less common than autism. For example, in [Kolvin's \(1971\)](#) series of cases, autism was noted to be 1.4 times as common as childhood schizophrenia. Similarly, using DSM-III criteria, a prevalence study in North Dakota of schizophrenia presenting in childhood revealed that the disorder appeared to be far less common than autism ([Burd and Kerbeshian, 1987](#)).

Characteristics of four samples of children with schizophrenia from four studies using rigorous diagnostic criteria are presented in [Table 60.2](#). Reported sex ratios of the disorder have varied across studies. Both a slight male predominance, on the order of 2 males to 1 female ([Kolvin, 1971](#); [Russell et al., 1989](#); [Volkmar et al., 1988](#)), and essentially equal sex ratios ([Eggers, 1978](#); [Jacobsen and Rapoport, 1998](#)) have been reported. It has been found that children with schizophrenia generally function in the low average to average range of intelligence ([Table 60.2](#)). In addition, in contrast to early reports of a high-socioeconomic-class association with autism ([Chapter 46](#)), there is some evidence that suggests that children with schizophrenia may be observed more commonly in both less educated and professionally successful families ([Table 60.2](#)).

	Study			
	Kolvin et al., 1971	Green et al., 1984	Volkmar et al., 1988	Russell et al., 1989
N	33	24	14	35
Male/female	24/9	15/9	10/4	24/11
Sex ratio (M:F)	2.66:1	1.67:1	2.5:1	2.2:1
Mean IQ	85	88 (±16.0)	82 (±12.5)	94 (±10.5)
Social class				
I and II	16%	0	25%	54%
III	37%	17%	33%	23%
IV and V	47%	83%	42%	14%

**Table 60.2. Sample Characteristics: Childhood Schizophrenia**

## CLINICAL DESCRIPTION

### Precursors of Schizophrenia

Various attempts have been made to identify possible childhood precursors of adult schizophrenia; both follow-back and prospective studies have been employed. Follow-back studies of the childhood histories of adult schizophrenic patients have suggested some early patterns of developmental difference from children who did not develop schizophrenia ([Goldstein, 1980](#)). Similarly, prospective studies that follow children presumably at greater risk for developing adult schizophrenia have attempted to identify various factors that may contribute to adult schizophrenia. Follow-up studies of children with psychiatric disorders have not shown a clear-cut pattern among children who go on to develop adult schizophrenia; although some differences are noted, it does not appear that shy, withdrawn children are particularly likely to develop adult schizophrenia. The applicability of both types of studies to childhood schizophrenia is somewhat questionable; the limited available data arise from case series.

### Premorbid Course in Childhood Schizophrenia

The developmental history of children with schizophrenia indicates significant deficits in language and motor functioning, as well as impairments in social functioning ([Alaghband-Rad et al., 1995](#); [Asarnow et al., 1995](#); [Hollis, 1995](#); [Nicolson et al., 2000](#)). In these samples, approximately one-half to three-quarters of the children had motor (e.g., delayed milestones, poor coordination); language (expression and comprehension); and/or social (e.g., social withdrawal, aloofness) disturbances years prior to the onset of psychotic symptoms. [Eggers and colleagues \(2000\)](#) observed that the first prominent signs of dysfunction in premorbid development in their sample seemed to be disturbances in adaptive social behavior and a tendency for withdrawal, suggesting that these be considered vulnerability factors. Attentional issues and hyperactivity also characterize the early history of many of these children ([Alaghband-Rad et al., 1995](#); [Asarnow et al., 1994a](#)). In several cases, a diagnosis of ADD/ADHD preceded the onset and diagnosis of schizophrenia. Depressive symptoms and conduct problems have also been noted ([Asarnow et al., 1994b](#); [Russell, 1994](#); [Russell et al., 1989](#)). A subset of children is reported to display early features common to the PDDs such as echolalia, hand flapping, and/or a pervasive lack of responsiveness ([Alaghband-Rad et al., 1995](#); [Asarnow et al., 1995](#); [Russell, 1994](#)). In addition to being a chronic disorder, childhood-onset schizophrenia is associated with a more severe premorbid course that has implications for school performance. [Alaghband-Rad and colleagues \(1995\)](#) reported that 50% of the children with schizophrenia in their sample had repeated at least one grade before the onset of psychotic symptoms, 65% had been placed in special education classes, and 30% were diagnosed with a specific learning disability. When compared to children with major depression, children with early-onset schizophrenia showed poorer levels of overall premorbid adjustment and greater premorbid impairment with regard to peer relationships, school performance, school adaptation, and interests ([Asarnow et al., 1995](#)). There is also evidence to suggest that the pattern of developmental disturbances varies with age of onset; relative to adolescent/adult onset schizophrenia, impairments of language, but not social or motor functioning, were more common for childhood-onset schizophrenia ([Hollis, 1995](#)). [Alaghband-Rad and colleagues \(1995\)](#) reported sex differences for premorbid developmental history, specifically with regard to motor abnormalities (boys more affected than girls), but [Hollis \(1995\)](#) did not find this.

### Onset of the Disorder

The age at which a diagnosis of childhood schizophrenia is made varies in association with the criteria used to a considerable extent; however, most studies that employ reasonably stringent criteria suggest that the disorder is very rarely observed before age 5. [Russell \(1994\)](#) reported that, in their sample, the mean age of onset of nonpsychotic disturbance was 4.6 years and the mean age of onset of psychotic symptomatology was 6.9 years, with diagnosis of the full disorder at 9.5 years. The onset of disturbance is generally described to follow at least three patterns: (a) acute onset, without apparent premorbid signs of incipient disturbance; (b) insidious onset, with a gradual deterioration in functioning; and (c) insidious onset with an acute exacerbation of disturbance ([Green et al., 1984](#); [Kolvin, 1971](#)). The most common pattern appears to be insidious onset of illness ([Alaghband-Rad et al., 1995](#); [Asarnow et al., 1995](#); [Eggers et al., 2000](#); [Russell, 1994](#)). The course is characterized by early emergence of various developmental, behavioral, and psychiatric problems, followed by gradual onset of psychotic symptoms (about 2 to 3 years later), and then eventual diagnosis of the full clinical syndrome, which tends to be chronic. Some sex differences have been reported. As with adult schizophrenia, males appear to have an onset of their disorder earlier in childhood ([Green et al., 1984](#)). In addition, boys have been reported to be more likely to show early signs of abnormal development and have an insidious onset relative to girls with the disorder ([Alaghband-Rad et al., 1995](#); [Asarnow et al., 1995](#); [Hollis, 1995](#)). Distinctions between “early onset” and “very early onset” in childhood and adolescence have been suggested ([Werry, 1996](#)).

### Thought Disturbance

Available research data on the phenomenology of thought disturbance in childhood schizophrenia are limited. Only a handful of studies have been conducted using reasonably stringent diagnostic criteria. (See [Caplan, 1994](#), for a review.) Additional problems are posed by the lack, at least until recently, of valid and reasonably standardized assessment instruments, as well as by the difficulties inherent in clinical assessment. However, the results of the few available studies are surprisingly similar ([Green et al., 1984](#); [Kolvin, 1971](#); [Russell et al., 1989](#); [Volkmar et al., 1988](#)). [Table 60.3](#) summarizes the results from several series of cases. Auditory hallucinations are consistently the most frequently reported symptoms, usually exhibited by about 80% of cases. Auditory hallucinations may include persecutory or command hallucinations, voices conversing, voices commenting about the child, and the like ([Russell et al., 1989](#)). Visual and somatic hallucinations are reported less frequently. Although delusional beliefs can be difficult to assess, available studies suggest that these are exhibited in at least 50% of cases. Delusional beliefs can involve persecution, somatic concerns, ideas of reference, or grandiose or religious ideas ([Russell et al., 1989](#)). The content of hallucinations and delusions appears to vary with age; younger children typically express less complicated hallucinations or delusional beliefs, and the content of the delusions or hallucinations may center on parents, fantasy figures, and animals ([Arboleda and Holzman, 1985](#); [Russell et al., 1989](#)). The reported presence of formal thought disorder varies considerably from study to study, reflecting the nature of specific definitions used. Magical or illogical thinking, incoherence, and loosening of associations are often observed and distinguish these children from their typically developing peers. However, levels of thought disorder may not differ significantly between children with schizophrenia and children with major depression, suggesting that although frequent, this feature is not specific to childhood-onset schizophrenia ([Tompson et al., 1997](#)). In addition, it is sometimes the case that children, particularly in the early phases of the illness, may not clearly exhibit a degree of thought disturbance sufficient to satisfy strict diagnostic criteria. Furthermore, assessment of thought disturbance can be particularly problematic in younger or developmentally delayed children ([Caplan, 1994](#); [Russell et al., 1989](#); [Volkmar et al., 1988](#)).



Study	Thought Disorder (%)	Delusions (%)	Hallucinations	
			Auditory (%)	Visual (%)
Kolvin et al., 1971	60	58	82	30
Green et al., 1984	100	54	79	45
Volkmann et al., 1988	93	86	79	28
Russell et al., 1989	40	63	80	13

**Table 60.3. Characteristics of Thought Disturbance in Childhood Schizophrenia**

## ETIOLOGY AND PATHOGENESIS

### Neurobiological Mechanisms

Although various lines of evidence suggest the involvement of biological processes in syndrome pathogenesis, no specific biological marker for the disorder has been identified. The work of [Bender \(1947\)](#) suggested that childhood schizophrenia, broadly defined, is commonly associated with signs of neurologic immaturity, such as delays in motor development and neurologic “soft” signs. Similarly, [Cantor \(1988\)](#) proposed that a constellation of hypotonia and hyporeflexia characterizes at least some children with schizophrenia. Studies of electrophysiologic activity have also provided some general, although nonspecific, evidence of CNS dysfunction. Differences have been noted in the electroencephalograms (EEGs) of children with schizophrenia as compared to children with various disorders ([Waldo et al., 1978](#)), and abnormalities in event-related potentials also have been observed ([Strandburg et al., 1984](#)). Autonomic nervous system (ANS) activity and reactivity has been an area of interest in adult onset schizophrenia. In the one study of ANS functioning in childhood schizophrenia, a pattern of abnormality was observed, similar to adult findings ([Zahn et al., 1997](#)). The results were interpreted to suggest an inability to allocate attentional resources so as to distinguish important from less important stimuli. Investigations of neurotransmitter systems have been uncommon and have not produced consistent results ([Werry, 1996](#)); some researchers have suggested a possible role for the cholinergic system in syndrome pathogenesis ([Cantor, 1988](#)). Cerebrospinal fluid monoamine metabolites were studied in relation to treatment response and revealed no significant changes in monoamine ratios and concentration, despite improved clinical symptomatology ([Jacobsen et al., 1997a](#)). Immune processes similarly have not been found to mediate clinical response to antipsychotic medications, but a relative failure of cellular immunity was observed in children with schizophrenia compared to children with ADHD and children with obsessive-compulsive disorder (OCD) ([Mittleman et al., 1997](#)). Three additional areas have been the subject of more focused study and are considered in the following.

### SMOOTH PURSUIT EYE MOVEMENTS

There have been numerous studies of smooth pursuit eye movements in adults with schizophrenia, with consistent findings of abnormalities. This measure is of particular interest because it may represent a physiologic marker sensitive at identifying genetic risk for schizophrenia ([Ross et al., 1999](#)). In studies of childhood-onset schizophrenia, the results have been similar to those of previous studies of adults. Compared to age-matched controls and ADHD children, children with childhood schizophrenia showed significantly greater smooth pursuit impairments, a lower amount of time overall spent engaged in tracking the target, and a higher rate of anticipatory saccades ([Jacobsen et al., 1996b](#)). In a study designed to address markers of genetic risk for childhood schizophrenia, two physiologic markers were examined: Anticipatory saccades during smooth pursuit eye movements and P50 auditory evoked response to repeated stimuli ([Ross et al., 1999](#)). In a previous report, these researchers showed that abnormal inhibitory gating of the P50 auditory evoked response to repeated stimuli is transmitted in families in adult-onset schizophrenia as a single gene trait, with a close link to the  $\alpha 7$  nicotinic receptor gene locus on chromosome 15q14 ([Freedman et al., 1997](#)). The same may be true for anticipatory saccades, although this evidence is preliminary and it is noted that other smooth pursuit eye movement measures have been linked to chromosome 6p ([Olincy et al., 1997](#)). The results of the current study showed P50 ratio abnormalities in all 10 children with schizophrenia in the sample, as well as abnormal and more severe elevations of anticipatory saccades in nine out of 10 of the children ([Ross et al., 1999](#)). For both markers, 15 of the 20 parents examined also showed these abnormalities. Elevated P50 ratios and anticipatory saccades may represent physiologic markers of some part of the genetic risk for childhood-onset schizophrenia. Additionally, there was evidence in this study to suggest that inheritance of these schizophrenia-risk genes from both parents may result in increased genetic risk for childhood-onset schizophrenia.

### BRAIN MORPHOLOGY

Structural differences in the brain have been examined in childhood schizophrenia using magnetic resonance imaging. Current findings represent the systematic investigation of researchers at the NIMH, which have been detailed in a series of reports ([Giedd et al., 1999](#); [Jacobsen et al., 1996a, 1997c,d](#); [Nicolson et al., 2000](#); [Rapoport et al., 1997](#)). It is important to note that these results are preliminary and need to be independently replicated; however, quantitative analysis of the brain has revealed several significant differences in this NIMH sample. Smaller total cerebral volume, increased ventricular volume, and reduced area of the thalamus were obtained in the childhood schizophrenia group compared to healthy controls, which is consistent with findings in adult-onset schizophrenia. Increased size of the corpus callosum and reduced cerebellar volume also were shown ([Jacobsen et al., 1997b,c](#)). Differences in the volume of the prefrontal lobes were not found ([Nicolson et al., 2000](#)), although there is some evidence from functional imaging studies that is suggestive of abnormalities in this region ([Bertolino et al., 1998](#); [Jacobsen et al., 1997e](#)). Lateral temporal lobe structures, particularly the superior temporal gyrus, were significantly enlarged in the group of children with schizophrenia compared to controls. Medial temporal lobe structures also have been examined. The volumes of the amygdala and hippocampus were not significantly different in the two groups, although there were discrepancies in symmetry patterns ([Jacobsen et al., 1996a](#)). Area measurements of the planum temporale also did not reveal significant differences ([Jacobsen et al., 1997d](#)), consistent with a pattern of relative sparing of some temporal lobe structures. Repeat scans have been obtained as part of a prospective longitudinal study ([Jacobsen et al., 1998](#); [Rapoport et al., 1997](#)). The results of these studies revealed a differential decrease in thalamic and temporal lobe structures and a differential increase in ventricular volume. In addition to structural differences, there is evidence to suggest progressive changes in the brain in childhood-onset schizophrenia that are present during adolescence and may taper off in adulthood ([Giedd et al., 1999](#)).

### GENETIC STUDIES

Genetic and family studies provide what is probably the most convincing evidence of some biological basis for the disorder. Rates of schizophrenia and related disorders among the first-degree relatives of children with schizophrenia are substantially elevated in comparison to those observed in the normal population, as is the case in relatives of adult schizophrenic patients ([Kallman and Prugh, 1971](#); [Kolvin, 1971](#); [Nicolson et al., 2000](#)). It is reported that approximately 10% of parents exhibit a schizophrenic disorder ([Kallman and Prugh, 1971](#); [Kolvin, 1971](#)). Genetic risk for childhood schizophrenia has also been associated with physiologic markers of liability observed in unaffected relatives (see the preceding). Additional abnormalities, other than schizophrenia, may represent a risk factor relevant to the early-onset of the disorder. [Alaghband-Rad and colleagues \(1998\)](#) report a preliminary finding of an increased rate of mental retardation in the siblings of children with schizophrenia, which may also be associated with sex chromosome abnormalities. Cytogenetic abnormalities have been examined in children with schizophrenia and also show an increased rate ([Kumra et al., 1998](#); [Nicolson et al., 1999b](#); [Usiskin et al., 1999](#)). In particular, there is support for an association between deletions on chromosome 22q11 (velocardiofacial syndrome) and schizophrenia. In these cases, the loss of gene product balance may represent another factor predisposing the child to greater risk or susceptibility to additional genetic and environmental events. A role for apolipoprotein E alleles, trinucleotide repeats, and human leukocyte antigen also has been examined in childhood-onset schizophrenia, with negative findings to date ([Fernandez et al., 1999](#); [Jacobsen et al., 1998](#); [Sidransky et al., 1998](#)). The nature of the underlying mechanisms that might account for the early onset of schizophrenia in some individuals remains uncertain. General considerations include a higher genetic loading, as well as the complicated interaction of various factors in combination with some genetic predisposition ([Asarnow et al., 1986](#); [Werry, 1996](#)).

### Neuropsychological Function

Neuropsychological studies have revealed deficits in attentional capacities and the processing of information. Current findings chiefly represent the report of a series of investigations conducted by Asarnow and colleagues ([1994a, 1995](#); [Karatekin and Asarnow, 1998a,b](#); [1999](#)). They have found that rote language skills and simple perceptual functions are not impaired in children with schizophrenia, but they do show significantly reduced performance on measures of fine motor speed and tasks that place demands on attention and/or short-term memory. On a measure of conceptual reasoning and capacity to shift set (Wisconsin Card Sorting Test), the group

of children with schizophrenia made more perseverative errors than normal controls. They also showed a tendency to perform more slowly and make more errors on a guided visual search task (Rey's Tangled Lines). [Kumra and colleagues \(2000\)](#) report a similar pattern of neuropsychological deficits in the areas of auditory attention, verbal memory, and mental flexibility. Deficits in some areas of spatial organization also were observed.

More detailed studies of performance on visual search tasks have been conducted, which are thought to tap into the presumed core attentional deficit in schizophrenia. Children with schizophrenia have been shown to perform more poorly on span of apprehension tasks ([Asarnow et al., 1994](#)). Although this finding could reflect delayed initiation of search, it appears to result from the use of a less efficient strategy (e.g., impairment in top-down control) when dealing with more complex information ([Karatekin and Asarnow, 1998b, 1999](#)). A slower reaction time on measures of visual attention and guided search have been interpreted to indicate a failure to adopt and maintain a major "set to respond" ([Zahn et al., 1998](#)). Taken together, the findings are suggestive of inadequate representation of context and use of self-guided hypothesis testing for control of action in children with schizophrenia ([Karatekin and Asarnow, 1999; Zahn et al., 1998](#)).

On measures of intelligence, children with schizophrenia are generally found to perform in the low average range. When groups of children with childhood-onset schizophrenia and high functioning autism were compared, they did not differ in their full-scale intelligence quotient (IQ) score or performance on the perceptual organization and verbal comprehension factors of the WISC-R ([Asarnow et al., 1994](#)). The childhood-onset schizophrenia group did show a significantly lower score on the distractibility factor. There is also a report of significant decline in full-scale IQ score in children with schizophrenia, when measured prior to and following onset of psychosis. This did not represent a deterioration of performance but rather a failure to make age appropriate gains as indicated by a comparison of raw scores on this test ([Bedwell et al., 1999](#)). This pattern of intellectual decline was interpreted as evidence of an ongoing or progressive pathologic process in childhood-onset schizophrenia.

### Psychological Processes

The interpretation of studies of children at high risk for schizophrenia is complicated because the disorder is low in frequency, even among those children presumed to be at greatest risk, and other factors may complicate the interpretation of results. There is no evidence at present to suggest that unusual psychological trauma accounts for the earlier age of onset in this group. In addition, no single personality pattern or pattern of premorbid adjustment appears to uniformly characterize preschizophrenic children. Various studies have reported an increased incidence of signs of neurosensory and neuromotor deficits in children with schizophrenia and those presumably at risk for the disorder ([Bender, 1947; Cantor, 1988; Fish, 1986](#)). Disturbances in attentional capacities have been noted in children with schizophrenia; these problems may precede the emergence of frank psychosis and may account, in part, for observed associations with various learning problems. Compared to autistic children, those with schizophrenia are much less likely to exhibit mental retardation ([Green et al., 1984; Russell et al., 1989; Volkmar et al., 1988](#)); however, problems in the assessment of retarded individuals may make it more difficult to establish the diagnosis with certainty in such cases. In particular, in addition to the presence of hallucinations, thought disorder is a characteristic feature of schizophrenia. Formal thought disorder (e.g., impaired organization and processing of thoughts) and impaired use of cohesion (e.g., ability to communicate their ideas) have been found to measure different aspects of thought disturbance ([Caplan et al., 2000](#)). However, this can sometimes be difficult to assess in young or delayed children. From a developmental perspective, these are areas of functioning that children are normally acquiring; children become more organized in their thinking and communication of ideas over time. In addition, it is reported that some children with schizophrenia have been experiencing psychotic symptoms for years that were not obvious to parents and teachers ([Russell, 1994](#)). Rather, the disorder may present outwardly as a significant behavioral disturbance suggestive of ADHD or conduct disorder. It has been proposed that when psychotic symptoms appear in early development, they may not be readily distinguished from normal experience. Thus, for some children, early in the course of the disorder, their psychotic symptoms may not be experienced as frightening, markedly disorganizing, or ego-dystonic ([Russell, 1994](#)).

### Family and Interpersonal Dynamics

Various studies have examined patterns of family interaction in the families of individuals with schizophrenia. The available research is primarily concerned with adult patients. The few studies available regarding family dynamics in childhood schizophrenia are difficult to interpret because fairly stringent diagnostic criteria were not used and many early reports were really about autism rather than schizophrenia. Available studies have emphasized disturbances in patterns of communication (e.g., in relation to inconsistent communications or communications with unusual patterns of affective expression, such as high levels of expressed emotion). Various unusual personality profiles have been noted in family members ([Kolvin, 1971; Singer and Wynne, 1963](#)), and it clearly is the case that family members are at greater risk for the disorder themselves ([Kolvin, 1971](#)). Unfortunately, most theoretic models have not been empirically tested, and the significance of reports of unusual patterns of familial interaction is unclear. In addition to the obvious possibility that unusual patterns of interaction cause schizophrenia in children, it is also quite possible that these abnormalities are reactions to the child's disorder or, alternatively, that they reflect the same underlying vulnerability exhibited more directly by the affected child. At the present time, the role of family dynamics in syndrome pathogenesis remains to be established.

### Environmental and Social Influences

In addition to genetic factors, a role for environmental events has been considered in the pathogenesis of schizophrenia. Studies of adult schizophrenic individuals suggest that complications in pregnancy, labor, and delivery may be more frequent and related to an earlier age of onset, but this has not been the finding in childhood-onset schizophrenia ([Nicolson et al., 1999a](#)). In addition, there does not appear to be a relationship between age at onset of puberty and onset of psychotic symptoms ([Frazier et al., 1997](#)). There is evidence that stressful life events may play a role in precipitating psychotic episodes in children ([Birley and Brown, 1970](#)). The study of cases in which the onset of the disorder follows a clear environmental precipitant are of some interest because they may clarify how psychological processes interact with biological predispositions for the disorder. Available data suggest that children with schizophrenia are more likely to come from lower socioeconomic status (SES) families. However, it is not clear to what extent SES suggests a role for the environment in pathogenesis, because lower SES also might be the result of parental psychopathology leading to a downward drift in occupational status. It is furthermore noted that, with adults, exposure to certain pharmacologic agents (e.g., stimulants) may produce hallucinations and a schizophrenic-like psychosis.

### ASSESSMENT

To date, no single biological marker for schizophrenia in childhood has been found, although this remains an important goal for future research ([Tsuang et al., 2000](#)). Because overt psychosis can be observed in association with organic mental disorders, it is important to conduct a careful medical history and examination, particularly if there are unusual or atypical features. If history or physical signs suggest substance abuse—particularly of stimulants or phencyclidine—then appropriate toxicological screening should be obtained. Given the infrequency of the condition, a complete neurologic examination including an EEG is indicated. Genetic testing, in some cases, may also be advised given the findings of a higher rate of cytogenetic abnormalities in children with schizophrenia that, notably, had been previously undetected.

Schizophrenic disorders in childhood are not typically associated with mental retardation, although various developmental and learning problems may be associated with the disorder. Psychological testing is helpful both in documenting current levels of intellectual and adaptive functioning and in pointing out areas of specific strength or weakness important for educational programming ([Chapter 43](#)). In addition to information provided by formal intellectual assessment, projective tests should be administered. It has been reported that many children with schizophrenia did not exhibit disorganized thinking at interview ([Russell, 1994](#)). Thus, projective tests may provide important information about the severity of thought disorder and psychotic thinking, supplementing information obtained from other sources. Several assessment instruments have been developed explicitly for the assessment of psychosis or thought disorder in childhood ([Caplan, 1994; Caplan et al., 1989; Puig-Antich and Chambers, 1983](#)). Communication assessments should be obtained, particularly in cases where communication problems are prominent.

### DIFFERENTIAL DIAGNOSIS

The diagnosis of schizophrenia in childhood is probably most readily made in older children and adolescents. For children less than 10 years of age or those with developmental disabilities, difficulties in communication, changes in conceptions of reality, and the nature of symptom expression may complicate the diagnostic picture. The diagnosis should be made only after careful evaluation and after it is clear that the symptoms do not reflect the presence of an organic process (e.g., the effects of substance abuse or seizure disorder). Generally multiple informants must be interviewed to obtain an adequate history, and usually several sessions with the child are required to obtain an adequate mental status examination. The differentiation of schizophrenia from affective disorders can be difficult because mood disturbance can be observed in schizophrenia to some degree. Prominent mood disturbance that is prolonged and not brief relative to the total duration of the apparent schizophrenic illness suggests the need to consider the various affective disorders and schizoaffective disorder in the differential diagnosis ([American Psychiatric Association, 1994](#)). Occasionally, patients with an OCD may exhibit ideas that are difficult to distinguish from delusions, although usually the individual recognizes the irrational nature of such ideas. Hallucinations are occasionally reported as an isolated symptom: A diagnosis of childhood schizophrenia should not be



made in such cases. Sometimes only the subsequent course clarifies the diagnosis. Assessment procedures are summarized in [Table 60.4](#).

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Historical information
1. Early development and characteristics of development
2. Age and nature of onset
3. Medical and family history
Psychological/communicative examination
1. Assessment of intellectual level (IQ)
2. Assessment of adaptive behavior
3. Projective testing
4. Assessment of communication skills (particularly if communication problems are evident)
Psychiatric examination
1. Evaluate thought disturbance, hallucinations, delusions, etc.
2. Evaluate associated affective problems
3. Note unusual features of course/presentation
Medical evaluation
1. Physical examination for signs of associated medical conditions
2. Evaluate possible substance abuse
3. Neurologic consultation (including EEG)
4. Toxicology screen if indicated

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**Table 60.4. Evaluation Procedures: Childhood Schizophrenia**

Schizophrenia can be observed in association with various other psychiatric and developmental problems, for example, conduct disorder, learning disabilities, mental retardation, and even autism; comorbid conditions should be noted when present ([Russell et al., 1989](#)). Adherence to a multiaxial approach aids in this process. In individuals with mental retardation, autism, or developmental language disorders, a diagnosis of schizophrenia should be made only if it is clear that the apparent disturbance in thinking does not reflect communication impairments. Although the bulk of available evidence has suggested that childhood schizophrenia and autism are unrelated, a few investigators have speculated on possible continuities between autism and schizophrenia; and DSM-III-R as well as DSM-IV, in contrast to DSM-III, do allow both diagnoses to be made ([American Psychiatric Association, 1987, 1994](#)). The change in DSM-III-R reflects awareness that presumably autism does not protect an individual from also developing schizophrenia; however, individuals with both conditions do not seem to be common ([Volkmar et al., 1988](#)). The diagnosis of PDD-NOS should not be made concurrently with a diagnosis of schizophrenia. Clinicians should be particularly alert to the possibility of a diagnosis of schizophrenia when multiple different diagnoses have been given over time, or concurrently.

## TREATMENT

As with other aspects of the disorder, studies of treatment are few in number and limited in various respects. Apparent variations in the natural history of the disorder ([Eggers, 1978](#)) pose further problems for assessing treatment studies. Practice parameters for assessment and treatment have recently been published ([McClellan and Werry, 1994](#)). To a considerable extent, treatment programs should attempt to address the specific patterns of strength and weakness exhibited by the individual child; treatment modalities also depend on the stage of illness (i.e., the presence of active psychotic symptomatology). Typically, multiple treatment modalities are needed, including medications, educational and family interventions, and supportive psychotherapy. Inpatient treatment may be indicated depending on the clinical situation.

The few available reports suggest that major tranquilizers have a role in treatment and may diminish the positive symptoms of the disorder (e.g., hallucinations and delusions) ([Kydd and Werry, 1982](#)). However, concerns about the possible long-term side effects of these medications suggest the need for informed consent, careful monitoring, and periodic reevaluation ([McClellan and Werry, 1994](#)). In general, no specific major tranquilizer is clearly superior, and the choice of medication should be guided by the particular constellation of problems exhibited. Particularly for children and adolescents whose disturbance does not respond to more usual agents, the "atypical" antipsychotic clozapine may be considered; although considerable interest has centered on this agent in adult "nonresponders," data in children are more limited ([Frazier et al., 1994](#); [Jacobsen et al., 1994](#); [McClellan and Werry, 1994](#)). In the only controlled trial of clozapine in childhood-onset schizophrenia, clozapine was found to be superior to haloperidol for both positive and negative symptoms in children nonresponsive to typical neuroleptics ([Kumra et al., 1996](#)). However, almost half of the patients have had to discontinue use of clozapine because of seizures, hematologic abnormalities, or treatment nonresponse over time. A "second generation" atypical antipsychotic medication, olanzapine, was subsequently studied in an open trial ([Kumra et al., 1998a](#)). The results were suggestive that this medication was effective in decreasing psychosis in at least some of the neuroleptic-nonresponsive children. However, many children in the sample remained poor responders to all forms of treatment. The authors suggest that given the relatively safe side-effect profile of olanzapine and enhanced therapeutic effects for some children in their study, it represents a good "first-line" agent for schizophrenia but not any other disorders in childhood. The agent is new and clinical experience reflecting its use in the pediatric population is limited ([Kumra et al., 1998a](#)). Evidence regarding the use of other classes of medications is even more restricted. Given the potential for stimulant medications to induce a psychotic illness, their use is probably contraindicated.

Examination of withdrawal dyskinesias (WD), tardive dyskinesia (TD), and extrapyramidal (EPS) side effects in children and adolescents who were in the early years of their treatment with neuroleptics showed that almost half presented with WD or TD at some point ([Kumra et al., 1998b](#)). There appeared to be an association between TD/WD and greater premorbid impairment, severity of illness, and duration of neuroleptic exposure. However, the abnormal movements seen in these patients were generally not severe, involved mostly the orofacial region, and improved over time. The incidence of EPS was lower in patients who received clozapine and the long-term use of this medication did not appear to be associated with development of TD. Recommendation was made for using the minimum effective dosage of antipsychotic medication in all cases, attempting to reduce gradually the dose of antipsychotic medication after the patient has been stable for 1 year. In addition, observing children and adolescents with psychotic disorders medication-free was found to be clinically informative as continued neuroleptic treatment was no longer warranted in some cases (e.g., owing to remission or an error in diagnosis) ([Kumra et al., 1998b](#)).

Studies of psychotherapy with this population are difficult to interpret given changes in diagnostic practice. It does appear that supportive psychotherapy may be of benefit to some children ([Cantor and Kestenbaum, 1986](#)). The usefulness of intensive, insight-oriented therapy is much less clear. Family interventions should aim to increase levels of appropriate family involvement and interaction to support the child's development and enhance communication among family members. Behavior modification procedures may be useful in reducing levels of maladaptive behaviors and increasing the availability of the child for educational and other interventions.

Many children with schizophrenia exhibit associated problems in development and learning that must be addressed through the provision of a comprehensive educational program. The disorder can have a deleterious impact on the acquisition of basic developmental and adaptive skills. Special education should be provided to address associated learning and developmental problems. It is important that the treatment program provided be integrated and that all the various professionals involved be aware of the need for close communication and the provision of a consistent program.

## OUTCOME AND FOLLOW-UP DATA

As with other aspects of the disorder, only limited data are available regarding the course and outcome of childhood schizophrenia, if the disorder is strictly defined. [Bennett and Klein \(1966\)](#) reported a follow-up of [Potter's \(1933\)](#) series of rather well-documented cases of children with schizophrenia. In these cases, the outcome of a 30-year follow-up was quite poor; only one case had made a satisfactory readjustment, although the histories of prolonged treatment in institutional settings may have worsened ultimate outcome.

[Eggers \(1978\)](#) conducted a 20-year follow-up of 57 children who exhibited schizophrenia before age 14. Unfortunately, the study is somewhat limited because the accuracy of early diagnosis and clinical information is questionable in these cases. About 20% of cases had apparently experienced complete remission, about 30% had improved, and the remaining 50% had had moderate or poor outcomes. The worst prognostic feature was an early onset (before age 10) in children with premorbid personality difficulties; all such cases had a poor outcome at follow-up. In a more recent report (mean follow-up period 38 years after onset), [Eggers and colleagues \(2000\)](#) also noted that the individuals in their sample showed a change in diagnostic subtype at different points in the total course of their disorder, indicating marked variability in the production of psychotic symptoms in the course of childhood-onset schizophrenia. [Kydd and Werry \(1982\)](#) reported on 15 children originally diagnosed as schizophrenic between 1971 and 1981. At follow-up, all cases were re-diagnosed using DSM-III. Of the 15 cases, 11 appeared unequivocally to exhibit schizophrenia, with the remaining four cases exhibiting a schizophreniform disorder. Earlier onset was associated with poorer prognosis; about half of the more strictly diagnosed schizophrenic cases exhibit chronic difficulties. In a more recent study, [Asarnow and colleagues \(1994b\)](#) found that most children (14/18) continued to meet criteria for schizophrenia 1 year after hospitalization. Of the remaining children, two showed schizoaffective disorder, one was in remission, and another showed no evidence of psychosis (but was diagnosed with conduct disorder, dysthymia, and ADD). At final assessment, between 2 and 7 years after hospitalization, a continued diagnosis of schizophrenia was present for 11 of 18 individuals. Patterns of outcome were also examined and showed substantial

variability when global adjustment was considered. Several of the children (8/18) showed relatively severe impairment throughout the follow-up period, and 10 out of 18 showed more substantial improvement.

As with adults, the use of psychotropic medications appears to be of particular benefit in relation to the “positive” symptoms of the disorder, although the dearth of studies in this area makes this observation only tentative at best. [McKenna and colleagues \(1994\)](#) reviewed 71 cases of children and adolescents referred for evaluation of what was presumed to be schizophrenia; only 19 children appeared to meet stringent criteria for the condition. As with adult schizophrenia, “negative” symptoms in childhood schizophrenia appear to be less responsive to pharmacologic intervention. There is some suggestion that the presence of more affective symptoms or a more schizoaffective clinical picture is associated with a more favorable course ([Eggers, 1989](#)). In general, it would appear that better outcome is related to acute onset, older age at onset, better premorbid adjustment, and well-differentiated symptomatology.

## RESEARCH DIRECTIONS

Research on essentially all aspects of the disorder is critically needed. Given the apparent wealth of information on childhood schizophrenia as broadly defined during the 1950s and 1960s, it is somewhat paradoxical that the available information on this disorder is now clearly less than that regarding autism. The relationship of childhood schizophrenia to adult forms of the illness remains an important topic for research; although essential points of phenomenologic similarity are apparent, the relative infrequency of the condition in childhood, the severity of the disorder and reason for its earlier age of onset, and the apparent relationship of subsequent outcome to early-onset suggest areas of ongoing research as well. The study of childhood schizophrenia would appear to be of considerable interest in the extent to which it may clarify aspects of syndrome etiology and pathogenesis. Studies of clinical populations are generally important for illuminating aspects of the disorder's natural history, neurobiology, treatment, and diagnosis. The applicability of current criteria for schizophrenia to children remains controversial; younger children may not as readily fulfill adult diagnostic criteria for the disorder, when rigidly applied. Misdiagnosis of the condition remains common ([McKenna et al., 1994](#)). The development of more precise and developmentally appropriate diagnostic criteria should facilitate research. Limited efforts to subtype affected individuals have been made ([Cantor, 1988](#)); the reliability and utility of such schemes remain to be established, but clearly, such work is critical ([Tsuang et al., 2000](#)).

Neurobiological studies have been relatively few in number; explication of underlying biological substrates of the disorder remains an extremely important area for future research ([Caplan, 1994](#); [Werry, 1996](#)). It will be important to explore further and determine the genetic basis for childhood schizophrenia and the factors associated with its earlier onset. Carefully designed follow-up studies are also needed to identify those factors most strongly related to ultimate outcome.

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### CASE ILLUSTRATION

Kristine was the second of two children born to upper-middle-class parents after a term pregnancy complicated by maternal viral illness and a prolonged labor. At birth, she was noted to be in good condition. She was a somewhat fussy baby whose motor and communicative development appeared to proceed appropriately. She appeared to be normally related and enjoyed social interaction with her parents and older sister. She was enrolled in a nursery school program at age 4 and subsequently in regular kindergarten and first grade classes; although she was noted to be somewhat shy, no other concerns were expressed about her cognitive or emotional development. At age 6 years 11 months, she exhibited an episode of acute hallucinations and delusions. This episode had been preceded by a period of several weeks during which she had seemed increasingly withdrawn and was observed talking to herself. The hallucinations were predominantly auditory in nature, but were not well described, and apparently consisted of hearing single words; delusions revolved around fears of being kidnapped. Kristine became markedly anxious and developed complex rituals and compulsive behaviors apparently involving some aspect of her delusional fears. She was hospitalized, but extensive medical investigation failed to reveal a specific medical explanation for her difficulties. A positive family history of schizophrenia in a maternal aunt was noted, and a presumptive diagnosis of schizophrenia was made. Low doses of neuroleptics led to partial remission of the hallucinations and delusions, although she remained quite withdrawn. After her hospitalization, she began twice weekly psychotherapy and was transferred to a private school where she received extensive special education.

She was seen for comprehensive reevaluation at age 10. At that time, psychological testing revealed a full scale IQ in the borderline retarded range with associated deficits in adaptive behavior. Projective testing revealed marked thought problems. Communication assessment revealed significant difficulties in communication, with marked difficulties in social interaction, use of irrelevant language, and neologisms. On psychiatric examination, Kristine was an attractive girl with a generally bland affect. Her initial comment on meeting the examiner was to say, “love you.” Associations were loose, with marked tangential thinking and irrelevant associations. She denied auditory hallucinations but did admit to seeing “monsters” on occasion; a relatively elaborate delusional system was present. Some obsessional thinking was also in evidence, as were a few peculiar mannerisms.

Kristine continued to have marked difficulties in adolescence. Although she received a comprehensive educational intervention program, deficits in academic performance and cognitive skills increased in severity. In late adolescence, the onset of marked behavior problems led to another hospitalization and subsequent placement in a residential institution.

#### Comment

This case illustrates various points at which childhood schizophrenia differs from autism: The onset, in this case, was after some years of apparently normal development, the disorder was characterized by prominent delusions and hallucinations, and there was a family history of schizophrenia. Children with an onset of schizophrenia in childhood have a less favorable prognosis than those with onset in adolescence.

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# 61 NEUROLOGIC DISORDERS

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Child psychiatrists and neurologists can benefit from a shared neuropsychiatric perspective that connotes sufficient familiarity with each other's field so as to enable each to optimally meet their patients' needs and to foster appropriate interdisciplinary communication.

A major requirement for the psychiatrist is to be sensitive and responsive to indications of neurogenic substrates that may underlie what may at first appear to be purely psychopathologic phenomena. The psychiatrist should also be prepared to diagnose and formulate treatment plans for psychiatric complications of primary neurologic disorders. (For information on neuropsychiatric signs, symptoms, and syndromes, see [Chapter 30](#); for information on acquired brain disorders, see [Chapter 33](#).)

This chapter deals with psychiatric aspects of the following representative neurologic disorders: epilepsy, brain tumors, and neurodegenerative and neuromuscular disorders. It is hoped that the perspective developed herein will be of value to the contemporary child and adolescent psychiatrist who, in the face of accumulating clinical and research data, is called on to marshal greater sophistication regarding diagnostic and therapeutic challenges at the neurology–psychiatry interface.

## EPILEPSY

Epilepsy is a condition characterized by sudden, recurrent, and transient disturbances of mental functions or body movements that result from excessive discharging of groups of brain cells ([Smith and Darlington, 1998](#)). As such, epilepsy does not refer to a specific disease but rather to a group of symptoms that have many different causes in different individuals. The underlying causes are static in some individuals and progressive in others. All of the underlying causes of epilepsy have in common the quality of causing cerebral neurons to become excessively excited. The statistically more common causes of this neuronal hyperexcitability generally involve either structural abnormality of the brain or biochemical aberrations of a metabolic, infectious, or other physical etiology. This broad spectrum of etiologies clearly includes both genetically based and acquired varieties of brain pathology, as well as potential combinations of these.

Reviews of the literature on the psychogenic precipitants of epileptic seizures ([Williams and Bergtraum, 2000](#); [Williams et al., 1993](#)) clarify that numerous studies in both experimental animals and patients have substantiated the capacity of emotional stress to precipitate epileptic seizures in neurologically vulnerable individuals. Hence, there is an important role for the psychiatrist not only in helping patients and their families to cope with the troubling psychiatric consequences of epilepsy, but also in potentially intervening in some cases with regard to the stresses that may bring on seizures.

The signs and symptoms of epilepsy are manifold. Among the most common manifestations are episodes of partial or complete loss of consciousness, localized or generalized muscular spasms or jerks, or apparently purposeful behavior performed while awareness is depressed. The multifaceted modes of clinical manifestation of epilepsy and the fact that electroencephalogram (EEG) recordings are not always diagnostic, lead to another major diagnostic and therapeutic challenge at the neuropsychiatric interface: recognizing and treating psychogenic nonepileptic seizures.

### Epidemiology

Estimates based on epidemiologic studies indicate that up to 1% of the population in the United States has epilepsy and that new cases appear at an annual rate of 40 per 100,000 (Hauser et al., 1991). Furthermore, the rate is highest in children younger than 5, with another peak of incidence at the age of puberty. Thus, it is clear that children and adolescents merit special consideration with regard to developmental issues that interact with the psychiatric complications of epilepsy.

There is evidence that socioeconomic factors play an important role in the development of epilepsy. For example, the incidence was found to be higher in black (1.96%) than white (0.95%) children living in New Haven, CT ([Hauser and Hesdorffer, 1990](#)). However, the cause of this difference is uncertain: The relative importance of perinatal factors, trauma, nutrition, other environmental influences, or genetics is unknown.

The relative role of inheritance in epilepsy is controversial. It is often difficult to distinguish social and economic factors from primary genetic predisposition, because poor nutrition and inadequate perinatal care frequently run in families. However, several studies indicate that, in patients with generalized epilepsy, close relatives have a twofold to fourfold increase in incidence of convulsive disorders, strongly suggesting a genetic component of vulnerability.

### Classification of Epileptic Seizures

A revised International Classification of Seizures based on a more contemporary understanding of seizure pathophysiology, which has served to improve professional communication, is summarized as follows ([Alarcon, 1998](#)):

- I. Partial seizures (seizures beginning locally)
  - A. Simple partial seizures (consciousness not impaired)
    1. With motor symptoms
    2. With somatosensory or special sensory symptoms
    3. With autonomic symptoms
    4. Compound forms

- B. Complex partial seizures (with impairment of consciousness; temporal lobe or psychomotor seizures)
  - 1. With impairment of consciousness only
  - 2. With cognitive symptomatology
  - 3. With affective symptomatology
  - 4. With "psychosensory" symptomatology
  - 5. With "psychomotor" symptomatology (automatisms)
  - 6. Compound forms
- C. Partial seizures secondarily generalized
- II. Generalized seizures (bilaterally symmetric and without local onset)
  - A. Absence seizures
  - B. Myoclonic seizures
  - C. Infantile spasms
  - D. Clonic seizures
  - E. Tonic seizures
  - F. Tonic-clonic seizures
  - G. Atonic seizures
  - H. Akinetic seizures
- III. Unilateral seizures
- IV. Unclassified epileptic seizures (because of incomplete data)

Space limitations preclude more detailed consideration of these clinical subtypes here, but it clearly behooves the psychiatrist working with a seizure patient to be cognizant of the specific seizure manifestations, associated clinical and prognostic features, and specific medication requirements as determined by the patient's specific seizure type.

## PSYCHOLOGICAL AND PSYCHIATRIC ASSESSMENT

### Cognitive Functioning

An early review of several studies in this area ([Stores, 1981](#)) indicated that, although many children with epilepsy function normally at school, proportionately more of them have learning problems as compared with nonepileptic children. In the Isle of Wight study, for example, which concentrated on 9- to 11-year-old children, over twice as many of those with epilepsy showed serious specific reading retardation when compared with nonepileptic children.

Developmental considerations are important. [O'Leary and associates \(1981\)](#) administered a neuropsychological test battery to 48 children age 9 to 12 with tonic-clonic seizures. The children with seizures of early onset (before age 5) were significantly impaired, compared to the children with seizures of later onset, on eight of the 14 measures in the battery. The deficits were seen on tasks whose requirements included attention and concentration, memory, complex problem solving, and motor coordination.

Seizure type, duration, and degree of seizure control are also pertinent in the findings of some studies. [Farwell and coworkers \(1985\)](#) did detailed neuropsychological testing of 118 epileptic children, age 6 to 15, and a control group of 100 children without seizures. The Wechsler full-scale IQ of seizure patients was significantly lower than that of control subjects and was related to seizure type. Children with minor motor or atypical absence seizures had the lowest average full-scale IQ. All seizure types, except classic absence alone, were associated with below-control intelligence. Intelligence was also correlated with degree of seizure control. A highly significant inverse correlation between years with seizures and intelligence was found. Finally, children with seizures had been placed in special education or had repeated a grade in school almost twice as frequently as control subjects, and their academic achievement was behind grade placement more often than that in the control group.

[Seidenberg and colleagues \(1986\)](#), who examined the academic achievement scores of 122 children with epilepsy, supported the essential features of the preceding study. As a group, these children were making less progress than expected for their age and IQ level. Academic deficiencies were greatest in arithmetic, followed by spelling, reading comprehension, and word recognition. Here also, clinically relevant variables included age of seizure onset, lifetime total seizure frequency, and presence of multiple seizures.

The study by [Ellenberg and associates \(1986\)](#) is illuminating lest one falls prey to the erroneous assumption that childhood epilepsy necessarily engenders intellectual deterioration. They compared the full-scale IQs at 7 years of age of children who had experienced one or more nonfebrile seizures, with the IQs of their seizure-free siblings who were tested at the same age in a large longitudinal study. Among 98 children with seizures, the mean score on IQ tests at 7 years was not significantly different from the mean score of their siblings. Mental retardation was more common among the children with seizures, but children who had had neurologic abnormalities before the first seizure accounted for the excess of retardation. It seems likely that sampling differences account for the differences in findings between this and previously cited studies, with the study by Ellenberg and associates drawing a larger proportion of "mild" cases (i.e., better controlled) than the previous studies.

More recent studies, utilizing more comprehensive neuropsychological assessments, have substantiated deficits in both verbal and visual attentional skills in children with epilepsy ([Williams et al., 1998](#)). Furthermore, the finding of diffuse cognitive dysfunction across all seven cognitive domains assessed was documented in a sibling-controlled study of epileptic children with complex partial seizures ([Schoenfeld et al., 1999](#)). A large, retrospective study, involving 251 children with epilepsy, pointed to multiple factors impacting on the degree of cognitive impairment, including age of onset and duration of epilepsy, seizure frequency, and number of antiepileptic drugs used ([Bulteau et al., 2000](#)).

Anticonvulsant drugs, especially in combination, have been shown in a variety of studies to interfere in significant ways with cognitive function, including attention, concentration, memory, motor and mental speed, and mental processing ([Reynolds, 1985](#)). These sometimes-subtle effects are easily overlooked but may accrue to the point of substantial impairment, particularly for children in learning situations ([Gerber et al., 2000](#)).

Other studies have shown that most epileptic patients can be controlled on single-drug therapy and that there is often little difference in antiepileptic efficacy between the major antiepileptic drugs within designated seizure categories ([Voorhies, 1988](#)). Consequently, the relative influence of each drug on cognitive function may prove to be a most important factor in the choice of an anticonvulsant drug. In this regard, it should be noted that several studies have shown significant associations between long-term use of phenytoin and phenobarbital and cognitive deterioration in children. By contrast, carbamazepine has been noted in some studies to be less prone to contribute to cognitive impairments in both short- and long-term use in both normal volunteers and seizure patients. This finding needs to be qualified, however. [Forsythe and colleagues \(1991\)](#) found, in a controlled, prospective study of newly diagnosed childhood epilepsy, that carbamazepine in moderate dosage adversely affected memory, but phenytoin and sodium valproate did not; valproate had the fewest side effects.

More recent controlled studies have suggested a more sanguine outlook when anticonvulsants are carefully monitored. [Mandelbaum and Burack \(1997\)](#) did a 12-month prospective follow-up study of 43 children with a variety of seizure types. Analysis of the children's performance after 6 and 12 months of antiepileptic therapy showed no significant deterioration attributable to medication. Other studies of children whose seizures were controlled at therapeutic doses of antiepileptic drugs (AEDs), similarly found no evidence of AED-induced cognitive impairment ([Aldenkamp et al., 1998](#); [Bates et al., 1998](#)). Nevertheless, the clinician must be vigilant in identifying subgroups of children who may be particularly vulnerable to cognitive and/or behavioral side effects of particular AEDs or combinations of AEDs ([Aldenkamp et al., 1998](#); [Bourgeois, 1998](#)). In all likelihood, the greater sophistication in AED serum monitoring, coupled with more sophisticated methodology and study design account for the more favorable outlook of more recent studies ([Cochrane et al., 1998](#); [Espie et al., 1999](#)).

In light of the preceding observations, one should not assume that all cognitive impairment of youngsters with epilepsy is owing to either static cerebral pathology or the side effects of anticonvulsant medication. [Siebelink and coworkers \(1988\)](#) studied 21 children with epilepsy having subclinical epileptiform EEG discharges in the waking state, using telemetered EEG studies during psychological testing. The authors found a total IQ for the children with epilepsy below that of control populations, which was accounted for by that subgroup of children who exhibited discharges during the test; those who did not show discharges at that time were unimpaired. These findings have been replicated and amplified by [Aldenkamp and associates \(1999\)](#), who similarly identified a subgroup of children with epilepsy and learning disabilities who had difficult-to-detect seizures, demonstrating that uncontrolled seizures can impair academic functioning even when the seizures are of short duration and have subtle symptoms. These studies have implications both for the interpretation of neuropsychological test results in children with epilepsy and also



for the drug treatment of those who continue to exhibit subclinical EEG discharges when overt seizures have been controlled.

It can be discerned readily from the preceding that primary attention should be given to periodic formal monitoring of the cognitive functioning of seizure patients, with particular view to picking up signs of progressive cognitive impairment that might otherwise be overlooked. It is certainly within the purview of a psychiatrist treating such a patient to ascertain that such monitoring has occurred and that therapeutic serum anticonvulsant levels have been documented and to discuss the possible need for a change in anticonvulsant regimen when indicated by the patient's level of cognitive functioning with the treating neurologist ( [McConnell and Duncan, 1998](#)).

### **Behavior and Personality**

The literature on personality changes in epilepsy has been prolific, long-standing, and controversial. According to some estimates, for example, 30% to 40% of patients with temporal lobe epilepsy (complex partial seizures) experience persistent psychiatric symptoms that, more frequently than uncontrolled seizures, become the most incapacitating aspect of the illness ( [Tucker and McDavid, 1997](#)). Multiple clinical reports in adult temporal lobe seizure patients have delineated characteristic features, including deepened emotions, changes in sexual function, aggressivity, development of intense religious or philosophic interests, circumstantiality, and interpersonal viscosity.

A study in children with unilateral temporal lobe seizure foci failed to disclose characteristic differences in cognitive or personality features between right and left temporal lobe seizure patients ( [Camfield et al., 1984](#)). Yet, when the two groups of unilateral seizure patients were combined, 10 of 27 patients (five with left focus, five with right focus) were seen to have personality maladjustment on formal assessment. Furthermore, the group as a whole showed significantly lower neuropsychological test functioning than normally adjusted children.

Developmental considerations may help understand the evolution of certain psychopathologic correlates of the seizure state over time. Thus, [Flor-Henry \(1983\)](#) has studied severe psychopathologic syndromes (psychosis) seen in adult seizure patients. He found that schizophreniform psychosis in these patients was related to pathology in the dominant hemisphere, whereas depressive psychosis (and neurosis) was related to pathology of the nondominant hemisphere. In relation to this, [Lindsay and colleagues \(1979\)](#) did a long-term outcome study of 100 children with temporal lobe seizures. Mental deficiency, the hyperkinetic syndrome, and cataclysmic rage outbursts were prominent in childhood. Indeed, only 15 of the 100 probands were wholly free from psychological problems in childhood. Yet follow-up indicated that the occurrence of overt psychiatric disorder in adult life was relatively low: Of those survivors who were not gravely mentally retarded, 70% were regarded as psychiatrically healthy. Overt schizophreniform psychoses developed in 10% of the survivors. Males with continuing epilepsy and left-sided foci were at special risk: 30% of such patients became psychotic. In consonance with the noted findings of Flor-Henry and others, no patient coded as having a right-sided focus in childhood became psychotic by the time of follow-up 13 years later. Lindsay and colleagues emphasize the surprising and hopeful change from the overwhelming presence of psychopathology in the childhood sample to the predominance of relative psychological intactness by adulthood. They note that the majority of their patients received psychiatric intervention in their early years and emphasize the importance of providing such service in the appropriate management of epileptic children.

A study by [Hoare \(1984a\)](#) reflected further indications of the probably primary role of central nervous system (CNS) dysfunction in the psychiatric vulnerabilities of patients with epilepsy. Two groups of epileptic children, one newly diagnosed and one with chronic epilepsy, were contrasted with two comparable groups of diabetic children and with a nonpatient sample in order to evaluate the development of psychiatric disorder. The results confirm previous findings that children with chronic epilepsy are significantly more disturbed than children with chronic physical illness not involving the CNS and children in the general population. Children with newly diagnosed epilepsy were also significantly more disturbed than those with newly diagnosed diabetes and children in the general population. In both groups of epileptic children, those with focal EEG abnormalities and/or complex partial seizures were particularly vulnerable to psychiatric disturbance. The development of inappropriate dependency was also greater in the two epileptic groups of children than the comparison groups ( [Hoare, 1984b](#)).

Similar findings are reported by [Austin and associates \(2000\)](#) in a follow-up study that disclosed that patients with high seizure frequency at baseline and follow-up, particularly girls, developed higher rates of behavior problems than a comparison group of patients with asthma. [Schoenfeld and coworkers \(1999\)](#) reported increased social and school competence problems, particularly of the internalizing type, when comparing pediatric epilepsy patients with complex partial seizures to sibling controls.

An additional finding pointing to the increased risk of psychopathology among children with epilepsy is an epidemiologic study by [Hackett and colleagues \(1998\)](#). Additional specific risks in various comparative studies of epileptic children include hyperactivity, when the epilepsy is associated with severe intellectual disabilities ( [Jones and Cull, 1998](#)), and depression ( [Dunn et al., 1999](#)) in nonselected adolescent epileptic patients, as well as thought disorder and schizophreniform psychosis in pediatric patients with complex partial seizures ( [Caplan et al., 1997, 1998](#)).

In addition to the previously noted capacity of anticonvulsants to engender untoward cognitive side effects, these medications also can cause behavioral and emotional symptoms. The capacity of phenobarbital to generate or exacerbate attention deficit hyperactivity symptoms in children is well documented ( [Voorhies, 1988](#)). Additionally, [Brent and coworkers \(1987\)](#), in a study comparing 15 epileptic children on phenobarbital to 24 treated with carbamazepine, found a much higher prevalence of major depressive disorder in those on phenobarbital (40% versus 4%,  $P > 0.02$ ) and suicidal ideation (47% versus 4%,  $P > 0.005$ ), as determined by semistructured psychiatric interviews. The differential prevalence of depression between medication groups was only noted in those with a family history of a major affective disorder among first-degree relatives.

It should be noted that not all studies of psychopathology among children and adolescents with epilepsy find an increased incidence in these youngsters as compared with control groups ( [Kaminer et al., 1998](#)). Thus, [Kokkonen and associates \(1997\)](#) studied psychosocial outcome of young adults who had epilepsy in childhood. They found that the social handicaps found in some of these patients as young adults was largely associated with the neurologic and cognitive impairments accompanying the epilepsy, rather than the epilepsy itself or associated anticonvulsant medication.

Yet the preponderance of studies suggest that youngsters with epilepsy are at increased risk with regard to the development of psychiatric disturbance. Certainly not every youngster with epilepsy requires psychiatric attention, but a substantial proportion does stand to benefit therefrom. A psychiatrically sophisticated neurologist who routinely follows these youngsters and can make appropriate early referral for psychiatric assessment when needed is probably the best conduit for such intervention. Once this has been done, assessment should follow a standard age-appropriate format for the patient and family, with special focus on those areas of vulnerability noted in the preceding.

### **Psychogenically Precipitated Epileptic Seizures and Psychogenic Nonepileptic Seizures**

Based on the substantial evidence pointing to the enhanced cognitive and emotional vulnerability of youngsters with epilepsy, it is not surprising that one manifestation of this vulnerability would be a worsening of their presenting seizure symptoms under circumstances of emotional stress. It is important, however, for the clinician to be thoughtful and sensitive in exploring this area because of the many complexities that abound in both differential diagnosis and treatment.

[Williams and Bergtraum \(2000\)](#) reviewed several studies pointing to the role of environmental stress and emotional experiences as precipitants of true epileptic seizures. These included reports of the emotional activation of EEG abnormalities in patients with convulsive disorder, particularly those with sensory (simple partial) and complex partial seizures when exposed to stress interviews. Furthermore, the direct emotional activation of seizures has been documented in several animal species when seizure-prone animals were exposed to various forms of environmental stress. In addition, certain stressful cognitive tasks can also generate epileptiform EEG activation, though more commonly in patients with idiopathic generalized epilepsies ( [Matsuoka et al., 2000](#)). These stimulus-induced seizures are designated here as psychogenically precipitated epileptic seizures.

In contrast to the preceding, the term psychogenic nonepileptic seizures has been used to designate seizure-like phenomena of purely psychological origin produced by patients with varying degrees of conscious or unconscious intentionality and for a vast array of psychological reasons. Although reviewed in detail elsewhere ( [Williams, 1997](#)), it is worth noting the general categories of diagnostic classification to be considered in the process of assessment, including somatoform disorders, factitious disorders, and malingering.

Sometimes it may be extremely difficult for the clinician (neurologist, psychiatrist, or other) to be certain about the distinction between epileptic seizures (whether psychogenically precipitated or not) and psychogenic nonepileptic seizures, even after a thorough review of the history, physical examination, and laboratory findings. This is not only because of the protean manifestations of epileptic phenomena but also because many patients frequently possess an artful capacity to mimic epileptic

seizures on a conscious or unconscious basis.

Simultaneous videotape and EEG recording has been a significant aid in the differential diagnosis of epilepsy and pseudoseizures ( [Gates, 1998](#)). The procedure focuses one camera on the patient, who is wired with electrodes connected to an electroencephalograph, while a second camera is focused on the EEG write-out. The picture of the patient and the simultaneous EEG tracing appear on a split-screen video monitor. This combined recording is stored on the videotape for future analysis and interpretation. Studies using this technique have helped clarify that, even patients with clearly documented psychogenic nonepileptic seizures, there may be a coexisting neurogenic seizure disorder in from 15% to 50% of patients.

One limiting factor with the differential diagnostic capacity of video-EEG monitoring, despite its allowing a much longer observational period than a routine EEG, is that the patient may not experience a typical seizure event during the monitoring period. To augment the diagnostic yield of this valuable monitoring technique, a variety of suggestive procedures, including intravenous placebo infusion and hypnosis have been used ( [Walczak et al., 1994](#)). Placebo infusion elicits typical psychogenic events in most patients with psychogenic seizures. However, atypical events or epileptic seizures may occur in a minority of patients. This points to the need for an adequate monitoring period, corroboration with family or other observers that the elicited events are typical, and close collaboration between the neurologist and psychiatrist in conducting a comprehensive evaluation of both these overlapping areas.

Some brief guidelines may be outlined for psychiatric intervention in such situations. First, if the diagnosis is not entirely clear at the time of initial psychiatric consultation, as is most often the case in these circumstances, it is best to approach the patient and family with a candid acknowledgment of this. The role of the psychiatrist is explained as that of exploring the possibility that emotional factors may play a role in symptom formation, as they can with virtually any physical symptom (e.g., headache, peptic ulcer, hypertension, etc.). Second, a traditional psychiatric assessment of the patient and parents, separately and together, is then pursued, with particular focus on possible sources of stress or conflict that might contribute to symptom formation. It is best to conceive and discuss any psychodynamic or other formulation deriving from such an exploration in tentative, hypothetical terms, with due regard to the patient's self-esteem and especially avoiding any confrontational or accusatory stance with the patient or family. Third, if the combined outcome of neurologic and psychiatric assessments points to the probability that emotional factors do contribute to symptom genesis, then a treatment plan tailored to the individual circumstances of the child and family needs to be formulated ([Williams and Bergtraum, 2000](#); [Wyllie et al., 1999](#)).

### **Epilepsy and Aggressive Behavior**

There is controversy in the literature regarding the association between epilepsy on the one hand and aggression and violence on the other ( [Herzberg and Fenwick, 1998](#)). This continues to be so despite accumulating evidence pointing to the clinical validity of subacute postictal aggression ( [Gerard et al., 1998](#)) and to neuroimaging studies pointing to a neuropathologic substrate for the phenomenon ( [Woermann et al., 2000](#)). One well-controlled study of 83 children with epilepsy found an association between the presence of anterior temporal lobe epileptiform spike activity and increased aggression scores on the Achenbach Child Behavior Checklist ([Whitman et al., 1982](#)). The authors note, however, that such biological variables predictive of behavioral disorder accounted for only small amounts of the variance in their comparison of children with temporal lobe and generalized epilepsies. In this regard, they recommend that more consideration be given to delineating the "situation-centered" variables predictive of behavioral disorder in children with epilepsy.

Using a different research strategy, clinical studies of violent incarcerated adolescents by [Lewis and coworkers \(1985\)](#) have suggested that epilepsy, especially psychomotor (complex partial, temporal lobe) epilepsy, is more prevalent in young offenders than the general population. Furthermore, psychomotor epilepsy in this population was particularly associated with violence. The authors review some of the diagnostic difficulties inherent in establishing the diagnosis of psychomotor seizures in an individual where such a clinical diagnosis may interact with the perception of legal responsibility for a violent act. Despite some of the imprecision inherent in diagnosing psychomotor seizures, however, added circumstantial support of the diagnosis was lent by the very frequent history of severe CNS trauma, owing to such factors as perinatal difficulty, CNS infection, head injury, or a history of frank grand mal seizures. Of note also was the frequent association of reports of severe physical abuse in the histories of these youngsters. The latter association in particular certainly addresses the probable role of situational variables in fostering violence in youngsters biologically at risk by virtue of a greater predisposition to impulsivity and aggression. Most important, Lewis emphasizes in this and subsequent reports that a clinician called on to evaluate a violent delinquent may render a valuable service in identifying potentially treatable neuropsychopathology, so appropriate intervention can occur ( [Lewis, 1997](#)).

### **TREATMENT**

In view of the generally acknowledged enhanced psychological vulnerability of epileptic patients, particularly to the effects of anxiety and depression ( [Robertson, 1998](#)), it is clearly advisable to provide appropriate intervention when such symptoms are in evidence. In this regard, the elevated risk of suicide in epileptic patients is noteworthy. Psychiatric interventions with such patients should be geared to the nature of the presenting symptomatology, while taking note of the relevance of the patient's epileptic condition.

An important consideration in treating any child or adolescent with epilepsy and an associated psychiatric problem is the need to take into account family dynamics pertinent to the genesis of the problem, as well as family resources that can be mobilized in the service of effective treatment. This is particularly relevant in light of the frequently encountered problem of heightened dependency needs in such patients, noted in the preceding. Finally, the importance of maintaining open lines of communication between the neurologist and mental health practitioner to ensure appropriate psychobiological treatment integration cannot be overemphasized.

### **PSYCHOTHERAPEUTIC INTERVENTION**

A somewhat unique psychotherapeutic challenge in this population is that of treating psychogenically precipitated epileptic seizures and psychogenic nonepileptic seizures. There are many reports of successful psychotherapeutic or psychobiological treatment interventions for purposes of seizure control ( [Williams and Bergtraum, 2000](#)). It should be emphasized that appropriate differential diagnostic assessment is a prerequisite to appropriate treatment planning in this area. The various treatment approaches utilized may be summarized under the following broad headings: conditioning techniques, psychodynamic approaches, relaxation and hypnosis, and biofeedback. Often several such approaches may be usefully integrated in the service of maximizing therapeutic impact to facilitate symptom relinquishment and foster improved adaptation by the patient.

It should be noted for patients with refractory epileptic seizures, that seizure surgery, with recent improvements in technique and technology, has been offering some very promising results ([Engel, 1996](#)). Thus, [Meyer and colleagues \(1986\)](#) reported that of 50 child and adolescent patients receiving temporal lobectomy for medically refractory seizures, 78% became essentially seizure-free, and 88% benefited significantly from the operation in terms of improved seizure control. The importance of careful and extended presurgical seizure monitoring cannot be overemphasized, however. Our own clinical experience includes a patient who illustrates this point.

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### **CASE ILLUSTRATION**

R., a 9-year-old girl with an IQ of 117 and a history of gelastic (complex partial) seizures dating from the first year of life, was referred for seizure surgery because of a progressive worsening of her seizures despite numerous anticonvulsant medication trials. Seizure surgery was not performed, despite a diffusely abnormal electroencephalogram (EEG), because a circumscribed seizure focus accessible to surgical removal could not be delineated. While in the hospital, her histrionic and manipulative style was observed, as was the fact that her seizures were more numerous when her parents were present, reaching a peak of more than 300 a day. In light of no further deterioration in her previously abnormal EEG, the consulting psychiatrist postulated a hypothesis of either psychogenically precipitated epileptic seizures or psychogenic nonepileptic seizures, or both, superimposed on a preexisting epileptic seizure substrate. After conferring with the neurologist, an intervention of intensive psychotherapy was initiated, including individual, parental, and family sessions, behavior modification, and hypnosis. Within 2 weeks, seizure frequency had diminished to zero, and R. was discharged from the hospital. She immediately returned to school and continued to be seizure-free as she was followed over 6 months in supportive outpatient psychotherapy. Her anticonvulsant medication was gradually tapered, and she continues seizure-free off all anticonvulsant medication for a 10-year period. In retrospect, the hypothesis of spontaneously remitted epileptic seizures and superimposed psychogenic seizures has been confirmed by the patient's clinical response to treatment. The absence of video-EEG monitoring during this hospitalization made it impossible to define whether psychotherapy had been primarily beneficial in alleviating psychogenically precipitated epileptic seizures, psychogenic nonepileptic seizures, or both.

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### **Psychopharmacologic Considerations**

The need for the clinician to be aware of potentially cognition-impairing effects of various anticonvulsants, particularly phenytoin and phenobarbital, has been noted. Additionally, the frequently observed capacity of phenobarbital to generate hyperactivity, irritability, and aggressivity in children should be borne in mind ( [McConnell and Duncan, 1998](#)). Consequently, it is clearly important to advocate the lowest reasonable dose of the least toxic anticonvulsant consonant with good seizure control. Carbamazepine, for example, has been reported in a number of studies to manifest mood stabilizing, as well as anticonvulsant properties ( [Evans et al., 1987](#)). However, it has been recognized that negative psychiatric consequences of carbamazepine also may be encountered ( [Pleak et al., 1988](#)); therefore, careful clinical



monitoring is in order.

Another developmentally important consideration in children and adolescents is their different rates of metabolizing anticonvulsants. Children generally require higher milligram per kilogram doses of anticonvulsant than do adults by virtue of their metabolizing the drugs more efficiently. There is a shift toward the adult rate in early adolescence. Consequently, an anticonvulsant dose that yielded therapeutic blood levels in late childhood may be transformed insidiously into a toxic dose in early adolescence if not monitored closely. An associated consideration is the advisability of immediately checking a youngster's serum anticonvulsant level if there is any sudden change in cognitive, emotional, or behavioral functioning, or a worsening of seizure control. Questions of compliance in taking anticonvulsant medication always should be considered ([Shope, 1988](#)).

In many epileptic patients, even when the anticonvulsant regimen is optimized, there may still be indications for the additional use of psychotropic medication for comorbid psychopathology. Benzodiazepines have a valid place in this regard when minor tranquilization is needed. The value of intravenous diazepam or lorazepam in status epilepticus is well known, so there is clearly no danger of its lowering the seizure threshold, but the capacity to enhance sedation by interaction with a coexisting anticonvulsant or to generate behavioral disinhibition should be noted ([Stewart et al., 1990](#)).

Although there is a theoretical basis of concern about stimulants lowering the seizure threshold based on animal studies involving high dosages, clinical experience suggests that methylphenidate and other commonly used stimulants may be a safe and effective treatment for certain children with seizures and concurrent attention deficit disorder, provided that titration is pursued judiciously ([Stimmel and Dopheide, 1996](#)).

Major tranquilizers do have a place in the treatment of interictal psychoses in patients with epilepsy ([McConnell and Duncan, 1998](#)). It should be noted, however, that several neuroleptics, particularly chlorpromazine and clozapine, have a significant capacity to lower the seizure threshold and induce seizures. Of the neuroleptics currently available, molindone, haloperidol, and risperidone appear to be the safest in this regard.

Most tricyclic antidepressants also have the capacity to lower the seizure threshold and induce seizures ([Wroblewski et al., 1990](#)). Although the serotonergic antidepressants as a group have less of a tendency to lower the seizure threshold, several can interact with anticonvulsants; therefore, close monitoring of blood levels of anticonvulsants is warranted. Clinically, one must balance the potential benefits in combating depressive symptoms against the potential impact of possible drug interactions or lowering of the seizure threshold.

There is clinical evidence of the potential benefit of adding propranolol to the medication regimen of epileptic patients with uncontrolled aggressive outbursts that have not been controlled by optimal anticonvulsant medication adjustment ([Connor and Steingard, 1996](#); [Silver et al., 1999](#); [Williams et al., 1982](#)). Although this has not yet been subjected to controlled study, open clinical experience to date suggests that a significant proportion of such patients demonstrate improved behavioral control of aggressive behavior and irritability on propranolol. As long as titration is gradual, with appropriate monitoring of ECG, pulse, and blood pressure and with cognizance of the relevant contraindications to using adrenergic blocking medication, side effects are minimal and well tolerated.

## Prevention

Efforts at prevention of epilepsy are best addressed to the most common and remediable etiologies. These would include improved perinatal care, genetic counseling of individuals with family histories of epilepsy, and early intervention with those infectious, traumatic, or metabolic aberrations known to predispose to epilepsy. Efforts at prevention of the psychiatric complications of epilepsy include judicious use of anticonvulsant medications to minimize adverse side effects, as well as appropriate education of the patient and family to enable early recognition and appropriate early treatment of psychiatric complications should they arise. Finally, educating the public about epilepsy is important to minimize the unwarranted stereotypic stigmatization and discrimination that have long burdened these patients and their families.

## BRAIN TUMORS

The incidence of intracranial tumors in youths below the age of 15 years ranges from two to five in 100,000 per year, which ranks these tumors second only to leukemias as neoplasms in children. Brain tumors make up roughly half of all solid tumors in children. Although supratentorial tumors constitute the majority of brain neoplasms in adults, infratentorial tumors predominate in children. Generally, infratentorial or posterior fossa tumors (i.e., those of the cerebellum and brain stem) are more silent than supratentorial tumors, and psychiatric symptoms such as personality change are less common. Instead, children with brain tumors tend to show more signs and symptoms of increased intracranial pressure, often with insidious onset and without localizing signs ([Herskowitz and Rosman, 1982](#)). The majority of intracranial neoplasms in children are primary tumors of glial origin, whereas metastatic tumors and meningiomas are quite rare. Thus, the location, presenting symptoms, and representation of the various types of brain tumors in youth differ from those of adults.

The etiology of most nonmetastatic intracranial tumors is unknown; therefore, prevention is not possible. Craniopharyngiomas and teratomas are of congenital origin, arising from developmental malformations. Intracranial sarcomas are increased by cranial irradiation. Some tumors are associated with certain systemic conditions (e.g., Wiskott-Aldrich syndrome, renal transplantation, and leukemia) and neurocutaneous syndromes (e.g., ataxia-telangiectasia, neurofibromatosis, and tuberous sclerosis) ([Evans, 1987](#)).

## Clinical Features

The presenting symptomatology of brain tumors is largely dependent on tumor location and rate of growth. Because of the predominance of infratentorial and midline locations, most brain tumors in children tend to lead to obstruction of cerebrospinal fluid circulation (CSF), resulting in increased intracranial pressure. The symptoms and signs of increased intracranial pressure (IICP) are somewhat variable and are presented in [Table 61.1](#). It should be noted that IICP has multiple etiologies (e.g., metabolic encephalopathy, intracranial infection, pseudotumor cerebri, and trauma) and is not pathognomonic for brain tumor. Headache is rarely localizing and is not qualitatively different in these brain tumors from that seen with other conditions. Transient accommodation of the brain and skull to IICP is thought to account for the remitting and exacerbating nature of the headache, which is often worse after periods of recumbency or with straining. Similarly, vomiting is often mild, variable, and intermittent.

1. Headache: Bifrontal or occipital; often mild; worse in the morning, after recumbency, and with coughing or Valsalva; progressively persistent.
2. Vomiting: Often mild, not usually projectile; intermittent; occurs on rising; with or without headache; progressively more frequent.
3. Personality changes: Restlessness and irritability, memory loss, academic impairment; later, lethargy, apathy, depression, somnolence, and altered consciousness.
4. Papilledema (more common in children after suture fusion).
5. Diplopia, strabismus (cranial nerve VI palsy), impaired upward gaze and proptosis ("setting sun"); cranial nerve III palsy.
6. Bulging fontanelle and delayed fontanelle closure (infancy).
7. Widened sutures on x-ray and increased head circumference (3-2 years).
8. Altered vital signs: Increased blood pressure, increased or decreased pulse, decreased and irregular respirations.
9. Diffuse slowing on EEG.
10. Generalized seizures.

**Table 61.1. Signs and Symptoms of Increased Intracranial Pressure in Children, in Approximate Order of Presentation**

Neuropsychiatric symptoms are common but nonspecific (e.g., deterioration in school functioning) ([Kaye and Duffner, 1998](#)). Personality changes can be seen, such as quietness and decreased motoric activity (in an attempt to lessen headache), restlessness and irritability (often owing to headache as well), and diminished levels of consciousness, leading to coma. Presenting symptoms other than those owing to IICP are discussed for each tumor type.

Besides the neurologic and psychiatric history and examination, intracranial tumors are diagnosed by skull radiography and especially by computed tomography (CT) and nuclear magnetic resonance imaging (MRI) of the brain.

Characteristics of various brain tumors are presented in [Table 61.2](#). Classifications of childhood brain tumors are arbitrary and have undergone change in recent

years ([Becker, 1985](#); [Swaiman, 1994](#)). The classifications are still rather controversial, partly because of the unusual patterns of growth seen in these tumors, which are comprised of embryonic or poorly differentiated cells. Further details concerning these tumors are discussed in the following, especially the psychiatric symptomatology and the effect of treatment on neuropsychological functioning.

**Table 61.2. Characteristics of Brain Tumors in Children**

### Infratentorial Tumors

Infratentorial tumors are those of the cerebellum, fourth ventricle, and brain stem. For all of these tumors, IICP is a prominent feature and may be the only cause of symptoms early on. Other presenting symptoms are owing to compression of brain stem structures and include progressive cranial neuropathies, often involving ocular and facial muscles; nystagmus, which is nonlateralizing; spasticity; ataxia; cervical pain; and head tilt.

Astrocytomas and medulloblastomas are the two tumor types of the cerebellum and have approximately equal incidences. Cerebellar astrocytomas (primarily pilocytic) are slow growing and mostly noninvasive. The onset of symptoms is insidious, usually beginning many months prior to diagnosis with mild vomiting and/or headache. Increased intracranial pressure is caused by compression of the fourth ventricle and cerebellar-pontine angle cisterns ([Laurent and Cheek, 1986](#)). Nystagmus, ataxia, unilateral hypotonia, and intention tremor also may be present. The course is usually benign. Treatment consists of resection; CSF shunting, if required; and radiation therapy, depending on malignancy and extent of the resection. Medulloblastoma, in contrast, is rapid growing and highly malignant. This tumor arises from the midline cerebellar vermis and may seed into the fourth ventricle and other structures. The onset of symptoms is acute, with vomiting, headache, and axial ataxia presenting several weeks to 2 months prior to diagnosis. Involvement of the reticular activating system may lead to an altered state of consciousness known as a coma vigil or akinetic mutism. Treatment consists of excision and chemoradiotherapy (owing to universal recurrence and inability to resect the entire tumor). The prognosis is not good; however, the 5-year survival rate has improved to about 50% with aggressive surgery and high-dose irradiation ([Cohen and Duffner, 1994](#)).

Fourth ventricle tumors include ependymomas and choroid plexus papillomas. Ependymomas, which may arise in any ventricle, predominate in the fourth ventricle in children and may lead to early CSF flow obstruction. Increased intracranial pressure is the presenting condition and, when the tumor is infratentorial, may be accompanied by nystagmus, head tilt, and ataxia. Following excision and irradiation, survival rates range from 15% to 100%, depending on the malignancy of the ependymoma and whether seeding has occurred ([Cohen and Duffner, 1994](#)). Choroid plexus papillomas arise primarily in the fourth or lateral ventricles, are highly vascularized, and produce CSF, which results in IICP. Additional symptoms reported in one 12-year-old boy by [Blackman and Wheeler \(1987\)](#) consisted of somatic complaints, separation anxiety, school refusal, fearfulness, and crying episodes for a year. This child also had mild ataxia and impaired coordination since the age of 2 and had a head circumference at the 98th percentile. CT scan made the diagnosis during the second psychiatric hospitalization. Tumor resection resulted in amelioration of the depressive and anxiety symptoms.

Brain stem tumors are primarily gliomas of the pons. Increased intracranial pressure may not be present. Common symptoms are cranial nerve deficits, ataxia, spasticity, and gait disturbance and a classic triad of progressive cranial neuropathies, pyramidal tract signs (hemiparesis), and cerebellar pathway signs (truncal and extremity ataxia and nystagmus) have been described ([Cohen and Duffner, 1994](#)). Although the duration of symptoms is usually brief, one case with a 6-month history of headaches precipitated by postural change has been reported ([Novak and Moshe, 1985](#)). Radiation is the treatment of choice, as surgery does not alter the course. Prognosis is very poor, even with irradiation and chemotherapy.

### Supratentorial Tumors

Supratentorial tumors include cerebral and parasellar or midline neoplasms.

Cerebral hemisphere tumors can be in any lobe, the basal ganglia, and/or the lateral ventricles and can be of various types, such as those listed in [Table 61.2](#). Increased intracranial pressure is not usually found until late in the course, unless the tumor is rapidly growing and malignant (e.g., glioblastomas, in which the duration of symptoms is only about 6 weeks) ([Cohen and Duffner, 1994](#)). With low-grade tumors such as pilocytic and anaplastic astrocytomas, the duration of symptoms is often several years. Personality changes may be some of the earliest manifestations but may be subtle and difficult to ascertain in young children. Hence, these tumors may grow quite large before detection. These personality changes may include intermittent irritability, listlessness, depression, and decrements in school performance, memory, attention, social awareness, and personal hygiene, although these are less well documented in children than adults. In the child with academic or behavioral deterioration, funduscopic examination is important, as it may show papilledema if a brain tumor is the underlying cause ([Herskowitz and Rosman, 1982](#)). Headache is also an early complaint. Aggression is sometimes seen with temporal lobe tumors. Seizures (which can be generalized tonic-clonic, complex partial, and/or focal motor) and focal neurologic deficits such as motoric weakness, hemiparesis, aphasia, alexia, agraphia, and hemianopsia tend to be later signs that result in diagnosis. In most cases of cerebral tumor, total resection is not possible, and excision is often followed by irradiation. Prognosis varies with tumor type: For low-grade astrocytomas, 5-year survival after excision and irradiation is 50% ([Laurent and Cheek, 1986](#)).

Parasellar tumors are those arising in proximity to the sella turcica. Craniopharyngioma is the most common of these, arising from remnants of the pharyngeal duct near the pituitary stalk. This leads to endocrine dysfunction, and infiltration of the hypothalamus leads to hypothalamic dysfunction. Growth retardation and delayed sexual maturation are frequent but are difficult to appraise early on. Obesity, somnolence, visual and olfactory hallucinations, and alterations in body temperature and blood pressure may occur. Later signs may be those of IICP and visual disturbances owing to optic chiasm compression. Treatment is by resection, with irradiation if resection is subtotal. The prognosis is good; however, these patients have both transient and permanent endocrinologic abnormalities requiring treatment. Pituitary adenomas are rare in children and are manifested by growth retardation and visual loss. Optic nerve and chiasm gliomas are not uncommonly associated with neurofibromatosis. Visual defects are the primary presentation. Treatment is irradiation and possible chemotherapy. In infants, optic gliomas are usually aggressive, and the prognosis is poor, whereas in older children the glioma is often benign and self-limited ([Kanamori et al., 1985](#)). A rare case of a disseminated parasellar germinoma involving the basal ganglia in a 13-year-old boy included early psychotic symptoms, with delusions and thought disorder, obsessive-compulsive features, aggression, mood lability, and suicidal ideation ([Mordecai et al., 2000](#)).

Midline tumors are those in the third ventricle, hypothalamus, or thalamus, in addition to the parasellar region. Pinealoma is the only brain tumor in children with a marked sex difference, having a male:female ratio of about 3 to 4:1. Pinealomas may be in the pineal region or in the posterior third ventricle ([Laurent and Cheek, 1986](#)). Symptoms include those of IICP, endocrine disturbance, and visual defects. Diabetes insipidus, growth retardation, delayed puberty, or precocious puberty may be present. Parinaud syndrome (paralysis of upward gaze with bilateral ptosis owing to pressure on the superior colliculi) is characteristic but may also be a sign of IICP ([Cohen and Duffner, 1994](#)). Other nonspecific signs (e.g., ataxia, nuchal rigidity, central deafness, and nystagmus) may be seen. Resection and/or irradiation are the recommended treatments. Colloid cysts of the third ventricle are rare. Movement of the tumor by flexion of the head results in intermittent obstruction of CSF flow, leading to intermittent symptoms of sudden headache, vomiting, and somnolence. Many of these tumors, although occurring in childhood, are not diagnosed until adulthood. Hypothalamic gliomas are rare and cause a characteristic diencephalic syndrome in infants. The infant appears alert and euphoric but becomes markedly emaciated. It is differentiated from failure to thrive by the activated state of the child. Hypothalamic gliomas in later childhood may present with accelerated growth and precocious puberty. Radiation therapy is the only treatment possible. Thalamic tumors are also rare. These often mimic cerebellar tumors but may have more characteristic signs, such as rhythmic alternation of extremities, athetosis, and posturing.



## Psychiatric Sequelae of Brain Tumors and Brain Irradiation

Consideration must not only be given to the presenting neuropsychiatric symptoms of brain tumors but also to those symptoms secondary to the sequelae of the tumor after treatment and the treatment itself. Standard neurologic examinations have little prognostic value for quality of life in children with brain tumors ( [Milstein et al., 1985](#)). Until recently, as survival rates have increased, very few studies had been done to assess functioning after treatment by means other than neurologic examinations. Several studies, using global and specific measurements, have shown many surviving children functioning at near normal and some achieving high levels of education, although substantial proportions of this population, ranging from 40% to 60%, have intellectual and emotional disturbances ( [Bamford et al., 1982](#)). Emotional disorders that have been reported include psychotic symptoms, immaturity, depression, anxiety, and oppositional and antisocial behavior ( [Danoff et al., 1982](#)). Many early investigations that reported functional deterioration did not or were not able to separate important variables, including tumor characteristics, age of the child, and treatment modalities ( [Mulhern and Kun, 1985](#)). Tumor location is often divided grossly into infratentorial or supratentorial locations for these studies. Impairments from infratentorial tumors are variable, with predominant visual-motor problems and about a 30% to 60% incidence of neuropsychiatric deficits, behavior disturbance, and need for special education ( [Mulhern and Kun, 1985](#)). These latter types of impairments are much more common with supratentorial tumors, with about a 50% to 85% incidence ( [Kun et al., 1983](#)). In five children with temporal lobe tumors, [Mulhern and Kun \(1985\)](#) found impulse control problems and special education needs in all, with more severe deficits than in adults with temporal lobe tumors. Further subdivision on the basis of location yields sample sizes too small for generalization.

Age at diagnosis and treatment appears to be positively correlated with better functioning if diffuse CNS damage has occurred, whereas younger children with focal injuries have better recovery than do older children ( [Eisner, 1981](#); [Mulhern and Kun, 1985](#)).

Increased intracranial pressure has been postulated to be responsible for some of the long-term intellectual deficits with brain tumors; however, it does not seem to have a lasting impact after treatment ( [Mulhern and Kun, 1985](#)).

The treatment modalities of cranial irradiation and CNS chemotherapy have been shown to adversely affect intellectual functioning. Children with acute lymphocytic leukemia (ALL) provide a useful comparison group for investigating the effects of CNS irradiation and chemotherapy in children with brain tumors, allowing separation of irradiation and chemotherapy effects from those of the tumor and neurosurgery. Cranial irradiation is a standard treatment for ALL, and many children with ALL develop a postirradiation syndrome within 4 to 8 weeks of treatment (somnolence, anorexia, and lethargy). Longer-term changes consist of cerebral atrophy, distractibility, memory and attention deficits, and declining IQ and achievement test scores, all of which are greater with higher radiation dose and combined irradiation-chemotherapy in the younger child ( [Cohen and Duffner, 1994](#); [Halberg et al., 1992](#); [Mulhern and Kun, 1985](#)). Nonverbal and information-processing skills may show cumulative deterioration ( [Anderson et al., 2000](#)). Even without radiation, CNS chemotherapy causes neurocognitive deficits, especially in academic achievement, that may take 3 to 4 years to appear ( [Brown et al., 2000](#)). Children with brain tumors are thought to be at greater risk for these effects, as radiation dosages for brain tumors are much higher than those for ALL. Indeed, more severe sequelae have been demonstrated, with either pervasive intellectual deterioration or more selective performance and mathematic deficits ( [Duffner et al., 1985](#); [Kun et al., 1983](#)). These effects have become more noticeable and problematic as rates of survival have increased, especially because these adverse consequences are more prone to occur in younger children, tend to worsen over time, and may take 2 to 5 years to be fully noted despite initial improvement in the first 6 months after treatment for older children ( [Duffner et al., 1985](#); [Mulhern and Kun, 1985](#); [Packer et al., 1989](#)). Irradiation also poses the additional adverse effects of long-term growth hormone suppression with resulting decelerated growth, hypothyroidism, hearing deficits, leukoencephalopathy, and oncogenesis (especially of sarcomas) ( [Cohen and Duffner, 1994](#); [Duffner et al., 1985](#)).

The diagnosis of brain tumor in a child is clearly devastating to the family. Often there is little time to optimally adjust to this news before the operation and treatment are begun. With increased survival, the family frequently must cope with a "changed" child, and joy over the child's survival can be challenged and tempered by the sorrow over his or her cognitive, behavioral, and emotional deterioration. This may occur even in the child with an IQ in the normal range ( [Cohen and Duffner, 1994](#)). As with other chronic disease, the parents tend to become unrealistic and overprotective ( [Danoff et al., 1982](#)). They may infantilize their child and avoid discussion of such issues as tumor recurrence and death. Marital and intrafamilial stress is not uncommon. Long-term psychiatric sequelae of brain tumors in childhood include cognitive deficits, school failure, social difficulties, and unemployment and appear more severe with supratentorial tumors and with cranial irradiation ( [Glauser and Packer, 1991](#); [Kaye and Duffner, 1998](#); [Mostos et al., 1991](#)). Educational and psychological services, such as special education, psychotherapy, and national or local brain tumor groups provide much-needed support for the child and family.

## Conclusions

Brain tumors in youth are primarily infratentorial, and initially presenting psychiatric symptoms are not as common or obvious as in adults. When present, these changes are often subtle and include irritability, declining school performance, memory impairment, decreased attention, depression, and changes in sleep (somnolence). Symptoms of IICP are often the primary presentation. Children with academic and behavioral deterioration deserve a neurologic examination, and the index of suspicion for intracranial mass should be high if headache, vomiting, alterations in consciousness, visual disturbances, cerebellar signs, or neurologic deficits are present. With recent increases in survival rates, long-term sequelae of intellectual, emotional, and behavioral deterioration have been noted. In general, these neuropsychiatric deficits are worse for supratentorial tumors, in younger ages at diagnosis and treatment, and after cranial irradiation. IQ, achievement, and memory testing should be done before and after treatment and followed for several years thereafter to assess long-term sequelae of cranial irradiation. Educational and emotional support services for the child and family can have a beneficial impact on adjustment after treatment.

## NEURODEGENERATIVE AND NEUROMUSCULAR DISORDERS

For convenience, this section covers neurodegenerative and neuromuscular disorders together. As with brain tumors, the classification of these disorders is controversial, and neurocutaneous disorders are sometimes considered under nervous system tumors rather than under degenerative diseases ( [Menkes, 1995](#)) ( [Chapter 1](#), [Chapter 6](#), [Chapter 7](#)). [Table 61.3](#) lists major categories of these disorders and selected diseases for each. Only those diseases that have reported psychiatric manifestations are discussed here.

Neurodegenerative disorders
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possible autoimmune disorders and/or slow virus infections. Varying degrees of intellectual impairment are present; and spasticity, optic atrophy, ataxia, weakness, and seizures are common. In most cases, treatment is purely symptomatic and supportive.

*Krabbe's disease* (globoid cell leukodystrophy) is a cerebral storage disease resulting in demyelination. It usually begins suddenly at 4 and 6 months of age and rapidly progresses to death, although later onset and slower progression have been observed ( [Menkes, 1995](#)). Restlessness, irritability, stiffness, seizures, and peripheral neuropathy are seen.

*Metachromatic leukodystrophy* (MLD) has different forms with different enzyme defects and different ages of onset, from infancy to adulthood. In the late infant or young child, early hypotonia gives way to spasticity; impairment of speech and intellectual deterioration gradually develop, accompanied by gait disorders, coarse tremor, athetoid movements, decreased deep tendon reflexes, and later, seizures ( [Swaiman, 1994](#)). Death ensues within several months of diagnosis. In the juvenile form of MLD, onset begins between 5 and 7 years with ataxia and movement disorders and progresses slowly to spasticity.

*Multiple sclerosis* (MS) usually begins in the young adult; however, rare cases of MS have occurred in children. Few children have symptoms before 10 years of age; most occur after puberty. As with adults, the course is unpredictable and characterized by waxing and waning symptoms, and the symptoms are similar: Ataxia, weakness, blurred vision, paresthesias, dizziness, headache, mood lability, and incontinence can occur ( [Menkes, 1995](#)). Diagnosis is made retrospectively and is bolstered by CSF abnormalities (especially pleocytosis and elevated g globulins), abnormal evoked potentials, and visualization of demyelinated lesions on CT or MRI. The etiology remains unknown, but MS is thought to be autoimmune in nature, perhaps because of a slow virus infection in early life. We have encountered cyclothymia as the presenting feature of this disorder in a 10-year-old girl who was admitted to our child psychiatry inpatient service with an initial diagnosis of bipolar affective disorder. The patient's complaints of visual disturbances were originally interpreted as probable conversion symptoms until fundoscopic examination yielded evidence of optic neuritis and led to the appropriate underlying neurologic diagnosis.

*Schilder's disease* (diffuse cerebral sclerosis) is an acute and progressive demyelinating disorder similar to MS, with onset often between ages 5 and 12. Intellectual impairment, emotional lability, gait disturbance, and seizures are early manifestations. Late signs include visual complaints, aphasia, deafness, and cranial nerve palsies, followed by death within 1 to 2 years ( [Menkes, 1995](#)).

### System Degenerations

These disorders involve progressive degeneration, primarily of one neurologic system ( [Swaiman, 1994](#)).

#### BASAL GANGLIA

*Parkinson's disease* has a rare juvenile form, with the onset around age 10 to 15 years of resting (pill-rolling) tremor, bradykinesia, rigidity, and intellectual deterioration. Treatment is the same as the adult form, but death usually occurs by the third decade.

*Huntington's disease* can begin as early as age 3, although only 1% to 3% of cases have their onset in childhood ( [Hecht, 1987](#)). The inheritance is autosomal dominant, and the juvenile form is usually inherited from the father. Commonly, the disease presents with dementia, personality change, and seizures between ages 5 and 10, followed by marked rigidity and hypokinesia. Chorea-athetosis may be absent. Death occurs within 3 to 10 years after onset.

*Dystonia musculorum deformans* is a genetic disorder marked by increased muscle tone with gait disturbance and abnormal foot positioning, torticollis, and/or dystonic movements, without intellectual impairment. Onset may occur around age 5. The limbs become distorted but relax during sleep. Fixation and incapacitation may ensue. These cases are not infrequently misdiagnosed as conversion disorder or hysteria, but the reverse can happen as well, with psychogenic dystonia sometimes resulting in inappropriate medical therapy or even surgery (such as insertion of straightening rods or thalamotomy) ( [Fahn and Williams, 1988](#)).

*Wilson's disease* is an inborn error of copper metabolism resulting in degeneration of the basal ganglia and hepatic cirrhosis. Initial presentation is hepatitis and/or dystonia and gait disturbance in mid-childhood ( [Menkes, 1995](#)). Intellectual impairment, affective lability, anxiety, and psychotic symptoms are common, as are tremor, dystonia, dysarthria, ataxia, and choreoathetosis. Oral chelating agents and restriction of dietary copper lead to only partial recovery.

#### CEREBELLAR

*Friedreich's ataxia* presents with gait disturbance, with peak onset between ages 10 and 13. By age 20, most cases are nonambulatory. Dysarthria and hearing loss can occur, but cognitive deficits are unusual. [Hart and coworkers \(1986\)](#) reported on two children with Friedreich's ataxia who had deficits in information-processing speed, sustained attention, and memory, which occurred early in the disease course and progressively worsened.

#### BRAIN STEM AND SPINAL CORD

Degenerations in these structures, such as *Werdnig-Hoffmann disease* and *familial juvenile amyotrophic lateral sclerosis*, have no characteristic psychiatric symptoms.

### Neurocutaneous Diseases

These diseases, sometimes referred to as phakomatoses, involve both skin and nervous system, which have a common embryonic ectodermal origin. The etiologies are of hereditary enzyme and metabolic defects.

*Neurofibromatosis* (von Recklinghausen's disease) is characterized by café au lait spots and numerous Schwann cell tumors along nerves and nerve trunks. Giant neurofibromas may cause overgrowth, of the limbs, trunk, and face ("elephantiasis") ( [Swaiman, 1994](#)). Intracranial tumors are seen, with optic gliomas common in children; and acoustic neuromas, ependymomas, and meningiomas frequent. Mental retardation is often present to varying degrees, visual-spatial integration deficits may be present, and hyperactivity and learning disorders are common ( [Varnhagen et al., 1988](#)). The inheritance is autosomal dominant, but about half of the cases represent new mutations of a gene on chromosome 17 ( [Martin, 1987](#)).

*Tuberous sclerosis* may present with infantile spasms as early as 6 months, hypopigmented nevi (ash-leaf spots), adenoma sebaceum, shagreen patches (large, raised, leathery areas of the skin), subungual fibromas, and retinal tumors. There are cardiac, renal, and pulmonary lesions. Sclerotic nodules that may calcify are found in the brain: These are responsible for developmental delay or arrest, hypersarrhythmia on the EEG, and mixed seizures. Psychiatric symptoms vary: Although some children may have average intelligence, most show significant cognitive impairment, and up to 50% are autistic ( [Harrison et al., 1999](#)). [Hunt and Dennis \(1987\)](#) have shown that normal intelligence is associated with the absence of infantile spasms, whereas the majority of cases with a history of infantile spasms show mental retardation and/or classic symptoms of autism. Aggression, obsessions, hyperactivity, psychotic symptoms, and impaired social behavior and communication are prominent; these behavioral features are often the most pressing concern of the parents. The inheritance of tuberous sclerosis is autosomal dominant with variable penetrance ( [Menkes, 1995](#)).

*Sturge-Weber syndrome* is manifested by a facial port-wine nevus in the distribution of the trigeminal nerve. Intracranial angiomatous malformations may give rise to focal seizures and hemiplegia, beginning by age 1. Symptoms progress relentlessly, with seizures becoming intractable. Most children are mentally retarded, and dementia becomes severe.

*Von Hippel-Lindau disease* occasionally presents in children as ataxia and spasticity, with hemangioblastomas of the cerebellum, kidney, liver, pancreas, and epididymis. Symptoms of posterior fossa tumor occur.

*Ataxia-telangiectasia* is a cerebellar degenerative disorder with characteristic telangiectasias of the conjunctivae and, less commonly, of the ears, face, and chest. These appear around age 4 and are usually preceded by ocular motor apraxia (difficulty breaking visual fixation without turning the head), ataxia, and intention tremor. Later, dysarthria, choreoathetosis, and intellectual deterioration are seen ( [Evans, 1987](#)). Many of these children have immune dysfunction with recurrent infections and a tendency to develop leukemias and lymphomas. Incapacitation occurs in adolescence, and death occurs in the early twenties.



## NEUROMUSCULAR DISORDERS

The predominant feature of neuromuscular disorders is weakness, with a variety of manifestations. The child psychiatrist should be familiar with these disorders, because they may have associated psychopathology or may be misdiagnosed as psychosomatic disorders. Two types of neuromuscular disorders are considered here: polyneuropathies and muscular dystrophies.

### Polyneuropathies

*Guillain-Barré syndrome*, juvenile myasthenia gravis, and most other polyneuropathies do not have characteristic psychiatric symptoms. However, because of their presenting symptoms, they are not uncommonly discounted as being conversion disorders. Guillain-Barré syndrome is a postinfectious inflammatory neuropathy, usually preceded by a viral infection or immunization. Within a few weeks of an upper respiratory or other infection, the child typically begins to complain of paresthesias in the extremities, which can have a glove-and-stocking distribution. Difficulty in walking and ataxia follow, and weakness rapidly ascends from the lower to upper extremities. Maximum deficits occur over several weeks to a month, followed by a plateau phase, then rapid recovery over several months ([Menkes, 1995](#)). Early in the course, the child may be seen as malingering to avoid school or other responsibilities. In many cases, respiratory insufficiency can occur, necessitating ventilatory support. Most patients recover with no sequelae. [Buschbacher \(1995\)](#) reported a documented case of a 15-year-old girl who was diagnosed and treated for Guillain-Barré syndrome and, within a few months, made a full recovery. Subsequently, she had recurrent episodes of a conversion disorder manifesting identical symptoms of her original Guillain-Barré syndrome. Each episode was associated with substantial stress at school or home.

*Juvenile myasthenia gravis* may also be misdiagnosed as psychosomatic. The symptoms are generally the same as in adults and usually occur after the age of 10 years. Ptosis and diplopia are hallmark signs, with waxing and waning fatigue that may be worse at certain times of day ([Menkes, 1995](#)).

### Muscular Dystrophies

The muscular dystrophies are inherited diseases characterized by progressive, symmetrical muscle weakness and atrophy of unknown etiology. *Duchenne muscular dystrophy* and *Becker type* are both x-linked recessive conditions; about one-third of Duchenne cases represent new mutations ([Martin, 1987](#)). The gene whose dysfunction is responsible for Duchenne muscular dystrophy has been located and its protein product, dystrophin, identified ([Hoffman et al., 1987](#)). Dystrophin is absent from the muscle tissue of boys with Duchenne muscular dystrophy. Duchenne-type dystrophy appears between 2 and 4 years of age, with delayed motor milestones, whereas the Becker type has its onset between 5 and 11 years of age ([Menkes, 1995](#)). Although the Becker type has a slower progression, both lead to wheelchair confinement and a decreased life span. *Fascioscapulohumeral* and *myotonic muscular dystrophies* are autosomal dominant disorders. The fascioscapulohumeral type occurs between 6 and 20 years of age and primarily affects the shoulder girdle and face. The myotonic type occurs from infancy to adulthood and involves distal and facial muscles, cataract development, gonadal atrophy, and myotonia.

Varying degrees of mental retardation of unknown pathogenesis have been reported in Duchenne, myotonic, and congenital muscular dystrophies, but less so in the Becker and fascioscapulohumeral types ([Menkes, 1995](#)). In Duchenne muscular dystrophy, estimates of mental retardation range from as low as 20% to as high as 50%, whereas in Becker's muscular dystrophy mental retardation is less well defined and thought to be about 5% ([Cotton et al., 1998](#); [Melo et al., 1995](#)). With increasing age and physical disabilities, increasing, decreasing, and static intellectual impairment have all been observed. [Whelen \(1987\)](#) reported average IQ but significantly lower verbal fluency and immediate verbal and nonverbal memory. [Sollee and associates \(1985\)](#) noted that IQ deficits in young Duchenne patients are primarily in language and attentional-organizational tasks, not in visual-motor tasks; these impairments improved to average with age. A similar trend was observed in a study that controlled for motor skills ([Cotton et al., 1998](#)). The Duchenne subjects, as compared to a healthy control group, had poorer functioning on neuropsychological tests that measured complex attention, verbal fluency, nonverbal memory, and visuospatial cognition. Results suggested that cognitive abilities in the Duchenne group might be specific rather than global.

In a comparison study of children with Duchenne's muscular dystrophy and spinal muscular atrophy, a more characteristic profile was found for children with Duchenne ([Billard et al., 1992](#)). Their WISC-R verbal IQ was significantly lower, whereas performance IQ was in the average range. Their left-handedness was more prevalent and language-based cognitive abilities, such as reading and verbal memory, were impaired. [Billard and associates \(1998\)](#) further examined the reading abilities and processing skills of three groups of children: Duchenne muscular dystrophy, spinal muscular atrophy, and those receiving mainstream education. The Duchenne children exhibited a reading-age that was significantly lower, and they were substantially impaired in reading single non-words, similar to that seen in dysphonetic dyslexia. [North and coworkers \(1996\)](#) described patients with Becker's muscular dystrophy who had no muscle weakness but presented with intellectual impairment and psychiatric disturbance, including learning disabilities, poor judgment, and depression.

The controversy over the extent of existence of cognitive impairment and the possible extent of CNS involvement in various forms of muscular dystrophy remains. It is questioned whether the absence of dystrophin in Duchenne patients affects the brain. [Kim and coworkers \(1995\)](#) found that dystrophin is deficient in synapses in the brain of Duchenne patients and that this deficiency might be related to cognitive impairment.

Children with all forms of muscular dystrophy are at risk for developing psychiatric disturbance such as anxiety, depression, and dysthymia disorders; the incidence is high regardless of normal or impaired intellectual functioning ([Melo et al., 1995](#)). [Fitzpatrick and coworkers \(1986\)](#) found frequent diagnoses of dysthymic disorder and major depressive disorder in boys with Duchenne, especially older boys. Another study showed that boys with muscular dystrophy, as compared to those with primary psychiatric disorder and chronic fatigue syndrome, related their depression and anxiety primarily to their physical condition ([Wood et al., 1994](#)). Social isolation, depression, and prolonged grief reactions are common; parents exhibit denial, preoccupation with the child, difficulty responding to death issues, guilt over the genetic transmission of the disease, and marital difficulties ([Witte, 1985](#)). The parents' initial expectations of a rapid course are greeted with false hope, because the disease progresses slowly; however, the parents' grief and conflict reemerge as walking becomes more difficult. Communication within families about muscular dystrophy presents difficulties for both parents and child, and the child is often the silent partner in decision-making ([Fitzpatrick and Barry, 1986](#)). Proper management includes education, genetic counseling, facilitation of communication, involvement of the child in making decisions, and social and emotional support such as that provided by the Muscular Dystrophy Association.

## CONCLUSIONS

Neurodegenerative and neuromuscular disorders encompass many diverse diseases with an array of neuropsychiatric effects. There may be no specific neuropsychiatric symptoms (e.g., Becker-type muscular dystrophy), mild intellectual impairment (e.g., neurofibromatosis), depression (e.g., Duchenne muscular dystrophy), severe cognitive disability (e.g., Huntington's chorea), or autism (e.g., tuberous sclerosis). The etiology and pathogenesis for many of these disorders are insufficiently understood, and often treatment is only supportive. For those disorders for which specific treatment is available (e.g., Wilson's disease), the treatment may not reverse some of the neuropsychiatric deficits. As most of these diseases are inherited, genetic counseling is an essential part of treatment. Education about the disorder, enhancement of family communication, and provision of social and emotional supports are beneficial and necessary components of management. Psychotropic medication may be considered for clearly defined symptom alleviation.

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## 62 DEPRESSIVE DISORDERS IN CHILDREN AND ADOLESCENTS

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### HISTORICAL PERSPECTIVE

Many doubted the existence of depression and mania in children and adolescents for years because it was felt that children, for theoretical reasons such as “immature personality structures,” could not experience extremes of mood ([Schulterbrandt and Raskin, 1977](#)). Yet case reports on despondency and depression in children and adolescents date back to the early 17th century. Melancholia was reported in the middle of the 19th century. In the 20th century, the existence of depression in children was doubted through the 1960s because it was felt that children's immature superego would not permit the development of depression ([Weller and Weller, 1984a,b](#)). However, this thinking changed following the Fourth Congress of the Union of European Pedopsychiatrists in Stockholm, Sweden, in 1970. The theme of this meeting was “Depressive States in Childhood and Adolescence.” As a direct result of this conference, the Union of European Pedopsychiatrists concluded that depression in childhood comprised a significant portion of mental disorders in children and adolescents ([Akiskal and Weller, 1989](#)).

Subsequently, the publication of the book, *Depression in Childhood: Diagnosis, Treatment and Conceptual Models*, by [Schulterbrandt and Raskin \(1977\)](#) increased the awareness of childhood depression in North America. This book was based on findings by a group of clinicians, researchers, and scientists sponsored by the National Institute of Mental Health. They reviewed the existing literature on affective disorders in childhood and came to the conclusion that depression in children and adolescents can be diagnosed using adult criteria if adjusted to assess symptoms in an age-appropriate manner.

The third edition of *Diagnostic and Statistic Manual of Mental Disorders* (DSM-III), DSM-III-R, and DSM-IV have used the same criteria to diagnose depression in adults and children. A few minor adjustments were made in the criteria to account for differences in age and stage of development between adults and children. For example, in the criteria for depression, failure to gain expected weight can substitute for significant weight loss in children. Also, the duration of symptoms required to diagnose dysthymia is 1 year in children and adolescents instead of the 2 years required for adults.

### PHENOMENOLOGY OF DEPRESSIVE DISORDERS IN CHILDREN AND ADOLESCENTS BASED ON THE DSM-IV CLASSIFICATION SYSTEM

The DSM-IV diagnostic criteria for depressive disorders are the same for children and adolescents as adults, with small exceptions stated as notations to the criteria. There is evidence to support the use of DSM criteria for major depression in adolescents ([Roberts et al., 1995](#)).

#### Major Depressive Disorder

The DSM-IV defines a *major depressive episode* as a syndrome in which at least five of the following symptoms have been present during the same 2-week period:

1. Depressed mood (for children and adolescents, this can also be an irritable mood)
2. Diminished interest or loss of pleasure in almost all activities
3. Sleep disturbance
4. Weight change or appetite disturbance (for children, this can be failure to achieve expected weight gain)
5. Decreased concentration or indecisiveness
6. Suicidal ideation or thoughts of death
7. Psychomotor agitation or retardation
8. Fatigue or loss of energy
9. Feelings of worthlessness or inappropriate guilt

At least one of the symptoms must be diminished interest/pleasure or depressed mood. The symptoms must cause significant distress or impairment of functioning in social, occupational, or other important areas. The syndrome should not have been precipitated by the direct action of a substance or the result of a medical condition and should not be better explained by bereavement or schizoaffective disorder. Also, a major depressive episode should not be superimposed on schizophrenia, schizophreniform disorder, delusional disorder, or a psychotic disorder not otherwise specified.

The disorder can be rated as mild, moderate, or severe. Also, it can be noted to be with or without psychotic symptoms that can be mood congruent or incongruent. It also can be determined to be in full or partial remission. When the episode has lasted 2 consecutive years, the depression should be diagnosed as chronic.

A depression also may be determined to have melancholic features. Either a loss of pleasure in almost all activities or a lack of reactivity to usually pleasurable stimuli should be present. Additionally, at least three of the following are required:

1. A quality of depressed mood that is distinctly different from the kind felt when a loved one is deceased
2. Depression worse in the morning
3. Waking up 2 hours earlier than usual
4. Observable psychomotor retardation or agitation
5. Significant weight loss or anorexia
6. Excessive or inappropriate guilt

Depressive or manic episodes also can present with *catatonic features* requiring at least two of the following as delineated in DSM-IV.

1. Motoric immobility in the form of catalepsy or stupor
2. Motor overactivity that seems purposeless and not in response to external stimuli
3. Extreme negativism or mutism

4. Voluntary movement peculiarities such as posturing, grimacing, stereotypy, and mannerisms
5. Echolalia or echopraxia

The *seasonality of mood disorders* also can be specified. To diagnose a seasonal mood disorder, there should be a regular temporal relationship between depression and/or mania, and a particular period of the year. A full remission or switching from depression to mania should occur within that characteristic time of the year. An individual should manifest at least two episodes of mood disturbance in the last 2 years. Finally, seasonal episodes should substantially outnumber nonseasonal episodes. It is difficult to diagnose seasonal affective disorder in children, because they experience the recurring universal stressor of starting school every fall. Also, a young child might present with an apparent seasonal depressive disorder but not yet have had any prior episodes. In this situation, a seasonal mood disorder could not be diagnosed because the required three episodes would be lacking. Manic or depressive episodes also can be considered as *postpartum* in women if the onset is within 4 weeks of their delivery according to DSM-IV criteria.

### Dysthymic Disorder

Dysthymic disorder can be diagnosed when there is a depressed or irritable mood that lasts a year or longer (in adults this must be 2 years or longer), and the affected youth is never symptom-free more than 2 months. In addition to a mood disturbance, to diagnose dysthymia at least two of the following symptoms must be present:

1. Appetite change
2. Sleep change
3. Decreased energy
4. Low self-esteem
5. Difficulty making decisions or poor concentration
6. Feelings of hopelessness

Dysthymia should not be diagnosed if there is a major depressive episode during the first year of the disturbance; if there is a history of manic, hypomanic, or mixed episode; if the disorder is during the course of a psychotic disorder such as schizophrenia; or if it is secondary to use of a substance or general medical condition.

Dysthymia is often referred to as depressive personality because it often starts in childhood, adolescence, or early adult life. The condition is chronic. It is called secondary dysthymia if it coexists with anorexia nervosa, anxiety disorder, rheumatoid arthritis, somatization disorder, or psychoactive substance dependence. If the age of onset is prior to 21, it is called early onset. In children and adolescents, predisposing factors include attention deficit hyperactivity disorder (ADHD), conduct disorder, specific developmental disorder, and a chaotic home environment. Children who have dysthymia and subsequently develop a major depressive episode have a so-called “double depression.” [Kovacs and associates \(1984\)](#) report dysthymic children to be at risk for developing depression and mania on follow-up.

### Atypical Depressive Features

A depression also may be identified as having atypical features. Characteristics of this subtype are mood reactivity, exclusion of melancholic and catatonic subtypes, in addition to two or more of the following for a period of at least 2 weeks:

1. Increase in appetite or significant weight gain
2. Increased sleep
3. Feelings of heaviness in arms or legs
4. A pattern of longstanding rejection sensitivity that extends far beyond the mood disturbance episodes and results in significant social or occupational impairment

### Depressive Disorder Not Otherwise Specified

In the DSM-IV, this category includes disorders with features of depression that do not meet the criteria for a specific mood disorder or adjustment disorder with depressed mood. Examples include depressive episode superimposed on residual schizophrenia; a recurrent, mild depressive disturbance that does not meet criteria for dysthymia; or non–stress-related episodes that do not meet the criteria for a major depressive episode. DSM-IV should be consulted for further details as to the diagnostic criteria for depressive disorders not otherwise specified.

## PREVALENCE AND EPIDEMIOLOGY

Varying prevalence rates have been reported for depression. Such differences may result from different populations sampled—community, ambulatory psychiatry patients, pediatric outpatients (nonpsychiatric), or pediatric or psychiatric inpatients. Also, the refinement of structured and semistructured instruments for use in diagnosing child and adolescent psychiatric disorders that occurred in the 1980s made assessment more accurate ([Weller and Weller, 1986](#); [Weller et al., 1984a](#)).

Epidemiologic studies done in the United States have reported the incidence of depression to be 0.9% in preschoolers, 1.9% in school-age children, and 4.7% in adolescents ([Kashani and Sherman, 1988](#)). A study by [Lewinsohn and colleagues \(1993\)](#) of a randomly selected sample of 1,710 Oregon high school students yielded a percentage of female and male high school students with one current or lifetime episode of unipolar depression of 22.3% and 11.4%, respectively. The percentage of female and male students with two or more episodes was 4.9% and 1.6%, respectively. An epidemiologic study conducted by [Garrison and coworkers \(1997\)](#) in southeastern United States in adolescents aged 11 to 16 years found that the 1-year incidence for major depression was 3.3% and that the 1-year incidence of dysthymia was 3.4%.

Studies also have been done in specialized pediatric populations. For example, 40% of patients on a neurology ward with unexplained headaches suffered from depression ([Ling et al., 1970](#)), and 7% of general pediatric inpatients had depression ([Kashani et al., 1981](#)). In samples drawn from psychiatric settings, 28% of patients in a child psychiatry clinic ([Carlson and Cantwell, 1980](#)), 59% of psychiatric inpatients ([Petti, 1978](#)), and 27% of adolescent inpatients ([Robbins et al., 1982](#)) could be diagnosed as depressed. Thus, the occurrence of depression, as these studies indicate, is not rare. It is commonly encountered in psychiatric practice.

## CLINICAL DESCRIPTION

The age of a child and his or her psychological sophistication can play a major role in the depressed child's clinical presentation. Most children do not use language to effectively communicate information until age 7. Thus, it can be more difficult to diagnose depression prior to this age. However, attention to nonverbal communication such as facial expression and body posture can assist in making the diagnosis in younger children in whom verbal communication is not well developed ([Poznanski, 1982](#)). [Spitz and Wolf \(1946\)](#) and [Bowlby \(1960\)](#) have described anaclitic depression in institutionalized children who were separated from their primary caretakers. The children had listlessness, withdrawal, weeping, refusal to eat, and sleep disturbances. Unfortunately, there is a paucity of well-designed studies of depression in preschool-age children ([Akiskal and Weller, 1989](#)).

As children attend school and begin to better utilize language as a vehicle for exchanging information, they are better able to describe their feelings. Also, the school is another source of information on the child's functioning. As children are in school 6 to 7 hours a day, teachers can observe the child's behavior, as well as compare it to that of their peer group.

As discussed, depression runs in families. Very often at least one of the parents of a depressed child is also depressed. This can present a problem in evaluating a child. The concern is that depressed parents may tend to overreport problems in their children because they are inclined to view everything in a negative fashion ([Weller and Weller, 1990a](#)). Conversely, they also may underreport symptoms in their children when their own symptoms make them more preoccupied with themselves and less aware of things going on around them. School-age children are able to be interviewed and give detailed information as to their emotions and behaviors. Often the child reports sadness, suicidal thoughts, and sleep disturbances that his or her parents are not aware of ([Weller and Weller, 1990b](#)). However, parents may be more likely to report behavioral changes such as irritability, moodiness, “whininess,” and loss of interest. Also, parents often are more aware of duration of symptoms. Thus, it is important to interview both parent and child ([Weller et al., 1984a,b](#)).

Depressed children look sad, are tearful, have slow movements, and speak in a monotone voice, in a hopeless and despairing manner. They describe themselves in



negative terms, such as, "I'm dumb," "I'm stupid," "I'm a bad girl," and "Nobody loves me." Their school performance deteriorates, and they tend to drop out of favorite extracurricular activities such as baseball, soccer, and scouting. Somatic symptoms often occur, the most common being stomachaches and headaches ([Weller and Weller, 1989](#)). [McCauley and associates \(1991\)](#) reported a direct correlation between the frequency of somatic complaints and severity of depression. In late childhood, depression more often includes low self-esteem. The child may report disappointment with self, apathy, irritability, anxiety, and inability to concentrate. Self-endangering behavior and suicide attempts are quite common.

([Carlson and Kashani 1988b](#)) studied the frequency of different symptoms at different ages. Depressed mood, lack of concentration, insomnia, and suicidal ideation occurred equally in all groups. With increased age, there was decreased occurrence of depressed appearance and somatic complaints and increased occurrence of anhedonia, diurnal variation, hopelessness, psychomotor retardation, and delusions. [Ryan and coworkers \(1987\)](#) reported that depressed appearance, somatic complaints, psychomotor agitation, separation anxiety, and phobias were most common in depressed children. Anhedonia, hypersomnia, hopelessness, weight change, and drug abuse were increased in adolescents as compared to children. In adolescence, the clinical picture comes to resemble more that of the adult. Hopelessness and feeling that things will never change are reflected in the increased prevalence of suicide. There is a dramatic increase of suicide attempts and completion of suicide with puberty ([Akiskal and Weller, 1989](#)). Also, the prevalence of depression among females increases at puberty in comparison with males.

Recent research has focused on gender and cultural issues and depression in youth. A prospective, 10-year, longitudinal study of preadolescents into young adults ([Hankin et al., 1998](#)) found that the most critical time for gender difference to emerge was the period between 15 and 18 years. This was the age period when there was a peak in the increase of the overall rates of depression and onset of new cases of depression. The rate of depression increases dramatically for both genders and the rate of depression in females rises to double the prevalence rate for males. No gender differences were noted for depression symptom severity or recurrence. Researchers are beginning to examine the role of culture in juvenile depressives. Several studies have pointed toward the role of stress of acculturation in the increased incidence of depressive symptoms and suicidal ideation among Latino youth ([Hovey and King, 1996](#); [Roberts and Chen, 1995](#)). [Seigel and colleagues \(1998\)](#) in an epidemiologic study of youth (ages 12 to 17 years) in Los Angeles County using the Children Depression Inventory (CDI) found that Latinos reported more symptoms of depressed mood, independent of socioeconomic status, as compared to whites or African-American or Asian-American adolescents. They also found clear social class effects on the report of depression; as income decreased, the average level of depression increased. Clearly, more extensive controlled studies of subpopulations of depressed adolescents are needed. This work is necessary not only to understand the phenomenology of depression in various populations in our country, but also to further develop more appropriate treatment strategies that incorporate consideration of cultural.

## ETIOLOGY AND PATHOGENESIS

### Biological Mechanisms and Hypothesis

#### *Neuroimaging and Electroencephalography*

Increasingly sophisticated neuroimaging techniques have been developed over the past three decades. With neuroimaging, psychiatric disorders can be studied safely and longitudinally in children and adolescents who are at risk for a particular disorder or already have a disorder. Neuroimaging allows for individuals with these disorders to be compared to individuals with normal development ([Hendren et al., 2000](#)). Studies such as magnetic resonance imaging (MRI), functional magnetic resonance imaging (fMRI), magnetic resonance spectroscopy (MRS), and magnetoencephalography (MEG) are best suited to study structural, physiologic, and developmental brain abnormalities in youth and perform repeated measures because they do not involve ionizing radiation or radioactive isotopes ([Hendren et al., 2000](#)). Positron emission tomography (PET) has unique utility as it enables the study of receptor volume and the effect a compound may have on receptors; however, it is problematic to use PET in children and adolescents because it requires complex equipment (a cyclotron) and uses radiation.

Despite a multiplicity of neuroimaging studies in childhood autism, schizophrenia, attention deficit/hyperactivity disorder, and obsessive-compulsive disorder, to date there have been few neuroimaging studies in depressed youth. [Steingard and associates \(1996\)](#) examined 65 hospitalized depressed latency age children and adolescents. (The average age was 13 years. There were 12 children 12 years or younger and 44 adolescents over 12 years.) They used brain magnetic resonance imaging to compare depressed subjects with 18 hospitalized psychiatric controls without a depressive disorder. Depressed youth had a significantly smaller ratio of frontal lobe volume:total cerebral volume, and a significantly larger ratio of lateral ventricular volume:total cerebral volume than controls. The investigators suggest that the alterations in frontal lobe and lateral ventricle volumes is a promising finding and may suggest a role for the frontal lobes in the development of early-onset depression.

[Tutus and colleagues \(1998\)](#) studied adolescents with major depression using brain single-photon emission tomography (SPET), to examine the possible changes in cerebral perfusion and potential association between perfusion indices and clinical variables. The group used technetium-99m hexamethyl propylene amine oxime brain SPET. Fourteen adolescent outpatients (ages 11 to 15 years) with DSM-IV major depression and eleven age-matched normal control (ages 12 to 15 years) were studied. Significant differences between the perfusion index (PI) values of the untreated depressed patients and those of the controls were found, indicating relatively reduced perfusion in the left antero-frontal and left temporal cortical areas. No significant differences in regional PI values were found between the treated depressed patients and controls. Their findings suggest that adolescents with major depression may have regional cerebral blood flow deficits in left antero-frontal and left temporal cortical regions and greater right-left perfusion asymmetry compared with normals. These abnormalities in perfusion indices show a trend toward returning to normal values with improvement in depression, suggesting that they could be a state-dependent marker for adolescents with major depression.

Electroencephalographic (EEG) activity was studied in 16 depressed and nondepressed female adolescent suicide attempters and 22 normal controls ([Graae et al., 1996](#)). Suicide attempters differed from normal controls in alpha asymmetry. Normal adolescents had greater alpha (less activation) over the right than the left hemisphere. Suicide attempters had a nonsignificant asymmetry in the opposite direction. Nondepressed attempters were different than the depressed attempters because they accounted for most of the abnormal asymmetry, particularly in the posterior regions. Attempters with major depression tended to show alpha asymmetry in the anterior sites and nondepressed attempters tended to show alpha asymmetry in the posterior sites. Moreover, the alpha asymmetry over the posterior regions was related to the ratings of suicidal intent but not severity of depression. The alpha asymmetry in the suicidal adolescents was similar to that seen for depressed adults in its abnormal direction but not its regional distribution. The investigators suggested that the EEG asymmetry distinction between the depressed and nondepressed attempters might point toward a behavioral and physiologic separation between the two subgroups of suicide attempters.

The number of neuroimaging studies of adolescents with depression is currently limited; however, the field of neuroimaging in children and adolescents is advancing quickly, and there will likely be more studies of adolescents with depressive disorders in the near future. These studies may help to elucidate possible structural and physiological abnormalities in juvenile depression.

#### *Neuroendocrine Abnormalities*

Neuroendocrine abnormalities have been observed in depressed adults. Several such abnormalities have been studied to determine if they are biological markers of depression. Theoretically, there are two major types of markers, state and trait. A state marker is present or "positive" during an episode of illness and becomes absent or "negative" as the illness remits. The dexamethasone suppression test (DST) is considered a state marker ([Carroll, 1985](#)). The DST is positive in 50% of well-diagnosed endogenously depressed adults. Usually the marker normalizes as response to treatment occurs ([Greden et al., 1983](#); [Leekman, 1983](#)).

Cortisol hypersecretion, as well as dexamethasone nonsuppression, are reported in both prepubertal children and adolescents. [Weller and Weller \(1988\)](#) have reviewed the use of the DST in children and adolescents. Overall, 54% of depressed children and adolescents studied have abnormal DSTs; the abnormality seems more robust in prepubertal children (70% sensitivity) compared with that in adolescents (43%). Possible explanations for this finding include the possibility that prepubertal onset depression is more severe than adolescent-onset depression, and that neuroendocrine systems of prepubertal children are more intact, as they have not yet been influenced by drug abuse or the surge of sex hormones. [Weller and coworkers \(1985\)](#) also have shown that clinical outcome in some prepubertal depressed children is associated with DST results. Children who have normalized their DST at 5 months tended to be clinically well, whereas those with an abnormal DST at 5 months tended to be depressed.

Other biological markers that have been studied in adults, such as growth hormone, have recently begun to be studied in children. An initial study showed that depressed children have hyposecretion of growth hormone in response to an insulin challenge and hypersecretion of growth hormone during sleep ([Puig-Antich et al., 1987](#)). In a review, [Brent \(1992\)](#) reported that similar growth hormone responses could be found in nondepressed children of depressed biological parents, suggesting that these may be related to biological traits predisposing to depression. A more recent study ([De Bellis et al., 1996](#)) examined neuroendocrine changes in prepubertal depressives and examined nocturnal secretion of adrenocorticotropin (ACTH), cortisol, growth hormone (GH), and prolactin in these children compared with controls. Prepubertal depressed children had lower cortisol secretion during the first 4 hours after sleep onset compared with controls. ACTH, prolactin, and GH secretion did

not differ between groups. Lower nocturnal ACTH concentrations in depressed inpatients versus depressed outpatients were found, as well as in depressed sexually abused children versus nonabused children. Lower GH secretion in depressed females than in depressed males was noted. The investigators point out potential differences between nonstimulated (nocturnal) and challenge studies of neuroendocrine secretion, as the nocturnal neuroendocrine studies did not reveal expected group differences. [Dorn and colleagues \(1996\)](#) examined ACTH and cortisol response to ovine corticotropin-releasing hormone (oCRH) tests at baseline and following a cognitive stressor. Boys had higher measures of ACTH than girls, regardless of depression status, and cortisol levels were similar in both groups. The authors suggest that the gender differences in hormone responses may be related to differences in peripheral metabolism of ACTH, resulting in changes in immunoreactivity but not bioactivity or a different set point of the hypothalamic-pituitary-adrenal axis. The pattern of ACTH and cortisol responses to oCRH and 24-hour excretion of free cortisol was normal in adolescents with depression; the authors hypothesize this reflects normal negative feedback mechanisms at this age or that most of these patients suffer from atypical rather than melancholic depression. A study of the nocturnal serum melatonin profile in depressed children and adolescents and a control group found significantly higher melatonin in depressed subjects without psychosis than those with a psychosis and control subjects ([Shafii et al., 1996](#)).

Recent research also has examined possible abnormalities of the neurotransmitter systems. [Nobile and associates \(1999\)](#) has found that human platelet 5-HT uptake, is influenced differentially in nondepressed and depressed children by a common genetic variant of the promoter region of the 5-HTT. Depressed children had decreased transporter activity but this difference was almost entirely sustained by children who carried a specific genotype (the *l* variant for the 5-HTT promoter region). [Birmaher and associates \(1997\)](#) found that before the onset of affective illness, high-risk children had the same pattern of neuroendocrine response to L-5-HTP challenge as did children with major depression. The finding on serotonin in this theory may represent a trait marker for depression in children. The catecholamine theory of depression hypothesizes that norepinephrine levels are decreased in depressed patients. This has been studied by [McKnew and Cytryn \(1979\)](#), who reported that urinary 3-methoxy-4-hydroxyphenylethyl glycol (MHPG) was decreased in depressed children. Replication with larger samples is needed, and recent research in depressed children has focused more on abnormalities of the serotonin system.

#### *Steep Studies*

Studies of depressed adults have found shortened rapid eye movement (REM) latency, increased REM density, abnormal slow waves, and a decrease in sleep efficiency ([Kupfer et al., 1985](#)). In children and adolescents, preliminary studies are contradictory ([Emslie et al., 1990](#); [Puig-Antich, 1987](#)). Additional studies may be necessary to clearly determine what abnormalities of sleep architecture are present in depressed children and adolescents.

#### *Genetic Studies*

Several studies of depressed adults suggest a genetic component in the etiology of affective disorders. Such studies can be grouped in three general categories:

1. Twin studies have found that concordance for affective disorders in monozygotic twins is 76% compared with 19% in dizygotic twins. When monozygotic twins are reared apart, the concordance rate drops to 67%, possibly indicating that environmental factors may play a role in the expression of genetic factors ([Akiskal and Weller, 1989](#)).
2. In studying children of depressed adults, there is increased occurrence of affective disorders compared to other psychiatric disorders ([Weissman et al., 1984](#)).
3. Studies of parents and other relatives of affectively ill children and adolescents have found an increased age-adjusted morbidity risk for affective disorders, compared with the risk to the relatives of affectively ill adults ([Puig-Antich et al., 1988](#); [Strober et al., 1987](#)).

#### *Parent–Child Relation Model*

In this model, depression is conceptualized as resulting from poor parent–child interaction. Depressed adults report low paternal involvement and high maternal overprotection during their early childhood. As children's well being is dependent on the well being of the significant adults in their lives, the importance of environmental influences on affectively ill children cannot be ignored. Poor relationships with parents, siblings, and peers are common in affectively ill children and adolescents. An affectively ill child often has an affectively ill parent. In an ongoing study by [Weller and colleagues \(1990\)](#), affectively ill children have reported abuse or neglect by their affectively ill parent(s). Thus, in evaluating an affectively ill parent, it may be advisable to inquire about the well being of their children. [Hammen and associates \(1991\)](#) reported a significant temporal association between depression in mother and child. According to their findings, children with substantial stress exposure who concurrently had symptomatic mothers were significantly more depressed than those who were exposed to comparable levels of stress only.

In a review of children of depressed adults, [Beardslee and colleagues \(1983\)](#) found that, as infants and preschoolers, these children had higher rates of perinatal complications and cognitive and emotional delays and an inability to separate from their parents. As school-age children, they had depression, hyperactivity, school problems, enuresis, and excessive rivalry with peers and siblings for attention. During adolescence, problems with defiance, rebellion, withdrawal, and conflicts and disagreements with parents were reported. [Weissman and associates \(1984\)](#) reported a threefold increased risk for manifesting a DSM-III diagnosis in children (6 to 18 years of age) of depressed adults. The most commonly observed diagnoses were depression (13%), ADHD (10%), and separation anxiety (10%). Risk factors included both parents being depressed; early onset of illness in the parent; and divorce, separation, or widowhood of the parents. [Keller and colleagues \(1986\)](#) studied children of unipolar depressed parents. Severity and chronicity of parental depression were associated with current impaired adaptation in the child, presence of a DSM-III diagnosis in the child, and marital discord associated with a maladaptive child. In addition, depression in the mother was more strongly associated with psychopathology in their children than was depression in the father.

[Breslau and coworkers \(1987\)](#) studied 333 mother–child dyads. Depressed mothers tended to view their children as more symptomatic and reported increased risk for all psychiatric syndromes. Thus, information obtained from depressed parents about their children may be systematically biased. Again, this indicates the importance of directly interviewing a child and not relying on parents as the sole source of information about the child. Only one study ([Beardslee and Podorefsky, 1988](#)) has reported an optimistic view of children of depressed parents. In this study, 15 of 18 adolescents who were followed for 2½ years were doing well. Those doing well at follow-up were characterized by self-understanding, problem-solving ability, and commitment to relationships, being action-oriented, and having the ability to think and act separately from their parents. Many of these adolescents were caring for their affectively ill parents.

#### *Cohort Effect*

The cohort effect discussed earlier in regard to bipolar illness also applies to depressive illness. Thus, an increase in the incidence of depression in children can be expected in the future owing to this cohort effect.

[Klerman and coworkers \(1985\)](#) and [Gershon and associates \(1987\)](#) have reported a progressive increase in the lifetime cases of major depression over the last 70 years. They report high rates of disorders among relatives, with a younger age of onset and decreased age of onset in successive recent cohorts. The implications are that somehow genetics and environment have interacted to produce an increased incidence and increased severity of mood disorders in succeeding generations.

#### *Sociologic Model*

According to this model, depression is seen as the result of social structures that deprive people of desirable roles. The inability of our social institutions to cope with current social stressors leads to the development of depression in some individuals ([Klerman, 1976](#)).

### **Psychological Theories of Depression in Youth**

#### *Psychoanalytic Theory*

[Freud \(1917/1966\)](#) and [Abraham \(1911/1966\)](#) theorized that depression was due to the real or imaginary loss of a loved object. As it is difficult to objectively study the loss of an imagined loved object, there is little research to substantiate these theories.

#### *Life Stress Model*

This model assumes life stressors or changes in the environment that necessitate readjustments cause depression. Some have theorized that depressive symptoms in children are often a reaction to family turmoil ([Lefkowitz and Burton, 1978](#)). [Poznanski and Zrull \(1970\)](#) reported a high incidence of parental aggression, punitive



discipline, marital discord, and scapegoating or rejection in the families of depressed children.

It is often difficult to know whether the stressor produced the illness or the illness contributed to the manifestation of the stressor. For example, in a depressed child who also has a learning disability, it is incorrect to always assume that one caused the other. Often a biopsychosocial approach in treating the depression alleviates both the depression and coexisting problems.

#### *Behavioral Reinforcement Model*

In this model, it is theorized that inadequate or insufficient positive reinforcers cause depressive behaviors and feelings. Diminished reinforcers produce crying, irritability, and latency of response in young children ( [Ferster, 1973](#)). Limited social skills in depressed children may lead to positive reinforcers being unavailable to them. Social skills training may help alleviate this problem ( [Puig-Antich, 1987b](#)).

#### *Learned Helplessness Model*

In this theory, a depressed person perceives his or her behavior as independent of reinforcers. This perception leads to a sense of hopelessness and subsequently to “giving up” ( [Seligman, 1975](#)). The helplessness is caused by motivational, cognitive, and emotional deficits.

#### *Cognitive Distortion Model*

In this model a negative view of self, the world, and the future (the cognitive triad) are the basic causes of depression; however, it is not clear whether these distorted cognitions are the root or result of depression. Negative view of self (low self-esteem) is very common in children with all types of psychiatric disorders and is not unique to depression. A negative view of the future has been reported in a group of outpatient-depressed children. Although this theory is provocative, well-designed studies to assess its validity are needed.

#### *Self-Control Model*

This model assumes that depressed individuals have deficits in self-reinforcement, -evaluation, and -monitoring. Often they focus on the short- rather than long-term consequences of their actions. They often misattribute personal success to external forces and personal failure to themselves.

### **Assessment**

#### *Structured and Semistructured Interviews*

Diagnostic interviews can be useful in performing a psychiatric evaluation. Structured interviews such as the Diagnostic Interview for Children and Adolescents (DICA) ( [Reich, 2000](#)) and semistructured interviews, such as the K-SADS ( [Chambers et al., 1985](#)) can be used at baseline and follow-up to obtain additional information in a standardized fashion. The Childhood Depression Rating Scale-Revised (CDRS-R) of [Poznanski and associates \(1984\)](#) is a modified version of the Hamilton Depression Rating Scale that rates severity of depression based on information obtained from child, parent, teacher, and clinician. The Childhood Depression Inventory (CDI) ( [Kovacs, 1981](#)), a self-report scale similar to the Beck Depression Scale for adults, is not a diagnostic instrument and should not be used to diagnose depression, as its face validity is poor (i.e., children easily identify appropriate or inappropriate responses). The CDI rates severity of depression.

In a study where the CDI and a DST were performed concurrently, 62% of depressed children had both a positive DST and CDI scores indicative of depression. Only 7% of psychiatric controls and 0% of normal subjects had both tests positive ( [Fristad et al., 1988a](#)). Perhaps the use of both measures simultaneously yields more useful information than either test alone.

#### *Newer Structured Interviews*

The need to provide rapid, accurate, and comprehensive psychiatric diagnoses in children and adolescents in both clinical and research settings has led to the development of “second generation” structured interviews. The Children’s Interview for Psychiatric Syndromes (ChIPS; [Weller et al., 2000](#)) is a concise, comprehensive interview to assess 20 major Axis I psychiatric disorders, including major depression and dysthymia, and psychosocial stressors such as abuse, in youth 6 to 18 years of age using DSM-IV criteria ( [Fristad et al., 1998b](#)). The ChIPS and its parent version, the P-ChIPS, work well both in a clinical setting and in a nonclinical, community sample ( [Fristad et al., 1998b,c,d](#); [Teare et al., 1998a,b](#)). The ChIPS was designed to improve on methodology provided by first generation structured interviews, such as the DICA and the Kiddie-Schedule for Affective Disorders and Schizophrenia. The ChIPS and P-ChIPS are designed to be administered by trained lay interviewers. They require approximately 1 to 1½ hours for the parent and child interviews. It identifies syndromes with a degree of accuracy similar to the DICA, an existing structured interview with proven reliability and validity. The ChIPS sensitivity was superior to DICA’s sensitivity although the DICA’s specificity was higher than that of the ChIPS ( [Teare et al., 1998a](#)). The ChIPS and P-ChIPS have been effectively used in a large, inner-city psychiatric outpatient setting. The interview has also been used effectively in a minority population (Rowan et al., 1998, [1999](#)). Thus, the ChIPS can be used in busy clinic setting or private office to maximize resources and provide a quality control for diagnoses. Depressive disorders, one of the most common diagnoses in clinical psychiatric settings and a major health concern in children and adolescents, can be efficiently and accurately diagnosed through the ChIPS for both clinical and research purposes.

#### *Laboratory Studies*

A child should always have a medical evaluation as part of a complete psychiatric evaluation. Organic etiologies that might mimic a depressive disorder must be ruled out ( [Table 62.1](#)). Conditions thought to mimic depressive disorders fall into several major general categories: infections, medications, endocrine disorders, tumors, neurologic disorders, and miscellaneous disorders. Initial work-up should include a complete blood count (CBC) with differential, to rule out infection and anemia. Electrolytes, BUN, creatinine clearance, creatinine, and urine osmolality should be assayed to rule out kidney disorders. Liver function tests should be performed. Thyroid function tests (T<sub>3</sub>, T<sub>4</sub>, and TSH) are necessary to rule out thyroid disease and are also a necessary part of a lithium work-up. EEG evaluation may be needed for patients with a history or presentation suggestive of seizure disorder or episodic behavior. Also, an ECG must be done prior to starting a tricyclic antidepressant ( [Table 62.2](#)).

Medication	Neurochemical/Genetic	Endocrine	Infection	Other
Tricyclic antidepressants	Serotonin	Diabetes	Atypical mycobacteria	Acromegaly
MAOIs	Pheochromocytoma	Cushing’s disease	Tuberculosis	Diabetes insipidus
SSRIs	Subarachnoid hemorrhage	Adrenal pheochromocytoma	Brucellosis	Cocaine
Antipsychotics	Cardiac arrhythmias	Hypothyroidism	Chlamydia	Amphetamine
Antibiotics	Malaria	Hyperthyroidism	Coccidioidomycosis	Opium
Anticonvulsants	Botulism	Hyperparathyroidism	Chlamydia	Sickle cell anemia
Antipsychotics		Hypoparathyroidism	Atypical mycobacteria	Hypernatremia
Antipsychotics			Atypical mycobacteria	Hypernatremia
Antipsychotics			Chlamydia	Hypernatremia
Antipsychotics			Chlamydia	Hypernatremia
Antipsychotics			Chlamydia	Hypernatremia
Antipsychotics			Chlamydia	Hypernatremia

Reprinted from W. K. Mullen, J. L. Mullen, & C. D. Bellack (eds), *Psychiatric Assessment*, 2nd ed. New York: Guilford Press, 1998.

**Table 62.1. Conditions Mimicking Depression in Children and Adolescents**





self-perception. If the perception of low competence is not correct, therapy can enhance realism in self-perception.

### *Play Therapy*

Play therapy can provide a route of nonverbal communication in younger children or children who are developmentally delayed (especially, speech delayed). Particular attention should be paid to repetitive, reckless activity and issues of loss and retrieval. Play therapy also provides a way of discharging stress via motor activity. Displaced aggression with toys and the acting out of omnipotent fantasies through sadistic or dangerous superhero themes also may be observed in play therapy. Play therapy can be done individually or with a group. It provides the children an opportunity for success. The child is allowed to express feelings and eventually deal with them. The therapist also provides a healthy model for identification.

### *Insight-Oriented Therapies*

The prepubertal child (6 to 12 years) usually is resistant to the lifting of defenses, does not understand the concept of “going back in order to go forward,” and often prefers outside solutions rather than intrapsychic solutions; however, insight-oriented therapies may be successfully employed in older children. Indications for insight-oriented psychotherapy include nonpsychotic depression, behavior problems owing to intrapsychic conflict rather than conduct disorder, adequate intelligence, and achievement of formal operational thinking. Therapy typically starts with a supportive phase, progresses to empathy, and eventually goes to a collaboration/self-observing phase. The therapist gives verbal interpretations of anxiety and affect, as well as of impulsive acts. During the course of therapy, the focus goes from current relationships with significant others to transference relationships.

Play therapy or talk therapy may be used in prepubertal children. If play therapy is to be used, the child is assigned his or her own cubicle to keep play materials that are not to be shared with others. Useful play items include paper, pencil, crayons, scissors, string, tape, doll family, animals, fences, and two or three cars. The onset of puberty may be traumatic to depressed children. Usually, there is a feeling of increased internal and external pressures. Often there is anxiety about distorted body image and complaints of loneliness and isolation. The therapist needs to be more flexible with adolescents, because frequent cancellations are common. Adolescents often prefer same-sex therapists, and it is important for the therapist to avoid being perceived as a peer. The therapist needs to be active, frank, and able to express herself or himself on values if asked. Limits must be set on dangerous behaviors. In late adolescence, the therapist can be less active and more of an observer.

### *Behavioral Therapies*

The necessity for response-contingent positive reinforcement is emphasized in this therapy. With children, therapy often focuses on skills, especially interpersonal skills, so that reinforcement can be elicited.

### *Life Stress Model*

The focus of therapy is either resolving or accepting life stressors that have led to depression. An example might be a child who has an adjustment disorder with depressed mood in response to the separation or divorce of the parents. In this model, the therapist helps the child to accept the decision of the parents to separate or divorce.

### *Cognitive Psychotherapy*

It is assumed that irrational beliefs and distorted cognitions are the essence of the depression. Thus, the therapist challenges the distorted cognitions and encourages the patient to practice alternate behavior. Depressive cognitions include a negative view of self, the world, and the future. Usually the depressed child or adolescent personalizes failure, magnifies negative events, and minimizes positive events and attributes. Possible predisposing factors include early parental loss, a parent with negative cognitions (e.g., a depressed parent), or a parent with rigid rules. The therapist focuses on identifying and correcting cognitive distortions and helps the patient reevaluate his or her thinking. Therapy is active and time limited. Initially, the goal is symptom relief, but eventually, the goal is to alter depressive beliefs. This therapy may be most useful in mildly depressed children. Normal intelligence and at least the beginning of formal operational thinking are necessary; therefore, cognitive therapy is of limited use in preadolescents. Younger children often have cognitive deficits rather than errors. However, 7- to 12-year-old children can misinterpret, distort, self-blame, and feel guilt and self-anger.

A diary may be helpful in cognitive therapy. Parents can serve as “checkpoints in younger children.” A 10-point scale—where 10 is the best you ever feel and 1 is the worst you ever feel—can be used by children. Pictures of sad and happy faces to identify feelings can be used to self-monitor. Parents can be involved in planning the activities that will occur as part of the therapy. However, children should be given choices as to the activities, and contingent reinforcers should be used. Assignments should be graded in difficulty and progress from an easy to a difficult task, in order to allow success. “Think aloud” is a technique that can be used with the parents' prompting. The strategies employed by the therapist may include persuasion, challenging cognition, examining evidence, exploring alternative explanations, and determining consequences. Children and adolescents may need shorter sessions than adults. Some noncompliance should be tolerated. Therapists should maintain ongoing family contact and be sure not to assign complex homework to those who are learning disabled.

[Trautman and Rotheram \(1986\)](#) have described a modified format for cognitive therapy to be used in adolescents. Formal operational thinking is associated with the ability to conceive of contradictory explanations for a given event. Thus, the first step is to identify feelings, measure their intensity, and compare one feeling experience with others. An antecedent affect leads to a belief that may lead to consequences for the child. The therapist must be very active in engaging the adolescent, emphasize “collaborating” with the patient, involve the family, develop a problem list, and use the subjective units of distress scale (SUDS). “The feeling thermometer” may be used with adolescents; the adolescent identifies the best and worst things that have happened to him or her. Homework is assigned concerning three problems that the adolescent had in 1 week, and eventually, the therapist helps the adolescent come up with alternative thoughts. Adolescents are taught to be assertive and use self-instructed thoughts. A list of instructions is developed to be used for crisis situations.

An advantage of cognitive therapy for adolescents is that it is flexible and allows the therapist to move readily between the roles of teacher, confidant, role model, collaborator, and expert. However, cognitive therapy works best in mildly depressed adolescents. It is less effective in severely depressed adolescents, who may need adjunctive medication management.

### *Group Therapy*

Group therapy is useful in developing social skills in depressed children. It allows children to express feelings in a safe and supportive environment. It requires an energetic and empathic therapist.

### *Parent Training*

The purpose of parent training is to teach parents to manage specific problem behaviors and to appropriately use positive or negative reinforcers. Parents also are taught to communicate with their children in an age-appropriate manner. In particular, parents learn how to talk and listen to their children.

### *Family Therapy*

[Poznanski and Zull \(1970\)](#) described lack of generational boundaries, severe marital conflict, projection of parental feelings onto the child, rigid or chaotic rules, and disengaged or enmeshed relationships in the families of depressed children. The family therapist discourages rejection and encourages increased affection and communication among family members. Also, other family members who are concurrently depressed and require treatment may be identified.

### *Remedial Education*

Depression is frequently associated with decreased academic performance. The aim of remedial education is to increase interaction with teacher and peers. Paper and pencil tasks (passive tasks) are discouraged, and more oral and manipulation tasks (active tasks) are encouraged. Rapid academic gains occur as depression lifts ([Quay and Werry, 1979](#)).

### Out-of-Home Placement

Removal from the home sometimes is necessary for short periods of time if home is chaotic, especially when a parent is concurrently depressed. Possible placements include a foster home for the less disturbed children, a group home for moderately disturbed children, and a residential home for the most disturbed children.

### Recent Research in Psychotherapy for the Treatment of Depression in Youth

Recent research on effective therapies for depressed youth have focused on cognitive-behavioral therapy, systematic behavioral family therapy, individual nondirective supportive therapy, group therapy, and interpersonal psychotherapy with adolescents.

A comparison study of cognitive-behavioral therapy (CBT), systematic behavioral family therapy (SBFT), and individual nondirective supportive therapy (NST) for the treatment of major depression was conducted at Western Psychiatric Institute in Pittsburgh, Pennsylvania. Subjects were 107 adolescents (13 to 18 years) in an outpatient clinic with Beck Depression Inventory score of 13 or more. The CBT in this study was adapted from the adult CBT approach of Beck, to the specific developmental needs of adolescents. Systematic behavioral family therapy in this study was a combination of functional family therapy and a problem-solving model of family therapy developed by Robin and Foster. The NST therapists in this study avoided giving advice, setting limits, or teaching specific skills. The goal was to establish, maintain, and build rapport; provide support; and aid the patient in affect identification and expression of feelings. In the active phase of treatment, which included 12 to 16 sessions provided over 12 to 16 weeks, CBT appeared more efficacious for treatment of major depression than SBFT or NST, and resulted in more rapid and complete treatment response ([Brent et al., 1997](#)). Continued depression was predicted by clinical referral to the study compared to those who responded to an advertisement without a referral. This was in part mediated by hopelessness. A higher level of self-reported depression predicted clinical remission. An initial higher level of interviewer-rated depression predicted poorer functioning. CBT's performance continued to be robust, even with adverse predictors ([Brent et al., 1998](#)).

A 2-year follow-up of rapid response to treatment (decline of >50% in Beck Depression Inventory from pretreatment to the beginning of the second session of psychotherapy) indicated that rapid responders showed better outcome at the acute phase of treatment, 1 year, and for some measures, 2 years of follow-up. Subjects were most likely to respond rapidly or not at all in the NST. This research group suggested that milder forms of depression may benefit from initial supportive therapy or short trials of more specialized forms of psychotherapy ([Renaud et al., 1998](#)). Predictors of a need for additional treatment were severity of index depressive episode, comorbid dysthymia in the acute phase, severity of depressive symptomatology, presence of disruptive disorders, and family problems in the follow-up phase ([Brent et al., 1999](#)). On 2-year follow-up of the CBT, SBFT, and NST groups, there were no long-term differential effects of the three psychotherapies. Twenty-one percent were depressed during at least 80% of the follow-up period. Severity of depression at baseline and presence of self-reported parent-child conflict at baseline and in the follow-up period predicted lack of recovery, chronicity, and recurrence of depression ([Birmaher et al., 2000](#)).

[Clark and associates \(1999\)](#) examined the effects of acute and maintenance group CBT for 123 adolescent (ages 14 to 18 years) with major depression or dysthymia. Adolescents were randomly assigned to one of three 8-week treatment groups: (a) 16 2-hour adolescent group CBT sessions; (b) adolescent group CBT with a separate parent group; or (c) a wait list control group. Subjects who completed CBT groups were assigned to one of three situations: (a) assessment every four months with "booster" sessions designed to address three factors in the maintenance of treatment gains: self-monitoring, lifestyle changes, and high levels of social support; (b) assessment only every 4 months; and (c) assessment every 12 months for a 2-year follow-up. The 8-week CBT treatment groups both had higher recovery from depression than the wait list group. Rates of recurrence during the 2-year follow-up period were lower than that reported for treated adult depression but booster sessions did not reduce the rate of recurrence. Booster sessions did appear to accelerate recovery among subjects who were still depressed after the 8-week CBT trial.

[Clarke and colleagues \(1995\)](#) have also investigated a group cognitive therapy intervention targeting a sample of high school students at risk for unipolar depressive disorder. One hundred and fifty adolescents with elevated scores on the Center for Epidemiologic Studies—Depression Scale (CES-D) but not with diagnosable depression on the Schedule for Affective Disorders and Schizophrenia for School-Age Children were randomized to a 15-session cognitive group prevention intervention or a usual treatment group. The cognitive group adolescents were taught cognitive techniques to "identify and challenge negative or irrational thoughts that may contribute to the development of future affective disorder." The average age of the subjects was 15 years. The sample was predominantly white and mostly female. The investigators reported that their cognitive group intervention showed a significant advantage over the control group; the total incidence of unipolar depressive disorder was approximately half that of the control group on 12-month follow-up.

[Mufson and colleagues \(1994\)](#) have studied the acceptability and efficacy of interpersonal psychotherapy for depressed adolescents (IPT-A). Interpersonal psychotherapy for depressed adolescents was investigated in two phases. In Phase I, a small group of five adolescents were enrolled in ITP-A therapy and treatment was modified to meet the treatment needs of the adolescents and was standardized into a manual. In Phase II, 14 depressed outpatient adolescents entered a 12-week open trial of ITP-A. Twelve of the 14 adolescents had major depression, one had dysthymia, and one adolescent had depression, not otherwise specified. All 14 adolescents were minorities. Average age of girls was 14 years and average age of boys was 15 years. They were rated at weeks 0, 2, 4, 8, and 12 with the Beck Depression Inventory, Hamilton Rating Scale for Depression, Symptom Checklist, and Social Adjustment Scale Self-Report. There was a significant decrease in depressive symptomatology and significant improvement in functioning over the 12-week course of treatment.

### Psychopharmacologic Treatment

With the introduction of new classes of antidepressants in the 1990s, research on antidepressants in children and adolescents shifted away from the tricyclic agents, and began to focus on the selective serotonin reuptake inhibitors (SSRIs). The first controlled study to demonstrate the efficacy of an antidepressant in youth was by [Emslie and associates \(1997\)](#). He randomized 96 latency age children and adolescents ages 7 to 17 years with nonpsychotic major depression in outpatient treatment to an 8-week trial of 20 mg of fluoxetine or placebo. The study was double-blind and further controlled via a 1-week, single-blind placebo run-in. Primary outcome measures were global improvement of the Clinical Global Impression Scale and the Children's Depression Rating Scale-Revised. There was significantly greater improvement in fluoxetine treated subjects; 56% of the fluoxetine group was much or very much improved, compared to 33% of the placebo group. There was no clear difference in responsiveness to fluoxetine or placebo based on age or sex. Complete remission occurred in only 31% of fluoxetine-treated patients and 23% of the placebo-treated patients. In this study, fluoxetine was considered superior to placebo for the acute phase treatment of major depression in children and adolescent outpatients with severe, persistent, nonpsychotic major depression.

There is a preliminary report of a second double-blind placebo-controlled trial of an SSRI reporting positive response in depressed adolescents. This study examined the efficacy of paroxetine, imipramine, and a placebo in the outpatient treatment of 275 adolescents ages 12 to 18 years with major depression. The study used up to 40 mg of paroxetine or up to 300 mg of imipramine over an 8-week trial. Paroxetine demonstrated significantly greater improvement compared with placebo in HAM-D total score, HAM-D depressed mood item, K-SADS-L depressed mood item, and CGI score. The response to imipramine was not significantly different from placebo for any measure ([Keller et al., 2001](#)). A third double-blind, placebo-controlled study, of venlafaxine and psychotherapy or placebo and psychotherapy administered over 6 weeks to 33 latency age children and adolescents ages 8 to 17 years with major depression showed a significant improvement over time; however, the improvement could not be attributed to venlafaxine. The investigators did find a low-side effect profile for the study medication. This study used a relatively low dosage of venlafaxine and the trial was shorter in duration than the Emslie or the Keller studies, which may have contributed to the lack of demonstrated efficacy.

There have been several promising open trials of SSRIs in youth. An 8-week open study of nefazodone in 28 children and adolescent outpatients ages 7 to 17 years led to a significant reduction in symptoms of major depression in 69% of the children and 89% of the adolescents ([Findling et al., 2000](#)). Nefazodone was generally well tolerated in this open trial. An open trial of sertraline ([Ambrosini et al., 1999](#)) in 53 adolescent outpatients with major depression had positive results. Subjects were treated for 10 weeks in the acute phase and if they responded they were treated for an additional 12 weeks. At 6 weeks, 65% of subjects were significantly improved and at 10 weeks, 84% were significantly improved. Significant improvement occurred early on and was maintained for 22 weeks. Maximal response in clinician ratings occurred by the tenth week of treatment. Furthermore, sertraline was generally well tolerated. [Nobile and coworkers \(2000\)](#) completed a 3-month open trial of paroxetine treatment in a small number of children and adolescents with dysthymia. Seventy-one percent of subjects had a satisfactory response. Overall, controlled trials of these antidepressants are necessary to further determine safety and efficacy.

The SSRI antidepressants appear to have a low side effect profile. Thus, they have a distinct advantage over tricyclic antidepressants (TCAs) with fewer anticholinergic side effects, limited cardiovascular toxicity, and a wide therapeutic index ([Leonard et al., 1997](#)). The side effects of all of the SSRIs are similar. The most common side effects are nausea, vomiting, diarrhea, agitation, disinhibition, jitteriness, headache, insomnia, and tremors ([Leonard et al., 1997](#)). It is unclear why the SSRIs, to date, have demonstrated better efficacy than the tricyclic antidepressants. The majority of positive studies of SSRIs have been done with outpatient



children or adolescents. Studies of tricyclic antidepressants in youth were generally done on inpatients. Although there has been a reduction in inpatient length of stay in the past 10 years, leading to more adolescents being treated as outpatients, it is possible the population as a whole studied with SSRIs was "less sick" or less depressed. Some studies, such as the [Emslie study \(1997\)](#), excluded psychotic major depression, symptoms of bipolar disorder, or a family history of bipolar disorder. Another possible explanation for the specificity of positive treatment response with SSRIs is that because there is a differential maturation of brain regulatory systems, serotonergic and tricyclic antidepressants may vary in their capacity to alter aberrant neuroreceptor and neurotransmitter function in juvenile depressives ( [Strober et al., 1999](#)).

A definitive way to carry out the psychopharmacologic treatment of a depressed child or adolescent does not yet exist. Several treatment approaches have been suggested, such as the Texas Children's Medication Algorithm Project ( [Hughes et al., 1999](#) ) and the medication strategy suggested by [Wagner \(2000\)](#). Commonly, an SSRI is suggested as a first medication and if this trial fails, a second SSRI may be tried. If there is again a medication nonresponse, other monotherapies, such as venlafaxine, bupropion, mirtazapine, or a tricyclic antidepressant, may be tried. Alternatively, augmentation with lithium, buspirone, or another antidepressant may be considered. If the patient remains nonresponsive, a combination of certain antidepressants might be used, and if the youth continues to remain significantly depressed, an MAOI and then electroconvulsive therapy (ECT) might be employed. More research is needed before the clinician can recommend a length of time a depressed child or adolescent should remain on antidepressants after symptoms resolve, or how long to taper the medication before it is completely discontinued.

There have been multiple controlled studies of TCAs. (For a description of treatment response to TCAs in depressed prepubertal children, please refer to [Table 62.4](#).) Yet none of these studies have shown efficacy in the treatment of major depression in youth. Moreover, there have been several reports of sudden death potentially related to the TCA desipramine ( [Elliott and Popper, 1990](#); [Riddle et al., 1991](#) ). Thus, the use of TCAs for treatment of depressed youth has decreased since the advent of newer classes of antidepressants. However, the use of TCAs in prepubertal and adolescent patients is reviewed here for several reasons. First, knowledge of TCA studies in depressed juveniles is important from a historical perspective. These were some of the first medications available to treat depressed children and multiple controlled studies have been done. Second, as previously stated, there may be a role for treatment with this class of antidepressants if a juvenile fails to respond to other classes of antidepressants. As noted in the following, there has been evidence for efficacy in open trials. Third, knowledge of response to TCAs in depressed young persons may guide the development of future medications for depression in youth.

Author	Drug	Site (inpatient)	N	Improvement (%)	Duration of Treatment (Wk)
Lucas et al., 1966	Ami	28-30	10	60	6
Unger et al., 1970	Amipri	--	10	80	--
Klein and Klein, 1972	Imipri	--	100	72	--
Wenberg et al., 1972	Amipri	25-25	19	85	3
Puig-Antich et al., 1979	Imi	105	13	46	5
Soler et al., 1981	Ami/Ami	--	11	100	12
Comery and Felt, 1983	Imi	25-200	21	67	9
Geller et al., 1983	Nor	--	8	100	16
Weller et al., 1983	Imi	50-150	16	75	6
Overall Total Average			208	75	

Ami=amipramine; imi=imipramine; Dri=desipramine; Nor=nortriptyline; Imi=1-imipramine and/or desipramine.

**Table 62.4. Treatment Response to Tricyclic Antidepressants in Depressed Prepubertal Children**

Although controlled trials have failed to show the efficacy of TCAs in youth, there is evidence for efficacy in open trials. TCAs may have some use if trials of other medications, such as SSRIs or atypical agents, fail. The therapeutic role of tricyclic antidepressants for depressed children and adolescents needs to be seriously weighed against lethality of overdose, the issues of possible sudden unexplained death, and the availability of safer and easier to monitor medications ( [Geller et al., 1999](#) ). Imipramine has been available for decades and has been frequently prescribed to treat childhood enuresis. [Weller and Weller \(1984b\)](#) reviewed the use of TCAs in depressed children. At that time, well-designed open trials of TRCs were examined. When data from all the open trials were combined, the overall response rate was 75%, and response rates ranged between 46% and 100%. The authors suggested that future studies include plasma drug monitoring to determine equivalency of treatment ( [Table 62.4](#) ).

There are two published double-blind placebo-controlled studies of imipramine in depressed children. The first study, by [Puig-Antich and associates \(1987\)](#), included 38 children in both the inpatient and outpatient setting. Twenty-two received placebo and 16 received imipramine. Imipramine was started at 1.5 mg/kg per day and increased to 5 mg/kg per day. The mean dose was 136 mg (4.35 mg/kg). Response rate was 56% in the imipramine treated group and 68% in the placebo-treated group. Those who responded to imipramine had higher plasma levels (284 ng/mL) than those who did not respond (145 ng/mL). [Preskorn and colleagues \(1987\)](#) used plasma level monitoring and DST results to study response to imipramine in 22 moderately to severely depressed prepubertal inpatients. The laboratory physician adjusted the plasma imipramine levels to be between 125 and 250 ng/mL. Children responded to imipramine better than placebo. When DST results were examined, depressed children with a positive DST responded better to imipramine than placebo, but those with a negative DST responded equally well to imipramine and placebo.

An important consideration when using tricyclic antidepressants is that they have the potential to cause cardiotoxicity. Thus, a child or adolescent's electrocardiogram (ECG) should be monitored throughout TCA treatment. [Elliott and Popper \(1990/1991\)](#) suggest that at a minimum clinicians obtain an ECG at baseline, at about 3 mg/kg per day and at a final dose not to exceed 5 mg/kg per day. Prolongation of the P-R interval should not exceed 0.21, and the QRS changes should not exceed 30% of baseline. Other limits that should not be exceeded include a heart rate of 130, a systolic blood pressure of 130, and a diastolic blood pressure of 85 ( [Ryan, 1990](#) ). Dosage adjustment with plasma monitoring may be needed to ensure compliance and avoid toxicity if these parameters are exceeded. [Preskorn and colleagues \(1988\)](#) reported increased neurotoxicity in children who had plasma levels higher than 500 ng/mL.

Monoamine oxidase inhibitors (MAOIs) have been suggested to be helpful in treating so-called atypical depression in adults. Monoamine oxidase inhibitors have been used sparingly in children and adolescents but they have been used in youth who have not responded to TCAs, before newer classes of antidepressants became available. The major problem with MAOIs is the necessity of a tyramine-free diet, which requires abstinence from chocolate, chicken livers, pepperoni, bologna, sausage, aged cheese, and Chianti wine, among other foods. Most adolescents are too impulsive, lack the discipline, or simply do not want to follow these dietary restrictions to make the use of MAOIs feasible. [Ryan \(1988b\)](#) treated 23 adolescents who had not responded to TCAs with an MAOI; 74% responded. Of these, 57% had a good response and were able to comply with the diet.

At this time many new antidepressants are under study, and much more research on antidepressants in the treatment of depression in youth is needed. Most moderately to severely depressed children and adolescents may be considered for medication treatment. Before starting medication, side effects, as well as anticipated beneficial effects, should be explained to the parent or guardian and the youth. The parent or guardian should also be notified before obtaining informed consent that the Food and Drug Administration (FDA) has not approved these medications for use in depressed children. Literature on effective pharmacologic treatments for depression in youth is continually changing and practitioners who prescribe medication for childhood depression may be kept updated on new studies through review of well-known psychiatry journals, as well as the more recently available journal and newsletters on pediatric psychopharmacology ( [Rowan, 2000](#) ).

#### Electroconvulsive Therapy

Electroconvulsive therapy (ECT) can be effective when other measures fail in severe depression ( [Bertagnoli and Borchardt, 1990](#) ); however, the data concerning safety and efficacy in children and adolescents are sparse. As stated earlier, the [American Psychiatric Association has published specific guidelines for obtaining consent and for the indications and administration of ECT \(1990\)](#). Two psychiatrists who are experienced in treating children and are not otherwise involved with the case should concur with the recommendation for ECT prior to the initiation of treatment.

#### Outcome and Follow-Up Data

[Weissman and colleagues \(1999\)](#) did a 10- to 15-year follow-up of 83 subjects with prepubertal-onset major depression, 44 control subjects with anxiety disorder and without major depression, and 91 normal controls. They found the clinical outcome of children with prepubertal depression includes a high risk of suicide attempts (threefold increased compared to normal controls and twofold compared to the anxiety control group) and bipolar disorder. [Pine and associates \(1998\)](#) prospectively

studied the longitudinal associations between adolescent and adult depressive and anxiety disorders. They found that adolescent anxiety or depressive disorders predicted a twofold to threefold increased risk for adulthood anxiety or depressive disorders. [Pine and colleagues \(1999\)](#) also studied the subclinical symptoms of major depression in adolescence and found that they strongly predicted an adult episode of major depression.

[Lewinsohn and associates \(1999\)](#) followed adolescents with major depression into adulthood age of 24 years. The study included several control groups. Major depression in adulthood was significantly more common in the adolescent group with major depression than either a nonaffective psychiatric and a no disorder control group. Adolescents with major depression had a high rate of nonaffective disorders in young adulthood but did not differ from adolescents with nonaffective psychiatric disorder. This study documented the significant continuity of major depression from adolescence to young adulthood. Major depression in childhood not only appears to confer a risk for major depression in adulthood, but also appears to be a particularly serious form of affective illness, as they converted to bipolar disorder more frequently than adults do ([Kovacs, 1996](#)). Although there appears to be a positive short-term outcome for these youth, they appear to have recurrent episodes of depression relatively early in their lives ([Kovacs, 1996](#)), as well as a higher switch rate to bipolar disorder. Overall, outcome is poor, as might be expected; however, there is some reason to believe that the course of depression in children and adolescents may improve in the future. Newer classes of antidepressants and an interest in rigorously studying several forms of psychotherapy in youth may lead to more effective acute and long-term treatments in juvenile depressed patients and better outcomes.

A complicating factor in the assessment and treatment of this disorder in children and adolescents is the frequent occurrence of comorbidity. [Kovacs and coworkers \(1984, 1988\)](#) reported that 79% of depressed prepubertal children had at least one other Axis I diagnosis: 38% had dysthymia, 33% anxiety disorder, and 7% conduct disorder. A follow-up study of depressed children found that 36% developed conduct disorder as a complication of depression ([Kovacs et al., 1988](#)). [Kashani and Sherman \(1988\)](#) studied depressed adolescents. In this report, 75% had comorbid anxiety, 50% oppositional disorder, 33% conduct disorder, 25% alcohol abuse, and 25% drug abuse. Those with onset of depression in childhood or adolescence are at risk to develop multiple episodes on follow-up ([Kovacs et al., 1984](#)) and often require rehospitalization ([Asarnow et al., 1988](#)). [Harrington and associates \(1991\)](#) reported that for depressed children to have comorbid conduct disorder was prognostically favorable for the outcome of depression in adulthood but not for conduct disorder. These patients had a similar likelihood of developing antisocial personality as adults when compared with patients who had conduct disorder without depression but who were much less likely to have depression as adults when compared with a group of patients who were depressed but did not suffer from conduct disorder. These findings suggest that depression may represent an early feature of conduct disorder rather than a separate comorbid illness. In a prospective longitudinal study of the course of depression in children and adolescents, [McCauley and associates \(1993\)](#) reported poorer overall outcome to be associated with increased levels of stress in the family environment.

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#### CASE ILLUSTRATION: PREPUBERTAL DEPRESSED CHILD

B.J., an 11-year-old boy, was admitted to the children's inpatient unit with a chief complaint of, "Nothing is working out for me." He had been failing in school for the last year, although he had previously been a B student. He complained of trouble sleeping (middle insomnia). His parents had not bought new clothes for him for the last 2 years "because he had not outgrown his old ones." He looked very sad, cried easily, and thought his peers were making fun of him because he was "ugly and stupid." He had started refusing to go to school, because he felt very tired in the morning and wanted to sleep more. His mood usually improved by the afternoon. His mother had found a note written by B.J. that said he was planning to jump in front of a car on a busy street "to end my misery," and then listed to whom his belongings should be given after his death. Neither B.J. nor his parents could identify any stressors.

His mother had a history of numerous depressive episodes that had responded to antidepressants. Her first episode of depression occurred at age 10 when she was hospitalized on a general pediatric service for aches and pains and being tired all the time. B.J.'s maternal grandmother had received ECT for depression that occurred after B.J.'s mother was born.

Initially, B.J. was treated with individual and family therapy. However, because of his severe vegetative symptoms, he was given imipramine, and he responded within 10 days.

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#### CASE ILLUSTRATION: ADOLESCENT DEPRESSION

Shelly was a 14-year-old girl hospitalized because she was refusing to take a bath, had no energy, was sleeping 16 hours a day, and had dropped out of school. She had made multiple suicide attempts. These attempts involved taking her mother's antidepressants, overdosing with alcohol and drugs, and trying to jump off the roof of a five-story building. She was quite annoyed that people would not let her die. She saw no hope for the future because, "Life is a bummer." She rarely interacted with anyone on the unit during a 3-day observation period.

Her parents gave a three-generation history of bipolar illness in their family. Lithium was recommended because this child was thought to be at high risk for suicide and mania; however, her physician refused to give lithium to the child because "it deposits in the bones." She was started on a tricyclic antidepressant and thiothixene 1 mg twice daily and discharged. A week after discharge, she was found in another state trying to "conduct business." She was admitted to a psychiatric hospital and treated with lithium carbonate and had a fairly good response. Her physician was now willing to consider lithium because Shelly had push of speech and flight of ideas and was very grandiose and hypersexual.

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## RESEARCH DIRECTIONS

Although there has been a dramatic increase in the number of studies of depressed children and adolescents in the past two decades, more research is needed. Further studies are needed to assess risk factors that may predict depression, and such studies must be done in a prospective longitudinal manner. In regard to treatment, additional studies are needed not only to determine if pharmacologic treatment is helpful but also to identify subgroups that may be most responsive to pharmacologic intervention. With the changes in FDA regulations, there has been an increase in efforts to perform double-blind and placebo-controlled studies in children and adolescents. The first studies demonstrating some positive effect of medication in childhood depressive disorders were published in the past 5 years. Much more research is needed, though, before the most effective somatic therapies and treatment algorithms for the treatment of juvenile depressions can be determined. Controlled studies assessing the effectiveness of the various psychotherapies in childhood depression also are needed. Studies using neuroimaging appear promising and may revolutionize our understanding of depressive disorders in youth. Research on neuroendocrine factors and sleep architecture need to be expanded and replicated. The elucidation of the human genome, which has received so much attention in the news, may focus attention on genes that are expressed in childhood onset depressive disorders one day. Both preclinical and clinical research has accelerated over the past decade; we may begin to see significant progress in the areas of prevention and treatment of childhood depression as pieces of the puzzle are gradually worked out.

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major depressive episode occur over the same period. Also, the absence of a major depressive episode, a mixed episode, or a manic episode during the first year of the disturbance is required. [Klein et al. \(1986\)](#) reported that 24% of the offspring of bipolar patients and 0% of the offspring of control subjects had cyclothymia as detected by the General Behavioral Inventory, which had excellent correlation with the interview-derived diagnosis.

*Bipolar disorder not otherwise specified*: In DSM-IV, this diagnostic classification includes disorders that have manic features or hypomanic features that do not meet the criteria for any specific bipolar disorder as described previously. Examples of these disorders would include a manic episode that was superimposed on schizophrenia or a delusional disorder or recurrent hypomanic episodes, but with no intercurrent depression.

For all the preceding categories, the diagnoses should not be made if the disturbance can be explained by schizoaffective disorder or (excepting bipolar disorder, not otherwise specified) superimposed on schizophrenia, schizophreniform disorder, delusional disorder, or psychotic disorder not otherwise specified. Further information and explanation of the diagnostic criteria for bipolar disorders and details regarding diagnostic subtypes can be obtained from DSM-IV.

## HISTORICAL NOTE

Mania in children has been reported by several prominent psychiatrists over the years, including [Esquirol \(1845\)](#), [Kraepelin \(1921\)](#), [Kasanin \(1931\)](#) and [Bleuler \(1934\)](#). Kraepelin believed that mania was rare in children but that the incidence increased by adolescence. He reported that approximately 0.5% of his manic patients had their first episode at age 10 years or younger. However, some child and adolescent psychiatrists, such as [Kanner \(1960\)](#) and [Anthony and Scott \(1960\)](#), doubted mania's existence in children. Anthony and Scott developed and applied very stringent criteria to diagnose mania in a study of psychiatrically ill children. They concluded mania was very rare in children. However, they went on to describe a patient who appeared manic as a child and who subsequently had his mania confirmed when followed up as a young adult.

[Davis \(1979\)](#) developed criteria to diagnose mania in children. However, these criteria were very nonspecific, including minimal brain dysfunction, enuresis, and electroencephalographic anomalies. He called this syndrome *manic depressive variant syndrome*.

Others have diagnosed bipolar disorders in children and adolescents using adult-type criteria adjusted to children's different maturational levels ( [Carlson and Strober, 1978](#); [Weinberg and Brumback, 1976](#); [Weller et al., 1986c](#)). [Weller et al. \(1986c\)](#) conducted an extensive review of all available case reports of psychotic or severely disturbed children by blindly reviewing cases to determine how many, if any, met DSM-III criteria for mania. From 1809 to 1982, 157 case reports were reviewed independently by the authors. In doing so, they noted that 50% of children who satisfied DSM-III criteria for mania had been previously reported as having conduct disorder, attention deficit hyperactivity disorder (ADHD), or schizophrenia. This finding suggests that mania may have been previously underdiagnosed or misdiagnosed in children and adolescents. Several possible explanations may account for the difficulty clinicians may have in diagnosing children and adolescents with bipolar disorder. These include bias against the diagnosis of mania in childhood, the low base rate of bipolar disorder relative to other psychiatric illnesses in children, symptom overlap between bipolar disorder and other, more prevalent childhood psychiatric illnesses, and developmental constraints and variability in symptom expression and presentation.

## PREVALENCE AND EPIDEMIOLOGY

No definitive epidemiologic study to determine the prevalence of bipolar disorder in childhood has been completed. Current estimates of the prevalence of bipolar disorder in childhood are based on small community surveys or retrospective data. [Carlson and Kashani \(1988\)](#) studied a community sample of 150 adolescents, 14 to 16 years of age, from Columbia, Missouri, using structured diagnostic interviews to ascertain a diagnosis of mania. They reported that 14% satisfied criteria for mania if duration was not taken into account, and 7.5% of adolescents met criteria for mania if severity was not taken into account. However, when severity and duration were both taken into account, only 0.6% were diagnosed manic. The prevalence of bipolar disorder during late adolescence is estimated to approximate that in adulthood. Bipolar disorder occurs in 1% of adults. Men and women are equally afflicted. The epidemiologic catchment area studies done on 10,000 young adults 18 to 24 years of age in New Haven, Connecticut, Baltimore, Maryland, and St. Louis, Missouri; reported a prevalence of 0.6% to 1% for manic episodes ( [Robins et al., 1984](#)). Onset of bipolar disorder before puberty is estimated to be rare. In a retrospective study involving adult patients with bipolar disorder, 0.5% reported onset of their illness to have been between 5 and 9 years of age; 7.5% reported onset between 10 and 14 years of age ( [Lish et al., 1994](#)). Furthermore, reports suggest that bipolar disorder before puberty may be more common in boys than in girls ( [Carlson et al., 2000](#); [Geller et al., 1998](#); [Varanka et al., 1988](#)).

Since World War II, the prevalence of mood disorders, including mania and depression as well as schizoaffective disorders, has been increasing ( [Gershon et al., 1987](#); [Klerman et al., 1985](#)). This observation has come to be known as the *cohort effect*. One study describes a parallel increase of affective disorders in children and adolescents ([Ryan et al., 1992](#)). If this trend continues, it can be anticipated that more children and adolescents will manifest mania in the future. Anticipation, a tendency for a disease phenotype to worsen (e.g., to be expressed at an earlier age and to run a more severe course) in consecutive generations is one possible explanation of this phenomenon.

## CLINICAL DESCRIPTION

Manic adolescents often have a clinical presentation similar to that of adults. Symptoms such as elated mood or irritability, push of speech, flight of ideas, grandiosity, sleeplessness, and bizarre behavior, including bizarre attire, typically are observed. Prepubertal children with bipolar disorder most frequently present with symptoms that are considered atypical in adults ( [Biederman et al., 2000](#); [Boring and Kovacs, 1992](#)). Mixed features, rapid cycling, psychosis, high rates of comorbidity, especially with externalizing disorders, and significant functional impairment in social, academic, and occupational spheres are common ( [Carlson, 1990](#); [Carlson et al., 2000](#); [Geller et al., 1998](#); [Kovacs and Pollock, 1995](#); [Sanchez et al., 1999](#)).

[Carlson \(1983\)](#) analyzed in detail how symptom patterns vary by age. She concluded that younger manic children (<9 years of age) present with irritability and emotional lability, whereas older children present with euphoria, elation, paranoia, and grandiose delusions. Hyperactivity, push of speech, and distractibility were present in both age groups. Similarly, [DeLong and Aldershof \(1987\)](#) suggest prepubertal bipolar children begin their illness with cycles in which dysphoria, hypomania, and agitation commonly are intermixed. The extremes of depression and manic excitement appear to become more common with the onset of puberty.

The first controlled phenomenologic study of bipolar disorder in childhood reported that children with bipolar disorder are significantly more likely than children with ADHD to endorse cardinal symptoms of mania, including elated mood, grandiosity, hypersexuality, decreased need for sleep, flight of ideas, racing thoughts, social intrusiveness, and increased goal-directed activity ([Geller et al., 1998, 2000](#)). Reports of mixed mania, rapid cycling, and psychosis were very common in their sample of 93 young children diagnosed with bipolar disorder. Approximately 55% of the bipolar sample endorsed mixed mania, defined as a temporal overlap in the onset and offset of mania or hypomania with major depression or dysthymia ([Geller et al., 2000](#)). Furthermore, 87% of the sample endorsed rapid cycling, defined as four or more mood disturbances a year. Of the children who experienced rapid cycling, 10% reported ultrarapid cycling, with mood disturbances lasting a few days to a few weeks, and 77% reported ultradian cycling, with mood disturbances occurring within a 24-hour period ( [Geller et al., 2000](#)). Sixty percent of the sample endorsed psychotic symptoms. Of these, grandiose delusions were the most common and occurred in 50% of the sample ( [Geller et al., 2000](#)). Approximately 25% of the sample admitted to serious suicidal ideation with intent and plan, which underscores the potential mortality associated with this disorder ( [Geller et al., 2000](#)). Longitudinal studies of children with bipolar illness are needed to determine the evolution of cycling patterns and long-term outcome for these children.

Some suggest that the presentation of mania in prepubertal children resembles that of treatment-resistant, chronically ill adults ( [Biederman et al., 2000](#); [Carlson et al., 2000](#); [Geller and Luby, 1997](#)). Thus, childhood-onset bipolar disorder may represent the most severe form of the adult illness. The question of whether the phenotypic differences reported between childhood-onset and adult-onset bipolar disorder result from developmental variations of the same genotype ( [Geller et al., 2000](#)) or genotypic differences ( [Schürhoff et al., 2000](#)) remains unsettled.

The diagnosis of bipolar illness in adolescence is much less controversial; the phenomenology of bipolar disorder in adolescents more closely resembles that of adult-onset bipolar illness ( [Bowden and Sarabia 1980](#); [Carlson et al., 2000](#); [Carlson and Strober, 1978](#); [Coryell and Norten, 1980](#)). Furthermore, follow-up studies have confirmed the diagnosis in many cases ( [Kimura et al., 1980](#); [Krasa and Tolbert, 1994](#)). Mixed manic features and psychotic symptoms, including mood-incongruent hallucinations, paranoid delusions, and thought disorder, frequently complicate the clinical presentation of mania in adolescence ( [Akiskal, 1995](#); [Ballenger et al., 1982](#); [Bashir et al., 1987](#); [Goodwin and Jamison, 1990](#); [McGlashan, 1988](#); [McElroy et al., 1997b](#); [Rosen et al., 1983](#)). There is evidence that suggests psychotic symptoms are more common in bipolar disorder when onset is in adolescence than when onset is after age 30 years ( [Ballenger et al., 1982](#)).

Bipolar disorder often first manifests itself as depression in childhood ( [Lish et al., 1994](#)). Reports suggest that a bipolar course develops in 20% to 30% of children with major depression ( [Geller et al., 1994](#)). Several risk factors have been associated with the development of a manic episode after an episode of depression,

including (a) a depression characterized by rapid onset, psychotic features and psychomotor retardation; (b) a family history loaded with mood disorders, especially bipolar disorder; and (c) a history of antidepressant-induced hypomania or mania ( [Akiskal et al., 1983](#); [Strober and Carlson, 1982](#)).

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#### CASE ILLUSTRATION

J.R. was 6 years of age when he was referred for inpatient treatment by a physician in a day hospital program because his behavior could no longer be tolerated. He presented with extreme hyperactivity and push of speech (including profanities and sexual comments in which he invited the female staff to have sex with him). After a careful evaluation, sexual abuse was ruled out. Previous treatment with Mellaril, Thorazine, Navane, Ritalin, Cylert, Dexedrine, and clonidine had been unsuccessful. He was silly and giggly. He made up songs, punned, and had clang associations. He spoke so fast it was hard to follow his thoughts. He believed he was Superman and could fly. He ran in front of cars, thinking he could stop them. An interview with his parents revealed that this child's father had been involved in legal trouble and had been jailed on several occasions. The father had been diagnosed as bipolar by the referring physician, who thought the father was medicating himself with psychoactive drugs. The father had stopped abusing drugs and had started on lithium, with dramatic improvement. His early history was very similar to J.R.'s current clinical presentation. J.R. was given a trial of lithium carbonate, and he responded fairly well. He was easier to manage and direct and was able to attend school.

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#### CASE ILLUSTRATION

B.J. was 13 years of age when he presented to the emergency department of a pediatric hospital. His chief complaint was "I wanted to take the dog for a walk last night, so I went to Six Flags (an amusement park) to go to the bathroom!" His speech was very difficult to interrupt. He had paranoid thoughts that his friend and classmates were after him and making fun of him. He also talked about "pumping up my BB gun 10 times before pulling the trigger and slashing my wrists because I want to die." He also said he was "wanting to be born over and over again." He was calling his female classmates and asking them to have sex with him and also "wanted to go to the desert to hunt lions." He was refusing to eat and had slept only 2 to 3 hours per night for 3 days. He paced constantly and made fun of the resident in the emergency department. His maternal grandmother had depression and a maternal uncle had been put in detention for behavior similar to this when he was an adolescent.

He reported he came to the hospital to make a death movie, and he was hearing male and female voices telling him to grow up and "to cut it off." When asked what "to cut it off" meant, he replied, "my penis." He had been involved in oral sex for the last month, and he thought this was a punishment. He thought he could fly and make people blind by staring at them. Drug use was denied, and a drug screen was negative. Treatment with Mellaril 200 mg subdued him somewhat. The family refused a trial of lithium carbonate because their pediatrician was worried about its possible long-term side effects.

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## ETIOLOGY AND PATHOGENESIS

Numerous etiologies have been considered in mania. The major hypotheses are briefly discussed.

### Psychoanalytic Theory

In psychoanalytic theory, mania is a reaction formation to depression. It is described as a defense against depression that involves a defensive projection where the patient focuses on the weaknesses of others to avoid thinking of his or her own weaknesses.

### Biological Theories

#### GENETIC

Although the etiology of bipolar disorder is unknown, studies support that there is a substantial genetic contribution. Twin studies consistently report that monozygotic twins are more concordant for affective illness than are dizygotic twins. [Numberberger and Gershon \(1982\)](#) reported the concordance of mania in monozygotic twins to be 65%, whereas concordance was 14% in dizygotic twins. Likewise, [Bertelsen and colleagues \(1977\)](#) reported the concordance for monozygotic twins to be 67%, whereas concordance for dizygotic twins was only 20%. Concordance rates for monozygotic twin pairs were higher for pairs in which one had bipolar illness (79%) than for pairs in which one had unipolar illness (54%); concordance rates between dizygotic twins pairs were similar for bipolar (24%) and unipolar (19%) illness. Thus, these studies strongly support a genetic contribution to the vulnerability for affective illness.

Current reports involving adoptees with bipolar illness suffer from methodologic difficulties and provide only limited support for the hypothesis that genes rather than environmental or family influences account for a large degree of the risk associated with the development of bipolar illnesses. Results from two small studies were inconclusive ([Von Knorring et al., 1983](#); [Wender et al., 1986](#)). A larger study by [Mendlewicz and Rainer \(1977\)](#) found that 31% of biological parents of adopted manic probands were affected, whereas only 2% of adoptive parents manifested the illness.

Family studies involving adults with affective illness consistently report that the rate of affective illness in relatives of affectively ill adult probands is two to three times the rate in relatives of appropriately selected case control subjects ([Gershon, 1990](#)). A study of the relatives of bipolar subjects by [Rice et al. \(1987\)](#) showed a 1.5% to 10.2% incidence of bipolar disorder, compared with an incidence of 0.2% to 1.8% in relatives of normal control subjects. Furthermore, family studies support at least a partial overlap in vulnerability for bipolar and unipolar affective illness because the most frequent affective disorder in relatives of patients with bipolar illness is unipolar illness ([Gershon, 1990](#); [Rice et al., 1987](#)). A multicenter pilot family study completed by the [National Child Study Institute of Mental Health Molecular Genetics of Psychiatric Disorders Initiative Group \(1993\)](#) reported that the lifetime probability for development of affective disorders (bipolar, major depression, or dysthymia) in children and adolescents is associated with the number of affected parents who had high density of affective disorder in extended families. The corresponding percentages were 16.7%, 23.5%, and 100% for 0, 1, and 2 affected parents, respectively.

There is evidence to suggest that childhood-onset bipolar disorder is associated with greater familial loading for affective illness than adult-onset bipolar disorder ([Todd et al., 1994](#)). In studying the parents of bipolar children and adolescents, several investigators reported an increased incidence of both unipolar and bipolar disorders ([DeLong and Aldershof, 1987](#); [Strober et al., 1988](#); [Varanka et al., 1988](#)). Further, if onset of illness is in the prepubertal years, the rate of bipolar disorder in family members is three times that of those with an onset after puberty ([Strober et al., 1988](#)). Furthermore, recent reports on children of adults with bipolar disorder suggest that a history of early-onset illness increases the risk that bipolar disorder will develop in offspring ([Chang et al., 2000](#)).

To date, linkage and association studies have failed to identify the genes associated with bipolar illness. Detailed genetic studies of well defined bipolar children and adolescents are lacking.

#### NEUROBIOLOGICAL STUDIES

Few studies have examined the neurobiological underpinnings of bipolar disorder in children and adolescents. Preliminary neuromorphometric reports have found enlarged ventricles and increased number of white matter hyperintensities in prepubertal children with bipolar disorder compared with control subjects ([Botteron et al., 1995](#)). Thalamic abnormalities also have been observed in adolescent patients with bipolar illness. A study using magnetic resonance spectroscopy found elevated lipid levels in the frontal lobes and elevations of glutamate in both frontal lobes and basal ganglia of children with bipolar disorder relative to control subjects ([Castillo et al., 2000](#)). There are no published studies examining neuroendocrine or neuroanatomic differences in children and adolescents with mania.

### Familial Environmental and Social Influences

As described earlier, mania tends to run in families. When child-rearing practices of manic parents were examined, many had poor parenting techniques ([Cytryn et al., 1986](#)). The episodic nature of the unreasonable behavior of these parents may be deleterious to the normal development of these children.

## LABORATORY STUDIES

There are no specific laboratory studies that can diagnose bipolar disorder in children and adolescents.

## RATING SCALES

It appears that the diagnostic accuracy for bipolar illness in children and adolescents improves when DSM criteria are applied ([Carlson et al., 1994](#)). Several structured and semistructured interviews may be used to help assess mania in children. The Diagnostic Interview for Children and Adolescents, Revised (DICA-R) (Reich W, Weiner Z, Diagnostic Interview for Children and Adolescents [DICA-R-C], DSM III-R Version, St. Louis, Washington University, Division of Child Psychiatry, unpublished manuscript, 1988), the Diagnostic Interview Schedule for Children (DISC) ([Costello et al. 1982](#)), and the Children's Interview for Psychiatric Syndromes (ChIPS) ([Weller et al., 1999](#)) are structured interviews that have sections to assess bipolar disorders. Several semistructured interviews also may be used, including



the Schedule for Affective Disorders and Schizophrenia for School Age Children (KSADS) ( [Chambers et al., 1985](#)), the Washington University Kiddie and Young Adult Schedule for Affective Disorders and Schizophrenia—Lifetime and Present Episode Version for DSM-IV (WASH-U-KSADS) ( [Geller et al., 1998](#)), and the Interview Schedule for Children (ISC) ( [Kovacs, 1978](#)). With standardized assessment instruments, bipolar disorder can be overdiagnosed if a comprehensive and careful evaluation is not completed.

Clinical rating scales may be used to track the severity of target symptoms of mania. Such rating scales for bipolar disorders are quite underdeveloped at present. [Strober et al. \(1987\)](#) used the Beigel Murphy Scale ( [Beigel et al., 1971](#)) in assessing severity of mania in adolescents, and reported the instrument to be helpful. [Fristad et al. \(1988\)](#) modified the Mania Rating Scale ( [Young et al., 1978](#)) for use in prepubertal manic children. A preliminary study found it was helpful in differentiating manic children from hyperactive children on the total score as well as on most individual items. The Child Behavior Checklist (CBCL) also has been reported to distinguish children with mania from those with ADHD ( [Biederman et al., 1995](#); [Geller et al., 1998](#)). However, the CBCL is not a diagnostically derived instrument, and differences in scores may reflect severity of illness rather than diagnostic differences ( [Carlson and Kelly, 1998](#)).

## DIFFERENTIAL DIAGNOSIS AND COMORBIDITY

In diagnosing a manic episode, other psychiatric and medical illnesses should be excluded. Medical conditions ( [Table 63.2](#)) may mimic mania, and patients with several other psychiatric illnesses sometimes can appear manic. Reports suggest that manic symptoms are common in both nonreferred samples ( [Carlson and Kashani, 1988](#); [Lewinsohn et al., 1995](#)) and psychiatrically ill children ( [Carlson and Kelly, 1998](#)). Manic symptoms in children may be a marker for severe psychopathology and impairment ( [Carlson and Kelly, 1998](#)). Careful diagnostic workup and long-term follow-up often are needed to clarify diagnosis in this age group ( [Carlson, 1998](#); [Carlson et al., 2000](#)).

Head	Endocrine	Neurologic	Toxic	Medications	Other
Encephalitis	Hypothyroidism	Seizures	Toxic	Stimulants	Acetaminophen
Influenza		Head trauma	Gloma	Isoniazid	Digoxin
Spina		Multiple sclerosis	Meningioma	Sympathomimetics	Amphetamines
AD		Stroke			Hallucinogens
		Metabolic disease			Anemia
					Hemodysia

Reprinted from M. M. Zandi, J. R. Costello, & J. Costello (1998). *Journal of Abnormal Child Psychology*, 26, 1-18.

**Table 63.2. Medical Conditions Mimicking Mania in Children and Adolescents**

### Medical Conditions

#### DRUGS

A diagnosis of mania should not be made until the child or adolescent is observed in a drug-free state. Some drugs reported to induce manic states include amphetamines, corticosteroids, sympathomimetics, isoniazid, and antidepressants.

#### ENDOCRINE DISORDERS

Hyperthyroidism may present with a manic-like state.

#### NEUROLOGIC CONDITIONS

Mania has been associated with head trauma, multiple sclerosis, stroke (especially with lesions involving the right hemisphere and the thalamus), and seizure disorders with a left temporal focus. Also, space-occupying lesions such as meningiomas, gliomas, and metastatic lesions in the thalamus have been implicated ( [Wise and Rundell, 1988](#)).

### Psychiatric Conditions

#### ATTENTION DEFICIT HYPERACTIVITY DISORDER

Symptom overlap between mania and ADHD can lead to diagnostic confusion ( [Carlson, 1998](#)). Psychomotor agitation, distractibility, aggression, poor school performance, restless sleep, and sexually inappropriate behavior are symptoms that frequently are associated with both ADHD and mania.

Reports suggest that childhood-onset bipolar disorder frequently is comorbid with ADHD ( [Geller et al., 1995](#); [Wozniak et al., 1995](#)). Age of onset of bipolar illness may be associated with varying rates of comorbid ADHD. [Faraone and colleagues \(1997\)](#) reported that adolescents with childhood-onset bipolar illness had the same rate of comorbid ADHD as children with prepubertal-onset bipolar illness. Both groups had higher rates of ADHD than adolescents with adolescent-onset bipolar illness. Furthermore, [Sachs and colleagues \(2000\)](#) reported that among adults with bipolar disorder, a history of comorbid ADHD was evident only in those with a history of early-onset illness (mean age of onset, 12.1 years).

Cross-sectionally, children with ADHD may appear to resemble those with mania, especially in the prepubertal age range. However, manic children have more affect and are euphoric or irritable. Children with ADHD often have low self-esteem and a much longer duration of symptoms. Psychotic symptoms are common in prepubertal manic children ( [Varanka et al., 1988](#)) but are not typically part of ADHD. The sleep disturbances and overactivation associated with ADHD are chronic and part of the child's baseline behavior. The sleep disturbance associated with mania usually is associated with onset of the mood disturbance. Age of onset for mania usually is after 7 years of age, whereas age of onset for ADHD usually is in the toddler years.

#### CONDUCT DISORDER

Conduct disorder frequently is a comorbid condition with bipolar disorder ( [Kovacs and Pollock, 1995](#)). Many children with mania have unruly, disruptive behavior suggestive of a conduct disorder. However, children with a "pure" conduct disorder do not have push of speech, flight of ideas, or delusions of grandeur. Also, the duration of symptoms is more chronic and less suggestive of an abrupt onset or clearly episodic course. A study by [Weller et al. \(1986c\)](#) found that children who clearly satisfied criteria for mania sometimes were diagnosed as having conduct disorder.

#### DRUG AND ALCOHOL ABUSE OR DEPENDENCY

Children and adolescents with mood disorders sometimes become involved with the use of alcohol or drugs in an attempt to treat symptoms attributable to their mood disorder. Most adolescents have easy access to drugs and alcohol. Special attention should be paid to rule out mania in prepubertal children presenting with drug abuse.

#### SCHIZOPHRENIA

Symptoms such as hallucinations and delusions are common in both mania and schizophrenia. Usually the onset of schizophrenia is insidious, whereas the onset of mania is more sudden. Both schizophrenic and manic adolescents can be delusional, and, at times, it may be difficult to differentiate paranoid schizophrenia from

mania. Assessment of premorbid personality may be helpful in differentiating the two disorders. In schizophrenia there may be either schizoid traits or bizarre conduct disorder, whereas in mania, there may be symptoms suggestive of ADHD or conduct disorder with significant affective symptomatology. A positive family history for bipolar disorder is more common in manic adolescents than in schizophrenic adolescents ( [Strober et al., 1988](#) ).

## TREATMENT

### General Considerations

Once a diagnosis of mania has been established after a thorough assessment, treatment must be planned and implemented. Psychotherapeutic and pharmacologic intervention may be required in children and adolescents. It is difficult to treat a full-blown manic episode on an outpatient basis. Hospitalization often is essential to ensure patient safety. In the hospital, firm limits must be set with the patient. Arguments, challenges, or questions regarding a patient's delusional system should be avoided until the medications take effect and the patient is more amenable to other therapeutic interventions. It is essential to educate parents and children about bipolar illness. Support groups organized through the National Alliance for the Mentally Ill are available in certain communities and can be of help to some patients and their families.

Several baseline laboratory studies need to be done before starting a child on lithium carbonate or any other psychotropic medication that can affect many organ systems. For example, lithium affects the neuroendocrine, central nervous, reproductive, and gastrointestinal systems. A good prelithium workup should include a complete blood count with a differential ( [Table 63.3](#) ). This is needed because lithium can cause neutrophilia, and carbamazepine and divalproex, mood-stabilizing medications sometimes used in lithium nonresponders or rapid cyclers, may cause suppression of white blood cells. Electrolytes, blood urea nitrogen, creatinine, creatinine clearance, urine osmolality, and liver function tests should be performed. Lithium is known to affect serum electrolytes, especially sodium. Also, lithium may affect kidney function tests; hence, a baseline creatinine clearance, as well as urine osmolality, should be determined and repeated periodically if lithium treatment lasts more than 6 months.

**Table 63.3. Pretreatment Screen for Mood Disorders in Children and Adolescents**

Thyroid function tests are required because lithium can alter thyroid function. Hypothyroidism is the most common abnormality. Because a hypothyroid person also may look depressed, baseline measurements of thyroid-stimulating hormone (most sensitive) and thyroxine and triiodothyronine resin uptake should be measured before lithium initiation, and should be repeated periodically throughout treatment. Liver function tests should be done in cases in which medications normally metabolized in the liver are being used.

A pregnancy test and serologic tests (for syphilis and acquired immunodeficiency syndrome) should be performed, especially in sexually active adolescents. Hypomanic and manic people are known for their hypersexuality; they may engage in sexual activity with multiple sexual partners in an indiscriminate manner. Pregnancy must be ruled out because mood-stabilizing agents have been associated with teratogenic effects. Before treating a manic child or adolescent with lithium or other medication, consent from the family and assent from the patient should be obtained, and known side effects should be explained.

### Psychopharmacologic Treatment

There is a dearth of controlled medication trials in children and adolescents with bipolar disorder. However, conventional wisdom suggests acute mania is best managed with an antimanic agent. Adjunctive medications such as benzodiazepines or antipsychotics may be used in the short term to help control symptoms of severe agitation, insomnia, and psychosis. It usually is wise to start medications at a low dose and to titrate them up to the minimally effective dose to minimize adverse effects and maximize compliance. Careful titration is particularly important in younger children because they are particularly prone to experience untoward effects ( [Campbell et al., 1991](#) ). Every effort should be made to manage both the acute and maintenance phase of this illness with as few agents as possible. Antidepressants may be necessary when depressive episodes occur, but use should be closely monitored.

### LITHIUM

Lithium has been extensively studied in adults with bipolar disorder. Furthermore, reports suggest that maintenance treatment with lithium may be effective in reducing the risk of suicide associated with bipolar disorder ( [Tondo and Baldessarini, 2000](#) ). Controlled trials of lithium for the treatment of explosive aggressive behavior in children and adolescents also have been conducted ( [Campbell et al., 1995](#) ). Most reports of its use in children and adolescents with bipolar disorder consist of case reports ( [Geller and Luby, 1997](#) ) and open-label trials ( [Kafantaris et al., 1998](#) ). A randomized clinical trial of lithium for the treatment of bipolar illness in adolescents included 25 adolescents who met DSM-III-R criteria for either bipolar I, bipolar II, or major depressive disorder with adolescent predictors of future bipolar disorder. These youths also had secondary substance abuse. Lithium was not more efficacious than placebo on measures of affective symptoms. No study has examined the efficacy of lithium in prepubertal children with mania, although safety ( [Hagino et al., 1995](#) ) and dosing ( [Hagino et al., 1998](#) ) have been studied.

The most common side effects of lithium are gastrointestinal upset, nausea, overeating, weight gain, enuresis, tremor, and acne. Diarrhea sometimes is an early sign of toxicity. Seizures may occur with lithium overdose. Children and adolescents often tolerate lithium carbonate better than adults. Lithium levels of 0.6 to 1.2 mEq/L are well tolerated. If there is no response at that level, the level might be increased to 1.4 mEq/L with careful monitoring and in the absence of significant side effects. [Weller and colleagues \(1986a, 1986b\)](#) have developed a dosing schedule based on weight, so that a child can be started on lithium and reach a therapeutic level quickly ( [Table 63.4](#) ). Children who have central nervous system impairment or mental retardation may be particularly sensitive to side effects from lithium and may have difficulty achieving a therapeutic effect.

A. Administer dosage according to chart below.  
 B. Take blood levels 12 hours after last dose (8:00 AM before morning dose) every other day (M, W, F) until two consecutive levels appear in the therapeutic range (0.6–1.2 mEq/L). If no clinical improvement is seen, increase dose to reach 1.4 mEq/L.  
 C. Administer Side Effect Rating Scale and Mania Rating Scale weekly.

Weight (kg)	8 AM	Dosage (mg) 12 noon	8 PM	Total daily
15–25	150	150	300	600
25–40	300	300	300	900
40–50	300	300	600	1,200
50–60	600	300	600	1,500

\*CAUTION: In dealing with organically impaired or retarded children, use lower doses. Adapted from Weller (B, Weller SA, Fristad MA, Lithium dosage guide for prepubertal children: A preliminary report. *J Am Acad Child Psychiatry* 25:92–96, 1986.

**Table 63.4. Lithium Protocol**



## CARBAMAZEPINE

Other medications have been less well studied than lithium in manic children and adolescents. Carbamazepine (Tegretol; Novartis, Summit, NJ) has been reported to be as efficacious as lithium in reducing symptoms of acute mania in adults with bipolar disorder ( [Simhandl et al., 1993](#); [Small et al., 1991](#)). Carbamazepine may not be as efficacious as lithium in maintenance treatment ( [Greil et al., 1997](#)). No published study has examined the efficacy of carbamazepine for either the acute or maintenance treatment of mania in children or adolescents. Furthermore, carbamazepine has been associated with the development of mania in children ( [Myers and Carrera, 1989](#); [Reiss and O'Donnell, 1984](#)).

Carbamazepine must be used carefully because it is known to cause bone marrow suppression with clinically significant neutropenia. Often, sore throat and fever are the first warning signs of a seriously low neutrophil count. Carbamazepine usually is started at 100 mg twice daily (15 to 30 mg/kg) and increased every fourth day. Therapeutic plasma levels are 5 to 11 mEq/L; biweekly blood counts to evaluate possible bone marrow suppression should be obtained for 3 months. Furthermore, dosage may require adjustment at 4 to 6 weeks owing to autoinduction of hepatic enzymes.

## DIVALPROEX

There is a substantial body of evidence to support the efficacy of divalproex in the treatment of adults with bipolar illness ( [Bowden et al., 1994](#); [Denicoff et al., 1997](#); [Pope et al., 1991](#)). Support for its use in children and adolescents is limited to case reports and small open-label trials ( [Papatheodorou and Kutcher, 1993](#); [Papatheodorou et al., 1995](#)). Fifteen adolescents and young adults were treated with divalproex sodium for 7 weeks for acute mania; improvement on the clinical severity scale for mania was reported in nine subjects ( [Papatheodorou et al., 1995](#)).

A starting dose of 250 mg twice daily may be used and increased gradually to a dose not to exceed 25 to 60 mg/kg/day. Blood levels between 50 and 120 mg/L are considered effective in adults. However, no controlled studies in children or adolescents have been published. The most common side effects are nausea, vomiting, and anorexia. Because hepatotoxicity has been reported, periodic liver function tests are necessary. Fatal hepatotoxicity has been reported in children; this risk is highest for children younger than 2 years of age and diminishes to close to zero after 10 years of age ( [Bryant and Dreifuss, 1996](#)). More recently, concerns have risen about a possible association between childhood valproic acid use and later risk for polycystic ovary disease in women ( [Irwin and Masand, 1998](#); [Isojarvi et al., 1993](#)). This serious untoward effect has been reported in patients treated for epilepsy, but has not been reported in those treated for bipolar disorder ( [Rasgon et al., 2000](#)). However, caution should be used when prescribing divalproex to any adolescent girl. It is recommended that before starting divalproex in an adolescent girl, a baseline weight, baseline waist/hip measurement, complete menstrual history, and evaluation for hirsutism be obtained.

## LAMOTRIGINE

There is growing support for the efficacy of lamotrigine in the treatment of adults with bipolar disorder ( [Calabrese et al., 1996](#); [Fatemi et al., 1997](#); [Kusumaker and Yatham, 1997a,b](#); [Sporn and Sachs, 1997](#); [Walden et al., 1996](#)). Its use in the treatment of mania in children and adolescents, however, is limited by its age-related association with Stevens-Johnson syndrome ( [Dooley et al., 1996](#); [Mackay et al., 1997](#); [Messenheimer et al., 1998](#)). Its use in the treatment of mania in children, as in epilepsy, is limited to adolescents 16 years of age and older who have failed other therapies.

## GABAPENTIN

Gabapentin has been used in adults in the treatment of acute mania ( [Ghaemi et al., 1998](#); [Knoll et al., 1998](#); [McElroy et al., 1997a](#); [Young et al., 1997](#)). Few studies have been conducted in children and adolescents. One case report recounts a favorable response to gabapentin plus carbamazepine in a 13-year-old boy with bipolar disorder ( [Soutullo et al., 1998](#)). Gabapentin has been reported to worsen aggressive behavior in children treated for seizures ( [Lee et al., 1996](#); [Tallian et al., 1996](#); [Wolf et al., 1995](#)).

## ANTIPSYCHOTICS

A study involving prepubertal children with mania suggests that psychotic symptoms as well as manic symptoms can be effectively managed without the use of antipsychotic agents ( [Varanka et al., 1988](#)). A more recent report involving adolescents with psychotic disorder, however, suggests that lithium alone may not be able to control psychotic symptoms in this age group ( [Kafantaris et al., 1998](#)).

Haloperidol has been effective in the treatment of aggression associated with disruptive disorders in children ( [Campbell et al., 1984](#)). Use of traditional antipsychotics is limited by untoward effects that interfere with compliance, including risk of tardive dyskinesia, weight gain, and sedation. Reports suggest that the novel antipsychotic agents risperidone, olanzapine, and clozapine may be effective in the treatment of mania in children and adolescents ( [Chang and Ketter, 2000](#); [Fras and Major, 1995](#); [Fuchs, 1994](#)). Extrapyramidal symptoms tend to be observed less frequently with these agents than with traditional antipsychotics, but weight gain and sedation continue to pose significant problems. Furthermore, the risk of agranulocytosis associated with clozapine limits its use in this age group.

## Electroconvulsive Therapy

Electroconvulsive therapy (ECT) has been used to treat severe bipolar disorder in children and adolescents. In a literature review, [Bertangoli and Borchardt \(1990\)](#) report that the few systematic studies were done before the early 1950s, and since then only individual cases have been reported. Data concerning safety and efficacy of ECT in children are sparse. A report involving 16 adolescents who received ECT for treatment-resistant bipolar disorder indicated that ECT was effective, well tolerated, and cost efficient in this age group ( [Kutcher and Robertson, 1995](#)). Furthermore, adolescents given ECT for severe bipolar illness do not appear to sustain cognitive impairment at long-term follow-up ( [Cohen et al., 2000](#)). The [American Psychiatric Association \(1990\)](#) has published specific guidelines regarding indications for, procedures of obtaining consent for, and administration of ECT treatments. Before referring children for ECT, two psychiatrists experienced in treating children and who are not otherwise involved with the patient should agree with the recommendation for ECT. ECT may be considered when other modalities of treatment have failed.

## OUTCOME AND FOLLOW-UP DATA

There are no systematic follow-up data on preschool- or prepubertal school-age children with bipolar disorder. Anecdotal reports suggest that these children do not "grow out of it" ( [Poznanski et al., 1984](#)). Several studies report follow-up data on adolescent-onset mania. Reports suggest that compared with adults, adolescents with bipolar disorder have a more prolonged early course and are less responsive to treatment ( [McGlashen, 1988](#); [Strober et al., 1995](#)). Long-term outcome, however, is reported to be similar to that of adult-onset bipolar disorder.

[Landolt \(1957\)](#) found that after 18 years of follow-up, 17% of patients with adolescent-onset of mania recovered, whereas 50% had a poor outcome. Data on the remaining subjects were inadequate to make a decision as to outcome. [Olsen \(1961\)](#) reported on 28 adolescents with a mean age of onset of 15 to 16 years. He divided his group into those who had had an acute onset and those with prior childhood psychopathology. A 25-year follow-up revealed a good outcome for 33% of the acute-onset group, whereas only 15% in the group with prior psychopathology had a good outcome. [Hudgens \(1974\)](#) and [Weiner et al. \(1979\)](#) reported the outcomes for their adolescent patients with mania to be social disability, substance abuse, multiple suicide attempts, and completed suicide. In a 20-year follow-up of adolescents with mania, [Carlson et al. \(1977\)](#) reported that 60% of subjects studied had a good social outcome, 20% had significant impairment, and 25% were chronically ill.

Another study reported good outcome in 35 adolescents with mania ( [McGlashen, 1988](#)). The author divided this sample into two groups: those with onset before 20 years and those with onset after 20 years of age. Patients with the earlier age of onset had more psychiatric symptoms and trouble with the law, and 70% had "psychotic assaultiveness." However, there was no difference in the suicide rate or frequency of hospitalizations between the two groups. Also, those with earlier onset performed significantly better regarding work and frequency of social contacts.

In a naturalistic follow-up study, [Strober et al. \(1990, 1995\)](#) found that adolescents who continued their lithium carbonate treatment had fewer relapses than those who stopped the treatment, which suggests that lithium may play a role in preventing relapses. In this study, only 2 of 54 adolescents failed to recover from their initial

episode over a 5-year period. Furthermore, rate of recovery was influenced by the polarity of the index episode; subjects with depression took longer to recover than subjects who had cycling, mixed episodes, or pure manic episodes.

Adolescents with bipolar disorder are at increased risk for suicide relative to children with other psychiatric illnesses ( [Brent et al., 1988, 1993](#)). Eleven of 54 adolescents with bipolar disorder made serious suicide attempts during 5-year follow-up ( [Strober et al., 1995](#)). Comorbid substance abuse further increases the risk for suicide in adolescents ( [Rich et al., 1986, 1990](#)).

## SUMMARY

Bipolar disorder in children and adolescents is less studied than adult-onset bipolar illness. Historically, several factors have made the diagnosis of bipolar disorder in childhood difficult. These include bias against the diagnosis of mania in children, the low base rate of mania relative to other psychiatric illnesses in children, symptom overlap between bipolar disorder and other, more prevalent childhood-onset psychiatric disorders, and developmental constraints and variability in clinical presentation. The validity and prevalence of bipolar illness in childhood remain controversial. Epidemiologic studies are needed to assess accurately the prevalence of this disorder in childhood. Current consensus is that the incidence of bipolar illness increases with puberty. Reports on the phenomenology of bipolar disorder in children and adolescents indicate that its presentation is highly variable, with a developmental trend toward increasing resemblance to the adult phenotype with increasing age of onset. Diagnostic accuracy seems to improve with adherence to DSM criteria. The course of bipolar disorder in childhood has received only limited study. Available studies suggest that bipolar illness is a relapsing, recurrent illness with substantial morbidity and mortality. Few studies that systematically investigate the use of pharmacologic agents in the treatment of bipolar illness in youth have been published. Justification for the use of mood stabilizers in children is based primarily on adult literature. There is a significant need for additional research in all aspects of childhood-onset bipolar disorder.

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## 64 SEASONAL AFFECTIVE DISORDER IN CHILDREN AND ADOLESCENTS

Javad H. Kashani and Joseph M. Pastor

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Light and its effect on the human body has been the subject of investigation for centuries. Lighting conditions at work, home, and play can affect our behavior ([Wetterberg, 1992](#)). Seasonal rhythms in human behavior have been observed since antiquity, and Kraepelin noted seasonal differences in mood and behavior in the early 1900s. Numerous investigators became interested in the relationship between environmental light and mental illness, and in [1984, Rosenthal and colleagues](#) reported their initial description of a pattern of recurrent depressive symptoms observed in women in their early twenties that they termed *seasonal affective disorder* (SAD). They noted that one-third of adult patients with SAD had onset in childhood, and later described children with seasonal symptoms who complained of sadness, anxiety, fatigue, irritability, changes in appetite, and social withdrawal. These symptoms were associated with seasonal changes in school behavior and academic difficulties, especially in individuals with family histories of mood disorders and alcohol abuse ([Kern, 1990](#); [Mghir and Vincent, 1991](#); [Sher et al., 1999](#)).

The relationship between mood and light has aroused continued interest in general psychiatry, and investigators have only recently begun to pursue the scientific study of SAD in children and adolescents. This chapter reviews the literature regarding the clinical description, epidemiology, and etiology of SAD. Treatment also is reviewed, and future directions for study are discussed.

### CLINICAL DESCRIPTION

Seasonal affective disorder (SAD) was initially included in the [American Psychiatric Association's \(1990\) Diagnostic and Statistical Manual of Mental Disorders](#), third revised edition (DSM-III-R) and was described as a mood disorder modifier, not as a separate diagnostic entity or course specifier ([Bauer and Dunner, 1993](#)). SAD was designated a course specifier in DSM-IV ([American Psychiatric Association, 1994](#)) and was defined as a cyclical pattern of recurrent major depressive symptoms in adults with a seasonal or temporal pattern. It was applied to unipolar, bipolar I, and bipolar II disorders and had a regular, seasonal onset in the absence of psychosocial stressors. In [1984, Rosenthal and colleagues](#) presented their definition of SAD as recurring fall and winter depressive episodes without related stressors. Asymptomatic periods occurred in the spring and summer with two seasonal episodes in consecutive years, one of which met DSM-IV criteria for a major depressive disorder (MDD). Rosenthal et al. excluded any other Axis I pathologic process. To qualify for the seasonal specifier, a pattern of recurrent depression with initiation and termination of symptoms had to occur within a 60-day window from year to year. The remarkable feature, therefore, was the temporal nature of the disorder itself ([Blehar and Rosenthal, 1989](#)). In DSM-IV, the seasonal specifier also requires that full remissions or cycling into mania or hypomania occur regularly throughout the year and that within the last 2 years before the onset of SAD, two major depressive episodes had occurred demonstrating a seasonal aspect to the episodes. Other Axis I diagnoses do not necessarily exclude the application of the seasonal specifier ([Bauer and Dunner, 1993](#)).

The diagnosis of SAD is based on clinical evaluation. The practitioner must consider chief complaint, present and past psychiatric history, family data, and especially the temporal relationship between symptoms, environment, and psychosocial stressors. Patients with SAD usually complain of atypical depressive symptoms, such as increased appetite, carbohydrate craving, increased weight, and increased sleep, that are worse in the evening. These symptoms occur in 65% to 80% of patients with SAD and 15% to 30% of patients with MDD who present as outpatients. Other symptoms include premenstrual difficulties, interpersonal dysfunction, and work distress ([Terman et al., 1989](#)). Patients with SAD may meet criteria for major depression, but the symptoms are described as seasonal ([Pendse et al., 1999](#)).

Onset of SAD typically is in September through November, with hypersomnia, hyperphagia, and excessive fatigue occurring with a change in the amount of natural light. A later onset can occur if the fall is mild and sunny ([Mayor et al., 1991](#)). The most severely symptomatic months for patients are January and February, with remissions usually occurring in April and May. The lifetime prevalence of anxiety disorders in SAD is high—generalized anxiety disorder, simple and social phobia, as well as personality disorders are prevalent. Patients may have unipolar, bipolar I, bipolar II, or dysthymic disorders ([Terman et al., 1989](#)), although most are unipolar ([Sakamoto et al., 1995](#)). Other comorbid conditions include disruptive behavior disorders in children. Patients with SAD complicated by other psychiatric disorders are more severely depressed and can present with suicidal ideation. Two-fifths of SAD patients have pure SAD, two-fifths have complicated SAD, and one-fifth remit over 8 years. There are no data to suggest that having bipolar disorder and SAD worsens the long-term course of the SAD ([Schwartz et al., 1996](#)). In fact, some patients with SAD can become hypomanic secondary to use of a selective serotonin reuptake inhibitor ([Sakamoto et al., 1995](#)).

Seasonal affective disorder symptoms are more frequent with increasing distance from the equator and decrease with short geographic visits to lower latitudes. Symptoms usually are more intense with increasing latitude ([Partonen and Partinen, 1994](#); [Terman et al., 1989](#)). Patients living in an extreme northern latitude show an increase in severity of symptoms ([Rosenthal et al., 1984](#); [Sourander et al., 1999](#)). Geographic location and its impact on day length, daily hours of sunshine, and temperature are other variables that appear to affect patients with SAD.

Most children and adolescents are undiagnosed ([Sourander et al., 1999](#)). In children and adolescents, seasonal symptoms may be due to school pressures and not expressly SAD because it is difficult to eliminate the effects of the academic school year on patients ([Milman and Bennett, 1996](#)). Children show depressed mood of 2 weeks or longer, fatigue, sleep disturbance, carbohydrate craving, headache, school problems, and social withdrawal. Parents report that the children are irritable, tired, have more school problems, seem sad, cannot sleep, and have more somatic complaints, changes in appetite, carbohydrate craving, and increased motor activity. Parents also observe more crying, increased anxiety, social withdrawal, and tantruming. Symptoms that are similar to those of adults in the winter include depressed mood and decreased activity.

Most children do not present with complaints of excessive sleep, which is a commonly reported symptom in adults with SAD ([Swedo et al., 1995](#)). SAD is considered a valid type of mood disorder in children, and one of the major reported symptoms is disruptive behavior. Adolescents have a higher prevalence than children; it is estimated that 1 million adolescents have SAD ([Sourander et al., 1999](#)). SAD is a milder syndrome in children, yet it leads to functional impairment and results in social and academic difficulties at school. The children frequently are unaware of the problem and they perceive the disorder as external, with multiple complaints from teachers and parents ([Rosenthal et al., 1986](#)). It is debated whether pediatric patients with SAD may be predominantly bipolar II or unipolar ([Sher et al., 1999](#)).

The Seasonality Scale Index is a reliable measure to assess patients, yet SAD is a clinical diagnosis and should be made through obtaining a detailed history ([Magnusson et al., 1997](#)). The Seasonal Pattern Assessment Questionnaire is another instrument used in research protocols for identifying patients with SAD, but its psychometric properties are open to question ([Raheja et al., 1996](#)).

### EPIDEMIOLOGY

The lifetime prevalence of SAD is 1% to 2% of the general population, 0.4% to 0.9% in patients with MDD, and 1% in patients with dysthymic disorder. The combined prevalence of SAD and subclinical SAD is 3.1% over 2 consecutive years and 2.4% over 3 consecutive years, with an increased prevalence in twins ([Partonen and Lonnqvist, 1998](#)). [Carskadon and Acebo in 1993](#) and [Swedo et al. in 1995](#) reported that 3% to 5% of school-age children may have SAD. [Sourander et al. \(1999\)](#) observed that SAD occurs in 4% to 12% of adults, 1.9% of 9-year-olds, and 5.5% of 19-year-olds, with an increased prevalence in northern latitudes. The female-to-male ratio has been reported to be 4:1 to 6:1 ([Kasper et al., 1989](#); [Rosenthal et al., 1984](#)), although others have reported the sex ratio to be equal ([Carskadon and Acebo, 1999](#)). Some note up to a 69% frequency of a mood disorder in first-degree relatives of patients with SAD ([Rosenthal et al., 1984](#)). In general, approximately one-third of patients have onset in childhood, with higher rates in girls than in boys ([Sourander et al., 1999](#)). Patients with SAD report increasing symptoms with age and at puberty. Some postulate a relationship between puberty and increased rates of SAD in girls and of temporally related depressive episodes



in adult women during their reproductive years ([Swedo et al., 1995](#)).

## ETIOLOGY

Humans and other mammals have biological functions occurring on an approximately 24-hour cycle that are regulated by hormones through various anatomic structures and pathways in the central and peripheral nervous systems (circadian rhythms). These cycles are manifested in aspects of physiology, such as temperature, hormone production, and sleep–wake cycles. Hormones such as melatonin and cortisol have diurnal rhythms, affect sleep, and are influenced by light and temperature. Without exposure to light, the human body adjusts itself to an approximate 24-hour period, depending on the individual and the environment ([Wetterberg, 1992](#)). Because it is well known that certain mammals hibernate during shortened periods of light and experience seasonal physical changes owing to the environment, it has long been postulated that similar changes in the photoperiod have effects on human physiology and behavior.

Several hypotheses are reviewed in the literature for SAD. The first is the phase shift hypothesis (Lewy and Sack, 1986), which postulates that there is a primary disturbance of delayed timing of circadian rhythms in patients with SAD. Exposure to bright light is thought to terminate the delayed portion of nocturnal melatonin secretion. Others note that dim light also results in delayed melatonin secretion ([Glod et al., 1997](#)). Patients with SAD may have a particular vulnerability to a decreasing photoperiod in the winter months. There appears to be a correlation between later onset of daily light and depressed mood, which suggests that some individuals may have an exaggerated need for environmental light ([Oren et al., 1994](#)). Patients may for some unknown reason be more sensitive to changes in natural light because of biological phenomena or other mechanisms ([Guillemette et al., 1998](#)).

The second hypothesis ([Czeisler et al., 1987](#)) postulates that the amplitude, not the 24-hour phase of circadian rhythms, may be reduced and that light works by increasing the amplitude of hormone production and regulatory functions. There is little evidence that this phenomenon occurs with temperature, yet there is some research that supports this hypothesis with respect to hormonal changes in the human body.

The third hypothesis ([Teicher et al., 1990](#)) suggests that in SAD there is a primary regulatory disturbance, called *entrainment*, which refers to the body's ability to align itself with the environment. It is postulated that a reduction in the degree of entrainment is corrected by bright light. Serotonergic projections to the suprachiasmatic nucleus may control the rhythm, yielding decreased entrainment.

The fourth hypothesis maintains that SAD results from decreased absorption of quanta of light, perhaps due to a retinal pathologic process, and that bright light works by increasing the number of photons of light absorbed. A general change in the photoperiod appears to be mediated by retinal and hypothalamic structures in the central nervous system ([Partonen and Partinen, 1994](#)). Some suggest that bright light works by changing the secretion of melatonin, which is mediated by afferents through the retinohypothalamic tract, suprachiasmatic nuclei, and upper cervical ganglion to the pineal gland, which secretes the hormone ([Oren and Terman, 1998](#)). Melatonin reaches its highest levels at night and decreases during the day, and it is thought that patients with SAD have a greater seasonal change in the pineal gland's secretion of the hormone. Others suggest that the ventrolateral and dorsomedial aspects of the suprachiasmatic nuclei are intimately involved in entraining the circadian clock and that in patients with SAD, these neurons are pathologically misaligned with the environment in the winter months ([Thompson et al., 1990](#)).

Neurotransmitters and hormones also have been implicated in the etiology of SAD. It is unclear if serotonergic potentiation is the cause of circadian rhythm phase shifting ([Terman and Terman, 1999](#)). Noradrenergic projections may be responsible for decreased amplitude and attenuation. Hormones involved in sexual development may modulate the brain's vulnerability to changing season and light schedules ([Swedo et al., 1995](#)). Neuroimaging research in patients with SAD shows decreased metabolic rates ([Cohen et al., 1992](#)). Studies of prolactin, thyroid hormone, and adrenocorticotropic hormone have not clearly defined the pathophysiology of SAD. Estrogens may effect the timing of depressive symptoms and disruptive behavior in children and adolescents, and effect changes in adult women ([Oren et al., 1994](#); [Partonen and Partinen, 1994](#)). Seasonal changes may occur in serotonin levels in fall and winter ([Bick, 1986](#)). Disturbed biological rhythms may be due to impaired transmission of serotonin or other neuropeptides along afferent pathways to the circadian pacemaker. Decreased dietary tryptophan may lead to decreased levels of serotonin, and depressive symptoms ([Partonen et al., 1997](#); [Thalen et al., 1995a, 1997](#)). Other mechanisms suggested include an immune response of peripheral blood lymphocytes, the involvement of visual-evoked responses, changes in rapid eye movement density, serotonin dysregulation, and retinal disease ([Terman et al., 1989](#)). In addition, the dopaminergic system might be involved, and some suggest that a deficiency of dopamine may exist in certain neuroanatomic pathways ([Oren et al., 1994](#)).

## DIFFERENTIAL DIAGNOSIS

Differential diagnosis in SAD includes that of any mood disorder, especially organic mood disorders, dysthymic disorder, cyclothymic disorder, adjustment disorders, substance abuse and dependence, personality disorders, pseudodementia, schizoaffective disorder, and disruptive behavior disorders in pediatric patients. Practitioners also need to consider chronic fatigue syndrome, thyroid disorders, sleep disorders, eating disorders, and depressive disorders related to the menstrual cycle ([Partonen and Partinen, 1994](#)). The evaluation includes a physical examination, neurologic evaluation if indicated, and complete psychiatric history.

## TREATMENT

In general, treatments may include medications, psychotherapy, sleep deprivation, light therapy, dawn simulation, aerobic exercise, and outdoor recreation. Adequate sleep hygiene, dietary modification, and standard interventions for mood disorders are used routinely ([Partonen and Partinen, 1994](#); [Wirz-Justice et al., 1996](#)). The efficacy of psychotherapy in the treatment of SAD has not been studied ([Partonen and Lonquist, 1998](#)). The use of selective serotonin reuptake inhibitors is effective, yet some patients may be reluctant to take medications and elect alternative treatment. Numerous controlled trials support the effectiveness of bright light therapy for the treatment of SAD in adults and patients with atypical depressive symptoms ([Terman et al., 1996](#)). Light therapy also can be used as an adjunct to antidepressant treatment and is efficacious in patients whose symptoms do not meet the full criteria for SAD ([Kasper et al., 1989](#)). Although there are few data in children and adolescents, active treatment with light has been found to be superior to placebo ([Swedo et al., 1997](#)). Phototherapy is now regarded as the treatment of choice, with a response in 1 to 2 weeks, although classic antidepressant therapy is effective as well.

Light therapy can be used at home, in the workplace, during travel, and in the clinic. The treatment occurs with a light “box” placed at eye level approximately 1 meter from the patient for 2 to 6 hours per day with full-spectrum fluorescent light, incandescent light, or cool-white fluorescent light ([Terman et al., 1989](#)). Treatment with a light box is convenient because the patient sits in front of the apparatus on a regular basis and is able to read, study, or work throughout the duration of the exposure to light. Some institutions have separate rooms with permanently installed phototherapy equipment and white walls to maximize reflection ([Partonen and Partinen, 1994](#); [Terman et al., 1989](#)). Treatment usually is well tolerated by patients, with minimal side effects, and the response is more rapid than with antidepressants ([Terman et al., 1996](#)). The efficacy of light therapy has to do with the spectrum of the light, its intensity, and the duration and time of exposure to the light source. These variables are discussed in the following paragraphs.

The wavelength of light, or spectrum, and its relationship to SAD symptoms has been little studied. Light boxes that contain white fluorescent bulbs are common. Most protocols use full-spectrum fluorescent light bulbs that emit small amounts of ultraviolet (UV) light. The amount of UV transmission varies with the source (incandescent bulbs emit less UV light), and the length of exposure to UV light is an important factor because UV light has been reported to produce retinal changes. Full-spectrum light with UV is effective on typical and atypical depressive symptoms, whereas UV-blocked light has been noted to be effective only on atypical symptoms ([Lam et al., 1991](#)). Others note no clinical advantage using full-spectrum UV light over light that is UV blocked and suggest that there is no increased clinical efficacy with either source of transmission ([Lam et al., 1992b](#); [Partonen and Partinen, 1994](#)). UV light usually is not necessary in the treatment of SAD and may be blocked during treatment as a precaution in patients with concerns regarding retinal status. Shorter to medium wavelengths of light (blue, green, and yellow) have been shown in some studies to be more effective than longer-wavelength white light ([Lee et al., 1997](#)). White, broad-spectrum light, however, usually is used for a robust clinical response.

The intensity and duration of light exposure are important factors in phototherapy. Bright light usually is more effective than dim light, and intensity has a direct relationship to the response of SAD symptoms. As the intensity of light increases, the classic symptoms of depression, as well as atypical symptoms, respond with increasing frequency. However, atypical symptoms have been noted in trials to respond less aggressively to an increase in the intensity of light ([Lee and Chan, 1999](#); [Levitt et al., 1996](#)). Bright light of 2,500 to 10,000 lux has been found in most studies to be effective as first-line treatment, and removal of the light therapy results in relapse within a 2- to 4-day period ([Rosenthal et al., 1985](#)). Treatment with 2,500 lux for 2 hours each morning for 1 week yields a 67% response rate, and if the patient is treated with 10,000 lux for 30 minutes each morning for 1 week, the response rate increases to 75% to 80% ([Avery et al., 1992](#); [Partonen and Lonqvist, 1998](#)). By the middle of 1987, 24 trials found that bright light of 2,500 lux was more effective than dim light ([Blehar and Rosenthal, 1989](#)). Most researchers recommend 10,000-lux white fluorescent light from a light box for 30 minutes each day for 2 weeks ([Terman and Terman, 1999](#)). A more intense light treatment requires less time of exposure at any time of day, and after 1 to 2 weeks of treatment, sessions can be decreased to 5 days per week. Two hours per day of treatment

with low-intensity light is difficult for some patients, and studies note that a 30- or 40-minute duration of very intense light is as efficacious and improves compliance (Avery et al., 1992; Terman et al., 1989).

The timing of light treatment continues to be a topic of debate, although most studies suggest that morning treatment is more effective than evening (Rosenthal et al., 1985). However, other studies have not reproduced the clear efficacy of morning therapy over other dosing times (Lee et al., 1997; Meesters et al., 1991, 1993b; Schwartz et al., 1996). Dawn simulation (exposing a sleeping individual to light) also has been reported to be effective and has been used in adult and pediatric patients with SAD but is associated with less immediate improvement compared with light box treatment (Avery et al., 1992; Lingjaerde et al., 1998; Meesters, 1998).

The mechanism of light treatment is unclear. Light is thought to effect many biological functions through a variety of anatomic pathways that are involved in circadian bodily rhythms. Bright light is received by the retina and, through the retinohypothalamic tract and the suprachiasmatic nucleus, effects melatonin production in the pineal gland. The duration of melatonin secretion appears to be more important than the amount of melatonin secreted (Rosenthal et al., 1985), and extending the photoperiod with light therapy can change the duration of melatonin secretion (Rosenthal et al., 1984). In general, the greater amount of light in the summer months correlates with increased duration of melatonin secretion, and decreased duration in the winter months correlates with a shorter photoperiod (Wehr et al., 1986). The change in hormonal secretion due to the extension of the photoperiod in patients with SAD alters the phase-response curve and advances circadian rhythms. Light treatment also may decrease urinary output of norepinephrine and its metabolites (Anderson et al., 1992). The effects of light therapy are presumed to be mediated primarily by the eyes, yet little work has been done on how light affects the skin and its production of hormones.

Common side effects of light therapy include irritability, headache, nausea, eyestrain, insomnia, hyperactivity, and "feeling wired." These side effects occur in the beginning of treatment and reportedly increase with the intensity of light used, although other studies are inconclusive. Light-induced mania is rare, as is worsening of depressive symptoms. Side effects may be worse with head-mounted units or light visors, although there is a lack of clinically significant side effects or ocular changes (Terman and Terman, 1999). Short-term side effects are self-limited and there are no known long-term effects (Kogan and Guilford, 1998; Levitt et al., 1993).

Light therapy has been noted in general to be efficacious in children and adolescents in the limited studies available (Sourander et al., 1999; Rosenthal et al., 1986), and treatment protocols are analogous to those in adults. Studies have shown that children respond to light therapy at lower intensities and shorter exposures. SAD symptoms decrease with light treatment, compliance with light therapy is high, and side effects are fewer in the pediatric population (Giedd et al., 1998).

Patients may complain of the inconvenience of sitting in front of boxes routinely, and thus it is important to recommend that patients work the treatment into their normal and daily schedule. Some begin treatment prophylactically and purchase units themselves because there is a low rate of reimbursement by insurance companies despite documented efficacy (Oren et al., 1991b). Predictors of response to treatment include hypersomnia, hyperphagia, and atypical depressive symptoms (Lam, 1994; Lam et al., 1992a; Oren et al., 1992). Comorbid anxiety disorders are associated with a positive response, whereas the response in patients with concomitant personality disorders is not as impressive. The efficacy of light treatment is stable over time, with patients continuing to respond well with subsequent yearly treatments. Most patients continue to do well, experience few side effects, and have a decreased risk for MDD (Oren et al., 1992).

## FUTURE DIRECTIONS

The definition of SAD as a distinct entity, rather than a specifier for recurrent major depression, needs to be reviewed. The prevalence, diagnostic norms, and criteria in children and adolescents require further study. The etiology of SAD and, specifically, biological markers and hormonal mechanisms involved in the disorder, provide ample opportunities for study as treatments, such as light therapy, continue to spark scientific interest. Long-term outcome studies would be beneficial to ascertain whether patients continue to improve over time, and work is needed to compare the efficacy of light treatment with other treatment modalities. Finally, the use of natural, outdoor light in lieu of light therapy and the effect of light spectrum in the treatment of patients with SAD are of great interest.

## RESOURCES

There are many manufacturers now offering a variety of light appliances for personal and institutional use. Companies can be found on the Internet using "seasonal affective disorder," "light therapy," or "light box production" as search engine key words.

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# 65 SUICIDAL BEHAVIOR IN CHILDREN AND ADOLESCENTS: CAUSES AND MANAGEMENT

Cynthia R. Pfeffer, M.D

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At the beginning of the new millennium, suicidal behavior in children and adolescents continues to be an important mental health problem in the United States and worldwide. Recognition of the high degrees of psychiatric and social morbidities associated with suicidal acts among youth has stimulated extensive development of empirical investigations to identify risk factors for youth suicidal behavior. Notably, in 1999, the Surgeon General of the United States published guidelines for a major national initiative to prevent suicidal behavior among youth ( [U.S. Public Health Service, 1999](#)). The 15 major issues identified in this initiative were classified as awareness, intervention, and methodology. Awareness that suicide is a public health problem for which reduction of stigma and enrichment of community resources for suicide prevention was emphasized. The recommendations included development of a National Strategy for Suicide Prevention involving public and private resources that will improve the recognition and treatment of suicide risk by primary care providers, eliminate barriers in insurance coverage for treatments, educate families about suicide risk, as well as implement effective crisis and peer support programs in schools to eliminate youth distress, use schools and workplaces as referral and access points, and provide support to those who lose a loved one to suicide. Advances of the science of suicide prevention were suggested as occurring through research to identify risk factors, evaluation of suicide prevention interventions, promotion of interagency collaboration to improve monitoring for suicidal behavior, and development of new suicide prevention techniques through reduction of access to lethal suicide methods.

The Surgeon General's report built on the aggressive impetus to eradicate youth suicidal behavior that began in the 1980s with the initiation of innovative research methodologies involving epidemiologic, cross-sectional, psychological autopsy, and longitudinal approaches. Furthermore, numerous scientific papers published in the 1990s have educated health care professionals and the community about the characteristics of this profound mental health problem. This chapter provides an overview of suicidal behavior among children and adolescents that is derived from the information gleaned from these studies.

The concept of suicidal behavior in children and adolescents includes thoughts about causing intentional self-injury or death (suicidal ideas) and acts that cause intentional self-injury (suicide attempt) or death (suicide). Based on research suggesting that the severity of depression, death preoccupations, and general psychopathology are directly proportional to the severity of suicidal behavior, researchers concluded that suicidal behavior among children and adolescents involves a continuum from nonsuicidal behavior to suicidal ideas, suicide attempts, and suicide ( [Brent et al., 1988](#); [Pfeffer, 1986](#)). The complexity of these concepts requires more research, especially with respect to potential differences in various phases of child and adolescent development. The chapter focuses predominantly on fatal and nonfatal suicidal acts, such as suicide and suicide attempts.

## HISTORICAL NOTE

In the early 19th century, when Goethe's classic *The Sorrows of Young Werther* was published, an epidemic of youth suicide occurred. This epidemic was attributed to imitation of the book's hero, who shot himself after the breakup of a love relationship. Subsequently, the book was banned in Europe. In 1910, the Vienna Psychoanalytic Society convened a special meeting to evaluate risk factors for youth suicidal behavior ( [Pfeffer, 1986](#)). This historic meeting was organized because of concern about the significant rise in youth suicide and the anxiety that ensued about how to prevent suicide. Among the participants at this meeting were Drs. Federn, Freud, Rank, Steckel, and Tausk. Sigmund Freud, for example, was concerned that the most significant influence on youth suicide was conflict with loved persons. He proposed that intensive study of specific suicidal individuals would elucidate dynamic aspects of childhood suicidal behavior. Others offered suggestions about how to decrease stresses, such as school pressure, that may enhance risk of youth suicidal behavior. Many advocated the need to develop systematic techniques to study youth suicidal behavior.

A rapid rise in suicide among male 15- to 24-year-olds in the United States began in the late 1960s, peaked in 1977, and has decreased in the last few years. Clusters of youth suicide were recognized in the 1980s. As a result, extensive public attention was directed toward developing suicide prevention methods, particularly in schools. Subsequent research of such school suicide prevention programs redirected the thinking about the most effective and safe strategies for such suicide prevention efforts ( [Shaffer et al., 1991](#)). The importance of developing well conceptualized approaches to decreasing youth suicide was highlighted in the 1986 Health and Human Services–National Institute of Mental Health Task Force Conference on Youth Suicidal Behavior. Participants at these conferences included international research experts on youth suicidal behavior. Recommendations made at the close of these conferences ( [Alcohol, Drug Abuse, and Mental Health Administration, 1989](#)) included the need to:

1. Define suicide and report suicide in national and local databases more consistently.
2. Develop research to identify the multifaceted elements of youth suicidal behavior.
3. Evaluate the efficacy of treatments for suicidal youth and those at risk.
4. Support and plan appropriate suicide prevention methods.
5. Educate those providing health care about identification, treatment, and prevention of youth suicidal behavior.
6. Collaborate in the public and private sectors to prevent youth suicide.

National attention to this major mental health problem was stimulated by the publication in 2000 of the Surgeon General's guidelines to prevent youth suicide ( [U.S. Public Health Service, 1999](#)).

## EPIDEMIOLOGY

The most recent year for complete death records of the United States Vital Statistics is 1998 ( [Murphy, 2000](#)). The age-adjusted rate of suicide for 15- to 24-year-olds was 11.1 per 100,000, a rate less than the rate of 11.3 per 100,000 for all ages. It also is less than the rates of 11.4 per 100,000 in 1997 and 12.4 per 100,000 in 1979 for 15- to 24-year-olds. The age-adjusted suicide rate for 15- to 24-year-olds is in marked contrast to the age-adjusted suicide rate of 0.8 per 100,000 for 5- to 14-year-olds. Notably, the rates for 5- to 14-year-olds have doubled since 1979. Suicide among 15- to 24-year-olds is the third leading cause of death and in 5- to 14-year-olds, it is the sixth leading cause of death. In 1998, among 15- to 24-year-olds, there were 4,135 suicides. In the same year, among the 5- to 14-year-olds, there were 324 suicides.

Suicide rates in the United States in 1998 are highest among white males of all ages. The age-adjusted suicide rates for white males are followed by those for nonwhite males, white females, and nonwhite females. In 1998, age-adjusted male suicide rates per 100,000 among 15- to 24-year-olds involve the following: whites, 19.3; blacks, 15.0; Hispanics, 13.4. These rates are higher than the female rates. In 1998, the 15- to 24-year-old age-adjusted female suicide rates per 100,000 were



as follows: white, 3.5; Hispanics, 2.8; and blacks, 2.2.

Suicide caused by firearms accounted for 56.7% of all firearm injury deaths in 1998. Approximately 12% of all firearm deaths were in youths younger than 20 years of age.

Birth cohort effects have been identified for youth suicide in several countries ( [Klerman, 1988](#)). A birth cohort is defined as a group of people born within a specific period. Reports from Canada, the United States, and Australia suggest that each successive birth cohort has a higher youth suicide rate than previous birth cohorts. In addition, these reports suggest that suicide rates increase as individuals in a cohort age.

Period effects also have been identified for youth suicide rates. Period effects represent trends that are associated with phenomena occurring within specific periods. Notably, there has been a marked increase in suicide rates of 15- to 24-year-olds, specifically among individuals born after World War II ( [Klerman, 1988](#)). These individuals became known as the baby boom cohort. Other psychological problems, such as violence, substance abuse, and severe depression, also were significantly higher in these individuals.

Reliable national data for suicide attempts do not exist because there is no national registry for suicide attempts. However, the epidemiology of suicide attempts among children and adolescents is gradually being elucidated. It is estimated that approximately 8% to 10% of adolescents report a suicide attempt at least once in their lifetime. Approximately 1% of preadolescents in the general community report suicide attempts ( [Pfeffer et al., 1986](#)). The ratio of attempted suicide to suicide in children and adolescents has been estimated to be 50:1 ( [Andrus et al., 1991](#)).

The rates of suicide and suicide attempts for community samples are significantly lower than those found among psychiatric patients. For example, approximately one-third of preadolescent and young adolescent psychiatric inpatients attempted suicide before hospitalization ( [Pfeffer, 1986](#)). Suicide among children and adolescents who had a history of psychiatric hospitalization occurs approximately nine times more often than among children and adolescents in the community ( [Kuperman et al., 1988](#)).

## CLINICAL DESCRIPTION

Suicidal ideation and acts are episodic events that can be clearly defined and characterized to have a discrete onset and duration ( [Pfeffer, 1986](#)). Intent to cause harm to oneself is an essential ingredient in defining suicidal behavior. Suicidal intent may be explicit and strong or it may be ambiguous. Evaluating intentionality often is a difficult clinical task, especially among preadolescents. For example, a 9-year-old boy, who was seriously despondent after his dog died, threatened to stab himself with a knife during an argument with his mother. He denied that he had thoughts of wanting to kill himself but stated that he wanted to upset his mother. In this case, the intent was not clear, but the overt behavior was potentially life threatening. In contrast, a 15-year-old girl ingested 127 aspirin tablets after she broke up with her boyfriend. She wanted to kill herself because she felt she “had nothing to live for.” In this case, suicidal intent was clearly stated.

Because intentionality often is difficult to define in children and adolescents, it is helpful for a clinician to consider that self-injurious acts in children and adolescents are potentially suicidal. This enhances the possibility that a clinician will more readily plan appropriate life-sparing interventions.

Suicidal behavior involves a wish to cause death. However, understanding that death is final is not an essential ingredient in considering a child or adolescent as suicidal. Concepts about death develop in parallel with children's development ( [Pfeffer, 1986](#)). Although appreciation of the finality of death may not occur until adolescence, some suicidal adolescents do not have mature concepts of death. Furthermore, children's concepts of death may vary. For example, a 7-year-old may understand that because his pet bird has died, it will no longer be alive. However, this youngster may not understand that if he dies he will never be alive again. Fluctuations in a child's understanding of death also may occur. A child may realize that death is final at one time but when severely stressed—for example, by the divorce and arguments of his parents—such a child may believe that death is reversible. Using these concepts, it is evident that very young children, such as preschoolers who do not appreciate the finality of death, can be considered to be suicidal if they wish to carry out a self-destructive act with the goal of causing death.

Children and adolescents plan and carry out suicidal acts using a variety of potentially lethal methods. These include shooting, hanging, ingestion, suffocation, stabbing, running into traffic, burning, and drowning. In the last few decades, firearms have been the most common means of youth suicide ( [Brent et al., 1987](#)). Furthermore, young women attempt suicide more frequently than do young men. Women predominantly use methods that involve ingestion of lethal substances to attempt suicide, whereas men use firearms to commit suicide. The differences in suicidal methods used by men and women may account for why suicide is higher among men.

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### CASE ILLUSTRATION

The following case exemplifies some of the characteristics of a teenage suicidal episode. Sam, a 16-year-old boy, attempted to hang himself in his bedroom but was discovered by his father, who untied the noose. Sam left an extensive note on his desk describing how depressed he felt in the past few months. He had failed two school midterm examinations and was not accepted on the varsity swim team. He was very disappointed about these events. In addition, he had arguments with his girlfriend, who wanted to date other boys. The night of his suicide attempt, Sam attended a party and drank excessively. He became loud and argumentative. His girlfriend reprimanded him about his behavior. She told him that she was ashamed to be with him and returned a bracelet he had given her for her birthday. Sam was in despair about the tensions with his girlfriend. He returned home A.M. and wrote a long note explaining why he wanted to kill himself. After he completed the note, he tried to fall asleep but continued to be very upset. Gradually, he formulated a plan to hang himself on Sunday morning while his family was at church. However, that morning Sam's father did not go to church and discovered Sam in the process of attempting suicide.

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### CASE ILLUSTRATION

The following vignette highlights suicidal behavior in a preadolescent. Susan was 9 years of age when she took a dose of her mother's antidepressant medication. That day she was with her father, who was drinking heavily. He became angry because she was playing the radio too loudly. He hit Susan with a belt and demanded that she stay in her room. She felt devastated by the harsh discipline of her father and thought that it would be best for her to die. She remembered that she had heard her mother talk about wanting to kill herself, especially after arguments with her father. Susan knew that her mother was depressed and identified with her mother's sadness. She quietly left her room, hoping that her father would not hear her, and went to the medicine cabinet where her mother's medication was kept. She took the 10 pills that were in the bottle and returned to her room. Approximately 1 hour later, her mother returned. She was told by her husband how poorly Susan had behaved and how he had sent her to her room. Susan's mother felt concerned and went to speak with Susan. She found Susan in her bed and very drowsy. After attempts to arouse Susan, her mother called the pediatrician, who advised bringing Susan to the emergency department. On the way to the hospital, Susan told her mother that she had taken her mother's medication and felt that she wanted to die. She was admitted to the pediatric intensive care unit for treatment of the overdose.

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Both illustrations depict serious suicide attempts in children and adolescents. Both youngsters wanted to die and carried out their planned suicide attempts after experiencing intense stresses, depression, anger, rejection, and criticism. Each youngster could have died if he or she had not been discovered in the process of carrying out the act.

## ETIOLOGY AND PATHOGENESIS

Suicidal behavior is a complex symptom that is influenced by sociocultural factors and by the presence of psychiatric symptoms and psychiatric disorders, stressful life events, and problems in social adjustment. To conceptualize better the components of youth suicidal behavior, a multiaxial approach, similar to that of the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) classification for psychiatric disorders, may be used as a model of integrating the multifocal attributes associated with suicidal behavior in children and adolescents. Notwithstanding the fact that suicidal behavior is a symptom and not a psychiatric disorder, the factors involved with the incidence and prevalence of youth suicidal behavior can be outlined in accordance with five axes: (a) primary psychiatric disorders, (b) developmental and personality disorders, (c) biological factors, (d) stress, and (e) social functioning.

An important concern in understanding risk for youth suicidal behavior is that there are few studies of representative community samples of children and adolescents that evaluate the varied manifestations of suicide attempts. Most comparative studies of youth suicide and suicide attempts use psychiatric patients who attempted suicide. As a result, distinctions between suicide and attempted suicide have not been definitively identified. Rather, most studies suggest similarities between youth suicide and suicide attempts.

### Primary Psychiatric Disorders

Studies using psychological autopsy methodology to examine factors associated with youth suicide and cross-sectional research methods with children and adolescents who report suicidal ideation and acts validate the significant correlations of suicidal behavior with mood disorders and disruptive disorders among prepubertal children and adolescents, and the significant relationship of alcohol and substance abuse disorders with suicidal acts in adolescents. The San Diego

Suicide Study (Fowler et al., 1986; Rich et al., 1986) highlighted the comparison of 150 people who committed suicide when older than 30 years of age and 133 who committed suicide when younger. The youth suicide victims had a higher prevalence of drug abuse, noted by the finding that 53% of the younger suicides abused substances. Substance abuse was chronic and prevalent for approximately 9 years among these younger suicide victims. The most frequently abused substances were alcohol, marijuana, and cocaine. The younger suicide victims had a lower prevalence of mood and organic mental disorders. Another psychological autopsy report of 140 adolescent suicide victims and 131 community age- and sex-matched control subjects suggested that psychopathology, especially substance abuse, regardless of whether it was comorbid with mood disorder, distinguished risk for suicide among older versus younger adolescents (Brent et al., 1999). Specifically, this study indicated that adolescents who committed suicide when older than 16 years, compared with those who were younger than 16 years when they committed suicide, had higher prevalence of psychopathology, specifically substance abuse, and higher suicidal intent. This study could not identify why men committed suicide at significantly greater rates than women.

The New York Psychological Autopsy Study (Shaffer, 1988) described psychiatric factors associated with sex differences for 173 youth suicide victims. Using odds ratios, a statistic that defines the likelihood that a given factor will have a specific effect to increase risk for suicide, this study reported that among young men, a history of a suicide attempt (odds ratio = 22.5), presence of major depression (odds ratio = 8.6), and presence of substance abuse (odds ratio = 7.1) were the strongest correlates for adolescent suicide. The strongest correlates for female adolescent suicide were presence of major depression (odds ratio = 49.0) and a history of a suicide attempt (odds ratio = 8.6).

A case-control study (Brent et al., 1993a) comparing 67 adolescents who committed suicide with 67 demographically matched adolescents in the community reported that the presence of major depressive disorder increased risk for suicide by 27 times. Risk was increased nine times by presence of bipolar disorder, 8.5 times by presence of substance abuse, and six times by presence of conduct disorder. Most (82%) of the suicide victims had a mood disorder, and it was notable that when substance abuse coexisted with a mood disorder, there was a 17 times greater risk for suicide. History of previous suicidal thinking or suicide attempts were associated with suicide in this study. Although the suicide rate for young children is low, it has been reported (Hoberman and Garfinkel, 1989) that in a sample of suicide victims involving 21 (3.2%) youth younger than 15 years of age, 208 (31.7%) adolescents, 15 to 19 years of age, and 427 (65.1%) young adults, 20 to 24 years of age, the youngest group had the highest rates of antisocial behavioral, and the adolescents and young adults had higher rates of mood disorders.

Community samples of children and adolescents who attempted suicide have not been sufficiently studied and compared with those who committed suicide. One report of 302 adolescents in the community indicated that of those who made medically serious suicide attempts, approximately 16% had a diagnosis of cannabis abuse or dependence at the time of the suicide attempt, compared with approximately 2% of nonsuicidal adolescents (Beautrais et al., 1999). Other psychiatric disorders comorbid with cannabis abuse or dependence were significant risk factors for suicide attempts. A population-based study of 630 adolescents indicated that for boys, but not for girls, sexual orientation was associated with suicidal intent and suicide attempts (Remafedi et al., 1998). Bisexual and homosexual adolescents had higher risk for suicide attempts. Using data from 1,007 young adults followed for 21 years in the New Zealand Birth Cohort Study, the 2.8% who were classified as being gay, lesbian, or bisexual exhibited greater symptoms of depression, generalized anxiety, conduct disorder, suicidal ideation, and suicide attempts than the other young adults in this longitudinal study (Fergusson et al., 1999). This developmental study clearly suggested that gay, lesbian, and bisexual youth have more extensive psychiatric problems than those without these sexual orientations.

Although studies of children and adolescents who report nonfatal suicidal behavior suggest that mood disorders, particularly major depressive disorder, are significant correlates of suicidal behavior, clear differences between suicidal depressed youth and nonsuicidal depressed youth have not been identified (De-Wilde et al., 1993). In an effort to identify which aspects of major depressive disorder were associated with suicidal behavior, a study of 64 adolescent psychiatric inpatients reported that suicidal ideation or acts were significantly associated with severity of depressed mood, intensity of negative self-evaluation, increased level of hopelessness, poor concentration, and high levels of anhedonia (Robbins and Alessi, 1985). Furthermore, the number of suicide attempts reported was related to intensity of depressed mood, presence of alcohol abuse, heightened negative self-evaluation, and presence of substance abuse. The seriousness of suicidal intent was documented to be associated with increased degree of depressed mood and elevated degrees of negative self-evaluation. Finally, the lethality of suicide attempts was found to be correlated with increased level of depressed mood, elevated negative self-evaluation, intense states of anhedonia, presence of psychomotor agitation, and presence of alcohol or substance abuse.

The findings from another study of 200 adolescent psychiatric inpatients supported the significant correlations of mood disorders and alcohol abuse disorder with suicidal acts. Specifically, this study identified that adolescent boys who had a history of a suicide attempt and alcohol abuse were most likely to report a recent suicide attempt (Pfeffer et al., 1988). In contrast, adolescent girls who were most likely to report a recent suicide attempt had histories of a mood disorder, alcohol abuse, and aggressive behavior. Notably, among adolescent psychiatric inpatients, symptoms of depression and hopelessness accounted for a significant risk of suicide attempts (Dori and Overholser, 1999).

Research with psychiatric patients and youth in the community provided validating information about the significant association of mood disorders and youth suicidal behavior. For example, among child and adolescent psychiatric outpatients with a diagnosis of major depressive disorder, more than 70% reported suicidal ideation or attempts (Myers et al., 1991). Although these suicidal tendencies were recurrent, they did not increase in severity over a 3-year follow-up. Future suicidal tendencies were best predicted by irritability or anger, past history of suicidal thinking or behavior, and older age. Another study used a two-stage approach with self-report surveys and research interviews of 1,542 adolescents, 12 to 14 years of age, who were living in the community (Garrison et al., 1991). Prevalence of moderate to severe suicidal ideation was 4% in boys and 8.7% in girls. Prevalence of suicide attempts was 1.9% in boys and 1.5% in girls. Presence of major depressive disorder imparted an almost seven times greater risk for suicidal ideation and an almost 10 times greater risk for suicide attempts. Another community study interviewed 1,710 older adolescents and reported the significant relation of depression to suicide attempts (Lewinsohn et al., 1993). Furthermore, when the effects of depression were controlled, the greater the number of other risk factors, such as externalizing and internalizing behaviors, lifetime history of psychiatric disorders, school problems, health problems, and gender, the greater the likelihood of reporting a suicide attempt. Aggression, substance abuse, and cigarette smoking were high-risk behaviors noted in community samples. Most studies of community samples use adolescents who attended school. In contrast, when 576 runaway youths were interviewed, 37% reported a history of a suicide attempt (Rotheram-Borus, 1993). Although girls were more likely to have attempted suicide, depression was significantly associated with suicide attempts in boys and girls. These reports substantiate the effects of similar risk factors for suicidal behavior in community and psychiatric patient samples.

Follow-up investigations are important for identifying risk factors for suicidal behavior. In a 5-year follow-up study of 180 consecutively psychiatrically discharged adolescent inpatients, it was observed that approximately 25% of the youths attempted suicide and no one committed suicide in the follow-up period (Goldston et al., 1999). This study pointed out that the first year after discharge was the period of highest risk. The strongest predictor of a suicide attempt was the number of prior suicide attempts. Mood disorder alone was not a predictor of a posthospitalization suicide attempt, but in conjunction with a history of a suicide attempt, mood disorder predicted a future suicide attempt.

### Developmental and Personality Disorders

Although there is evidence that youth with specific developmental disorders that impair their learning skills are at heightened risk for suicidal behavior, research suggests that IQ is not a predictor of youth suicidal behavior. With attention focused on understanding the effects of comorbid psychiatric disorders, it has become apparent that elements related to personality disorders may be significant correlates of youth suicidal behavior. For example, psychiatric symptoms such as impulsivity and intense aggression, which may be indicators for the future development of adult personality disorders such as narcissistic, antisocial, and histrionic personality disorders, have been reported to be positively associated with suicidal behavior in prepubertal children and adolescents. This is illustrated by a psychological autopsy study conducted in Finland (Marttunen et al., 1991) of 53 adolescents who were 13 to 19 years of age when they committed suicide, which highlighted the prevalence of personality disorders. It was reported that 94% had a mental disorder. The most prevalent disorders were mood disorder (51%), alcohol abuse (26%), and personality disorder (32%), especially antisocial personality disorder. A reanalysis of the San Diego Suicide Study suggested strong evidence for high prevalence of comorbidity with depression, substance abuse, and antisocial personality disorder (Rich and Runeson, 1992). Furthermore, 41% of the 133 adolescents in the San Diego Suicide Study had symptoms of a borderline personality disorder. The investigators raise the important question of whether Axis I and Axis II psychiatric disorders are independent in relation to youth suicide.

A report of 453 consecutive Israeli male suicides, 18 to 21 years of age, suggested that an appreciable number (18.6%) had no DSM-III-R Axis I disorders and that approximately 9% had no DSM-III-R Axis II psychopathology (Apter et al., 1993). The most prevalent diagnosis (53.5%) among these suicide victims was major depressive disorder. Notable was the finding that narcissistic or schizoid traits were quite prevalent among youth suicide victims. This report also suggested the importance of focusing on aspects of personality in understanding youth suicidal behavior.

One of the first reports to identify an association between personality disorder and adolescent suicide attempts noted that among 53 adolescent psychiatric inpatients, those with borderline personality disorder had the most frequent and severe suicide attempts (Friedman et al., 1983). In another report, impulsivity and aggression were noted to be associated with suicidal behavior among adolescent psychiatric inpatients (Apter et al., 1998). Higher scores on the subscale of the Kiddie Schedule



for Affective Disorders and Schizophrenia that indicate degree of suicidal tendencies were found for adolescents with conduct disorder than for adolescents with major depressive disorder. It is possible that features of conduct disorder that may predict adult personality disorder may be important links to youth suicidal behavior. Another study reported that adolescent psychiatric inpatients with borderline personality disorder had significant rates of suicidal behavior, specifically among the girls but not among the boys ([Pfeffer et al., 1988](#)).

A study evaluated the prevalence and severity of assaultive behavior, impulsivity, and personality disorders in 37 adolescent psychiatric inpatients who attempted suicide and adolescent psychiatric inpatients without a history of suicidal behavior ([Brent et al., 1993a](#)). Significantly more suicide attempters than nonsuicidal adolescents had symptoms of personality disorder, especially borderline personality disorder. However, no discernible differences were found in prevalence or severity of assaultiveness or impulsivity among the two groups of adolescents.

Although reports of prepubertal suicidal children have not focused on disturbances in personality traits, subgroups of suicidal children have been identified. For example, prepubertal children who report both suicidal and violent behaviors had significant deficits in impulse control ([Pfeffer, 1986](#)). Features of violence, suicidal behavior, and impulsivity may be ingredients for future development of personality disorders. In contrast, a group of prepubertal children were described who were suicidal and not violent. These children were predominantly depressed and had good impulse control. The relationship between indicators of personality disorders and suicidal behavior remains to be identified in this subgroup of suicidal children.

## Biological Factors

Elucidation of biological correlates of suicidal behavior in prepubertal children and adolescents is emerging and appears, in general, to concur with features described for suicidal adults. Positive correlations in adults between suicidal and violent behaviors, impulsivity, and aberrations in neurotransmitter systems, especially those involved with serotonin metabolism and regulation, have been described. [Asberg \(1976\)](#) was the first to report low levels of 5-hydroxyindoleacetic acid (5-HIAA) in cerebrospinal fluid (CSF) of violent suicidal adults. Suicide occurred more frequently among those individuals than among those with higher levels of CSF 5-HIAA. These results were validated in numerous other studies suggesting associations between aberrant serotonin system functioning and  $\alpha_2$ -adrenoceptors in postmortem brain samples, especially frontocortical regions, and low levels of 5-HIAA in CSF samples of suicidal adults. In addition, some studies of suicidal adults report lower levels of homovanillic acid, a dopamine neurotransmitter metabolite, in the CSF of suicidal compared with nonsuicidal adults. Following evidence that brain regions of depressed suicide victims have an altered density of the serotonin 2A (5-HT<sub>2A</sub>) receptors, one study evaluated the allele and genotype frequencies of the 102T/C polymorphism of this serotonin receptor ([Du et al., 2000](#)). This study suggested a significant association between suicidal ideation in adults with major depressive disorder and the 102C allele in the 5-HT<sub>2A</sub> receptor gene.

Research using similar complex methods to study youth is underway. At the 1995 annual meeting of the Society for Biological Psychiatry, Greenhill reported preliminary results of a study of suicidal adolescent psychiatric inpatients with a diagnosis of conduct disorder that found low levels of CSF 5-HIAA in these adolescents. A report of peripheral plasma markers suggested that platelet imipramine binding may be lower in depressed prepubertal children and adolescents with a history of suicidal behavior than among youth with no history of suicidal behavior ([Ambrosini et al., 1992](#)).

Studies of other peripheral markers have suggested significant correlations with suicidal behavior. [Pfeffer and colleagues \(1998a\)](#) were among the first to report the relationship between suicidal behavior in prepubertal children and peripheral serotonin markers. This study of 75 prepubertal psychiatric inpatients and 35 prepubertal, nonsuicidal, community control subjects indicated that whole-blood tryptophan content was significantly lower among inpatient children with a recent suicide attempt than among the nonsuicidal children or the inpatients with suicidal ideation. Furthermore, psychiatric inpatient children with a mood disorder had a significantly higher platelet serotonin content than inpatients without a mood disorder. Another study of nine adolescent psychiatric inpatients with a history of repeated suicide attempts and 10 adolescent psychiatric inpatients without suicide attempts indicated that the suicide attempter adolescents had significantly lower platelet benzodiazepine receptor densities than the non-suicide attempter adolescents ([Soreni et al., 1999](#)). This result was similar to that in adults with generalized anxiety disorder and posttraumatic stress disorder.

Neuroendocrine functioning, especially hypothalamic–pituitary–adrenal (HPA) axis functioning, which is a system associated with stress responses, has been studied regarding associations with suicidal behavior. Elevated levels of plasma cortisol were identified as associated with suicidal behavior in adults. Some reports for youth indicate a possible role for excessive HPA function among suicidal youth. For example, it was reported that an elevated level of plasma cortisol during the dexamethasone suppression test occurred among suicidal compared with nonsuicidal prepubertal child psychiatric inpatients ([Pfeffer et al., 1991b](#)). It also was reported that adolescents who reported suicide attempts during an episode of major depressive disorder had elevated levels of plasma cortisol before sleep onset ([Dahl et al., 1990](#)). This was not observed for adolescents with major depressive disorder who did not have a history of a recent suicide attempt. The role of HPA activity, especially as a potential indicator of response to stress, in the risk of youth suicidal behavior requires additional research.

Approaches using chronobiological methods have yielded important information on the biological correlates of youth suicidal behavior. For example, unlike prepubertal children, adolescents with major depressive disorder who reported a recent suicide attempt had aberrations in sleep architecture before the onset of sleep, compared with no aberrations among depressed adolescents with no history of suicidal behavior ([Dahl et al., 1990](#)). Follow-up assessments of neuroendocrine functioning in depressed adolescents suggest that sleep-related growth hormone secretion may predict future episodes of major depression and suicide attempts. Specifically, a prospective study of 34 depressed and 43 nondepressed adolescents who were followed up in young adulthood suggested that adolescents with at least one episode of major depression during the follow-up and who exhibited at least one suicide attempt secreted significantly more growth hormone during the first 4 hours of sleep ([Coplan et al., 2000](#)).

## Stress

Extensive research results suggest the significant relationship between youth suicidal behavior and stressful life events. Prepubertal children who reported suicidal ideation or suicide attempts had higher rates of cumulative stressful life events than nonsuicidal prepubertal children. The varied types of stressful events include deaths and separations of relatives, births of siblings, illness, hospitalization, and multiple family moves. Similarly, adolescent suicide attempters, compared with depressed nonsuicidal adolescents and normal adolescents, have more lifetime and recent stressful life events.

Family disruptions and discord stemming from excessive arguments and overt violence, loss of relatives due to marital separations or divorce, and problems in family interpersonal relations due to parental psychiatric illness are important aspects of stressful circumstances experienced by suicidal children and adolescents. A comparative psychological autopsy study reported that adolescent suicidal victims, compared with nonsuicidal adolescents, had more exposure to suicidal behavior among relatives or friends ([Shafiq et al., 1985](#)). For example, 60% of the suicide victims, compared with 12% of the nonsuicidal adolescents, had parents or adult relatives with suicidal tendencies. The parents of these suicide victims had more emotional problems and absence from home, and they abused their children more than did parents of nonsuicidal adolescents. This study highlights the importance of identifying youth who may have psychological problems after the suicidal behavior of a relative.

The New York Psychological Autopsy Study reported that a family history of suicide imparted a fivefold greater risk of suicide on adolescent boys and an almost threefold greater risk for suicide on adolescent girls ([Shaffer, 1988](#)). It also was reported that among adolescents who committed suicide and adolescents who reported suicidal ideation or suicide attempts, there was a significantly high prevalence of mood disorders, antisocial personality disorders, and suicide among relatives ([Brent et al., 1988](#)). In one of the first systematic, controlled family studies of prepubertal suicidal children, [Pfeffer et al. \(1994\)](#) reported high rates of suicidal acts, violence, substance abuse, and antisocial personality disorders among first-degree relatives of prepubertal children who reported suicidal ideation or suicide attempts. Family discord also was marked among these suicidal children. Similar results were obtained in a study that followed these prepubertal children into adolescence ([Pfeffer et al., 1998b](#)). It indicated that adolescents who reported a lifetime history of a suicide attempt had higher rates of family discord, suicide attempts of mothers, and parental substance abuse.

Violence, especially physical and sexual abuse, has been described as a strong risk factor for youth suicidal behavior. A report suggested that 159 adolescent suicide attempters evaluated in an urban hospital emergency service, who were compared with a similar number of nonsuicidal adolescents evaluated in the same emergency service, were three to six times more likely to have had contact with the Department of Social Service Registry for suspicion of physical abuse ([Deykin et al., 1985](#)). The Department of Social Service found the estimated proportion of suicide attempts that could be explained by a history of abuse was 12%. This study lends support to the idea that physical abuse is a significant risk factor for youth suicidal behavior. In a community sample of 776 children and adolescents that was followed for 17 years, it was observed that adolescents and young adults with a history of childhood maltreatment were three times more likely to become depressed or attempt suicide ([Brown et al., 1999](#)). Furthermore, the effects of history of sexual abuse conferred an eightfold greater risk for repeated suicide attempts in adolescents and young adults. Among 127 adolescent psychiatric inpatients, a history of childhood abuse was associated with internalizing and externalizing behaviors in suicidal

patients; however, a greater prevalence of internalizing disorders was identified in suicidal adolescents without a history of childhood abuse ( [Grilo et al., 1999](#)).

The effects on risk for suicidal behavior of severe family adversity during childhood, including economic strain, marital disruption, poor parent–child attachment, and exposure to sexual abuse, were highlighted in a 21-year longitudinal study of a community sample of 1,265 children born in New Zealand ( [Fergusson et al., 2000](#)). The effects of these factors on risk for suicidal behavior in late adolescence and young adulthood were mediated by mental health problems and stressful events in adolescence. The strong effects of child maltreatment were highlighted among 335 African-American women who were psychiatric inpatients ( [Thompson et al., 2000](#)). History of physical or sexual abuse, emotional abuse, and emotional and physical neglect combined with a current diagnosis of posttraumatic stress disorder significantly increased the risk for suicide attempts.

Another facet of family disturbance was reported using a follow-back research design to study the relation between prenatal, birth, and neonatal factors and youth suicidal behavior ( [Salk et al., 1985](#)). Fifty-six factors in the prenatal, birth, and neonatal records of 52 adolescents who committed suicide and records of a similar number of matched nonsuicidal control subjects were studied. Respiratory distress for more than an hour after birth, no antenatal care before 20 weeks of pregnancy, and chronic physical disease of the mother differentiated those who committed suicide from those who did not. An implication of this study is that early stress may enhance vulnerability to suicide, especially among adolescents who have a history of severe stress or psychopathology during adolescence. However, the mechanisms leading to such vulnerability require identification.

## **Social Functioning**

Problems with social adjustment involving interpersonal relations with family, peers, and others are important characteristics of suicidal children and adolescents ( [Pfeffer, 1986](#)). Poor social adjustment among suicidal youth is apparent even after a child or adolescent no longer reports suicidal tendencies. This feature, in large measure, accounts for the long-term vulnerability of children and adolescents to suicidal acts.

Social adjustment in children and adolescents is influenced by presence of adequate social supports and predictability of the environmental milieu, especially regarding relationships with parents, siblings, and other helpful adults and peers. Suicidal children and adolescents frequently lack available empathic individuals who could offer guidance and an avenue for the child or adolescent to vent distressing ideas and feelings. An unpredictable social support network combined with a suicidal youth's perceptions of inadequacies in mobilizing social support intensify the suicidal youngster's sense of isolation, anxiety, poor self-esteem, rejection, and hopelessness. Perceived hopelessness is among the significant factors that intensify suicidal risk for youth. Hopeless feelings frequently impair a suicidal youngster's ability to remain motivated to solve problems and cope with adversity.

Specific styles of coping among suicidal children and adolescents limit their flexibility and resourcefulness in coping with intense emotions and in problem solving ( [Pfeffer, 1986](#); [Schwartz et al., 2000](#)). When prepubertal suicidal children encounter ambiguities or difficulties in interpersonal situations, they lack skills to respond with beneficial actions that solve their dilemmas. Rather, suicidal children and adolescents characteristically respond either impulsively or tend to withdraw into states of paralytic rumination or fantasy. Ego mechanisms of defense, empirically studied among suicidal children and adolescents, involve denial of affects or events ( [Pfeffer, 1986](#)). This may account for observations suggesting that suicidal youths' reality testing may be impaired. Another ego defense mechanism is reaction formation, which operates to modify painful affects and distressing thoughts by turning their perception into perception of the opposite. Compensation, another ego defense mechanism among suicidal children, involves risk-taking behavior or overt exaggeration of one's skills. This defense defuses a youngster's sense of inadequacy. Suicidal adolescents also cope by involving specific ego mechanisms that are similar to those observed for suicidal preadolescents. In general, suicidal adolescents turn feelings against themselves, especially aggressive impulses. They also use aspects of reaction formation. These styles of coping are among the important factors that compromise suicidal children and adolescents in maintaining adequate levels of social relatedness. Combined with this, social functioning of suicidal children and adolescents also is impaired by life in an inadequate or unpredictable social milieu.

The role of cultural affiliation rather than ethnic identity as a risk factor for suicidal behavior is an important issue requiring extensive research. To illustrate this, varying rates of suicide within ethnic groups have been observed. A community study of 3,094 Native Hawaiian adolescents indicated that stronger cultural affiliation rather than ethnicity was significantly predictive of adolescent suicide attempts ( [Yuen et al., 2000](#)). Similar concerns have been observed for African Americans, in whom increased suicide rates may be related to migration, cultural affiliation, and changing social supports.

## **LABORATORY STUDIES**

### **Biological Assessment**

There is no distinct biological test to measure risk for youth suicidal behavior. The most fruitful areas of clinical investigation lay in understanding the relation between alterations in serotonin metabolites and neuroendocrine functions and risk for suicidal behavior. However, practical applications have not been determined.

### **Psychological Assessments**

Assessment of youth suicidal behavior involves comprehensive interviews with a suicidal youngster and the parents. Significant issues for assessment are the presence of current and past suicide intent, which involves the balance between the wish to live and the wish to die. It includes appraisal of past and current suicidal ideation and suicide attempts, accessibility of suicidal methods, and underlying risk factors, such as psychopathology, cognitive distortions (e.g., hopelessness), problematic coping styles involving impulsive behavior, and environmental stress factors. Assessment of a suicide attempt should focus on the type of method used, its potential for lethality, the degree of planning, and the likelihood that discovery of the act is possible. These assessments are complicated, especially when interviewing prepubertal children about their suicidal ideation and acts ( [Jacobsen et al., 1994](#)). Some of the aspects that influence the information obtained in a clinical interview are the child's cognitive and emotional state and the degree of parental psychopathology. It is important that clinical interviews be conducted at the time of maximum suicidal risk and repeated subsequently until the risk of suicidal behavior is diminished. The most important aspect of the assessment is to determine the degree of immediate danger for a youngster. If a youngster's situation is unpredictable, consideration of emergency psychiatric hospitalization is warranted.

Essential to the evaluation is to determine the current psychological status of a youngster with respect to type and degree of psychopathology and degree and quality of coping mechanisms, such as adequacy of judgment, degree of impulse control, intensity of hopelessness and helplessness, and ability to communicate. An important clue for an impending suicide attempt is preoccupation with death ( [Gothelf et al., 1998](#)). A youngster may be at lower risk for suicidal acts if he or she has good judgment, high impulse control; low levels of hopelessness and helplessness; and ability to communicate openly and honestly about feelings, worries, and thoughts of suicide. However, it must be appreciated that the status of these variables may change rapidly, so that repeated, comprehensive discussion with a suicidal youngster is necessary.

Assessment of family and environmental conditions is essential. It is important to determine whether a family can provide a consistent, stable environment or whether there is high degree of stress, violence, and psychopathology, as well as unavailability of relatives. Positive social supports are critical in diminishing suicidal risk among children and adolescents ( [Wagner et al., 2000](#)). If an environment does not provide sufficient beneficial social supports, a potentially suicidal child or adolescent needs to be removed from such an environment and placed in a situation, such as a psychiatric hospital, that promotes psychological growth and deters suicidal tendencies.

Clinical assessment may be aided by the use of specialized rating scales that measure suicidal risk factors. A recently developed, reliable and valid screening measurement is the Child-Adolescent Suicidal Potential Index (CASPI), which involves 30 items answered in a yes-no format ( [Pfeffer et al., 2000a](#)). The total score provides an estimate of suicidal risk. Another method involving nonverbal assessment of children and adolescents is the use of human figure drawings ( [Zalsman et al., 2000](#)). All such screening measures should be used in conjunction with in-person interviews to evaluate if the screening risk assessment is valid.

## **DIFFERENTIAL DIAGNOSIS**

The two most significant issues in a differential diagnosis of youth suicidal behavior are to identify whether the destructive act is self-intended rather than accidental, and to identify whether there is a high or low level of risk that an injury will occur. The first issue is a qualitative issue and involves a systematic assessment of intent and the specific circumstances in which a self-destructive act occurred. The second issue is a quantitative one and involves a comprehensive evaluation of the intensity of and interactions among risk factors. Psychiatric issues to be considered are the severity of depressive symptoms, presence of hypomania or manic symptoms, and mixed or rapid cycling states of manic or depressive symptoms. Other symptoms to consider are the severity of irritability, agitation, delusional symptoms, and other psychotic symptoms, including poor reality testing and hallucinations. These issues often are difficult to define distinctly. It is more helpful, in



situations where a definite conclusion cannot be made, to consider a youngster to be suicidal and at significant risk rather than to minimize the clinical condition. By using this approach, a plan for treatment may be conceptualized and implemented rapidly.

## TREATMENT

The most salient acute issue in treatment is to decrease the likelihood that self-inflicted injury or death could occur. Availability of psychiatric services involving outpatient and emergency services and inpatient or residential programs is important in lowering suicidal risk. Psychiatric hospitalization may be recommended if observation and intensive therapeutic intervention are warranted when a child or adolescent manifests an unpredictable condition. The hospital offers an environment with structure and a consistent, high-availability of staff to provide immediate, around-the-clock interventions. Hospitalization also is a way of removing a youngster from an environment that may be too stressful or disorganized. In contrast, outpatient or partial hospitalization should be used when the child or adolescent is not imminently likely to carry out a suicidal act or if there is sufficient family support to identify and respond to a suicidal crisis.

Emergency treatment involves certain principles that should be consistently followed. A suicidal child or adolescent should not be discharged from an emergency service without verification of the patient's clinical condition with the parent and discussion of making unavailable such lethal methods as guns, firearms, or medications ([Kruesi et al., 1999](#); [McManus, et al., 1997](#)) and limiting access to alcohol or drugs. The family's experience with the emergency service staff will affect compliance with future treatments ([Rotheram-Borus et al., 1996](#)).

Psychotherapeutic intervention involves developing a trusting atmosphere for truthful communication. It is essential that a therapist maintain an empathic but objective concept of a suicidal youngster. A therapist's collusion with the perceptions of despair, misery, or hostility of a suicidal child or adolescent impairs treatment progress. Discussion that may elucidate the operation of new and effective coping strategies is essential. This can be achieved by incorporating a cognitive orientation into the treatment process.

Delineation of the motivations for a suicidal act is an important feature of treatment. Such motivations may be conscious or outside the immediate awareness of a suicidal youth. Common motives for enacting suicidal behavior involve despair over loss of a special person such as a breakup with a boyfriend or girlfriend or separation of parents. These situations often are characterized by intense guilt on the youngster's part, who thinks he or she may have caused the loss. Other motivations involve anger or feelings of revenge in response to frustrations, deprivations, or perceived wrongdoing. Often a youngster impulsively threatens suicide in an effort to instill guilt in those who are closely involved in the youngster's life situation. Another motivation is based on psychotic functioning. Some psychotic youngsters feel so demoralized and in such psychic pain that they wish to relieve themselves of the pain by committing suicide. Such a response may be impulsive and occur without warning.

Treatment of suicidal children and adolescents is complex and often requires simultaneous use of multiple modalities. In addition to dynamic, supportive, and cognitive psychotherapy, psychopharmacologic modalities may be indicated. There are no controlled studies of treatment of suicidal children and adolescents, although initial efforts are now underway to develop and study controlled treatment conditions for factors that increase suicidal risk. For example, cognitive-behavioral therapy for depressed adolescents has been more effective than family or supportive therapy ([Brent et al., 1997](#)). However, 2 years after the treatment, there were no differences in outcomes with cognitive-behavioral, family, or supportive treatment ([Birmaher et al., 2000](#)). Interpersonal therapy, which addresses interpersonal conflicts involving loss, interpersonal role disputes, role transitions, and interpersonal deficits, has been found to be more effective for depressed adolescents than a control treatment ([Mufson et al., 1999](#)). This treatment may be helpful for depressed suicidal adolescents with interpersonal conflicts. However, its efficacy with suicidal adolescents has not been studied. Dialectical-behavioral therapy is the only psychotherapy that has been effective in reducing suicidal behavior in adults ([Linehan, 1993](#)). This treatment requires investigation with suicidal adolescents. Family therapy may be useful to decrease family discord and enhance effective family problem solving and conflict resolution. A home-based family intervention with a time-limited framework was found to have limited efficacy for children and adolescents who did not have a major depressive disorder ([Harrington et al., 1998](#)).

Studies of the efficacy of psychopharmacologic treatment of suicidal children and adolescents are needed. Lithium has been found to decrease by almost ninefold the recurrence of suicide attempts in adults with major depression or bipolar disorder ([Tondo et al., 1997](#)). Discontinuation of lithium was associated with a sevenfold increase in suicide attempts and a ninefold increase in rates of suicide ([Tondo et al., 1997](#)). Use of lithium with suicidal children or adolescents requires careful monitoring by a responsible adult because an overdose may be lethal. Other mood stabilizers also may be effective, but studies to demonstrate this are needed. In addition, antidepressants such as selective serotonin reuptake inhibitors may be promising for treating suicidal children and adolescents. Although the efficacy of fluoxetine, which is one of this class of medications, has been demonstrated in depressed children and adolescents ([Emslie et al., 1997](#)), such medications have not been demonstrated to be effective specifically for suicidal children or adolescents. Use of such medications requires consistent and detailed monitoring for possible activation that may be associated with suicidal risk.

Psychopharmacologic treatment to reduce the risk of underlying conditions associated with suicidal behavior involves use of various medications specific to the diagnosis or symptomatology. Caution in use of medications that may disinhibit some children and adolescents, such as psychostimulants, benzodiazepines, or phenobarbital, is required.

Treatment of parents and other relatives may be indicated, especially if such people have a direct influence on a suicidal youngster, and if they have impairing symptoms of psychopathology that contribute to the adverse family milieu. Parental marriage counseling, individual psychotherapy, or focused psychopharmacotherapy may be required for certain relatives. Detailed assessment and treatment of psychiatric symptoms and disorders of family members is necessary to promote a stable atmosphere for the youngster.

Collaborative efforts with other professionals who are involved with a youngster must be organized. For example, it may be essential to consult with the school psychologist or guidance counselor, who may speak with a youngster during school hours. Collaboration with the school may help stabilize a youngster in the school and community environment.

## APPROACHES FOR PREVENTION OF YOUTH SUICIDAL BEHAVIOR

Although rates of suicide among youth in the United States are high, the development of suicide prevention strategies can be inferred by the results of prospective studies of youth suicidal behavior. For example, it is evident from prospective research that prepubertal children and adolescents who report suicide attempts are at significant risk for recurrent suicidal acts ([Pfeffer et al., 1991a, 1993](#)). Prepubertal psychiatric inpatients who attempted suicide are six times more likely to attempt suicide in adolescence than are children in the general community. Prepubertal psychiatric inpatients who report thoughts of suicide are at a fourfold higher risk for attempting suicide within less than 10 years than children in the general community. Adolescent psychiatric inpatients who attempt suicide are at significant risk for a repeat suicide attempt within 6 months of follow-up ([Brent et al., 1993b](#)), and for suicide in less than 10 years ([Pfeffer, 1986](#)). The data on suicidal children and adolescents also are supported by studies showing that prepubertal children who have a major depressive disorder are at risk for suicide attempts in adolescence ([Kovacs et al., 1993](#)) and suicide in young adulthood ([Rao et al., 1993](#)). Suicide prevention efforts should focus on identifying such high-risk children and adolescents and monitoring them repeatedly for early signs of risk for a suicidal act. An important issue is to develop community case-finding approaches by means of screening procedures and referral of those children and adolescents found to be at risk for suicidal behavior. Educating mental health, medical, and school professionals, clergy, and others who are in contact with children and adolescents to identify children and adolescents at risk for suicidal behavior is an important suicide prevention strategy.

Efforts to prevent youth suicidal behavior require public health model strategies in school settings. These may focus on encouraging adolescents to seek help if they recognize symptoms of suicidal risk. New approaches to this are required because evaluations of prior school programs concluded that their efficacy was not adequate to prevent suicide, change adolescents' attitudes about suicide, or enhance help-seeking behavior. Some reports suggested that adolescents who had a history of suicidal behavior were especially upset and had negative feelings about suicide prevention curricula ([Shaffer et al., 1991](#)).

Clusters of youth suicide, notable during the early 1980s in the United States, became phenomena of intensive research investigation ([Gould and Shaffer, 1986](#), [Hafner and Schmidtke, 1989](#); [Phillips and Carstensen, 1986](#)). It became apparent that factors promoting youth suicide clusters involved imitation of suicidal acts. Clusters were defined as incidents of youth suicide that occurred either in the same defined period or the same defined location. Complex research methods determined that there was a significant increase in adolescent suicide within 2 weeks after media presentations of stories of actual or fictional youth suicide. Guidelines for media coverage have been developed and may be useful in decreasing risk for suicide after media presentations ([Gould and Kramer, 1999](#)).

Another aspect of the problem of imitation of youth suicide involved developing methods for communities to respond to a youth suicidal death so that risk for other

youth suicidal acts is lessened. Guidelines were established that included organizing community networks consisting of school professionals, mental health professionals, police, religious leaders, and parents to be available to respond to the community crises of a youth suicide. Organized liaison approaches with the media are essential to promote helpful media coverage of the event and to prevent confusion, fear, and risk for other youth suicidal acts stimulated by the presence of news reporters and presentations of the story of a youth suicide. Focus on other vulnerable youngsters is another aspect of responding to the crisis of a youth suicide. Discussions with friends and acquaintances of the adolescent who committed suicide are helpful. Research suggests that risk for suicidal behavior may be low among friends and acquaintances of an adolescent suicide victim within 6 months of the suicidal death ( [Brent et al., 1992](#); [Hazell et al., 1993](#)). However, the risk for development of a major depressive disorder and posttraumatic stress disorder is high among such friends and acquaintances.

Suicidal behavior involves a legacy that runs in families. Prepubertal children who report suicidal ideation and suicide attempts have higher rates of suicidal acts among first-degree relatives than nonsuicidal children ( [Pfeffer et al., 1994](#)). Research has identified that psychiatric morbidity is significantly greater for children and adolescents who suffered the suicide of a parent or other close relative ( [Cerel et al., 1999, 2000](#); [Pfeffer et al., 1997, 2000b](#)). Programs are needed to identify such bereaved children and adolescents and to offer them targeted intervention to prevent adverse outcomes resulting from the sequelae of a suicide in the family.

Finally, strong evidence suggests that availability of guns and firearms is significantly associated with youth suicide risk ( [Shah et al., 2000](#)). Intense efforts to prevent youth suicide require a national focus to advocate for better restrictions on the availability of guns and firearms, especially to children and adolescents. Furthermore, clinical assessments of suicidal risk among children and adolescents should incorporate inquiry about the presence of such lethal weapons in the home or whether there are other avenues for access to a gun or firearm. Decreasing such accessibility is an important suicide prevention action.

## CONCLUSION

Knowledge about risk for youth suicidal behavior has expanded. As we enter the new millennium, future efforts to investigate suicidal behavior should use new research methodologies that may lead to identification of the interactions between environmental factors and constitutional and biological factors that are associated with risk for suicidal behavior. Such studies may be relevant for developing effective treatment strategies to lower the risk for youth suicidal behavior. Research is needed to understand sociocultural factors involving risk and protective factors for youth suicidal behavior. Such research, when combined with research that focuses on biological factors or psychiatric phenomenology, may be helpful in reducing rates of suicidal behavior among youth.

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## 66 PREVENTION OF DEPRESSION AND SUICIDE IN CHILDREN AND ADOLESCENTS

Judy Garber, Ph.D. and Elizabeth McCauley, Ph.D.

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[Treatment versus Prevention](#)  
[Prevention Science](#)  
[Risk for Depression](#)  
[Programs for the Prevention of Depression in Children and Adolescents](#)  
[Clarke's Cognitive-Behavioral Group Preventive Intervention](#)  
[A School-Based Cognitive-Behavioral Preventive Intervention](#)  
[A Targeted, Family-Based Cognitive-Educational Prevention Program](#)  
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[Suicide Prevention Programs for Youth](#)  
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Depression is a common disorder with a chronic, episodic course marked by frequent recurrence, considerable impairment, and substantial health care costs (Birmaher et al., 1996a,b). According to a study of global mortality and disability associated with 107 disorders, depression ranked fourth in terms of its disease burden (Murray and Lopez, 1997). Although the prevalence of depression is low in prepubertal children (Fleming and Offord, 1990), the rates increase substantially during adolescence (Hankin et al., 1998). The point prevalence of childhood depression is between 1% to 2%, and adolescent depression is estimated at between 4% to 8%, with the average episode lasting between 6 to 8 months (Fleming and Offord, 1990; Kovacs, 1996; Lewinsohn et al., 1993, 1994a). An estimated 20% of adolescents will have a depressive episode by 18 years of age (Lewinsohn et al., 1993). Depression is recurrent, with as many as 40% of children and adolescents experiencing a second episode within 2 years, and nearly 75% within 5 years (Kovacs et al., 1984a,b; Lewinsohn et al., 1994a). Most will experience another episode in their adult life (Kandel and Davies, 1986; Rao et al., 1995), and most cases of recurrent adult depression had their initial onsets during adolescence (Pine et al., 1998). Finally, depression is associated with substantial impairment both during and after the episode, with difficulties in school, interpersonal relationships, occupational adjustment, tobacco and substance abuse, suicide attempts, and a 30-fold increased risk of completed suicide (Birmaher et al., 1996a; Brent et al., 1988a, 1993; Harrington et al., 1990; Riggs et al., 1995; Rohde et al., 1994). Thus, depression is an important public health problem that should be the focus of prevention efforts early in development.

Several reviews of the prevalence, course, outcome, etiology, and treatment of child and adolescent depression and suicide are available (e.g., Birmaher et al., 1996a, 1986b; Garber and Flynn, 2001; Hammen and Rudolph, 1996; and see Chapter 62, this volume). The purpose of this chapter is to describe programs for preventing depression and suicide that target children and adolescents and to review the empirical studies examining their efficacy. [For reviews of prevention of depression in adults, see Gillham et al. (2000) and Munoz (1993)].

### TREATMENT VERSUS PREVENTION

Prevention of mental health problems is important for several reasons. First, the cost of disorder to the individual and society in terms of personal suffering and lost productivity can be extensive. Second, in the long run, prevention may be more cost effective than continued treatment. Third, it is likely that over time there will not be enough mental health professionals available to treat all those individuals in need of services at any given time. (Coie et al., 2000). Thus, services for the prevention of mental health disorders are needed in addition to treatment.

Whereas treatment is concerned with the reduction of symptoms in individuals already manifesting a disorder, prevention is defined as lowering the risk of the onset of new cases of a disorder. With regard to depression, which tends to be episodic, prevention also can involve reducing the recurrence of symptoms after an episode has remitted (Compas et al., 1997; Munoz, 1993). That is, depressed individuals who return to normal functioning become "new cases" if they experience a future episode of clinical depression, and therefore also are candidates for preventive interventions.

Although efficacious psychosocial and pharmacologic treatments for adolescent depression have been demonstrated (Kaslow and Thompson, 1998), any single approach helps only approximately 50% to 60% of those treated (Brent et al., 1997; Emslie et al., 1997). Moreover, chronicity, comorbidity, and parental depression all render efficacious treatments less likely to work well (Birmaher et al., 2000; Brent et al., 1998; Clarke et al., 1992; Emslie et al., 1998). Therefore, prevention of depression, particularly among high-risk individuals, may be more cost effective than waiting for the condition to appear and then trying to treat a full-blown depressive episode. The question is not whether prevention is more or less important than treatment; rather, both prevention and treatment may be appropriate, depending on the particular goals of the researchers and clinicians. "Diverse intervention efforts and a broad portfolio of approaches are central to adolescent mental health" (Kazdin, 1993, p. 136).

### PREVENTION SCIENCE

Because the old distinctions among primary, secondary, and tertiary prevention (Felner et al., 1983) are now considered ambiguous, they have been abandoned. For example, secondary prevention referred to early intervention for new cases as well as interventions for individuals with high levels of symptoms but who did not yet meet criteria for a clinical disorder. The Institute of Medicine (IOM) Report (Mrazek and Haggerty, 1994; Munoz et al., 1996) adopted Gordon's (1983) distinction among three types of prevention. *Universal* preventive interventions target entire groups or populations that have not been selected on the basis of risk. *Selective* preventive interventions target individuals with elevated risk for the disorder. *Indicated* preventive interventions target high-risk individuals who display some early precursor signs of the disorder or symptoms predicting the disorder. Targeted prevention includes both selective and indicated interventions and has been contrasted with universal prevention (e.g., Offord, 1996). Whereas universal interventions that lead to small effects in large numbers of people may have enormous benefits for society (Offord, 1996; Offord et al., 1998), targeted prevention may be more efficient, leading to greater prevention effects for each individual involved.

The science of preventive intervention research involves the careful study of the identification and epidemiology of disorders, etiology, risk, and protective factors, the design and implementation of controlled preventive intervention trials with random assignment and extensive follow-up, and the examination of the generalizability of these interventions in real-world community settings (Coie et al., 1993). The IOM (Mrazek and Haggerty, 1994) suggested that the preventive intervention research cycle is a recursive process in which the results of clinical and field trials are used to inform more basic science concerned with etiology and mechanisms of risk.

### RISK FOR DEPRESSION

Although affective disorders represent a significant psychiatric problem for the general adolescent population, specific subgroups are particularly vulnerable to development of mood disorders and associated problems (e.g., suicide, social isolation). The IOM committee recommended that a risk reduction approach should be taken. This involves targeting populations with elevated levels of known risk factors, reducing risk factors that are malleable, and increasing protective factors such as problem-solving skills, interpersonal relationships, and social support (Beardslee and Podorefsky, 1988; Compas et al., 1997; Petersen et al., 1997). Risk factors that consistently have been found to be associated with mood disorders include being the offspring of a depressed parent, having subsyndromal depressive symptoms, having a personal history of a mood disorder, comorbid disorders, or family dysfunction, and being female (e.g., Brady and Kendall, 1992; Goodman and Gotlib, 1998; Pine et al., 1998; Reinherz et al., 1993). A complete review of all of these risk factors is beyond the scope of this chapter (e.g., Garber and Flynn, 2001; Hammen, 2001; see Chapter 62, this volume); rather, we highlight some of the most likely vulnerability factors for depression during childhood and adolescence.

First, parental depression is one of the strongest risk factors for depression in children ( [Beardslee et al., 1998](#); [Goodman and Gotlib, 1998](#); [Kaslow et al., 1994](#)). Numerous studies have shown that offspring of affectively ill parents are at risk for psychopathology, with the most frequent diagnosis being mood disorders ([Beardslee et al., 1988](#); [Hammen, 1991](#); [Orvaschel et al., 1988](#); [Radke-Yarrow, 1998](#); [Weissman et al., 1987](#)). A meta-analysis showed that offspring of parents with affective disorders are approximately four times more likely to have a mood disorder than are children of nondepressed parents ( [Lavoie and Hodgins, 1994](#)). Life-table estimates suggest that by the age of 20 years, a child with an affectively ill parent has a 40% chance of having an episode of depression, and by 25 years of age this rate increases to 60% ([Beardslee et al., 1993a](#)). Offspring of depressed parents also are at increased risk for high levels of medical utilization, behavior and school problems, suicide attempts, and substance abuse disorders ([Hammen et al., 1990](#); [Kramer et al., 1998](#); [Weissman et al., 1987](#)). In addition, the risk for offspring depression is greater if the parent had an early-onset or recurrent depressive episodes, and if both parents have experienced depressive disorders ( [Warner et al., 1992](#); [Weissman et al., 1987, 1992](#)). Current parental depression also appears to affect the course of adolescent depression, through slower recovery, lower levels of functioning, and poorer response to cognitive interventions ( [Brent et al., 1998](#); [Hammen, 1991](#); [Keller et al., 1986](#); [Weissman et al., 1992](#)). Thus, children of parents with affective disorders are at great risk for psychiatric disorders, especially depression, and therefore, they are a logical and critical population to target for prevention.

Second, one of the strongest predictors of depressive disorders is prior depressive symptoms. [Compas et al. \(1993\)](#) suggested that depressed mood may be a marker of risk for subsequent depressive syndromes. Subsyndromal levels of depressive symptoms significantly increase the risk of having a full major depressive episode in adults ([Horwath et al., 1992](#); [Judd et al., 1997](#)) and adolescents ([Clarke et al., 1995, 2001](#); [Lewinsohn et al., 1999](#); [Pine et al., 1999](#); [Rueter et al., 1999](#)). [Pine et al. \(1999\)](#) reported that having depressive symptoms during adolescence predicted a twofold to threefold greater risk of an episode of major depression in adulthood. [Brent et al. \(2001\)](#) found that adolescents with depression who only partially recovered still had subsyndromal depression and functional impairment, and were at significantly greater risk for recurrence compared with those who recovered fully from their depression. Subsyndromal levels of depression also can have a negative impact on academic/occupational and interpersonal functioning ( [Wells et al., 1989](#)). Thus, targeting individuals with subsyndromal depression for preventive intervention could not only reduce their risk for development of major depressive disorders, but could ameliorate existing levels of distress and dysfunction ( [Munoz et al., 1994](#)).

Third, a prior history of a depressive disorder also is a significant risk factor for subsequent depressive episodes. Children with mood disorders have recurrent depressive episodes during adolescence ( [Emslie et al., 1997](#); [Kovacs et al., 1984a,b](#)) and adulthood ([Garber et al., 1988](#); [Harrington et al., 1990](#); [Weissman et al., 1999](#)), and adolescents have recurrences later in adolescence ( [Lewinsohn et al., 1994b](#); [McCauley et al., 1993](#)) and into adulthood ([Lewinsohn et al., 1999](#); [Rao et al., 1999](#); [Weissman et al., 1999](#)). Recurrence rates have ranged from 45% to 72% over 3 to 7 years ( [Emslie et al., 1997](#); [Harrington et al., 1990](#); [Kovacs et al., 1984b](#); [Lewinsohn et al., 1999](#); [McCauley et al., 1993](#)). Such recurrent episodes are associated with negative functional outcomes in school, work, and interpersonal relationships ([Rao et al., 1999](#)). Moreover, the longer depressive episodes last, the more difficult they are to treat ( [Judd et al., 1998](#); [Thase and Howland, 1994](#)). Preventive interventions may reduce the risk for subsequent episodes of depression as well.

Finally, several other risk factors are known to be associated with the onset of depression, including female sex, negative cognitions, dysfunctional family environments, and comorbidity. The risk of onset increases in girls relative to boys starting during adolescence ( [Hankin et al., 1998](#); [Nolen-Hoeksema and Girgus, 1994](#)). Negative cognitions about the self and future have been found to predict changes in depressive symptoms in children, adolescents, and adults (e.g., [Metalsky et al., 1993](#); [Nolen-Hoeksema et al., 1992](#); [Robinson et al., 1995](#)). Family environments characterized by high levels of discord, criticism, and lack of involvement and supervision increase the risk of depression onset and recurrence, and interfere with recovery ( [Asarnow et al., 1993](#); [Birmaher et al., 2000](#); [Garber et al., 2001](#); [Kaslow et al., 1994](#); [Weissman et al., 1992](#)), and also may predict the occurrence of comorbid conditions such as disruptive disorders and substance abuse ( [Blum, 1987](#); [Fendrich et al., 1990](#)). Finally, comorbid conditions such as anxiety and disruptive disorders are associated with the onset of depression and interfere with the course of recovery from depression ( [Brady and Kendall, 1992](#); [Brent et al., 2001](#); [Hamilton and Bridge, 1999](#); [Reinherz et al., 1993](#); [Sanford et al., 1995](#)).

Identifying factors that are associated with the onset and recurrence of mood disorders, of course, does not necessarily mean that these variables play a causal role in the development of the disorder. Nevertheless, it makes sense to use these factors to help identify individuals who are at the greatest risk for the disorder, and therefore should be the focus of prevention efforts. Moreover, these risk factors also can provide some guidance about the nature of the preventive intervention that might be needed. For example, if the presence of negative cognitions presumably is a risk for the development of depression, then preventions that aim to alter such cognitive styles should reduce the occurrence of depressive symptoms.

## PROGRAMS FOR THE PREVENTION OF DEPRESSION IN CHILDREN AND ADOLESCENTS

Despite the declaration in 1984 that "In general, the onset of clinical depression cannot be prevented" ( [Lobel and Hirshfeld, 1984](#), p. 4), some evidence now exists that psychosocial interventions can reduce the occurrence of depression in youth. Three different groups of investigators have developed programs to prevent depression in children and adolescents ( [Beardslee et al., 1993b](#); [Clarke et al., 1995, 2001](#); [Seligman, 1998](#)). These programs vary with regard to the participant ages, targeted risk factors, and sample selection. Nevertheless, they each have shown some positive preventive effects.

### Clarke's Cognitive-Behavioral Group Preventive Intervention

Clarke and colleagues have developed a cognitive-behavioral intervention for the prevention of depression in adolescents, which is based on the Coping with Depression for Adolescents (CWDA) course used effectively to treat depression in adolescents ( [Clarke et al., 1999](#); [Lewinsohn et al., 1990](#)). This cognitive intervention is derived from the theoretical model of [Lewinsohn et al. \(1985\)](#) and includes many features of [Beck's \(1976\)](#) cognitive therapy. Treatment sessions are conducted in a classlike fashion, with the group leader teaching adolescents methods of controlling their depressed mood through cognitive restructuring techniques. Each adolescent is given a workbook ([Clarke et al., 1990](#)) that contains brief readings, structured learning tasks, short quizzes, and forms for homework assignments. Because it is presented as a class rather than therapy, the Coping with Stress (CWS) course is less stigmatizing than traditional psychotherapy, an issue that is particularly relevant with adolescents.

The intervention approach has been modified from cognitive therapy for depressed adults ( [Beck et al., 1979](#); [Ellis and Harper, 1961](#)) and teaches participants how to identify and challenge negative and unrealistic thoughts. [Clarke et al. \(1990\)](#) simplified these techniques for use with adolescents. Cartoon strips with popular characters (e.g., Garfield the Cat, Bloom County) are used to illustrate both the negative thoughts that contribute to depression, as well as the more realistic counterthoughts that offset the irrational beliefs. Through a series of progressively more advanced exercises, participants are taught how to apply cognitive techniques to their own personal thoughts, with the goal of learning to generate their own, effective counterarguments to unrealistic or excessively negative beliefs.

Using this approach, Clarke and colleagues have conducted a series of prevention studies both in schools and with at-risk offspring of depressed parents. In an early school-based primary prevention program for adolescent depression, [Clarke et al. \(1993\)](#) compared a three-session educational (knowledge-only) intervention in ninth grade health classes (n = 361) to a randomly assigned control condition (n = 261), and found a short-term reduction in depressive symptoms among boys but not girls. However, this effect was not sustained over a 12-week follow-up period. Based on the behavioral model of depression ( [Lewinsohn et al., 1985](#)), a more intensive five-session intervention was developed to train adolescents to increase pleasant activities as a method of preventing depressive symptoms. However, compared with a randomized control group (n = 190), the behavioral intervention adolescents (n = 190) failed to exhibit any persistent reduction in depressive symptomatology ([Clarke et al., 1993](#)).

[Clarke et al. \(1995\)](#) next completed a randomized, controlled trial of a school-based prevention program aimed at reducing the incidence of unipolar affective disorders and symptoms in high school adolescents at risk for depressive disorders by virtue of their having subdiagnostic levels of depressive symptoms, defined as *demoralization* ([Roberts, 1987](#); [Roberts et al., 1990](#)). At-risk ninth grade adolescents were identified by a two-stage screening procedure ( [Kendall et al., 1989](#)) consisting of a schoolwide administration of a self-report screening instrument (Center for Epidemiological Studies-Depression Scale (CES-D), followed by a confirmatory structured diagnostic interview (the Schedule for Affective Disorders and Schizophrenia, Childhood Version) with those adolescents who had elevated screener scores. Adolescents with *Diagnostic and Statistical Manual of Mental Disorders*, third edition-revised (DSM-III-R) major depression or dysthymia were diverted to a nonexperimental treatment group, and the remaining 148 subdiagnostic, demoralized adolescents were randomly assigned to either (a) a 15-session, after school, cognitive-behavioral preventive intervention, modified from the CWDA course (n = 74); or (b) a "usual care" control condition (n = 74). All were followed at 6 and 12 months postintervention. Results indicated that the cognitive-behavioral therapy prevented progression from subsyndromal depression to full-blown major depression in the youth randomized to preventive intervention compared with the control subjects. Total mood disorder incidence rates at a median 13-month follow-up were 14.5% for the cognitive-behavioral therapy intervention group and 25.7% for the control subjects ( [Clarke et al., 1995](#)).

Finally, Clarke et al. (2001) recently completed a study of a cognitive-behavioral preventive (CBP) intervention compared with a usual care group among adolescent



offspring of parents being treated for depression in a large health maintenance organization (HMO). Potential participants were identified by reviewing the HMO pharmacy database and behavioral health records. Those adults in treatment for depression who had children between 13 and 18 years of age were sent letters from their health care providers inviting them to participate in the study. Interested parents were then interviewed to confirm their diagnosis of a mood disorder, and the children were evaluated with regard to their history and current level of depressive symptoms and disorders. A "demoralized" group was identified who had current subdiagnostic levels of depressive symptoms (i.e., a CES-D score <24) or a previous episode of major depression. These 123 adolescents were randomly assigned to either a usual care control condition or the 15-session, group depression prevention program, called the Adolescent CWS course, which is an abbreviated version of the adolescent depression treatment program. Survival analyses revealed a significant advantage for the CBP group (9.3%) versus the usual care group (28.8%) in the cumulative incidence of major depression at the 12-month follow-up. The odds ratio of depression at 12 months was 5.64 (usual care vs. CBP, 95% confidence interval = 1.56 to 20.39) when adjusted for sex, age, CES-D score, and previous depression history. (The latter two were the criteria for inclusion.)

Thus, this CBP intervention appears to be quite promising for the prevention of depression in high-risk adolescents. It is efficiently delivered in a group format and has produced significant change in both depressive symptoms and rates of disorders, although the effects of the intervention showed some waning with time. Future studies need to examine whether this intervention will generalize to more geographically, ethnically, and socioeconomically diverse populations.

### **A School-Based Cognitive–Behavioral Preventive Intervention**

A second cognitive–behavioral depression prevention program has been developed at the University of Pennsylvania ( [Gillham et al., 1995](#); [Jaycox et al., 1994](#); [Seligman et al., 1999](#); [Zubernis et al., 1999](#)). The Penn Optimism Program (POP; Gillham JE, Jaycox LH, Reivich KJ, et al., unpublished manual, University of Pennsylvania, 1990) consists of a cognitive and a social problem-solving component. The cognitive component is based on cognitive–behavioral therapy ( [Beck et al., 1979](#)) and the learned helplessness theory of depression ( [Abramson et al., 1978](#)). Children are taught the cognitive model of emotion and behavior, identifying negative thoughts and thinking styles and disputing negative thoughts by generating alternatives and evaluating evidence for and against them. The social problem-solving component teaches children goal-setting, perspective-taking, information gathering, generating alternative actions, decision-making, and emotion control techniques. Both components use cognitive techniques; whereas the cognitive training section focuses on children's interpretations of problems, the social problem-solving component emphasizes children's actions to solve problems. A third aspect of the program focuses directly on coping with family conflict and other stressors and involves relaxation training, distraction, and seeking social support.

The POP was evaluated in a district-wide, school-based indicated prevention study that targeted 10- to 13-year-old children at risk for depression based on elevated self-reported depressive symptoms or self-reported parental conflict. Intervention groups comprised 10 to 12 children who met in 90-minute group sessions held after school over a 12-week period. Sixty-nine children from one school district were assigned to one of three prevention conditions: cognitive, social problem solving, or combined. They were compared with 49 matched control subjects from a neighboring district similar in demographic characteristics. All participants completed self-report measures of depressive symptoms and explanatory style before the intervention, postintervention, and at 6-month intervals for 3 years of follow-up. Parents and teachers reported on children's internalizing and externalizing behaviors.

Children who participated in the prevention groups reported significantly lower levels of depressive symptoms than control subjects through 2 years of follow-up. At the 2-year follow-up, 44% of control subjects reported moderate to severe levels of depressive symptoms Children's Depression Inventory (CDI) i.e.,  $\geq 15$  compared with only 22% of the prevention group ( [Gillham et al., 1995](#)). The effects on depressive symptoms did not endure beyond 2 years, however ( [Gillham and Reivich, 1999](#)). Significant improvements in explanatory style also were found for the prevention group through the 2-year follow-up period, and change in attributional style appeared to mediate the relation between the preventive intervention and change in depressive symptoms ( [Gillham et al., 1995](#)). Parents reported fewer externalizing symptoms at follow-up, and teachers reported significant improvements in classroom behavior in the prevention group compared with control subjects postintervention ( [Jaycox et al., 1994](#)).

Thus, the cognitive–behavioral prevention program developed at the University of Pennsylvania appears to reduce depressive symptoms in young adolescents over a 2-year follow-up period. Limitations of this study include the lack of random assignment to condition, the lack of assessment of depressive disorders, relatively small sample, and a high rate of attrition over the follow-up period.

Finally, using a similar group cognitive–behavioral prevention program with older adolescents (i.e., college freshmen), [Seligman et al. \(1999\)](#) randomized 231 at-risk college freshmen, with risk being defined as a negative attributional style, into either an 8-week group cognitive prevention program or an assessment-only control group. The prevention group had significantly fewer self-reported depressive symptoms compared with the control subjects at the 3-year follow-up. In addition, the prevention group had significantly fewer episodes of generalized anxiety disorder and a trend toward fewer major depressive episodes. Significant improvement in cognitions (i.e., attributional style, hopelessness, dysfunctional attitudes) mediated the effect of the intervention on depressive symptoms. A particularly important contribution of the studies by the researchers at the University of Pennsylvania has been their examination of mechanisms of change.

### **A Targeted, Family-Based Cognitive–Educational Prevention Program**

Beardslee and colleagues ( [1996](#), [1997a,b,c](#)) developed and evaluated a family based, cognitive–educational intervention for the prevention of depression. This was a selective prevention program in that it targeted nonsymptomatic adolescents who were at risk for the development of depression because of having at least one parent with an affective disorder. This intervention is theoretically compatible with cognitive–behavioral approaches ( [Beardslee and Schwoeri, 1994](#)) and aims to increase family understanding about affective disorders, improve parental focus on their children, and enhance child resilience ( [Beardslee et al., 1992](#), [1993b](#)).

Beardslee et al. ( [1997a,b,c](#)) compared two intervention programs: a clinician-based intervention administered to families on an individual basis, and a lecture program delivered to parents in a group format. The clinician-facilitated intervention consisted of 6 to 10 sessions in which a clinician worked with individual families. Most of the sessions were with the parents, one session was held with each child in the target age range, and a family meeting was held during which the clinician facilitated a family discussion of affective illness. The lecture condition consisted of two small group lectures for parents. Although children did not attend the lectures, parents were encouraged to talk with their children about their affective disorders.

The focus of the clinician-facilitated program was on (a) assessment of all family members; (b) educating parents and children about affective disorders and the potential effects of the illness on relationships with family members, particularly the impact of parental depression on children; (c) teaching information about risks and resilience in high-risk offspring; (d) linking this information to the individual's and family's life experience; (e) improving communication between parents and their children; and (f) enhancing the children's understanding of affective illness to help them develop concrete plans for coping in the future ( [Beardslee et al., 1992](#)). The lecture intervention also provided cognitive information about affective disorder, risks to and potential resiliency of offspring of depressed parents, recognizing distress in children, and enhancing children's adaptive capacities, but without the direct link to the individual experiences of family members.

Parents, recruited from an HMO in the Boston area, were invited to participate if they had at least one child between the ages of 8 and 15 years who had not been treated for an affective disorder, and if at least one parent had experienced an episode of affective disorder in the past 18 months. Thirty-seven families with 52 children were randomly assigned to either a clinician-facilitated, manual-based, psychoeducational preventive intervention or a standardized lecture-group discussion. Parents and children were assessed before the intervention, after intervention, and approximately 1 to 2 years after enrollment (or approximately 9 to 12 months after the intervention). [Beardslee et al. \(1996\)](#) showed that both interventions were feasible and well accepted by high-risk offspring and their depressed parents, had comparable acquisition of cognitive information, and were associated with positive changes in behavior and attitudes in both parents and children, and the degree of upset regarding child-related concerns decreased in both conditions from before to after intervention. Families in the clinician-facilitated group, however, reported significantly more positive behavior changes (e.g., increased communication with spouses and increased talking with their children about depression) and more positive attitude changes (e.g., increased understanding of their children's experience, increased closeness within the family) that were sustained over 3 years.

Positive intervention effects also were found on adolescent functioning both immediately posttreatment ( [Beardslee et al., 1993b](#)) and over a longer follow-up period of 18 months ( [Beardslee et al., 1997c](#)). Participants in the clinician-facilitated condition reported significantly greater levels of clinician-rated and self-reported change than did participants in the lecture condition. Greater parental benefit from intervention in terms of changes in illness-related behaviors and attitudes was associated with significant global change among children, including enhanced understanding of parental illness and improved communication with parents ( [Beardslee et al., 1997a,b,c](#)). In addition, children in the clinician-facilitated intervention reported significantly higher levels of global functioning. No differences were found for depressive symptoms, depressive disorders, behavioral problems, or self-worth, however.

The prevention study of Beardslee and colleagues ( [1997a,b,c](#)) had several strengths, including random assignment to intervention conditions, multiple outcome measures and informants, examination of possible detrimental effects, and evaluation of family functioning and other proposed mediators. A limitation of the study was

that it included a relatively small sample, thereby reducing power to detect differential effects of the interventions. The lack of a no-treatment control group made it difficult to interpret nonsignificant differences between the two conditions. Finally, the fairly short-term intervention might not have been sufficient to address the more chronic and severe consequences of parental depression. Nevertheless, the findings from this program of research indicate that providing parents with factual information about children's risk and resilience, and linking it to family members' affective illness experiences, can produce behavioral and attitudinal changes in parents that translate into more optimal functioning among children ([Beardslee and Gladstone, 2001](#)).

### Other Prevention Programs with Youth

Three other depression prevention studies are worth noting. [Petersen et al. \(1997\)](#) examined the effects of an intervention for preventing depression in a sample of 486 students in seventh grade. The sample was selected for elevated levels of depressive symptoms, stressors, and poor coping skills. Students were randomly assigned to either the intervention or no-treatment control group. The sixteen 40-minute sessions were conducted at the students' schools during the spring of their seventh grade year. Cognitive techniques included reducing irrational thoughts and increasing self-affirming thoughts. Problem-solving techniques included relaxation training, identifying goals, brainstorming about solutions, evaluating the consequences of decisions, and teaching assertiveness and other social skills. Measures of coping, depressive symptoms, and internalizing and externalizing symptoms were obtained before the intervention, immediately postintervention, and at 6- and 12-month follow-ups. Clinical interviews were conducted to assess depressive disorders.

Postintervention, prevention group participants reported fewer internalizing and externalizing symptoms and scored higher on measures of coping compared with control subjects. In addition, a surprising sex difference was found. Postintervention, the prevention group girls reported fewer depressive symptoms than control girls, whereas boys in the intervention group reported higher levels of depressive symptoms than control group boys. Thus, there actually were some short-term negative effects of the intervention for boys. There were no group differences for either girls or boys over the follow-up period, however. In addition, no differences were found between the intervention and control groups regarding clinical diagnoses of depression. Thus, Petersen and colleagues' preventive intervention had only short-term positive effects on reducing depressive symptoms in girls.

Using a longer cognitive-behavioral program, [Hains and Ellmann \(1994\)](#) found somewhat promising results, although their study should be interpreted with caution because of the small sample ( $n = 21$ ) and the absence of a control group at the 2-month follow-up. The stress inoculation training intervention consisted of thirteen 50-minute sessions and included a combination of group and individual sessions. High school students were taught (a) cognitive restructuring techniques for evaluating negative interpretations of problems by considering alternatives and examining the evidence for and against their beliefs; (b) problem-solving skills of generating multiple solutions, considering consequences, and choosing among different options; and (c) relaxation training consisting of deep breathing and progressive muscle relaxation techniques. Assessments of state and trait anxiety, state and trait anger, depressive symptoms, stress, and physical health were conducted before the intervention, postintervention, and at a 2-month follow-up. Records of students' grades and absences from school also were collected at these times. Participants were classified as high or low in emotional arousal based on their baseline scores on trait anxiety and anger.

Among the high-arousal students, program participants reported greater reductions in trait anxiety and depressive symptoms from preintervention to postintervention compared with control subjects, and the gains in the interventions group were maintained at the 2-month follow-up. Students in the wait-list group participated in the intervention between postintervention and 2-month follow-up, and similar postintervention results were found for high-arousal participants. In contrast, no intervention effects were found for the low-arousal group. Finally, no group differences were found with regard to grades or school absences.

Finally, using a large and ethnically diverse urban sample, [Kellam et al. \(1994\)](#) conducted a year-long universal reading enrichment program for first grade children. Children were randomized by classroom to either the regular reading program or the enriched curriculum throughout their first grade year. The enriched curriculum was designed to improve reading achievement by using a group-based assessment of mastery and a flexible corrective process that was tailored to individuals and was flexible in the timing and types of corrective techniques. Reading delays were used as the risk factor in this study.

[Kellam et al. \(1994\)](#) found a significant relation between depressive symptoms and achievement test scores, although the findings differed by sex. In boys who showed a gain in reading achievement at the level of at least the national average, depression fell from fall to spring compared with those whose achievement gain was lower. For girls, there also was a significant relation between gain in achievement and depressive symptoms, although this effect was not different as a function of the intervention. Kellam et al. explained these sex differences by suggesting that girls were more sensitive to teachers' feedback than boys and that girls tended to internalize blame more than boys, thereby maintaining depressive symptoms even in the intervention group.

### Summary and Future Issues

Despite earlier skepticism that depression could be prevented ([Lobel and Hirshfeld, 1984](#)), evidence is increasing that intervention programs can successfully forestall increases in depressive symptoms and possibly delay the onset of depressive disorders, although much more research is needed. Most programs have used combinations of cognitive-behavioral, problem-solving, and stress reduction techniques administered directly to the target children and adolescents in group formats. Less work has been done with families, although Beardslee and colleagues' ([1996, 1997a,b,c](#)) family education program has shown some positive effects. Although Clarke and colleagues ([Clarke et al., 1999; Lewinsohn et al., 1990](#)) did not find a benefit of an added parent intervention component compared with the adolescent-alone intervention, they did not test the effects of a cognitive-behavioral program delivered to the entire family at the same time. Prevention programs that integrate various components, such as cognitive-behavioral and family communication skills training, should be developed and tested. In addition, depression prevention programs should focus on the development and enhancement of competencies and resilience. By so doing, these programs not only can prevent disorder and dysfunction, but can build prosocial skills that may serve a protective function.

Whether conducting efficacy or effectiveness trials, prevention researchers should rely on rigorous experimental methods similar to those recommended for treatment outcome research (e.g., [Chambless and Hollon, 1998](#)), including control groups or alternative interventions, random assignment, large enough samples to achieve adequate statistical power, development and use of intervention manuals, evaluation of treatment fidelity, use of psychometrically adequate assessment measures, attention to the problem of differential attrition across conditions, assessment of possible mediators, use of multiple outcome measures, examination of the clinical significance of the findings, and consideration of possible negative effects of the intervention.

Most depression prevention studies have used a control or comparison group and some form of random assignment. The issue of whether the individual, family, classroom, or school is the unit of randomization needs to be addressed further, and more sophisticated nested analyses need to be conducted. Multiple measures and informants have been used to assess outcomes, although how best to combine this information and how to handle discrepancies across informants is an unresolved issue for the broader field of developmental psychopathology. In general, studies have used self-report measures of depressive symptoms as the primary outcome measure. Although change in self-reported depressive symptoms remains an important goal and should continue to be included in prevention studies, an evaluation of diagnosed depressive disorders also should be conducted. In addition, other psychopathology should be assessed, both because comorbid disorders can affect the impact of the preventive intervention on depressive outcomes, and because some powerful preventive interventions might actually reduce other problem behaviors in addition to depression.

Future prevention researchers will continue to grapple with the question of whether programs should be universal, indicated, or selective. The answer to this depends on the current state of knowledge about risk factors, the specific aims of the investigator, and the resources available. These approaches can be linked hierarchically and sequentially ([Compas et al., 1997](#)). Universal approaches can reach the most individuals, although the intensity of the intervention generally is limited. To show that the intervention has had an effect, it often takes very large numbers, particularly if the goal is to reduce onset of diagnosed depressive disorders. Selective interventions can be added for those individuals who are identified on the basis of some known vulnerability factor(s). Existing programs have shown some success with risk groups identified on the basis of parental history of depression, negative attributional style, stress, and emotional arousal. As more information is obtained about risk factors, and prevention programs are developed specifically to target these variables, it will be possible to provide more cost-effective services to those who are most in need. Because prior depressive episodes and high levels of depressive symptoms also are known predictors of future depression, indicated prevention programs with such individuals probably are the place to begin. Once programs are successful with these highest-risk individuals, prevention programs can be expanded more universally.

The issue of sex differences in the effects of preventive interventions needs to be taken seriously. Several studies have found different outcomes as a function of participants' sex (e.g., [Clarke et al., 1993; Kellam et al., 1994; Petersen et al., 1997; Reivich, 1996](#)), and the direction of these findings has not been consistent across studies. Why are some interventions working better or worse for boys versus girls? How do the children's age and developmental level interact with sex in the different prevention approaches? Does using same- versus mixed-sex intervention groups affect outcomes? The mechanisms underlying why sex moderates the effect of the



intervention on depressive outcomes need to be examined.

Another important issue concerns how enduring are the effects of prevention efforts. Most studies have conducted preintervention and postintervention assessments and then follow-up evaluations for another 2 to 3 years. Although the short-term effects have been encouraging, evidence of long-term effects is lacking. The contribution of booster or continuation sessions should be explored as a possible means of extending the positive initial effects of preventive interventions. Why the effects wane over time needs to be understood. Are participants forgetting what they learned? Do they lose their motivation to implement their acquired skills? Are the interventions just not powerful enough to sustain long-term benefits? What is the impact of development on the long-term effects? A related question is what is the best developmental period in which to intervene to produce the most enduring effects during the period of greatest risk for depression?

Several important practical questions also need to be addressed with regard to implementation of the various preventive intervention programs. For group interventions, what is the best size of the group? Most programs have included 8 to 10 participants in a group. Is that too large? How many therapists are needed? For groups with younger children or with children with comorbid disruptive behaviors, might two therapists be needed? Who are the best therapists? Can teachers, case managers, nurses, or nonprofessionals implement the interventions successfully? The extent to which the prevention program is structured and manualized will partially determine who can best implement it. What are the best settings in which to conduct preventive interventions: schools, psychiatric clinics, pediatric offices, HMOs? Some of this will depend on whether the prevention program is universal, selective, or indicated, although it is possible to execute any of these types of interventions in multiple settings. Finally, what are the essential components of preventive interventions? That is, can these prevention programs be boiled down to the absolute key ingredients so they can be broadly and cost-effectively disseminated? What kinds of innovative methods can be used to reach the most people (e.g., the Internet, public service announcements)?

Another critical and as yet unanswered question is to what extent existing prevention programs are applicable to diverse populations. In general, studies aimed at preventing depression have been conducted with white, middle class samples [although see [Kellam et al. \(1994\)](#) for an exception]. The IOM ([Mrazek and Haggerty, 1994](#)) recommended that external validity of prevention programs when implemented by other investigators and personnel across different settings, samples, and geographic locations should be examined. Moreover, not only are studies needed that test whether interventions generalize to different ethnic and socioeconomic groups, but more basic work needs to be done exploring whether the same or other factors make individuals from different social, economic, and ethnic backgrounds vulnerable to depression. Based on this work, existing prevention programs would need to be revised to reflect and accommodate these sociocultural differences.

Finally, prevention research also needs to investigate the mechanisms that underlie change. Seligman and colleagues ([Gillham et al., 1995](#); [Seligman et al., 1999](#)) have shown that changes in negative cognitions mediate the link between the intervention and reduced depressive symptoms. One of the exciting benefits of prevention studies is that they provide a means of testing theory by allowing an ethical and ecologically valid means of directly manipulating factors that are hypothesized to be part of the causal chain underlying the disorder. Researchers need to use prevention studies as an opportunity to study mechanisms as well as a means of reducing suffering.

## SUICIDE PREVENTION PROGRAMS FOR YOUTH

Rates of suicide and suicide risk behaviors among adolescents have risen dramatically over the last four decades ([Allison et al., 1995](#); [Andrews and Lewinsohn, 1992](#); [Berman and Jobes, 1995](#); [Brent, 1995](#); [Garrison et al., 1993](#); [Lewinsohn et al., 1994b](#); [McIntosh, 2000](#)). In 1956, the rate of suicide among adolescents (15 to 19 years of age) was 2.3/100,000; by 1996, these rates had increased to 9.7/100,000, an increase of 322% ([McIntosh, 2000](#)). Increases also occurred in the rates of suicide among children 10 to 14 years of age (from 0.4 to 1.6/100,000). Suicide is now one of the leading causes of death among adolescents, along with accidents and homicides (*Morbidity and Mortality Weekly Report*, 1998). Moreover, for every completed suicide, there is an estimated 8 to 25 suicide attempts ([Eggert et al., 1995b](#); [King, 1997](#)). The [Centers for Disease Control's \(CDC\) \(1990\)](#) survey of 11,631 high school students found that 27.8% of the students had thoughts of suicide, 16.3% had made a specific plan, and 8.3% had made a serious suicide attempt. Thus, adolescent suicide has become a major public health concern.

The most dramatic increases in rates of suicide among young people 15 to 24 years of age occurred between the mid-1950s and the late 1970s (1977: 13.6/100,000). Although the rate of suicide has remained fairly stable since then, it also has remained high; the average suicide rate between 1990 and 1996 was 13.1/100,000 ([McIntosh, 2000](#)). Furthermore, suicide rates for adolescents (15 to 19 years of age) and children (10 to 14 years of age) have increased consistently since the 1950s. Completed suicide continues to be a much more prominent and concerning problem for boys, whereas girls are responsible for more suicide attempts. When risk rates are computed separately by sex, the suicide rates for boys jump to 20/100,000, whereas the rates for girls are 3.6/100,000 (based on 1996 data; [McIntosh, 2000](#)). The ethnic demography of youth suicide has changed, however. In the past, suicide occurred predominantly in white boys with dramatic peaks in some, but not all Native American groups ([LaFromboise and Howard-Pitney, 1995](#)). In the 1990s, the suicide risk in the United States rose steadily among African-American boys and young men, particularly those living in urban settings, such that rates among this group are now comparable with the those of the white population ([McIntosh, 2000](#)).

### Factors Contributing to Increased Rates of Youth Suicide

Although not conclusive, factors thought to contribute to the dramatic increase in suicide among young people include changes in population density, increased prevalence of depressive, conduct, and substance abuse disorders, increased family instability, increased exposure to stressors, and ready access to firearms ([Berman and Jobes, 1995](#); [Brent et al., 1987, 1991](#)). The 1950s through 1970s mark the "baby boom" period, with dramatic increases in the birth rate in the United States. It is theorized that individuals who are part of large birth cohorts have to compete more for limited resources than members of smaller birth cohorts. There is some support for this theory based on analysis of historical trends, which indicates that members of larger birth cohorts are exposed to increased levels of stress and untoward outcomes such as increased violence and suicide [see review by [McIntosh \(2000\)](#)].

Increases in rates of depressive, conduct, and substance abuse disorders that have occurred since the 1950s affect the adolescents as well as their parents ([Klerman, 1990](#)). This trend certainly influences risk for suicide. Psychological autopsy studies have documented that up to 90% of youth who commit suicide had a diagnosable psychiatric disorder at the time of their death, although only a subset of these young people (33% to 50%) had ever had mental health treatment ([Brent and Perper, 1995](#); [Brent et al., 1988a, 1993a](#)). Alcohol use frequently is associated with youth suicide and seen as a potential trigger because it increases depression and hopelessness while reducing impulse control and problem-solving skills ([Shaffer et al., 1996](#)). Furthermore, parental psychopathology is more common among suicidal youth compared with community control subjects, particularly parental history of depression, substance abuse, or suicide attempts ([Brent et al., 1996](#)). [Brent and Perper \(1995\)](#) demonstrated that parental psychopathology increased a young person's risk for suicide even after controlling for familial transmission of psychopathology.

Exposure to suicide creates an additional risk for suicide. Suicide is more common in young people with a family history of suicide ([Brent et al., 1996](#)). However, suicide also is more likely to occur in young people who have been exposed to suicide through the death of a friend or classmate, or even through the media ([Brent et al., 1988a](#); [Gould et al., 1989](#)). Adolescent suicide also has been characterized by cluster or group suicides, phenomena that are particularly sensitive to exposure to suicide. Cluster suicides occur after exposure to a suicide; for example, the death by suicide of one youth in a high school is followed by the suicides of a number of young people in the community. Group suicides, on the other hand, represent the decision by a group of young people to take their lives together.

Family stability, another key risk factor for youth suicide ([Lewinsohn et al., 1993, 1996](#); [Thompson et al., 2000](#)), has changed dramatically over the last four decades with the marked increase in divorced or working parents. Living with a single parent, living without a parent, and father-absent families have been associated with increased suicide risk behaviors ([Garrison et al., 1991](#); [King et al., 1995](#)). Suicide-vulnerable teens frequently describe parents as unsupportive but also critical and controlling ([Allison et al., 1995](#); [Martin and Waite, 1994](#)). Studies of adolescent suicide risk behaviors indicate that parents tend to be either unaware of their teen's suicidal behaviors or report much lower rates than the teen reports ([Garrison et al., 1991](#); [Joffe et al., 1988](#)). Adolescent suicide attempts occur most frequently during the late afternoon/evening, between school and bedtime, a time that may be unstructured or unsupervised ([Nakamura et al., 1994](#)). In a study of female suicide attempters, 78% of the attempts were precipitated by family fights ([Rotheram-Borun and Trautman, 1988](#)). Parent-child discord has been identified as the most common precipitant for suicide and suicidal behaviors ([Brent et al., 1988b](#); [Hawton et al., 1982](#)).

School stressors, dropout status/truancy, or poor grades also have been linked with suicide risk behaviors ([Gispert et al., 1987](#); [Levy and Deykin, 1989](#); [Lewis et al., 1988](#)). Specifically, lower grades are related to increased risk of suicide attempts ([Garrison et al., 1991](#)), suicides often follow long school absences ([Shaffer, 1988](#)), and negative attitudes toward school capabilities are associated with suicidal ideation ([Butler et al., 1994](#)). [Gould and colleagues \(1996\)](#) concluded that youth who are "drifting" with lack of affiliation to school or work are particularly at risk for suicide.

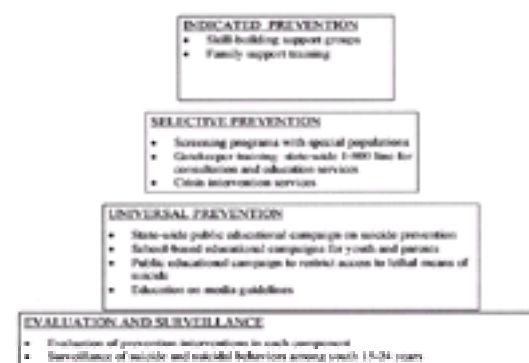
Finally, a number of epidemiologic and case-control studies have documented the relation between access to a firearm and suicide. Suicide is more common, particularly among adolescents, in areas with less restrictive gun control legislation in place ([Sloan et al., 1990](#)) and among those with access to guns in their homes

([Brent and Perper, 1995](#); [Kellermann et al., 1992](#)). Since the early 1980s, firearms have become the most common means of committing suicide among young people. In 1996, gunshot wounds accounted for 62% of all suicides among 10- to 19-year-olds in the United States.

However compelling, these factors do not adequately account for the fact that suicide rates have dramatically increased in boys while remaining stable among adolescent girls. [Berman and Jobes \(1995\)](#) point to the fact that boys are more likely to become involved in substance use and may be more likely to use alcohol when depressed, anxious, or angry. Furthermore, boys more commonly commit suicide when under the influence of alcohol. Others speculate that differences in support systems, coping styles, and willingness to seek help continue to differentiate male and female adolescents ([McIntosh, 2000](#)). Studies of social support and suicide risk reveal that both suicidal and depressed youth endorsed less social support from their family and friendship networks ([Asarnow, Carlson, and Guthrie, 1987](#); [Mazza and Reynolds, 1998](#)). Also, when social support was increased in preventive interventions, suicide risk behaviors and depression decreased ([Eggert et al., 1995a](#)).

### Approaches to Suicide Prevention

As noted earlier, prevention science defines a continuum of approaches ranging from *universal* programs that benefit the general population, to *selective* programs that target at-risk populations, to *indicated* prevention programs that focus on high-risk individuals. [Figure 66.1](#) outlines these approaches as they apply to youth suicide prevention. The CDC (1992) has identified a series of strategies to be used in suicide prevention with the assumption that effective prevention efforts will need to incorporate a number of different strategies simultaneously. These strategies fall into two conceptual categories: (a) those designed to increase recognition of youth at risk and facilitate referral to mental health services, and (b) those designed to address risk factors related to youth suicide. The CDC (1994) also has responded to the contagion aspect of youth suicide by developing guidelines for the media so that coverage of suicide is not sensationalized by glorifying the victim or providing morbid or dramatic details of the death.



**Figure 66.1.** Recommended youth suicide prevention programs and strategies. (Reprinted with permission from Eggert LL, Thompson EA, Randal B, et al.: *Youth suicide prevention plan for Washington State*. Olympia, WA, Washington State Department of Health, 1995.)

The strategies outlined by the CDC include school and community gatekeeper training, educational, screening, and peer support programs, crisis services/hotlines, means restriction, and interventions after a suicide. The 1992 CDC document underscored a series of problems with youth suicide prevention efforts at that time. The central problem was the failure to gather adequate data on efficacy and outcome. The report highlighted related problems such as an over-reliance on general educational programs that appeared to have limited efficacy, inadequate attention to other, more promising strategies such as means restriction, and failure to tie suicide prevention programs into other risk-reducing programs, such as alcohol and drug abuse programs. The limited data on efficacy of these efforts are summarized in the following sections.

### Initial Wave of Youth Suicide Prevention Efforts

The alarming increase in youth suicide in the late 1970s led to the development and implementation of a wave of suicide prevention programs. The most common approach involved a combination of gatekeeper training and general education about suicide. Shaffer and colleagues ([Garland et al., 1989](#); [Shaffer and Garland, 1986](#)) conducted a survey of 435 of these programs. Gatekeeper training efforts were directed toward school personnel, parents, and students through education about suicide that was offered in universal, didactic programs; that is, it was offered to the student body in general. Students were trained as gatekeepers because young people are more likely to talk to a peer than a parent or other adult when feeling suicidal ([Tierney et al., 1990](#)). The programs were designed to increase awareness of suicide as a problem, and to teach students, teachers, and parents how to recognize youth who might be at risk and refer them to appropriate resources. Most programs operated from a model that emphasized suicide as a response to stress rather than focusing on suicide as a sign of mental illness. Many of the programs were quite limited in scope, some providing a single didactic session for the teachers covering warning signs and how to make a mental health referral. Seventy-six percent of the programs were 3 hours in length or less.

[Shaffer and colleagues \(1988\)](#) also conducted an in-depth evaluation of the efficacy of a sample of these school-based programs, including representation of both the stress and mental health models of suicide risk ([Shaffer et al., 1990, 1991](#)). They found that most students, even before exposure to the program, had a good sense of warning signs, felt that disclosure of suicidal ideation should be referred to a responsible adult, and that seeking mental health services was the desirable response. These students in general were positive about the prevention programs, but demonstrated only limited changes in knowledge and attitude about suicide from before to after the intervention assessment. A subset of youth, 5% to 20% of students, however, expressed the view that suicide was a reasonable solution to problems, would not reveal a peer's confidential communication of suicidal ideation or intent, and would not seek mental health help. These students did not demonstrate a significant change in attitudes postintervention. Moreover, students who reported having made a previous suicide attempt reported a negative response to the program. An 18-month follow-up of a subset of these students also failed to demonstrate significant program effects ([Vieland et al., 1991](#)). The investigators questioned the efficacy and cost effectiveness of these brief, universal, school-based, general education programs, particularly those that presented suicide as a response to normal adolescent stressors rather than an indication of significant emotional distress. They argued instead for taking a case-finding approach that would identify youth with emotional problems and facilitate their involvement with appropriate mental health interventions. In a review of suicide prevention efforts, [Shaffer and colleagues \(1991\)](#) emphasized the importance of focusing prevention efforts specifically on adolescent boys who had made a previous suicide attempt or who were depressed.

Other outcome studies ([Spirito et al., 1988](#)) found some support for programs that increased knowledge about suicide warning signs and mental health referral sources, but found no change in the proportion of youth who would ask a teacher, counselor, or parent about how to contact a mental health professional outside of school, and no increase in the proportion of youth who indicated they knew how to get help outside of school. [Spirito and colleagues \(1988\)](#) revealed improvements in coping skills after participation in a program that educated youth about helpful and harmful ways of coping with stress or suicidal ideation—participants were less likely than control subjects to believe social withdrawal was an effective response, reported less engagement in wishful thinking or blaming others, and reported decreased feelings of hopelessness after participation. None of the studies found evidence that programs triggered increases in suicidality among exposed teens, and [Shaffer et al. \(1991\)](#) noted a positive consequence of the programs, in that 3% of the participants identified themselves as in need of help and used the assessment/intervention opportunity to seek help from school personnel.

Although Shaffer and colleagues acknowledged the importance of the gatekeeper training components of these programs, they noted that this aspect of these school-based programs has not been carefully evaluated. In their 1992 report, the CDC cited only two studies that had evaluated gatekeeper training. In each of these studies [see [McConahay \(1990\)](#) and [Tierney \(1988\)](#), reviewed in [CDC \(1992\)](#)], participants who completed a 2-day training program retained the skills at a 6-month follow-up evaluation, but did not report working with increased numbers of suicidal youth. One of these studies ([Tanney, 1989](#)) assessed a gatekeeper training program developed and widely disseminated in Canada, the Foundation Workshop, which evolved into LivingWorks. Based on the realization that few caregivers across multiple disciplines were receiving adequate training in how to recognize and manage suicidal behavior, [Tanney \(1989\)](#) developed a 2-day gatekeeper training workshop that built on the principles of the suicide prevention work of the 1970s. This program involved a 2-day training commitment and covered knowledge and attitudes about suicide as well as skill-building simulations. By the late 1980s, approximately 4,000 caregivers had been trained. Participant evaluations were positive, but systematic program evaluation was not reported.

During the late 1980s, there also was growing attention given to the importance of limiting the access of youth to lethal means of self-harm. The importance of means restriction has been exemplified by the decline in suicide rates in England secondary to changes made in the lethality of cooking gas. Asphyxiation had been the most



common means of suicide in England up to that time; suicide rates declined without evidence of at-risk individuals turning to an alternative means ( [Shaffer et al., 1988](#)). Similarly, as noted previously, the increase in youth suicide in the United States has been linked to the increased availability of firearms. Initial studies documented that suicide was less common, particularly among adolescents and young adults, in areas where more restrictive gun control laws were in effect ( [Brent and Perper, 1995](#); [Sloan et al., 1990](#)). As summarized by [Brent and Perper \(1995\)](#), firearms are found more common in homes of suicide victims than either psychiatric or community control groups, if a firearm is in the home there is an increased risk of its being used in a suicide regardless of storage method, and suicide victims seldom purchase a firearm or seek a firearm outside the home ( [Kellermann et al., 1992](#)). These findings as well as the rising numbers of accidental and violent child deaths secondary to firearms led to an educational campaign directed at pediatricians to increase their awareness of the risk of firearms in the home and to increase the likelihood that primary care providers would ask about firearms and educate parents about the risks and need for safe management of firearms. Again, program evaluation data are not available.

Crisis centers/services and hotlines that offer 24-hour-a-day access have been a mainstay of suicide prevention efforts for decades. These programs assume that many suicides are impulsive acts prompted by problems that may seem overwhelming in the moment but that are manageable if the person can get immediate and easy access to appropriate social and emotional support. This premise is particularly applicable to suicide in youth who, as revealed by psychological autopsy studies, are typically emotionally distressed individuals who make an impulsive suicidal act when faced with a stressful event that makes them emotionally aroused (e.g., angry or anxious) ( [Shaffer and Craft, 1999](#)). Studies of the efficacy of crisis services or hotlines suggest that adolescents find them more accessible than traditional mental health services, and young adult white women are the group most likely to make use of these services (CDC, 1990; see reviews by [Shaffer et al., 1988, 1989](#)). Most adolescents or college students who had contact with these services were not involved in other mental health services at the time of contact and most felt satisfied with the help they received, but girls were more likely than boys both to contact a crisis line and feel that the contact was useful. A subset of young people, however, reported that the response given was not helpful to them. Data on whether telephone hotlines lead to an actual reduction in suicide frequency appear mixed. Evidence of a reduction in suicide rates among young white women was revealed in a study of suicide rates in the United States comparing counties with and without suicide prevention centers ( [Miller et al., 1984](#)). [Leenaars and Lester \(1995\)](#) examined the association between the presence of suicide prevention centers in a community and changes in suicide rates in Canada during 1985 to 1991. No statistically significant trends emerged.

[Shaffer et al. \(1988\)](#) concluded that hotlines continue to be an important part of the suicide prevention armamentarium because of the need for immediate access to help. They argued that increased public education about the availability of services geared particularly at young men and better training of crisis line workers would improve their usefulness. Furthermore, they reported that compliance with follow-up treatment recommendations was greater when the crisis line worker actually made an appointment for the caller and contacted the caller if the appointment was not kept ( [Shaffer et al., 1988](#))

### Youth Suicide Prevention Efforts: 1990s

Prevention science in general underwent considerable growth and refinement during the 1980s and early 1990s, leading prevention specialists to recommend guiding principles for the next phase of prevention projects that included the need to test theory-based causal models, target high-risk populations, include a developmental framework, focus on both risk and protective factors, address risk broadly (e.g., address related problems such as substance abuse), be comprehensive enough to address effectively the complex issues underlying risk, and include rigorous attention to methodologic issues such as sampling, measurement, and statistical analyses ( [Coie et al., 1993](#); [Eggert et al., 1995a](#)).

In response to the limitations of prior suicide prevention efforts, several alternative school-based suicide prevention programs were developed and evaluated. [Ciffone \(1993\)](#) examined a brief universal program offered in health class to all sophomores in a school district near Chicago, in light of the concerns raised by Shaffer et al. ( [1988, 1990](#)). This program was similar to those included in the Shaffer evaluation in that it was brief, school based, and universal. The program differed, however, in that it made a clear link between suicide and mental health problems. Students participated in a before and after (30 days after completion) program assessment using tools similar to those used in Shaffer and colleagues' evaluation, but no random assignment to intervention/no intervention was included. Posttest results revealed that after participating in the group, significantly more young people indicated a willingness to seek help from an adult if a peer talked to them about suicide, seek help from a friend themselves if they had thoughts of self-harm, or seek help from a mental health professional. Furthermore, significantly more youth endorsed understanding the association between mental health concerns and suicide. However, 55% of the subset that viewed suicide as a possible solution to problems at baseline did not change their view after the intervention. Although concluding that the brief, universal, school-based suicide prevention program was useful for most participants, the investigator emphasized that programs need to state clearly that suicidal behavior is a symptom of mental health problems that in turn can be effectively treated.

More extensive suicide prevention programs also were developed. [Klingman and Hochdorf \(1993\)](#) tested a 12-week cognitive-behavioral suicide prevention program with 237 eighth grade students in Israel. This program combined elements of gatekeeper training and general education about suicide with peer support program elements designed to reduce risk factors associated with suicide, such as poor coping skills. It also increased students' awareness of, and ability to respond to, peers in distress. Students were randomly assigned to the intervention program or a control group, which was an education program to control for attention effects. The index program was administered to groups of 18 in 50-minute interactive sessions over a 12-week period. The program followed a three-phase intervention model beginning with an education component geared to help teens understand the nature of distress and the role feelings and thoughts can have in maintaining distress. The second phase focused on the acquisition of coping skills, including positive self-talk, empathy training, help-seeking behavior, and training in how to identify suicide risk in peers. The third phase involved rehearsal and implementation, which was built into every session and extended through homework assignments. Results revealed significant reductions in suicidality among students who participated in the program compared with control subjects; this difference was most evident among the boys. Suicidality was measured by the Index of Suicide Potential adapted for use with young adolescents in Israel. Posttest evaluations also revealed improvements in awareness of distress, coping skills, and information regarding youth suicide, including risk signs and support resources for program participants, compared with control subjects.

Another program was conducted with Israeli high school students. [Orbach and Bar-Joseph \(1993\)](#) developed an introspective, emotion-oriented program that encouraged students to discuss their own experiences while also learning coping and problem-solving strategies as a way to protect them from self-destructive behaviors. A "self-help and peer-help" approach was emphasized. Participants were 11th grade students in six high schools; five were general education programs and one was a special program for conduct-disordered youth. Across the 6 schools, 393 students were randomly assigned to either the program or control group. The suicide prevention program was administered over 7 weekly 2-hour sessions. Preintervention and postintervention measures were given to assess suicidal potential, ego identity, and coping. In four of the six schools, students in the experimental group demonstrated a significant decrease in suicidal tendencies compared with control subjects, with girls showing a bigger decline than boys. In three of the six schools, students who participated in the program also provided evidence of significant improvement on the measures of ego identity, cohesion, and coping. No changes in hopelessness were observed. Both Israeli programs were well accepted by participants; in the high school study ( [Orbach and Bar-Joseph, 1993](#)), the students in the conduct-disordered class indicated that the program was too revealing but also demonstrated a significant decline in suicide potential at posttest. Neither of these programs has been replicated, however. This limits our ability to understand the implications of this work in relation to more general suicide prevention efforts across cultural contexts.

A culturally specific suicide prevention program has been tested in the Zuni Pueblo ( [LaFromboise and Howard-Pitney, 1995](#)). Zuni leaders initiated a suicide prevention program that focused on life and social skills training in hopes of increasing high school students' ability to cope with factors that increased risk for suicide. The Zuni Life Skills Development curriculum was administered to an unselected group of 69 students during their language arts class, with three classes per week over 30 weeks. Fifty-nine students in the same school served as a no-intervention control group; students were not randomly assigned to conditions. A multimethod assessment approach that included self-report measures, behavioral observations, and peer ratings was used to evaluate suicide vulnerability, hopelessness, self-efficacy, and ability to engage in effective problem solving when presented with a suicidal situation involving a peer. Pretest and posttest (at 8 months) assessments revealed that the students exposed to the curriculum had more positive scores than the no-intervention group on measures of suicide probability and hopelessness. In addition, the intervention group showed greater ability to perform problem-solving and suicide intervention skills on the behavioral observation but not the peer rating outcome measures.

Each of these investigative groups points to the fact that their findings differed significantly from the results reported by Shaffer et al. ( [1988, 1990](#)) regarding the initial wave of suicide prevention programs. The investigators emphasized that their interventions were more comprehensive than earlier programs and focused on alternative responses to suicide rather than presenting suicide as a normative response to stress. School-based suicide prevention programs continue to be controversial, however, in part because many still follow a stress-related model, assume a universal rather than indicated prevention approach, and assess changes in attitudes rather than actual suicide-related behaviors ( [Mazza, 1997](#)).

Another group of researchers has developed and tested a series of programs, the Reconnecting Youth Prevention Research Programs, designed to reduce suicidal potential among high-risk high school students by providing a combination of social support with skills training. [Eggert et al. \(1995a\)](#) began working with youth at risk for dropping out of high school. Evaluation of these young people revealed that they differed from typical adolescents not only in terms of school attendance and

grades, but 40% endorsed more depression and suicide risk as defined by previous suicide attempt or high suicidal ideation (Eggert et al., 1994b). They then evaluated the efficacy of an in-depth assessment protocol, the Measure of Adolescent Suicide Potential (MAPS) (Eggert et al., 1994b), versus a one- or a two-semester school-based program Reconnecting Youth (RY) that incorporated social support and life skills training to decrease depression and suicidal risk behaviors, improve school performance and attendance, and decrease drug involvement. Participants were students identified as at risk for high school dropout because of low grades and poor attendance and who also endorsed depression or suicide risk behaviors on a second screen. The program was administered as a "for credit" class in the student's high school schedule and was delivered in small groups of 12 participants with teachers selected for their ability to be supportive and to help students form into a supportive and effective prosocial working group. Additional core components included regular monitoring of mood, school behaviors, and drug use as well as skills training in four areas: self-esteem enhancement, decision making, personal control (anger, depression, stress management), and interpersonal communication. The one- versus two-semester programs differed in that the second semester expanded students' involvement in activities at school to increase their school bonding, and extended the use of skills learned in the first semester to a broader range of situations in the students' lives. The MAPS is a 2-hour, computer-assisted interview followed by a crisis intervention/social connection protocol to ensure the safety of the suicide-vulnerable youth. In that component, students who revealed significant suicide risk were connected with a school counselor for additional in-school services, and parents were notified. One hundred five students were compared across the three experimental conditions as well as with a contrast group (n = 202) of nonrisk students from the same high schools.

This study used a three-group repeated-measures design; trend analysis served to assess patterns of change for youth in the three study conditions across three points in time (baseline, 5 months, and 10 months). All groups showed significant declines in suicide risk behaviors, depression, hopelessness, stress, and anger, as well as increases in self-esteem and network social support, suggesting that the MAPS assessment was effective for decreasing suicidal behaviors and related emotional distress factors. Increased personal control was observed only in the experimental RY groups and not in the MAPS-only condition. This study demonstrated the efficacy of the RY prevention classes and the therapeutic potential of MAPS. More recent work has explored the mediating effects of the RY prevention program (Thompson et al., 2000). It was hypothesized that intervention effects would be influenced by the direct and mediating effects of teacher social support on peer group support and perceived personal control. Personal control was enhanced by teacher and peer group support and in turn contributed to reduced depression and suicide risk behaviors. A structural equation model was used to test the direct and mediating effects of RY teacher social support on peer support and personal control, thereby, on suicide risk behaviors and depression. As hypothesized, teacher social support directly enhanced peer group support, with a stronger effect evident in the intervention groups versus the control (MAPS) group. Peer group support enhanced personal control and thereby indirectly decreased depression and suicide risk behaviors, and personal control mediated the link between intervention and depression in the two-semester RY group.

Based on these findings, Eggert and colleagues developed a condensed version of the RY class, Coping and Support Training (CAST), which included the key elements of peer group and teacher support, monitoring, and skills training. CAST is administered in groups of 6 to 8 students, with 10 sessions over a 5-week period. Eggert and colleagues (Randell et al., 2001; Thompson et al., 2001) then tested CAST versus the MAPS only (now referred to as Counselor's CARE, or C-CARE) versus "usual care" control subjects using a larger sample of suicide-vulnerable youth. The impact of the three groups on reduction of suicide risk behaviors and improvement of individual mediators (personal control, problem solving coping, and self-esteem) and family mediators (family support, family goals met, and family distress/conflict) was tested using repeated-measures design (preintervention, post C-CARE at 4 weeks, and post CAST at 10 weeks). Subjects were 460 suicide-vulnerable youth randomly assigned to one of the three groups (C-CARE, CAST, and usual care/control). Findings revealed significant decreases in suicide risk behaviors and emotional distress indicators for all three groups. Significant intervention effects were reported for the posited personal mediators (increased personal control and problem-solving coping) and posited family mediators (increased family support and family goals met) as well.

These results provided preliminary support for the brief C-CARE and CAST models. The CAST intervention was superior only in enhancing the posited mediators of personal control, problem-solving coping, family support, and family goals, effects that appear related to specific skills training in CAST. Given the efficacy of these brief intervention models, the RY group currently is testing Counselor's Care in contrast to C-CARE paired with Parents' Care (P-CARE) a brief parent intervention (Randell BP, personal communication, 2001). P-CARE provides a structured intervention for parents of suicide-vulnerable youth that is delivered in two home visits. Parents are given gatekeeper-like training in how to recognize suicide warning signs and ask their teens about suicide while showing care and concern. A second home visit provides training in communication and problem-solving skills. Results are not yet available from this ongoing project.

### **Additional Suicide Prevention Approaches**

In addition to the development and testing of specific preventive intervention programs, a broader level of responses to the issue of suicide prevention has been evident since the early 1990s. Grossman and Kruesi (2000) have categorized recent youth suicide prevention strategies into public health, grassroots, means restriction, gatekeeper training, mental health, hospital safety, and community action approaches. The following sections review findings on the current status of these various strategies.

#### *PUBLIC HEALTH*

The public health approach focuses on reducing suicide. This is clearly stated in the U.S. government's *Healthy People 2000* (U.S. Department of Health and Human Services, 1999) and 2010 campaigns as well as the active writing and public speaking campaign highlighting the need for a public health approach to suicide prevention, which the former Surgeon General, Dr. David Satcher, has actively pursued (Satcher, 1998).

#### *GRASSROOTS*

Complementing these efforts has been the growth of grassroots programs, which on both the state and local level have pulled together relatives of those who have committed suicide with academic and governmental institutions. These initiatives, as represented in the work of Suicide Prevention Advocacy Network, a nonprofit organization begun by the parents of a young woman lost to suicide in 1996, have been very active in promoting a national suicide prevention initiative and bringing the need for suicide prevention to the attention of politicians and policy makers (Grossman and Kruesi, 2000).

#### *MEANS RESTRICTION*

Growing out of the initial studies demonstrating the strong connection between ready access to a firearm and rates of completed suicides, efforts have expanded to educate the public in general, and parents of at-risk youth specifically, about firearms. These efforts have included a task force on handguns and youth suicide as well as an educational campaign sponsored by the American Academy of Pediatrics that focused on increasing awareness among pediatricians of the need to ask about access to firearms and educate about firearm safety as part of routine pediatric practice. Only a few studies have explored the efficacy of education campaigns. Brent et al. (2000) reported on compliance with the recommendation to remove firearms from the home among parents of 106 adolescents with major depression who were participating in a treatment of depression research project. Parents who reported having firearms in the home were educated about the strong risk of suicide among depressed youth and the increased risk of suicide given the presence of a firearm, and were encouraged to remove the firearms. Only 26.9% of the families followed through with this request, even though they were compliant with the many other demands of the research study. The authors concluded that the brief educational intervention used was not adequate and emphasized the need for education that includes a variety of firearm storage options and is directed specifically at the parent who is the primary gun owner. Another study (Kruesi et al., 1999; McManus et al., 1997) investigated education about means restriction provided in emergency departments (EDs). They found that although only a few centers (14%) documented providing means restriction education to parents of youth brought for care after a suicide attempt by drug overdose, education resulted in a significant increase in parents who secured medications.

#### *GATEKEEPER TRAINING*

Several programs since the early 1990s have focused on increased gatekeeper training efforts, typically in conjunction with other prevention strategies. A comprehensive training program has been developed and extensively used in Canada, the LivingWorks Applied Suicide Intervention Skills Training (ASIST) (Ramsey et al., 1996). This training program has been tested in two comprehensive youth suicide prevention initiatives, one in the state of Washington (Eggert et al., 1999) and the other in Australia (Turley and Tanney, 1998; Turley et al., 2000a, 2000b)

Based on a statewide needs assessment, Washington State implemented a three-pronged youth suicide prevention program, including a public education campaign, gatekeeper training for adult caregivers, and enhancements to existing crisis services. These efforts provide important information about how best to spend limited public health dollars. Gatekeeping training was done first with adult caregivers and then with high school students in four demonstration projects. Compared with extensive public education campaigns, gatekeeper training of both youth and adults resulted in greater gains in the desired program outcomes of increased public awareness about youth suicide prevention, knowledge of suicide warning signs, and ability to initiate prevention steps. Finally, enhancement of crisis services was the



most challenging aspect of the program. Training of crisis services workers was effective in increasing competencies in assessing level of suicide risk, but enhancing access to youth-friendly crisis services was difficult given the limited funding and high use of existing services.

In 1995, the Australian Commonwealth provided funding for field trials of two gatekeeper training programs developed by LivingWorks ASIST, a 1- to 3-hour Suicide Aware program and the 2-day Suicide Intervention workshop. These programs use a "train the trainer" model. Initially, 70 individuals were trained, who in turn completed 2,870 Suicide Intervention workshops and 3,972 Suicide Aware trainings. Thus, the program was highly successful in creating opportunities to increase suicide awareness in the target communities. Efficacy of the Suicide Intervention workshops was assessed by self-report measures administered before, immediately after, and 4 months after program completion. Targeted outcomes were gatekeepers' readiness to help a person at risk, suicide assessment and intervention knowledge, and attitudes and beliefs affecting the gatekeepers' suicide prevention role. Measures of readiness to help (self-report of comfort, competence, and confidence), intervention knowledge (19-item Intervention Knowledge questionnaire), and intervention beliefs (Suicide Beliefs Questionnaire assessing two factors: willingness to help and optimism–pessimism about effective outcome) all increased significantly from preworkshop to postworkshop assessments, and gains remained stable at the 4-month evaluation. Although the longer-term follow-up data were encouraging, only 30% of the initial sample and only subsamples of the overall group completed the Intervention Knowledge and Suicide Beliefs questionnaires ( [Turley and Tanney, 1998](#)). To assess further the efficacy of the workshop, a more comprehensive assessment was completed on 91 participants to determine changes in their capacity to identify evidence of suicide risk, accurately estimate level of risk, demonstrate knowledge of the intervention process, and identify and articulate a plan of action. A comparison group of 40 individuals from similar professional/support role backgrounds who did not attend the workshop also was included. Capacity to apply information/skills presented in the workshop was assessed using two videotaped simulations; participants were rated by evaluators on their response to the simulations in terms of understanding of issues, assessment of risk, and knowledge about how an effective intervention strategy might be applied. The experimental and control groups did not differ at baseline, but at the postworkshop assessment the experimental group demonstrated significant gains in their self-declared readiness to make a suicide intervention and in their competencies as reflected in responses to the simulations, whereas the contrast group demonstrated no change. The investigators concluded that this evidence supports the efficacy of the ASIST approach, but acknowledged the need for longer follow-up evaluations that assess the effectiveness of these programs on actually altering rates of suicide (Tanney B, personal communication 3/23/2001).

Another program, Community Action for Youth Survival, combined gatekeeper training with means restriction and postvention efforts ( [Grossman and Kruesi, 2000](#)), and targeted school and health care professionals. Evaluation of the program indicated that participants reported increases in knowledge of suicide prevention practices that were retained over a 4- to 5-month period and that led to changes in their practices with teens. For example, they were more likely to make follow-up plans for at-risk youth and to talk with parents about means restriction. Efforts to disseminate the program nationwide through an interactive educational CD-ROM suggest that this approach alerts viewers to the problem of who then needs direct training ( [Grossman and Kruesi, 2000](#)).

## MENTAL HEALTH

Studies continue to provide modest support for the efficacy of suicide prevention hotlines. [Becker \(1997\)](#) examined the effectiveness of suicide prevention center telephone interventions in reducing the suicidality of a sample of 129 participants, ranging in age from 14 to 66 years. Evaluative data were obtained at the beginning and end of the telephone intervention and again 1 week later. Findings revealed significant reduction in suicidality both at the end of the call and over the 1-week follow-up period. Unlike prior studies, no sex differences were found. Similarly, [Turley et al. \(2000a\)](#) evaluated the results of an active campaign to promote Lifeline Australia and enhance the competency of the phone counselors. Calls regarding suicide made up 4% to 8% of the calls made across the 42 call centers and increased 41% during the active campaign, but declined gradually when the campaign ended. A follow-up study of a subset of callers revealed overall satisfaction with the counselor's response; most of the callers sampled in the follow-up study reported following the options for further care suggested by the counselor.

However, in a review of 14 studies examining whether suicide prevention centers have an effect on suicide rates, [Lester \(1997\)](#) found that only seven studies provided support for a preventive effect. Methodologic differences and limitations across projects make it difficult to come to a final conclusion, but Lester argued that suicide prevention centers have a small positive effect on reducing rates of suicide. It appears that concerns similar to those raised by [Shaffer et al. in 1988](#) remain in relation to crisis telephone services ( [Seeley, 1997](#)). These include the need for better training and supervision of the staff and volunteers providing these services, and the need for policies guiding hotlines in the legal and ethical issues of suicide prevention and crisis intervention services ( [Seeley, 1997](#)). Even with these drawbacks, however, suicide prevention scholars continue to include crisis and telephone hotline services as one of the important elements in youth suicide prevention efforts ( [Goldney, 1998](#)). Because of the impulsive nature of most youth suicidal behavior, the ready access to a care provider who can serve as a deterrent remains a critical component of prevention services.

Other programs that fall in this category include emergency department (ED)- and hospital-based programs for youth who present with suicidal ideation or attempts. As stated previously, suicide attempts are common among adolescents. Moreover, youth that make a suicide attempt are at increased risk for future attempts as well as for eventually committing suicide ( [Shaffer and Piacentini, 1994](#)). There are, however, few systematic studies of the treatment of suicidal adolescents, in part because these youth and their families frequently fail to follow through with the treatment recommendations made and appointments scheduled at the time of the suicide gesture ( [Trautman et al., 1993](#)).

[Rotheram-Borus and colleagues \(1994\)](#) have developed and tested a two-part intervention designed to decrease risk of repeated suicide gestures/completed suicide among Hispanic adolescent girls who present in an ED/clinic having made a suicide gesture. The first component of their program is ED based and includes training of ED staff; an educational video for parents and teens to view while in the ED aimed at increasing their understanding about youth suicide, the need for treatment, and what to expect from treatment; and an on-call therapist who works with the family while in the ED and helps with their transition to outpatient treatment. The second component is a six-session, cognitive–behavioral treatment program developed for working with suicidal youth and their families. The program, Successful Negotiation/Acting Positively (SNAP), is designed to increase positive feelings and problem-solving skills among family members while also demonstrating that therapy can be an effective tool so as to increase the probability that these families will seek help again when problems arise. The first component, directed at the family's initial contact in the ED, has proven more effective than standard ED care as reflected in reports of greater declines in depression and suicidal ideation and greater adherence with participation in follow-up treatment among girls in the specialized ED care, as well as more positive attitudes regarding treatment among their mothers ( [Rotheram-Borus et al., 1996](#)). Furthermore, recent data suggest that both the suicidal adolescent girls and their mothers who were exposed to the specialized ED care continued to report lower levels of depression at the 18-month follow-up assessment than did those who received traditional ED care. The specialized ED care was particularly associated with a decline in distress among mothers of highly symptomatic girls. Number of therapy sessions attended post-ED visit did not affect outcome, but the efficacy of SNAP *per se* has not been fully evaluated because all subjects were included in this component of the program ( [Rotheram-Borus et al., 2000](#)).

Finally, in the mental health arena, screening youth to increase identification and referral of youth with mental health problems has been recommended as a more effective approach to suicide prevention than universal, school-based education programs ( [Shaffer and Craft, 1999](#); [Shaffer et al., 1988](#)). Screening programs, however, have not been implemented yet because of the complex issues involved. Given the fluctuating nature of adolescents coupled with the fact that at least some key disorders that should be screened for, such as depression, are episodic, there are questions about how best to determine the timing and frequency of screening as well as concerns over ensuring that services are readily available for youth if a screening program were initiated ( [Hayden and Lauer, 2000](#)).

## Future Directions in Suicide Prevention

[Berman and Jobes \(1995\)](#) recommended that as we move forward, suicide prevention must be viewed from a broad approach that includes primary, secondary, and tertiary prevention efforts. At each of these levels, they suggest strategies for addressing individual predisposition, social milieu, and "proximal agents," which refers to efforts to educate caretakers about the risks associated with firearms as well as medications. [Table 66.1](#) summarizes their approach. One example of this kind of broad-based perspective has been developed and tested in the Dade County School District ( [Zenere and Lazarus, 1997](#)). Their program involves a comprehensive model of suicide prevention adopted by the school board and administration, with components that range from a comprehensive curriculum integrated into the district's health education program beginning in kindergarten to trained school-based crises teams. The curriculum addresses several themes: self-awareness development, decision-making skills, drug information, and development of positive alternatives and assertiveness, conflict resolution, and stress management skills. Suicidal behavior is not formally introduced until the 10th grade, when it is discussed as one unit of a life management skills class that all students take for one semester. The focus is on prevention strategies. The program was evaluated by comparing school district data on the number of students who completed or attempted suicide from 1980 to 1988 with the same data from 1989 to 1994, the first 4 years in which the program was operating. [Zenere and Lazarus \(1997\)](#) reported a decline of 67% in the average annual number of suicides as well as a significant decline in known suicidal attempts. Rates of suicidal ideation, however, declined during the first 2 years of the program and then returned to levels seen at the time of the program's inception. The investigators found that during the second 2 years of their study (1992 to 1994), the highest levels of suicidal ideation were reported by students in grades 6 to 8, and attempts were most common in students in grades 7 to 9, suggesting that the presentation of specific suicide prevention material should be in early middle school rather than 10th grade.

	Primary Prevention	Secondary Prevention	Tertiary Prevention
Primary prevention	Depression management Algebra management Cognitive-behavioral therapy Problem-solving training Coping enhancement Critical thinking skills Help-seeking training Relaxation Counseling Outpatient treatment	Depression management Early detection/initial of parents Coping skills Supportive therapy Family therapy Case finding Cognitive therapy Cognitive-behavioral therapy Crisis intervention programs Case management follow-up	Gun safety training for parents and adolescents Suicide awareness among health care providers Parent/teen/peer educator education Medication limited Subsistence skills Decrease access to guns
Secondary prevention	Psychiatric treatment Substance abuse treatment	Psychiatric treatment Crisis intervention programs Case management follow-up	Substance abuse treatment Psychophysics for diagnosis Neuroleptics for psychosis

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**Table 66.1. A Conceptual Model of Prevention Strategies**

In sum, suicide prevention requires the broad-based approach outlined in this review. It is critical that ongoing efforts address not only suicidal or risk-taking thoughts and behaviors *per se* but the myriad of individual, environmental, and societal risk factors that contribute to the suicide vulnerability of today's youth. Protective factors also must be activated—hence the importance of enhancing social problem-solving and emotion regulation skills as well as the young person's sense of personal competence and access to family support. The next generation of youth suicide prevention efforts must continue to draw on a wide range of prevention strategies that include universal, selective, and indicated prevention approaches.

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## 67 ANXIETY DISORDERS

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Anxiety disorders in children and adolescents are a prevalent, understudied, and perhaps underreported phenomenon. Estimates are that 10% to 20% of school-aged children exhibit symptoms of social withdrawal, anxiety, isolation, hypersensitivity, depression, and self-consciousness ( [Anderson et al., 1987](#); [Bernstein et al., 1990b](#); [Costello, 1989](#); [Kashani and Orvaschel, 1988](#); [McGee et al., 1990](#); [Orvaschel and Weissman, 1986](#); [Werry, 1986](#)). Anxiety disorders are associated with a negative impact in multiple domains such as low self-esteem, social isolation, inadequate social skills, and problems in academic work ( [Klein and Last, 1989](#); [Strauss et al., 1989](#)). Youth with anxiety disorders often experience debilitating physical symptoms such as headaches, stomachaches, and irritable bowel syndrome ( [Livingston et al., 1988](#)). Not only do child anxiety disorders have the potential to persist over time ( [Cantwell and Baker, 1989](#); [Keller et al., 1992](#)), but there is evidence that they tend to worsen over time ( [Strauss et al., 1988](#)). The presence of anxiety disorders in childhood also may confer a general risk for other forms of psychopathology, both concurrently, as well as later in life ( [Barlow et al., 1996](#); [Cole et al., 1998](#)). These observations highlight the importance of addressing these problems in children.

For most forms of psychopathology, knowledge about and research evidence from work with adults is better developed than that with children and adolescents. The temptation often is to extrapolate downward the information gained from work with adults. As is discussed shortly, with certain forms of anxiety, this has turned out to be an appropriate strategy, whereas with other forms of anxiety it has been less effective for understanding the problems as they affect youth. Until the publication of the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) in 1994 ( [American Psychiatric Association, 1994](#)), the study of childhood anxiety disorders was separate and fairly disconnected from the study of adult anxiety. At that point, separation anxiety disorder (SAD) became the only anxiety diagnosis specific to children alone, while the other childhood anxiety disorders were integrated into the lifelong “adult” diagnoses.

This approach to anxiety disorders “across the life span” makes sense in the context of increased efforts to identify early risk factors for anxiety. However, the practice of extrapolating down adult-derived conceptualizations to fit children (and thereby integrating these two separate fields) often fails to recognize the developmental trajectory that may underlie both children's pathology, as well as the more normative aspects of their development. This point is particularly salient when considering anxiety in children and adolescents because there are age-appropriate, nonpathologic manifestations of fear and anxiety that change over the course of development.

In the first section of this chapter, anxiety is broadly defined and an overview of developmental considerations for examining both normative and pathologic forms of anxiety in youth is offered. General epidemiologic data regarding prevalence, incidence, typical demographic features, and patterns of comorbidity are presented. The following are considered: SAD, social phobia, generalized anxiety disorder (GAD), specific phobias, panic disorder with and without agoraphobia, and school refusal [obsessive–compulsive disorder (OCD) and posttraumatic stress disorder (PTSD) are covered in separate chapters]. This is followed by an overview of treatment, including pharmacologic and psychosocial treatment studies. A discussion of etiologic theories of child anxiety disorders is beyond the scope of this chapter. Traditionally, these include biological risk factors, psychosocial risks, and learning theories. The reader is referred to [Chapter 25](#) for a review of these theories.

### DEFINITION AND DEVELOPMENTAL PERSPECTIVE

Anxiety may be broadly defined as “emotional uneasiness associated with the anticipation of danger” ( [Livingston, 1991](#)). In contrast to many other forms of psychopathology, anxiety is considered a normal emotion throughout development and plays both a protective and adaptive role ( [Eisen and Kearney, 1995](#); [Labellarte et al., 1999](#)). Fear usually is considered a normal reaction to a real or imagined danger or threat (e.g., fear of the dark in a young child) ( [Eisen and Kearney, 1995](#); [Morris and Kratochwill, 1991](#)). Indeed, neuroethologic models suggest that anxiety is necessary for “survival of the species” ( [Leckman and Mayes, 1998](#)). It is not uncommon to see fears as well as isolated subclinical anxiety symptoms develop and change over time in most children and adolescents, and transitory fears and anxieties are widely accepted as part of normal child development ( [Bernstein et al., 1996a](#); [Craske, 1997](#); [Last et al., 1996](#); [Silverman and Nelles, 1990](#)). These may include fears of harm to self or others, excessive worry about a particular situation, or separation anxiety ( [American Academy of Child and Adolescent Psychiatry, 1997a](#); [Bell-Dolan et al., 1990](#)).

A great deal of research has investigated developmental patterns of normal fear in children and adolescents ( [Evans et al., 1997](#); [Gullone, 2000](#); [Ollendick, 1983](#); [Ollendick et al., 1985](#); [Reed et al., 1992](#)). Results suggest that certain fears and anxieties are more common at some ages than others, although not as a function of age exclusively. Beginning in infancy, young children are fearful of stimuli in their immediate environment. By 12 months of age, fears begin to include strangers, strange places, and heights. In the preschool years, children become fearful of being alone, the dark, animals, and imaginary creatures. Once children enter school, it is common to see fears of supernatural phenomena, evaluative or social situations, and bodily injury/illness or natural disasters.

Because many childhood fears play a normal, adaptive, and protective role, the distinction between normal and abnormal child anxiety is not always clear ( [Bernstein et al., 1996a](#); [Klein, 1994](#)). The extent to which irrational fears or worries cause significant distress or impairment in academic or social functioning is important. The timing of symptoms also must be considered in a developmental context (e.g., mild separation anxiety means something different in a young child than in an older adolescent). In addition to using these factors to help differentiate normal from pathologic anxiety, it also is important to acknowledge that several symptoms associated with anxiety disorders may be present in individuals who do not qualify for anxiety diagnoses. In fact, some researchers and clinicians have supported the use of dimensional diagnostic systems as opposed to the categorical system provided in DSM-IV as a means of capturing the level of symptomatology present in a given individual.

Anxiety researchers also make distinctions among anxiety, fears, and phobias. In contrast to fears, phobias involve excessive, specific, persistent fear of a stimulus as well as significant avoidance or distress ( [American Academy of Child and Adolescent Psychiatry, 1997a](#)). Phobic reactions are disproportionate to the demands of the situation, impervious to reasoning, and often occur outside the normal developmental period of a fear (e.g., fears of monsters in an older child). Unlike fears and phobias, anxiety is more diffuse and lacking in specificity ( [Kendall et al., 1991](#)). Although some amount of anxiety is normative and adaptive in many situations, anxiety “disorders” involve irrational fear or worries that cause significant distress or interference in functioning.

There are many different types of anxiety symptoms and diagnoses. Notably, symptoms of anxiety also are seen in psychiatric and medical conditions in addition to those officially categorized as anxiety disorders ( [Table 67.1](#)). Anxiety symptoms and diagnoses present with a range of cognitive, behavioral, and physiologic symptoms. These include excessive fears and worries that can fall in a number of categories, including phobic avoidance, panic attacks, social or stimulus specific fears, and generalized vigilance/anticipatory anxiety ( [Table 67.2](#)).

Disorders usually first diagnosed in infancy, childhood or adolescence
Separation anxiety disorder
Selective Mutism
<b>*Adult diagnoses that include children</b>
Generalized anxiety disorder
Social phobia
Specific phobia
Obsessive-compulsive disorder
Posttraumatic stress disorder
Acute stress disorder
Panic disorder with and without agoraphobia
Anxiety disorder due to a general medical condition
Substance-induced anxiety disorder
Anxiety not otherwise specified
<b>Other disorders with anxious features</b>
Hypochondriasis
Somatization disorder
Adjustment disorder with anxious mood

**Table 67.1. DSM-IV Anxiety and Anxiety-Related Disorders**

Cognitive	Behavioral	Physical
Fearful	Restless	Cardiovascular (e.g., racing heart)
Nervous	Clingy	Respiratory (e.g., difficulty breathing)
Stressed	Dependent	Skin (e.g., sweating)
Fretful	Shy	Musculoskeletal (e.g., stiff neck)
Self-defeating	Withdrawn	Gastrointestinal (e.g., irritable bowel)
Difficulty concentrating	Reluctant	Headache
Inattentive	Avoidant	Dizziness

**Table 67.2. Signs and Symptoms of Anxiety**

## PREVALENCE AND EPIDEMIOLOGY

Overall, anxiety disorders are considered to be one of the most common psychiatric diagnoses in children and adolescents (Albano et al., 1996; Bernstein and Borchardt, 1991). In community samples, anxiety disorders have been shown to occur in 5% to 18% of children (Benjamin et al., 1990; Costello and Angold, 1995). In preadolescent children, the prevalence is 0.3% to 12.9%, depending on the disorder (Anderson et al., 1987; Craske, 1997; Kashani et al., 1989). For adolescents, prevalence ranges are from 0.6% to 7%, depending on the disorder (Bell-Dolan and Brazeal, 1993; Lewinsohn et al., 1993). Specific phobias, SAD, and GAD tend to be the most prevalent.

There is very limited information available with regard to sex, age, socioeconomic status (SES), and ethnicity as they relate to child anxiety disorders. With regard to sex, in general community samples, girls outnumber boys in terms of number of fears (Craske, 1997). This may, in part, be the result of socialization and parent modeling. In terms of anxiety diagnoses, in community samples, girls outnumber boys except in the diagnosis of OCD (American Academy of Child and Adolescent Psychiatry, 1997a). However, there appears to be less of a sex differential for children and adolescents in clinical settings (Last et al., 1992; Strauss and Last, 1993).

With regard to age, SAD has an earlier age of onset than the other anxiety disorders (Last et al., 1992). Younger children with SAD also tend to have more symptoms and different symptoms than older children (Francis et al., 1987). In contrast, older children with GAD have been shown to have more symptoms than younger children with GAD (McGee et al., 1990). These limited data also parallel the normative developmental trajectory presented earlier. There are no current data describing the relationship between age and other anxiety disorders.

Research on the connection between SES and ethnicity and their relationship to child anxiety has focused mostly on normal fears. For example, lower SES children have been shown to have more fears and more intense fears than higher SES children (Gullone, 2000). Some authors also have found differences in the development of normal fears in cross-cultural studies (Dong et al., 1994; Neal et al., 1993). For example, African-American children report fewer concerns about school- or embarrassment-related fears than white children. It also has been suggested that there may be some connection between SAD and lower SES (Last et al., 1992; Velez et al., 1989). There also may be higher rates of GAD in middle and upper class white children (Last et al., 1987a). Finally, African-American children may be less likely to present for treatment for school refusal (Last and Perrin, 1993). Again, we have no research with regard to SES and other anxiety disorders. Clearly, further study is needed to understand better these important issues.

## COMORBIDITY

As with adults, anxiety disorders in youth frequently occur simultaneously with each other and with other types of psychopathology. Rates of comorbidity among anxiety disorders tend to be somewhat lower in the general population—39% in children (Anderson et al., 1987; Kashani et al., 1990) and 14% in adolescents, (McGee et al., 1992)—than in clinic samples (50%) (Last, 1987). After comorbidity with another anxiety disorder, depression is the most commonly reported comorbid condition among youth with anxiety disorders. In their review of the relationship between anxiety and depression in children and adolescents, Brady and Kendall (1992) found that 15.9% to 61.9% of children identified as anxious or depressed had the other disorder as well. Differential diagnosis among anxiety and other childhood disorders is a significant concern as well. (This issue is discussed in detail for each specific disorder addressed later in this chapter.)

Anxiety disorders also can occur with externalizing disorders such as attention deficit/hyperactivity disorder, conduct disorder, and oppositional defiant disorder. One group of researchers has reported that approximately 20% of cases with an anxiety disorder also meet criteria for an externalizing disorder (Last et al., 1992; Last et al., 1987b). Epidemiologic data support the conclusion that anxiety and behavior disorders co-occur at beyond chance levels (Anderson et al., 1987; McGee et al., 1990).

## GENERAL ASSESSMENT ISSUES

In addition to a standard psychiatric assessment, a comprehensive anxiety evaluation should pay careful attention to a number of specific issues. These include (a) whether the anxiety is stimulus specific, spontaneous, or anticipatory; (b) the degree of avoidance in daily life caused by the anxiety symptoms; (c) social and familial context and reinforcers of symptoms; (d) temperament, quality of attachment, stranger/separation responses, and childhood fears; (e) medications or medical disorders that may cause anxiety symptoms; and (f) family history of anxiety disorders (American Academy of Child and Adolescent Psychiatry, 1997a).

Useful guidelines for both a general child psychiatric examination as well as an assessment specific to child anxiety can be found in the American Academy of Child and Adolescent Psychiatry's "Practice Parameters for the Psychiatric Assessment of Children and Adolescents" (American Academy of Child and Adolescent Psychiatry, 1997b) and "Practice Parameters for the Assessment and Treatment of Children with Anxiety Disorders" (American Academy of Child and Adolescent Psychiatry, 1997a). The medical workup for an anxious child should include a review of visits to physicians for the presenting symptoms, medications (prescription, over the counter, herbal and health supplements) taken by the patient that could produce anxiety symptoms, and physical conditions that may mimic anxiety disorders (e.g., documented hypoglycemic episodes, hyperthyroidism, cardiac arrhythmias, caffeineism, pheochromocytoma, seizure disorders, and migraine) (American Academy of Child and Adolescent Psychiatry, 1997a).

Once it has been established that a child or adolescent may be experiencing fear or anxiety beyond that expected for someone of his or her age or developmental level, the clinician must begin a more careful review of symptoms. In doing so, there are several issues that may be encountered. Children and young adolescents may be less likely to recognize that their fear response is excessive or to describe themselves as in "distress." In addition, children and young adolescents may show their anxiety in very different ways than adults (e.g., crying, tantrums, irritability, somatic complaints). Finally, children and young adolescents may have difficulty



reporting on their experiences. It is crucial to use developmentally appropriate questioning and examples and to seek additional sources of information (e.g., parents, observational measures).

A comprehensive clinical assessment also might include a structured or semistructured psychiatric interview to confirm anxiety diagnoses and other comorbid disorders. In addition, clinician rating scales, self-report scales, parent-report measures, and observational measures may help to determine the specific nature and severity of anxiety symptoms. The overall goal of these assessment measures should be to (a) provide reliable and valid assessment of symptoms across multiple domains, (b) discriminate symptom clusters, (c) assess severity, (d) incorporate multiple observations (e.g., parent and child), and (e) be sensitive to treatment-related changes in symptoms (March et al., 1997; Stallings and March, 1995).

### Diagnostic Interviews for Child and Adolescent Anxiety Disorders

A number of general diagnostic interviews for children and adolescents include sections/modules on anxiety disorders. These include the Schedule for Affective Disorders and Schizophrenia for School Age Children—Epidemiologic Version (K-SADS-E) (Orvaschel, 1995), the Schedule for Affective Disorders and Schizophrenia for School Age Children—Present and Lifetime Version (K-SADS-PL) (Kaufman et al., 1997), the Diagnostic Interview for Children and Adolescents—Revised (DICA-R) (Reich, 1997), and the National Institute of Mental Health Diagnostic Interview Schedule for Children (DISC) (Schaffer et al., 1996). Only the Anxiety Disorders Interview Schedule for DSM-IV—Child Version (ADIS-IV) (Silverman and Albano, 1996) is designed specifically to evaluate anxiety disorders in youth.

### Clinician Rating Scales

A number of clinician rating scales have been developed to assess child and adolescent anxiety disorders (Greenhill et al., 1998). These measures allow for clinician ratings of general symptoms of anxiety (e.g., social phobia, specific phobia, GAD). Symptoms of OCD and PTSD usually are not included. Many of these instruments are based on composite interviews of both child and parents, and include the Hamilton Anxiety Rating Scale (HARS) (Hamilton, 1959), the Pediatric Anxiety Rating Scale (PARS) (Riddle and Greenhill, 1997), and the Anxiety Rating Scale for Children—Revised (ARSC-R) (Bernstein et al., 1996b).

### Self-Report Measures

A number of self-report measures have been developed to assess the severity of both generalized anxiety and fearfulness in children and adolescents. In addition, many instruments exist that measure the severity of symptoms specific to individual disorders (e.g., social anxiety, posttraumatic stress reactions, obsessions and compulsions). Examples of general self-report measures include the Multidimensional Anxiety Scale for Children (MASC) (March et al., 1997), the Revised Children's Manifest Anxiety Scale (RCMAS) (Reynolds and Richmond, 1978), the State-Trait Anxiety Inventory for Children (STAIC) (Spielberger, 1983), and the Screen for Child Anxiety-Related Emotional Disorders (SCARED) (Birmaher et al., 1997), which also has a parent-report version. A number of more specific measures of anxiety deserve brief mention. These include the Fear Survey Schedule for Children—Revised (FSSC-R) (Ollendick, 1983), the Social Anxiety Scale for Children—Revised (SASC-R) (LaGreca and Stone, 1993), and the Social Phobia and Anxiety Inventory for Children (SPAI-C) (Beidel et al., 1995).

## SPECIFIC DISORDERS

### Separation Anxiety Disorder

#### DEFINITION AND CLINICAL DESCRIPTION

Separation anxiety disorder is the only childhood anxiety disorder retained in DSM-IV under “Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence” (American Psychiatric Association, 1994). The essential clinical feature of SAD is tremendous fear and anxiety regarding being apart from home or from primary attachment figures. The anxiety must be inappropriate for the child's age and developmental level and must last at least 4 weeks. It is particularly important to distinguish normal from abnormal separation anxiety, given that such anxiety is an age-appropriate phenomenon from approximately 7 months to 6 years of age (Albano et al., 1996; Bernstein and Borchardt, 1991; Rabian and Silverman, 1995). The reported prevalence of SAD is in the range of 2.4% to 5.4% in most studies (Anderson et al., 1987; Benjamin et al., 1990; Bowen et al., 1990; Costello, 1989).

Children with SAD may show distress when separation is anticipated or occurs, or will try to avoid separation situations altogether. Distress may take the form of terror or autonomic arousal. Children with SAD often resist separation by clinging, crying, begging, or complaining of somatic distress. The underlying fear is that harm may come to the attachment figure or the child, resulting in permanent separation. School refusal and excessive somatic complaints are the most common reason for parents and children to seek treatment.

#### DEVELOPMENTAL PERSPECTIVE

Separation anxiety disorder is found most often in prepubertal children, but can be diagnosed at any age before 18 years (Albano et al., 1996; Kashani and Orvaschel, 1988). As noted earlier, the distress or impairment in functioning related to separation must be greater than what would be considered developmentally appropriate. Subclinical SAD symptoms are thought to be relatively common in nonclinical samples (Bell-Dolan et al., 1990). Francis and colleagues (1987) found that the age of the child did differentiate both the specific symptomatology and the total number of symptoms in clinical samples of youth with SAD, with younger children showing more symptoms and greater distress.

#### DIFFERENTIAL DIAGNOSIS/COMORBIDITY/RELATED SYMPTOMS

The differential diagnosis of children presenting with difficulty separating from parents, avoidance of school or other situations involving separation, or excessive distress on separation should include, in addition to SAD, a number of anxiety as well as other disorders. The primary focus of the fear in SAD must be related to the threat of or actual separation from primary attachment figures. Occasionally, OCD may present looking like SAD, but usually other symptoms of OCD would be present.

Children with SAD often may be diagnosed with other comorbid anxiety disorders, as well as depression (Last et al., 1987b). Overanxious disorder (now GAD in DSM-IV), as well as specific phobia appear to be the most common comorbid anxiety problems (Last et al., 1987a). Children with SAD often present with a number of specific fears (e.g., monsters, the dark, animals), even if the fears are not serious enough to warrant a phobic diagnosis (Last, 1989). In addition, SAD is the most common anxiety diagnosis in samples of depressed prepubertal children (Keller et al., 1992; Kovacs et al., 1989). Other related features include somatic complaints (Last, 1991) as well as decreased participation in extracurricular activities and time spent with friends (Albano et al., 1996). Academic performance also may be affected because of preoccupation with separation during the school day, or refusal to attend school.

#### COURSE AND OUTCOME

There is not a great deal of research with regard to the long-term course of SAD (Cantwell and Baker, 1989; Keller et al., 1992). However, SAD is seen in general as having a variable course, with possible exacerbations at times of stress or transition. In some children with SAD, there appears to be complete recovery with little residual impairment. This may be particularly true for children in whom the disorder develops at younger ages (Black, 1995). Others may have a chronic course characterized by periodic exacerbations and recurrences or more persistent symptoms.

A later age of onset, comorbidity with other psychiatric disorders, family psychopathology, and missing more than 1 year of school may be associated with increased rates of chronic illness (Black, 1995). Moreover, if the anxiety symptoms interfere with the normal tasks of developing self-esteem and peer relationships, later developmental stages may be affected. Ongoing somatic complaints also may develop into a consistent pattern throughout childhood and adolescence and lead to multiple medical evaluations. Many authors have suggested that childhood SAD is a risk factor for the development of panic disorder or agoraphobia as an adolescent or adult (Battaglia et al., 1995; Biederman et al., 1993; Gittelman and Klein, 1984; Moreau and Follet, 1993). Children with SAD also may be at increased risk for depressive disorders and social phobia as adults (Black, 1995). Children of parents with panic disorder have more than a threefold increase in the risk for SAD (Weissman et al., 1984).

## TREATMENT

Individual, family-based, and group cognitive-behavioral treatment (CBT) appear to be successful in the treatment of SAD. Historically, individual psychodynamic psychotherapy has focused on mastering issues of separation, autonomy, self-esteem, and achieving age-appropriate independent behaviors. Parents may play an important role in the treatment, as they are encouraged to understand both the child's need for security and for independent behavior. It is important to explore each parent's own issues related to separation that may be unconsciously being played out with their own children.

Although there are no CBT programs designed specifically for treating SAD, a number of studies have successfully used CBT strategies to treat children with SAD as well as other anxiety disorders (i.e., GAD, social phobia and specific phobia) ([Barrett et al., 1996](#); [Cobham et al., 1998](#); [Dadds et al., 1997, 1999](#); [Kendall, 1994](#); [Kendall et al., 1997](#)). A systematic combined treatment program (CBT plus imipramine) was developed by Bernstein and colleagues ([Bernstein et al., 2000](#)) to treat children with school refusal (with comorbid anxiety and major depressive disorder), and this is discussed subsequently.

Specific treatment strategies have included (a) exposure (gradual exposure, systematic desensitization using relaxation); (b) contingency management (positive reinforcement, shaping, extinction); (c) cognitive strategies (problem solving, coping thoughts, self-talk); (d) modeling (demonstration of appropriate behaviors); and (e) family treatment (parent anxiety management, teaching parents to serve as cotherapists) ([Francis and Beidel, 1995](#); [Labellarte et al., 1999](#)). From a clinical perspective, the specific strategies used may vary slightly based on the child's SAD symptoms (e.g., avoidance vs. anticipatory distress) and developmental level. However, a combination of systematic desensitization with positive reinforcement (and cognitive strategies in older children) often is quite effective. For example, children are encouraged gradually to spend time apart from parents (e.g., going in another room, upstairs, to school) and are rewarded with stickers/prizes for successful efforts ([Francis and Beidel, 1995](#); [Labellarte et al., 1999](#)).

In general, psychotherapeutic interventions usually are initiated for SAD before pharmacotherapy is invoked. Children with significant distress and functional impairment who have not responded to specific psychotherapeutic interventions might be considered. Pharmacotherapy would be used only as an adjunct to behavioral or psychotherapeutic interventions. A child with SAD who had comorbid GAD or other anxiety disorders might benefit from the addition of medication to the multimodal treatment plan. Specific medication considerations are reviewed at the end of this chapter.

### Specific Phobia

#### DEFINITION AND CLINICAL DESCRIPTION

Specific phobia is a marked and persistent fear of circumscribed objects or situations (phobic stimuli), such as animals, blood, heights, closed spaces, or flying, such that the child/adolescent either avoids the stimulus whenever possible or endures it with intense anxiety ([American Psychiatric Association, 1994](#)). These fears are unrelated to fears of embarrassment in public or performance (as in social phobia) or fears of having a panic attack (as in panic disorder). Exposure to the phobic stimulus almost immediately provokes a significant anxiety response that may include a panic attack.

The fear must have persisted for at least 6 months and interfere with the person's normal routine, social relationships, or academic functioning, or there must be marked distress about having the fear. Children, unlike older adolescents and adults, may not realize that the fear is excessive or unreasonable. The reported prevalence rates of specific phobia range from 2.4% to 3.3%, with a higher prevalence usually reported for girls than boys ([Anderson et al., 1987](#); [Bird et al., 1988](#); [Kashani and Orvaschel, 1990](#); [Kashani et al., 1989](#)). However, there has been far more research conducted on the epidemiology of "normal" childhood fears.

Behaviorally, children with specific phobia may show avoidance either on exposure or in anticipation of exposure to the feared stimulus. They also are likely to scream, cry, or seek comfort in attachment figures. The cognitions of phobic children tend to focus on the belief that an encounter with the feared stimulus will result in harm to self or others (e.g., fearing being bitten by a dog or stung by a bee). Phobic children also tend to exhibit significant anticipatory anxiety. Physiologic symptoms in children with specific phobias may be somewhat consistent with panic-like symptoms such as increased heart rate, sweating, hyperventilation, shakiness, and upset stomach.

#### DEVELOPMENTAL PERSPECTIVE

Although much has been written on the natural history of "normal childhood fears," there is less research targeting clinical populations of children with specific phobia. It is generally believed that childhood phobias tend to parallel the onset of "normal" developmental fears with regard to age of onset and focus of the fear. Phobias of animals, darkness, blood, insects, and injury typically begin before age 7 years and have not been clearly linked to a traumatic event at onset ([Albano et al., 1996](#); [Marks and Gelder, 1966](#)). Other childhood phobias occur across a wide range of ages, with elevations between the ages of 10 and 13 years ([Strauss and Last, 1993](#)). Much of the work in this area, however, comes from retrospective studies of adults with specific phobia ([Liddell and Lyons, 1978](#); [Ost, 1987](#)).

#### COURSE AND OUTCOME

Specific phobias often begin in childhood and must be differentiated from normal developmental fears. There is some indication that childhood phobias improve over time without treatment; however, many children continue to have phobic symptoms even if they do not meet full diagnostic criteria ([Agras et al., 1972](#); [Ollendick, 1979](#)). Retrospective reports from adults also suggest some retention of phobic symptoms over time, with many adults reporting symptom onset for their phobias in childhood ([Ost, 1987](#)).

#### DIFFERENTIAL DIAGNOSIS/COMORBIDITY/RELATED FEATURES

In making a diagnosis of specific phobia, it is necessary that the fear not be related primarily to another anxiety disorder (e.g., fears of separation in SAD, fears of social humiliation in social phobia, or fears of having a panic attack in panic disorder). However, specific phobias commonly are comorbid with other anxiety disorders. Anxiety that is more generalized and diffuse may be better characterized as GAD. In addition, the clinician must be careful to differentiate the repetitive and ruminative quality of some phobias from obsessive fears associated with OCD or reexperiencing symptoms in PTSD.

## TREATMENT

The empirical literature for childhood specific phobias lags behind similar work in other childhood anxiety disorders. However, graduated *in vivo* exposure in combination with contingency management and self-control strategies appear to be the most promising treatment approaches to date ([Morris and Kratochwill, 1998](#); [Silverman et al., 1999b](#)). The exposure component involves gradual contact with progressively more distressing phobic stimuli. Exposure also can be presented imaginally as a starting point for treatment. The contingency management techniques involve modification of contingencies that relate to the child's phobic/avoidance behaviors (e.g., reinforcement and extinction). Finally, self-control strategies focus on self-evaluation and self-reward (e.g., cognitive coping strategies). A study by [Silverman and colleagues \(1999b\)](#) compared the relative efficacy of an exposure-based contingency management treatment and an exposure-based self-control treatment with an education support control group. Results showed substantial improvement for children in both of the exposure-based groups.

In general, the behavioral techniques are the first-line therapeutic intervention for most simple phobias. Pharmacotherapy has not been shown to be effective for specific phobias, and this is reviewed at the end of the chapter.

### Social Phobia

#### DEFINITION AND CLINICAL DESCRIPTION

Social phobia is a persistent fear of one or more social situations in which a person is exposed to unfamiliar persons or to scrutiny by others ([American Psychiatric Association, 1994](#)). Exposure to the feared social situations provokes anxiety, fear of embarrassment, and often panic attacks. Commonly feared situations include speaking in front of others, attending social gatherings, dealing with authority figures, performing in public, and speaking to strangers. Children and adolescents with social phobia avoid anxiety-provoking situations (resulting in interference in functioning) or endure them with marked distress. In addition, children and adolescents



may not recognize that their fear is excessive or unreasonable.

Social phobia has been estimated to occur in approximately 1% of children and adolescents ( [Anderson et al., 1987](#); [Kashani and Orvaschel, 1990](#); [McGee et al., 1990](#)). However, studies suggest that the prevalence is much higher in older populations and may be the most common anxiety disorder affecting adults ( [Kessler et al., 1994](#)). As a result, it is believed that social phobia in children and adolescents likely is underdiagnosed and undertreated because these children are labeled as “shy” rather than recognized as having clinical impairment ( [Albano et al., 1996](#)). In clinic samples, the prevalence of social phobia has been reported to be 14.9% ( [Last et al., 1992](#)). Children and adolescents with social phobia commonly fear that others will find some fault with them, considering them weird, unattractive, or stupid, or that they will do or say something foolish or embarrassing ( [Beidel et al., 1999](#)). Somatic symptoms are common and include racing heart, sweating, blushing, tremulousness, lightheadedness, and gastrointestinal distress ( [Beidel et al., 1991](#)). Socially phobic children and adolescents often have great difficulty in a number of school situations, and negative cognitions tend to focus on excessive concerns about embarrassment, negative evaluation, and rejection.

Although it is not fully described in this chapter, selective mutism also deserves mention. Selective mutism is characterized by persistent failure to speak in one or more major social situations when speaking is expected, despite speaking in other situations. Although selective mutism is not classified as an anxiety disorder in DSM-IV, research strongly suggests that it may be primarily a manifestation of social anxiety ( [Black and Uhde, 1995](#); [Black et al., 1992](#)). Shyness, fear of embarrassment, and social withdrawal are commonly seen in children with selective mutism. Systematic descriptions of children with selective mutism have revealed that nearly every child also meets criteria for either social phobia or avoidant disorder ( [Black and Uhde, 1995](#); [Dummit et al., 1997](#)). Despite early assumptions, children with selective mutism are not likely to have a history of early trauma or to be particularly oppositional ( [Black and Uhde, 1995](#)). The disorder is diagnosed in children who fail to speak in specific social situations for at least 1 month (not limited to the first month of school) and when the disturbance significantly interferes with educational or social functioning. For a comprehensive description of the assessment of a child with selective mutism, the reader is directed to other sources ( [Leonard and Topol, 1993](#)).

#### DEVELOPMENTAL PERSPECTIVE

Social phobia is most commonly diagnosed in adolescents, although cases of social phobia have been diagnosed in children as young as 8 years of age ( [Beidel and Turner, 1988](#)). Last and colleagues ( [Last et al., 1992](#); [Strauss and Last, 1993](#)) note the average age of onset in clinic-referred samples to range from 11.3 to 12.3 years. Social phobia may look quite different in younger and older children and adolescents ( [Beidel, 1998](#)). For example, younger children may have frequent tantrums or appear very “clingy” with parents or caretakers. They may refuse activities in school. Older children and adolescents are likely to shy away from family or social gatherings and peer relations altogether.

#### COURSE AND OUTCOME

Although research evidence is limited, it appears that when childhood social phobia goes unrecognized, there are significant and long-term negative consequences ( [Beidel and Morris, 1995](#)). As noted earlier, social phobia may result in school refusal behavior, premature termination of formal education, or failure to enter the workforce. In older adolescents, there is likely to be significant interference in occupational development or dating relationships.

#### DIFFERENTIAL DIAGNOSIS/COMORBIDITY/RELATED FEATURES

In making a diagnosis of social phobia, it is necessary to clarify the nature of the core fear as being specific to interactions with or performance in front of others. Children and adolescents with social phobia usually present differently than children with panic disorder (where the focus of fear is the panic attack itself), GAD (where social fears may be one of many disruptive fears), or specific phobia (where the fear is of objects or situations that do not involve social interaction). Making a differential diagnosis can be more complicated with SAD ( [Beidel and Morris, 1995](#)). Although there is a clear distinction between distress over separation from primary attachment figures and distress over approach or interaction with others, the reasons for avoidance in certain situations (e.g., school) are not always clear. The practitioner also must be conscious of obsessions or compulsions that may be fueling social avoidance (e.g., fear of contamination or harm coming to others) rather than the constellation of social fears that comprise social phobia.

Clinical correlates include depressed mood, higher trait anxiety, and increased substance use concerns ( [Beidel and Morris, 1995](#); [Strauss and Last, 1993](#)). Studies suggest that children and adolescents with social phobia have significantly poorer social skills than nonpsychiatric children ( [Beidel et al., 1999](#)). Many children and adolescents also may be perceived as oppositional because of their refusal to participate in developmentally appropriate activities.

#### TREATMENT

As noted earlier, a number of studies have successfully used CBT strategies in more generalized child anxiety treatment programs ( [Barrett et al., 1996](#); [Cobham et al., 1998](#); [Dadds et al., 1997, 1999](#); [Kendall, 1994](#); [Kendall et al., 1997](#)). These controlled treatment studies usually have included children with social phobia, as well as other anxiety disorders. Results from these studies tend to support the use of individual, family-based, and group CBT over comparison treatments ( [Beidel and Morris, 1995](#)). Interventions combining social skill-building as well as anxiety reduction techniques appear necessary for maximally successful outcome ( [Beidel et al., 1999](#)). [Beidel and colleagues \(2000\)](#) reported on 67 children with social phobia who were randomized to either a specific behavioral treatment program (designed to enhance social skills and decrease social anxiety) or an active but nonspecific intervention. Those who received the specific behavioral intervention were significantly improved across multiple dimensions, and the gains were maintained at 6 months' follow-up.

Pharmacotherapy should not be used as the sole intervention for children with social phobia. Medication might be part of a multimodal treatment plan, particularly after an intensive behavior intervention has been tried. Of the different medication options, the selective serotonin reuptake inhibitors (SSRIs) are the most likely first agents to consider. This is based on systematic efficacy studies of SSRIs in adults with social anxiety (e.g., paroxetine) and the most recent controlled trial of fluvoxamine in the successful treatment of social phobia in children and adolescents ( [Walkup et al., 2001](#)).

There is a small literature on the use of SSRIs in the treatment of children with selective mutism. In general, a behavioral treatment plan should be initiated first. If the child does not make sufficient progress, then adjunctive medication with ongoing behavioral treatment is considered. In an open study ( [Dummit et al., 1996](#)) 21 children with selective mutism were treated with fluoxetine, and 76% were improved. Improvement at week 9 was inversely correlated with age, suggesting that it is harder successfully to treat the child who has been symptomatic for a long time ( [Black and Uhde, 1994](#); [Dummit et al., 1996](#)). [Black and Uhde \(1994\)](#) reported that in 16 children with selective mutism who were treated in a double-blind, placebo-controlled study, fluoxetine was superior to placebo.

### Generalized Anxiety Disorder

#### DEFINITION AND CLINICAL DESCRIPTION

Generalized anxiety disorder is characterized by excessive and uncontrollable worry— *excessive* meaning that the worry is out of proportion to the facts of the situation, and *uncontrollable* meaning that once the individual starts worrying, he or she finds it very difficult, if not impossible, to stop. Therefore, the distinction between developmentally appropriate worry and the worry experienced by individuals with GAD is based less on the subject of the worry and more on the persistence and unrealistic nature of the worries in GAD. To be classified as having GAD, a youth needs to display a pattern of excessive worry that interferes with daily functioning and lasts for at least 6 months. Using these criteria, youth prevalence estimates range from 2.7% to 4.6% ( [Costello, 1989](#)).

Most often, the worry associated with GAD is not confined to one topic area. Typical worries for children and adolescents with GAD include worries about competence, approval, and appropriateness of past behavior. Future events and new or unfamiliar situations also are common foci for worries. There also may be excessive concerns about punctuality. Children with GAD often are seen as overly conforming and perfectionistic, and may be seen by adults as rigid or oppositional. Children and adolescents with GAD frequently seek reassurance from others, although this reassurance usually provides only fleeting relief from the oppressive worries.

Patients also are expected to present with symptoms of motor tension and vigilance, although in children only one such symptom is required to establish the diagnosis, whereas in adults three physical symptoms are required. Somatic complaints such as headaches, abdominal pain, and difficulties sleeping are common. Phenomenologically, patients report feeling keyed-up or on edge and feeling as if they are unable to relax. Particularly in children, irritability often is associated with the worry such that when more worried the child is more irritable.

## DEVELOPMENTAL PERSPECTIVE

Generalized anxiety disorder has only recently (beginning with DSM-IV) been diagnosed in children. Previously, children predisposed to excessive worry typically were classified as having overanxious disorder. Although children previously described as having overanxious disorder may not be identified with the current criteria, empirical evidence suggests that replacing overanxious disorder with GAD has had little impact on the identification and treatment of children whose anxiety is typified by excessive worry ([Kendall and Warman, 1996](#); [Tracey et al., 1997](#)). One advantage of the new system is that it facilitates study of the developmental progression of this pattern of symptoms.

Currently, we know very little about how or if the nature of the symptoms of GAD change throughout childhood and into adulthood. However, preliminary evidence suggests that the one developmental consideration included in the DSM-IV criteria for GAD (requiring fewer somatic/physical symptoms in children than in adults) may not be warranted ([Pimentel et al., 2000](#)). There is some evidence for the consistency of GAD presentations across middle childhood ([Tracey et al., 1997](#)). Also, in childhood, GAD is equally common in boys and girls, but starting in adolescence it is more common in girls ([Werry, 1991](#)). Finally, caution must be used in making inferences to children younger than 7 years of age because there is some evidence that cognitive changes that occur at approximately 7 or 8 years of age have an impact on worry processes ([Vasey et al., 1994](#)).

## DIFFERENTIAL DIAGNOSIS/COMORBIDITY/RELATED SYMPTOMS

The symptoms associated with GAD can be caused by an underlying medical condition. It is particularly important to investigate potential medical causes when autonomic symptoms predominate. Gastrointestinal problems, hyperthyroidism, pheochromocytoma, hypoglycemia, and lupus should all be considered if suggested by the history or physical examination. Excessive use of caffeine or other stimulants, particularly among adolescents, also should be ruled out before assigning the diagnosis of GAD.

A diagnosis of GAD is appropriate only when the focus of the worry is unrelated to worry better captured by another anxiety disorder. GAD can be differentiated from the other anxiety disorders in a number of ways. It can be differentiated from the anxiety associated with social phobia because the child with GAD would worry about their performance even if not being evaluated by others, whereas the child with social phobia would worry only when fearing negative evaluation by others. In children, it can be difficult to differentiate worry in GAD from obsessions associated with OCD because children with obsessions are less likely to report them as feeling ego-dystonic, and because children's worries may be seen as less reality based in the eyes of adults. Finally, when the anxiety that a child presents with is short lived and of relatively mild intensity, and when it follows a specific stressor, a diagnosis of adjustment disorder with anxious mood is more appropriate than a diagnosis of GAD.

## COURSE AND OUTCOME

Many, if not most, individuals with GAD report having been anxious for much of their lives, with anxiety most often beginning in childhood. This observation is consistent with the belief held by some that GAD is better conceptualized as a temperamental characteristic (trait) than as a major mental disorder. Also consistent with this belief is evidence that GAD most typically follows a chronic but fluctuating course, with symptoms worsening at times of stress. Obviously, for the symptoms to meet criteria for a disorder, there must be significant distress or interference.

## TREATMENT

A comprehensive assessment of the child should yield a targeted treatment plan that may use individual or family psychotherapeutic interventions. For some, a dynamic insight-oriented psychotherapeutic intervention may be successful. For many, CBT interventions are needed.

Cognitive-behavioral interventions designed for use with children and adolescents individually, as well as interventions that incorporate the family and interventions with groups of children, have been useful in treating GAD ([Barrett et al., 1996](#); [Cobham et al., 1998](#); [Kendall, 1994](#); [Kendall et al., 1997](#)). These interventions use cognitive restructuring techniques to target anxious cognitions, and relaxation training to target physiologic arousal. Behavioral experiments and *in vivo* exposure to anxiety-producing stimuli are used to reduce avoidance behaviors. Group treatments incorporate peer modeling to enhance skill building; similarly, family treatments focus on teaching parents to be effective models for their children. All approaches entail the use of positive reinforcement to increase desirable behaviors and reduce avoidance.

Few controlled trials exist for medications for children with GAD. The SSRIs have become increasingly more popular as first-line medications based on adult studies and one large, controlled trial in children. The Research Unit on Pediatric Psychopharmacology (RUPP) study ([Walkup et al., 2001](#)) reported that fluvoxamine was superior to placebo for children with GAD. A more detailed review of this study is provided at the end of the chapter.

## Panic Disorder With or Without Agoraphobia

### DEFINITION AND CLINICAL DESCRIPTION

Diagnosing children with panic disorder has been a much-debated topic (e.g., [Kearney and Silverman, 1992](#); [Nelles and Barlow, 1988](#)). As in adults, panic disorder refers to the experience of unexpected panic attacks accompanied by persistent apprehension about their recurrence or behavioral modifications in daily routine as a result of the attacks. A panic attack is a discrete period of intense fear or discomfort that is characterized by the presence of at least four somatic or cognitive symptoms. Somatic symptoms include shortness of breath, accelerated heart rate, chest pain, choking sensations, dizziness, tingling or numbing sensations, hot/cold flashes, sweating, trembling, and nausea. Cognitive symptoms include fear of dying, going crazy, and losing control. Panic attacks are typified by their abrupt onset, with all or most symptoms reaching their peak within 10 minutes.

Panic disorder may or may not be accompanied by agoraphobic avoidance. Agoraphobia is anxiety about being in places or situations in which escape might be difficult or embarrassing, particularly if one should experience a panic attack. Typical situations that are feared or avoided are being outside alone, being in a crowd, standing in line, bridges, modes of travel, shopping malls, and movie theaters. Agoraphobia usually results in a pervasive pattern of avoidance, but occasionally may result in the youth being able to endure to feared situation with intense distress, or only in the presence of a companion.

One of the main reasons that researchers have questioned the phenomenon of panic disorder in children and adolescents is a reliance on a cognitive theory of panic disorder that suggests that panic attacks and panic disorder result from "catastrophic misinterpretation" of bodily (i.e., somatic) sensations ([Clark, 1986](#)). Proponents of this model suggest that children and younger adolescents are not cognitively capable of making the kinds of internal, catastrophic attributions that characterize spontaneous panic attacks ([Nelles and Barlow, 1988](#)). They state that notions of external causation (e.g., "My heart is racing because I don't like my teacher") dominate children's cognitive responses, thus precluding the cascade of events that precipitates a panic attack.

Despite the controversy, given evidence from numerous case studies of panic attacks and panic disorder in children and adolescents ([Alessi and Magen, 1988](#); [Alessi et al., 1987](#); [Ballenger et al., 1989](#); [Biederman et al., 1997](#); [Hayward et al., 1989](#); [King et al., 1993, 1997](#); [Last and Strauss, 1989](#); [Moreau et al., 1989](#); [Vitiello et al., 1990](#)), the debate has turned more recently from questions of whether children and adolescents experience panic attacks and panic disorder, to questions about the phenomenology of panic attacks and panic disorder in children and adolescents. Estimates from community samples are that from 35.9% to 63.3% of adolescents have experienced panic attacks, with 0.6% to 4.7% of adolescents experiencing symptoms sufficient to meet DSM-IV diagnostic criteria for panic disorder. Although retrospective reports from adolescents and adults suggest that at least some subset of panic cases have their onset in childhood, there are as yet no reliable estimates as to the prevalence of the disorder in prepubertal children.

The most commonly reported symptoms among adolescents with panic attacks appear to be trembling, dizziness/faintness, pounding heart, nausea, shortness of breath, and sweating ([Kearney et al., 1997](#); [King et al., 1997](#)). Cognitive symptoms are reported less frequently than somatic ones ([King et al., 1997](#)). The most commonly avoided settings included those involving large groups of people unknown to the patient (e.g., restaurants, crowds, auditoriums) ([Kearney et al., 1997](#)).

## DEVELOPMENTAL PERSPECTIVE



Despite recent research, there still is some resistance to the idea that panic disorder is anything but very rare in children. For example, [Last and colleagues \(1992\)](#) suggest that the panic symptoms that occur before adolescence are most often an associated feature of another anxiety disorder. Panic attacks themselves, not even full-blown panic disorder, also are more likely in older children and adolescents ([Hayward et al., 1992](#)).

#### *DIFFERENTIAL DIAGNOSIS/COMORBIDITY/RELATED SYMPTOMS*

The most difficult distinction required when diagnosing panic disorder in youth is the difference between an unexpected panic attack, as required for a diagnosis of panic disorder, and situationally cued attacks that are typical of other anxiety disorders. This task becomes especially challenging given the cognitive predisposition of children, as described previously, to attribute their experiences to external, identifiable, situational factors. Careful noting of patterns in the circumstances surrounding attacks helps with this distinction because panic attacks occurring in the context of another anxiety disorder occur primarily in the presence of the target stimulus. In addition, presence of pervasive apprehension about having a panic attack supports the diagnosis of panic disorder rather than the diagnosis of panic attacks in the context of another anxiety disorder.

Panic attacks can be caused by a variety of medical conditions, including hyperthyroidism, hyperparathyroidism, vestibular dysfunctions, seizure disorders, and cardiac conditions. When panic is accompanied by palpitations, a discussion with the pediatrician as to the need for electrocardiography and cardiac consultation is encouraged. Appropriate laboratory tests and physical examinations should be used to rule out these causes before assigning a diagnosis of panic disorder.

#### *COURSE AND OUTCOME*

Although more direct research with children and adolescents is required, many researchers agree that the modal age of onset for panic disorder is in mid-adolescence, especially after age 14 years ([Von Korff et al., 1985](#)). Very little is known about the course and outcome for children and adolescents with panic disorder with or without agoraphobia. Based on retrospective reports of adults with these disorders, however, it appears likely that untreated, these disorders will persist into adulthood.

#### *TREATMENT*

Empirically supported psychosocial and pharmacologic interventions for panic disorder in youth have not been fully developed nor tested. However, preliminary evidence from a small, multiple-baseline–design study by [Ollendick \(1995\)](#) supports the efficacy of CBT for use with adolescents with panic disorder with agoraphobia. In this study, breathing retraining, applied relaxation, cue-controlled relaxation, positive self-instruction training, and cognitive coping procedures were applied in an interoceptive conditioning exposure paradigm. After teaching the adolescents the necessary coping skills, the adolescents were exposed to anxiety-provoking situations until their anxiety dissipated (up to 4 hours). Panic attacks were eliminated, agoraphobic avoidance was reduced, and self-efficacy for coping with future attacks was increased for all adolescents. Treatment gains were maintained at 6-month follow-up.

Currently, there are no systematic trials of the treatment of panic disorder in children. In adults, SSRIs have become the pharmacotherapeutic treatment of choice, and several SSRIs have approved indications for the treatment of panic in adults (see the discussion at the end of the chapter on medications for the anxiety disorders).

#### **School Refusal**

##### *DEFINITION AND CLINICAL DESCRIPTION*

School refusal is not a separate DSM-IV diagnostic category, and children with this symptom likely represent a heterogeneous group. [King et al. \(1995\)](#) have suggested the following criteria for clinically significant school refusal: “1) Severe difficulty attending school, often resulting in prolonged absence; 2) Severe emotional upset, including excessive fearfulness, temper outbursts, or complaints of feeling ill when faced with the prospect of going to school, 3) Staying home with the parent’s knowledge when the youngster should be at school; 4) Absence of antisocial characteristics such as stealing, lying, and destructiveness” (p. 3). These criteria highlight the need to differentiate school refusal behavior from truant behavior and cases in which other diagnostic categories (e.g., conduct disorder) may be more appropriate.

Some children outright refuse to leave home, whereas others may get part way to school only to rush home in distress, and still others make it to school only to spend much of the day in the nurse’s office, or only to make frequent calls home. When fear is maintaining the school refusal behavior, clinical reports suggest that fear may be of a variety of situations or specific events. Clinical reports also indicate that when school refusers are faced with attending school, they evidence marked physiologic changes, including muscular tension, breathing irregularities, headaches, stomachaches, and pallor ([Granell de Aldaz et al., 1987](#); [King et al., 1995](#)). Excessive dependency on the parent, and parents who present as overprotective and anxious themselves also are common. Reviews suggest that school refusal occurs in 1% of all school-aged children and in 5% of clinic-referred children ([Burke and Silverman, 1987](#); [King et al., 1995](#)).

##### *DEVELOPMENTAL PERSPECTIVE*

The nature of school refusal behavior is likely to change with age, as is the nature of the precipitating events. For example, fear of separation is more common in younger school refusers, whereas fear of teachers or other children is more common in older children; social-evaluative fears also are more common in older children. As children get older, and as the problem potentially becomes more entrenched, it becomes harder for parents physically to force children to attend school, so it is more common for older children with school refusal never to make it to school, rather than to go to school and spend time in the nurse’s office.

##### *DIFFERENTIAL DIAGNOSIS/COMORBIDITY/RELATED SYMPTOMS*

Children and adolescents with school refusal behavior most frequently meet diagnostic criteria for another major mental disorder. Among the most common diagnoses that accompany school refusal behavior are SAD, specific phobia, social phobia, and major depression ([King et al., 1995](#)). In addition, when there is acute avoidance of any situation, a consideration of abuse needs to be explored.

##### *COURSE AND OUTCOME*

Acute onset is more common in younger children (often following a change in routine school attendance such as an illness, move to new school, or a vacation). [Ollendick and Mayer \(1984\)](#) found that the onset of school refusal behavior coincides with entry into school and transitions from elementary to middle school (in the United States, between 5 and 6 years of age, and 10 and 11 years of age). In general, the longer the pattern of school refusal persists, and the more entrenched the pattern becomes, the harder it will be to treat successfully ([King et al., 1995](#)).

##### *TREATMENT*

Several randomized clinical trials have tested the efficacy of CBT interventions for children with severe school refusal behavior. One study ([King et al., 1998](#)) used a manual-based multicomponent treatment program to provide coping skills training and exposure to children, and to provide training to parents and teachers in behavior management strategies. Using the outcome criterion of 90% school attendance, this study found that 88.23% of the treated children had improved, whereas only 29% of the children assigned to a wait list condition had achieved this criterion. Another study ([Last et al., 1998](#)) compared a CBT intervention with a psychosocial placebo control condition. The CBT consisted of graduated *in vivo* exposure and cognitive restructuring training. The placebo control therapy included educational presentations, supportive psychotherapy, and a daily diary assignment to record thoughts and fears. Contrary to expectations, there were no differences in the efficacy of these two approaches. Posttreatment, both groups showed improvements in school attendance and other related outcome measures. Regardless of treatment received, younger children and children with higher rates of school attendance before treatment showed the greatest improvements after treatment.

Another approach is [Kearney and Silverman’s \(1990\)](#) prescriptive treatment approach, which tailors the components of CBT to specific features of children’s school refusal behavior. They identify the motivating conditions and look at removing the negative consequences associated with attending school (e.g., aversive social experiences) and the positive consequences of being at home (e.g., special time with a parent or friends). Open results are promising, but controlled trials are needed

([Kearney and Silverman, 1990](#)).

Until the early 1990s, with the intensive study of OCD, school refusal had been the most studied anxiety in children. The first controlled trials in children with anxiety problem were completed with school refusers. The original four placebo-controlled trials of tricyclic antidepressants for separation anxiety or school refusal reported contrasting results that have been attributed to issues of diagnostic heterogeneity, comorbidity, drug dosage, and absence of control of concurrent therapies ( [Berney et al., 1981](#); [Bernstein et al., 1990a](#); [Gittelman-Klein and Klein, 1971, 1973](#); [Klein et al., 1992](#)). Gittelman-Klein and Klein ( [1971, 1973](#)) reported that children receiving imipramine (100 to 200 mg/day) were significantly more successful (81%) in returning to school than those on placebo (47%). [Klein et al. \(1992\)](#) attempted to replicate their study but found that children who had failed behavioral treatment and who were then randomized to either imipramine or placebo did not significantly differ in outcome. [Berney and colleagues \(1981\)](#) reported that low-dose clomipramine (40 to 75 mg/day) was not superior to placebo in the treatment of 51 children (9 to 14 years of age) with anxiety symptoms (44% had comorbid depressive symptoms). In 1990, [Bernstein et al. \(1990a\)](#) reported that a comparative study of imipramine, alprazolam, and placebo in children did not show significant differences between active medication and placebo. Both alprazolam and imipramine were associated with greater improvement on some rating scales, but this did not approach significance.

In their 2000 landmark study, Bernstein and colleagues used an adapted version of [Last and colleagues' \(1998\)](#) manual to test the efficacy of CBT plus imipramine (CBT+IMI) versus CBT plus placebo (CBT+PBO) treatments for adolescent school refusers with comorbid anxiety and major depressive disorder. Sixty-three subjects entered, and 47 completed, this 8-week, randomized, controlled trial. School attendance improved significantly for the CBT+IMI group but not for the CBT+PBO group. At 8 weeks, 54% of the CBT+IMI group were in remission (defined as 75% school attendance), compared with 17% of the CBT+PBO group. They reported that CBT+IMI was significantly more efficacious than CBT+PBO in improving school attendance and in decreasing symptoms of depression ( [Bernstein et al., 2000](#)). This study was the first combined systematic treatment study completed in children and adolescents with an anxiety disorder, and provides a model for combined psychosocial and drug treatments.

As an aside, school phobias may develop in patients after the addition of a neuroleptic. This phenomenon was initially reported with haloperidol and pimozide and is likely pharmacologically mediated ( [Linet, 1985](#); [Mikkelsen et al., 1981](#)). Most recently, this was reported with an atypical neuroleptic, risperidone ( [Hanna et al., 1999](#)).

## GENERAL TREATMENT ISSUES

[Kendall \(1994\)](#) estimated that fewer than 20% of youth receive the necessary treatment for their anxiety disorder. This is due in part to the fact that children and adolescents with anxiety disorders are more “internalizing,” so their behavioral problems (and distress) may not be as obvious to others. In addition, many may wish to hide their symptoms from others. Despite the difficulties with providing treatment for all of the youth in need, researchers are beginning to document which interventions are efficacious for treating anxiety disorders in youth.

The American Psychological Association (Division 12) ( [Chambless et al., 1996](#); [Task Force on Promotion and Dissemination of Psychological Procedures, 1995](#)) has instituted a uniform set of criteria for standardizing the process of evaluating the empirical support for treatment efficacy. Following these criteria, treatments can be judged as well established, “probably efficacious,” or experimental. To be considered well established, treatments must have been shown to be more effective than pill placebo or alternate treatment, or equivalent to an established treatment in two or more group-design studies. Alternatively, positive outcomes from nine or more single-case studies that describe the sample carefully and use treatment manuals to compare the treatment with another treatment can be used as evidence. “Probably efficacious” treatments have been shown more effective than a no-treatment control group in two studies, or in two studies *from the same investigator* have been shown to be superior to an alternate treatment or equivalent to an established treatment. Alternatively, results from three or more single-case studies using treatment manuals and well described samples may be used as evidence. Any treatment that has not attained probably efficacious status is considered experimental.

Similarly, sophisticated evidence-based medicine guidelines offer another model for assessing the empirical treatment literature for psychiatric disorders. The reader is directed to the text by Sackett and colleagues and to the excellent “User’s Guide” series in the *Journal of the American Medical Association* ([Guyatt and Rennie, 1993](#); [Sackett et al., 1997](#)). Evidence-based models allow for the assessment of combined treatments in a multidisciplinary biopsychosocial model.

In two meta-analyses reviewing treatment outcome results for diverse psychotherapeutic procedures (i.e., psychodynamic, nondirective, family systems, behavioral, and cognitive-behavioral therapies), Weisz and colleagues ( [1987, 1995](#)) concluded that regardless of client age, therapist experience level, or target problem, behavioral treatments were more effective than nonbehavioral treatments. Based on this conclusion, [Ollendick and King \(1998\)](#) focused their application of the criteria for establishing empirically supported treatments for child anxiety disorders on behavioral and cognitive-behavioral procedures. To date, imaginal and *in vivo* desensitization, filmed and live modeling, and CBT that uses self-instructional training have all attained probably efficacious status when used to treat phobias in youth. Participant modeling and reinforced practice are considered well-established for treating phobias in youth.

For other anxiety disorders, CBT interventions with and without family involvement have achieved probably efficacious status ( [Ollendick and King, 1998](#); [Piacentini, 2000](#)). The bulk of the data that support this conclusion comes from Kendall and colleagues’ “Coping Cat” treatment manual ( [Kendall, 1994](#); [Kendall et al., 1997](#)) and from manuals derived from this approach (e.g., “Coping Koala” by Barrett and colleagues ( [Barrett et al., 1996](#)). These procedures entail using cognitive techniques to identify anxious feelings and thoughts and to recognize somatic symptoms of anxiety. Cognitive strategies also are used to cope with anxiety, as are behavioral techniques, including exposure, modeling, relaxation training, and contingent rewarding. Follow-up data from the “Coping Cat” manual suggest that treatment gains are maintained for as long as 1 to 3 years after treatment ( [Kendall and Southam-Gerow, 1996](#)). Group CBT with and without family components also has achieved probably efficacious status for treating anxiety disorders in general ( [Piacentini, 2000](#)).

Researchers also have begun to investigate patient characteristics that may influence treatment outcome in such a way as to help clinicians choose among “probably efficacious” treatments. Support has been found for the belief that when parental anxiety is present, interventions that involve the family directly are more efficacious than individual treatment for the child ( [Cobham et al., 1998](#)).

Even if parents are not participating in family therapy *per se*, parental involvement in treatment may be crucial to the success of the child’s treatment ( [Siqueland and Diamond, 1998](#)). First, parents can help children practice and generalize the skills learned in CBT. Next, parents’ beliefs and attitudes about their own and their child’s ability to cope with anxiety can help or impede treatment success. Finally, increased independence in their child may cause fear or uncertainty in parents. In response to such factors, some authors have included parental anxiety management and family management as components in manualized CBT for child anxiety ( [Barrett et al., 1996](#); [Cobham et al., 1998](#)).

In 1999, a systematic review concluded that no definitive data exist in support of the efficacy of psychopharmacologic agents in the treatment of anxiety disorders in youth (excluding OCD) ([Labellarte et al., 1999](#)). [Of note, the first and only large, systematic psychopharmacologic study of non-OCD anxiety disorders was published subsequently in 2001 ([Walkup et al., 2001](#))]. In a review of the “best available” evidence to date, [Labellarte and colleagues \(1999\)](#) concluded that methodologic flaws, such as failure to use standardized ratings scales to measure outcome and inclusion of subjects with poorly defined primary diagnoses, weakened the ability to draw empirically sound conclusions. In fact, the authors concluded that none of the studies completed to date met the minimal criteria for adequacy of pediatric psychopharmacologic trials proposed by [Riddle and colleagues \(1998\)](#).

One methodologic flaw that has the potential to skew conclusions is that in most of the pharmacologic studies published to date, a drug agent is viewed as the “primary” change producer, to the exclusion of considering the effect that concurrent procedures may have on treatment outcome ( [Kearney and Silverman, 1998](#)). This means that many studies that on the surface appear to be testing the effects of a pharmacologic agent alone are in fact testing a poorly operationalized combination of medication and psychotherapy. Following from this, it will be crucial to make explicit the procedures used in drug trials, in addition to prescribing and distributing the medication. A second and perhaps more important conclusion is that well controlled studies that explicitly examine the efficacy of combined treatment conditions are sorely needed, and until the study by Bernstein and colleagues was published in [2000](#), none existed. No treatment has yet attained “well-established” status for treating anxiety disorders in general (Ollendick and [King, 1998](#); [Piacentini, 2000](#)). Recent work comparing a CBT intervention with a nonspecific control treatment weakens the case for granting CBT procedures “well-established” status because the outcome from the CBT was equivalent to that from the nonspecific treatment control condition ( [Silverman et al., 1999a](#)). Supporters of CBT are quick to point out that the procedures used in the nonspecific treatment control condition in this study may have overlapped too much with some of the integral elements of the CBT. In addition, the target symptoms in the anxious children in this study were specific phobias, which may be more easily treated than other forms of anxiety in children. Further research comparing interventions with alternative treatment control procedures will clarify this issue.

## Overview of Pharmacologic Interventions



Studies of the specific classes of agents are reviewed briefly because they appear in other chapters of this text. The reader is referred to several excellent comprehensive reviews of pharmacologic treatment of pediatric anxiety disorders ([Riddle et al., 1999](#); [Velosa and Riddle, 2000](#)).

## BENZODIAZEPINES

Benzodiazepines, because of their sedative, muscle-relaxant, and anxiolytic properties, have been used to treat anxiety in children in small open and controlled trials.

[Bernstein and colleagues' \(1990a\)](#) comparative controlled study of alprazolam (mean daily dosage of 1.4 mg/day), imipramine, and placebo in children with school refusal did not show a significant difference between either medication over placebo. In the treatment of overanxious or avoidant disorder, 30 children and adolescents were randomized to either alprazolam (mean dosage 1.6 mg/day, range 0.5 to 3.5 mg/day) or placebo in a double-blind fashion. After 4 weeks, 88% of the completers on alprazolam improved, versus 62% on the placebo, but this was not statistically significant ([Simeon et al., 1992a](#)). [Grae and colleagues \(1994\)](#) reported a double-blind, placebo-controlled, crossover trial of clonazepam (0.5 to 2 mg/day) versus placebo in 15 children with mainly SAD. There was no difference in outcome between the clonazepam and the placebo groups.

Benzodiazepines may be a logical choice in the treatment of anxiety associated with medical procedures. In 13 pediatric patients who received low-dose alprazolam (0.125 to 1 mg) in an open fashion before bone marrow aspiration and spinal taps, there was a decrease in anticipatory and acute situational anxiety ([Pfefferbaum et al., 1987](#)). A double-blind placebo study of 0.2 mg/kg of oral midazolam in prepubertal children undergoing laceration repair showed a significant decrease in anxiety on active medication ([Hennes et al., 1990](#)). Currently, midazolam is available only as a parenteral injection solution.

In summary, benzodiazepines sometimes are used on a short-term basis for symptomatic treatment of anxiety. Typically, a SSRI would be the logical choice for an anxiety disorder requiring ongoing medication treatment, but a benzodiazepine might be initiated in conjunction with the SSRI if the anxiety was severe ([American Academy of Child and Adolescent Psychiatry, 1997a](#)).

## BUSPIRONE

No large, controlled trial of buspirone in children and adolescents has been published. Buspirone has been reported to be useful in the treatment of anxiety in two small open studies ([Kutcher et al., 1992](#); [Simeon et al., 1992b](#)). Buspirone (up to 50 mg/day for up to 9 weeks) has been used in an open fashion in 25 prepubertal children with anxiety and depression. The condition in six children worsened, and only three had sufficient benefit to continue on the drug after the end of the study ([Pfeffer et al., 1997](#)). Further study is required before conclusions can be made concerning its safety and efficacy in children with anxiety disorders.

## BETA BLOCKERS

Beta-adrenergic blocking agents (beta blockers) have not been systematically studied in children, although they occasionally have been used for children and adolescents with anxiety disorders or aggressive disorders. Beta blockers sometimes are prescribed to block the physiologic symptoms of anxiety in adults with performance anxiety or panic disorder. None of these agents has been studied in children, so because of the absence of data, they should not be considered until other options have failed ([American Academy of Child and Adolescent Psychiatry, 1997a](#)).

## TRICYCLIC ANTIDEPRESSANTS

The controlled trials of a tricyclic antidepressant for school refusal are reviewed earlier in the chapter. [Bernstein and colleagues' \(2000\)](#) report of CBT+IMI being effective for school-refusing adolescents suggests that such therapy should be considered for the school-refusing child with anxiety and depression. The increasing literature on the SSRIs ([Walkup et al., 2001](#)), and their preferable side effect profile over the tricyclic antidepressants, suggest that a SSRI should be chosen over a tricyclic antidepressant as the initial treatment.

## SELECTIVE SEROTONIN REUPTAKE INHIBITORS

Small open trials of children with mixed anxiety disorders showed promise for the SSRIs. [Birmaher et al. \(1994\)](#) reported a favorable response to fluoxetine in 21 children with anxiety disorders (social phobia, overanxious disorder, or separation anxiety) treated in an open fashion. Eighty-one percent showed a moderate to marked improvement in anxiety symptoms on a mean dose of 25 mg (range, 10 to 60 mg). [Fairbanks and colleagues \(1997\)](#) reported an open trial of fluoxetine for 16 children and adolescents with mixed anxiety disorders. During the 6- to 9-week trial, all patients had significant improvement in general functioning. The mean length of time for treatment response was 5 weeks. As reviewed earlier, there is a small open literature on the use of SSRIs in the treatment of selective mutism ([Black and Uhde, 1994](#); [Dummit et al., 1996](#)).

The first large-scale, systematic study of a SSRI was completed in 128 children and adolescents 6 to 17 years of age with either social phobia, SAD, or GAD ([Walkup et al., 2001](#)). Initially, 153 children were evaluated and underwent 3 weeks of open treatment with supportive psychoeducational therapy. Five children's condition improved with brief psychoeducation, and they did not go on to medication therapy. One hundred twenty-eight children were assigned to either fluvoxamine or placebo for 8 weeks. The fluvoxamine was increased every week by 50 mg to a maximum of 300 mg/day for adolescents and 250 mg/day for children younger than 12 years of age. The average dose of fluvoxamine was  $2.9 \pm 1.3$  mg/kg, and the average last dose was  $4 \pm 2.2$  mg/kg. Five children in the fluvoxamine group discontinued treatment because of adverse effects, and one withdrew for adverse effects in the placebo group. Abdominal discomfort was significantly more prevalent in the fluvoxamine group than in the placebo group, and there was a trend toward a greater frequency of increased motor activity in the fluvoxamine group. The children in the fluvoxamine group had greater reductions in symptoms of anxiety and higher rates of clinical response than the children in the placebo group. On the Clinical Global Impression Scale, 48 of 63 (76%) children in the fluvoxamine group had a response to treatment, compared with only 19 of 65 children (29%) in the placebo group ( $p < .001$ ). The authors conclude that fluvoxamine is an effective treatment for children and adolescents with social phobia, SAD, or generalized anxiety.

## FUTURE DIRECTIONS/CHALLENGES TO RESEARCH

The release of the first large, controlled efficacy study of a non-OCD childhood anxiety disorder has had an important impact on the field. It does not mean that pharmacologic treatment is the most effective intervention for anxiety disorders. The study exemplifies the need for the next step in research, the combination of disorder-specific CBT and medication. Are medications more effective than CBT, or is CBT more effective than medication? Is the combination of CBT plus medications more effective than either alone? As we move into the next century and develop specific treatments, the next challenge is to study unimodal versus combined treatments in a multidisciplinary framework.

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# 68 OBSESSIVE-COMPULSIVE DISORDER

Kenneth E. Towbin, M.D., and Mark A. Riddle, M.D.

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A 20-year retrospective of obsessive-compulsive disorder (OCD) certainly points up how much has been learned about OCD, but even more how much the study of OCD has advanced our understanding of the brain. It seems only fitting that study of obsessions and compulsions would lead investigators to understand basic brain structures and functions. After all, obsessions are among the oldest mental symptoms for which there are detailed descriptions, and a history of OCD reads like a history of psychiatry itself. Obsessions and compulsions were depicted as possession by the devil in 1467 in *Malleus maleficarum* (Kramer and Sprenger, 1928) and were described in the “Obsessi” of Paracelsus. In the 1600s, pious texts tell of extremes of religious doubting and “scrupulosity” or excessive devotion (Hunter and MacAlpine, 1963). Pioneers of psychiatry such as Esquirol (1838), Maudsley (1895), Freud (1909, 1953b), and Janet (Pittman, 1987) took up this troubling and fascinating disorder. At each time, the concept of OCD reflects the prevailing philosophy of thought, motivation, and free will.

The current *zeitgeist* features four discoveries that have promoted the explosion of OCD research since the 1980s. First came the discovery that medications that inhibit serotonin (5-HT) reuptake are effective for many patients with OCD. Subsequently, powerful techniques for observing structures and measuring regional brain activity under various circumstances were applied to understand how this happens. During this time, other investigations focused on the prevalence of OCD. By using even more reliable methods, it now appears that OCD is neither as rare as was first thought nor as common as the initial studies reported. Moving from pathophysiology to etiology, the fourth series of discoveries directed our attention to the genetic transmission of OCD. The effort to understand OCD has deepened our understanding of the prevalence, course, etiology, and pathology of these symptoms and along the way has broadened our knowledge of the neuroanatomy of voluntary functions and the heritability of complex behaviors.

## DEFINITIONS

*Obsessions* are unwanted thoughts, images, or impulses that the person realizes are senseless or unnecessary, intrude into his or her consciousness involuntarily, and cause functional impairment and distress. Despite this lack of control, the sufferer still recognizes that these thoughts originate in his or her own mental process. Because they arise in the mind, obsessions can take the form of any mental event—simple repetitive words, thoughts, fears, memories, pictures, or elaborate dramatic scenes.

*Compulsions* are actions that are responses to a perceived internal obligation to follow certain rituals or rules; they also cause functional impairment. Compulsions may be motivated directly by obsessions or by efforts to ward off certain thoughts, impulses, or fears. Often children report compulsions without the perception of a mental component. Like obsessions, compulsions are often viewed as being unnecessary, excessive or senseless, and involuntary or forced. Persons suffering from compulsions often elaborate various precise rules for the chronology, rate, order, duration, and number of repetitions of their acts.

These definitions reflect three critical concepts with relevance to the differential diagnosis. A most essential criterion is functional impairment as a consequence of symptoms. Two others carry from the classic definitions of Jaspers (1963) and Maudsley (1895): Persons feel that they are being forced or invaded by the symptoms, and they possess insight into the senselessness or excessiveness of their thoughts or acts. The validity of requiring patients to view their acts as unnecessary or their thoughts as senseless was challenged after observations that this objectivity may be inconstant. Insel and Akiskal (1986) and Lelliott and coworkers (1988) report on severely impaired patients who at times doubt the need to perform their rituals or who think their behaviors are senseless and who, at other times, are convinced of their necessity to the point of near or actual psychotic proportions. The criteria for OCD set out in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) (American Psychiatric Association, 2000) (Table 68.1) have been modified such that awareness of the senselessness or excess of the symptoms must be present only at some phase of the illness. For children, this criterion is dismissed altogether.

**A. Obsessions or compulsions:**  
Obsessions or compulsions (1, 2, 3, and 4):  
1. Recurrent and persistent ideas, thoughts, impulses, or images that are experienced at some time during the disturbance as intrusive and unwanted and cause marked anxiety or distress.  
2. The thoughts, impulses, or images are not simply excessive worries about real-life problems.  
3. The person attempts to ignore or suppress such thoughts, impulses, or images or to neutralize them with some other thought or action.  
4. The person recognizes that the obsessive thoughts or impulses or the compulsive acts are a product of his or her own mind (not imposed from without, as in thought insertion).  
**Compulsions:** (5 and 6)  
5. There are repetitive behaviors or mental acts that the person feels driven to perform in response to an obsession or according to rules that must be applied rigidly.  
6. The behaviors or mental acts are aimed at preventing or reducing distress or preventing some dreaded event or situation; however, these behaviors or mental acts either are not connected in a realistic way with what they are designed to neutralize or prevent or are excessive.  
**B. At some point the person has recognized that the obsessions or compulsions are excessive or unreasonable.** (Note: This does not apply to children.)  
**C. The obsessions or compulsions cause marked distress, are time consuming (take more than 1 hour a day), or significantly interfere with the person's normal routine, occupational (or academic) functioning, or social activities or relationships.**  
**D. If another Axis I disorder is present, the content of the obsessions or compulsions is not restricted to (e.g., preoccupation with death in the presence of an anxiety disorder; hoarding or repetitive checking with appearance in the presence of body dysmorphic disorder; preoccupation with having or sexual abuse in the presence of hypochondria; or guilty ruminations in the presence of a major depressive disorder).**  
**E. This is not the direct effect of a substance (e.g., a drug of abuse, a medication) or a general medical condition.**

Reprinted from American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. (DSM-IV). Washington, DC: American Psychiatric Association; 2000. pp 425-426.

Table 68.1. DSM-IV Criteria for Obsessive-Compulsive Disorder

Few studies differentiate between childhood-onset and adolescent-onset OCD. Therefore, in this chapter, “child,” “childhood,” and “children” are used to signify children *and adolescents*. Some studies have sampled adolescent subjects only. When this is so, the more exclusive term is employed.



## PREVALENCE AND EPIDEMIOLOGY

The prevalence of OCD in childhood or adolescence should be understood in the context of the prevalence of subclinical obsessions or compulsions in this segment of the population. [Evans and coworkers \(1997\)](#) have sent out mailings to parents of children less than 6 years old and find that urges to make things “just right” and preoccupations with symmetry and rules are very common in this unselected population. These concerns decline as children enter grade school age. To learn about the prevalence of obsessive or compulsive symptoms and to compare prevalences across development, [Zohar and Bruno \(1997\)](#) used self-report measures for a study of 1,083 children attending grades 4, 6, and 8. As predicted, many children confirmed the presence of some features. Sixty percent of fourth graders reported preoccupations with guilt about lying and engaging in checking behaviors, and 50% reported contamination and germ fears. By eighth grade, rates for these concerns declined to 40%, but 60% of eighth graders reported worries about cleanliness, and 50% noted intrusive rude thoughts. Mean scores across the age range in this population on the Maudsley Obsessive Compulsive Inventory were 11 to 12.5 out of a possible 30; like the findings of [Evans and coworkers \(1997\)](#) in a younger population, rates of behaviors and symptoms declined over time. A subgroup of eighth graders had high symptom scores (greater than two standard deviations from the mean) and reported high levels of anxiety. The authors suggest that this small group (4%) represents a clinically at-risk group because the large number of symptoms and the elevated state and trait anxiety are such a contrast to the decline in both in their ages.

Nearly 20 years after the Epidemiologic Catchment Area (ECA) survey ([Robins et al., 1984](#)), there continue to be important disagreements over the most reliable prevalence rate for OCD. Significant disparities and varied methods have yielded figures between 0.5% and 3%. Because it is safe to conclude that child and adult OCD are the same disorder, adult epidemiologic reports are relevant to understanding rates for children and adolescents.

The early work ([Rudin, 1953](#)), using weak methods by current standards, reported a prevalence of 0.05% for adult OCD. The scarcity of OCD was predominant until 1984, when ECA survey reported surprising prevalences of 1.2% to 3.29% ([Robins et al., 1984](#)). Subsequent examination of these high rates uncovered weak concordances for OCD. Among all the diagnoses in the ECA study, the concordance between diagnoses derived from lay interviewers and trained clinicians for OCD were the poorest ([Anthony et al., 1985](#); [Helzer et al., 1985](#), [Nestadt et al., 1994](#)). Lay interviews employing the Diagnostic Interview Schedule rely on simple “yes” or “no” responses to queries about the presence of broadly defined obsessions or compulsions. These are not reliable measures for actual *clinical cases* of OCD. [Helzer and coworkers \(1985\)](#) go on to say that results from community surveys, in which many subjects cluster at the “threshold of the diagnostic definition,” are unreliable because the response to a single probe carries too much weight. [Karno and coworkers \(1988\)](#) have reanalyzed ECA data, and prevalence rates are sustained despite the flawed method. False-negative results balanced false-positive results.

In an effort to learn more about this problem, [Nelson and Rice \(1997\)](#) used ECA methods and interviewed a community sample at two intervals separated by 12 months. The 1-year stability of the diagnosis of OCD was “very low.” Only 20% of subjects who reported “ever having symptoms” at time 1 reported “ever having symptoms” at time 2. These investigators concluded “the Diagnostic Interview Schedule diagnosis of obsessive-compulsive disorder possesses extremely limited validity.”

[Stein and coworkers \(1997\)](#) employed a different measure (the Comprehensive International Diagnosis Interview) and DSM-IV criteria. Again, rates derived from lay interviews revealed that 22% to 25% of adults expressed having obsessions or compulsions. However, when clinicians reviewed lay interviews, the rates dropped sevenfold (to 0.7%). These investigators also examined rates of “subclinical OCD” in which criteria were met for symptoms, but not impairment in DSM-IV. Rates for this subclinical syndrome were roughly equivalent to rates of clinical OCD (0.6%). Stein and coworkers, concluded, like [Karno and coworkers \(1988\)](#) and [Nestadt and coworkers \(1994\)](#), that lay interviews lead to many false-positive diagnoses.

The first prevalence rates reporting childhood clinical data ranged from 0.2% ([Berman, 1942](#); [Hollingsworth et al., 1980](#)), to 1.2% ([Judd, 1965](#)) (1.2%). [Flament and coworkers \(1988\)](#) conducted a rigorous study to discover the general adolescent population prevalence. Screening employed a modified Leyton Obsessional Inventory (LOI), and this was followed by direct clinical interviews of subjects with a high score. This produced a point prevalence of 0.35% and a lifetime prevalence of 0.40%. Weightings, such as those used in the ECA study, generated point and lifetime prevalence rates of 1% and 1.9%, respectively ([Flament et al., 1988](#)). Of the 18 adolescents diagnosed with OCD, 12 (67%) reported that symptoms resulted in high subjective interference, yet global assessment scores averaged 67 (“generally functioning pretty well”). To ascertain prevalence in a nonclinical population further, [Zohar and coworkers \(1992\)](#) performed detailed clinician-rated evaluations of 562 consecutive 16- to 17-year-old male and female inductees to the Israeli army. The OCD prevalence rate was 3.6%. Fifty percent of patients were identified as having “obsessions only.” This latter figure was substantially higher than that reported from clinical populations and casts doubt on the validity of the diagnoses generated by this method. Rates for compulsions compared favorably with those of [Flament and coworkers \(1988\)](#), however.

Overall, it appears that OCD is certainly more prevalent than was believed in the 1950s, but it is not as common as was first reported by the ECA. Adult epidemiologic studies offer prevalence rates of OCD from 0.5% to 3%. However, studies that rely on lay interviews and the Diagnostic Interview Schedule are likely to be unreliable. In addition, rates will change as different diagnostic criteria are employed ([Nestadt et al., 1994](#), [Stein et al., 1997](#)). Current valid studies place the prevalence closer to 1% ([Bebbington, 1998](#); [Stein et al., 1997](#)). Prevalence studies employing only structured or nonclinician interviews have a high risk of being unreliable and unsound.

Valid studies suggest that the sex distribution of OCD continues to reflect clinic samples—males and females are equally affected. Male patients appear to have an earlier age of onset ([Rasmussen and Tsuang, 1986](#); [Swedo et al., 1989a](#)). [Noshirvani and coworkers \(1991\)](#) report that male and female patients are equally represented in their sample, but 35% of male subjects have their onset between the ages of 5 and 15 years, compared with 20% of cohort females. Generally, symptoms exist an average of 5 to 8 years before patients reach clinical attention ([Flament et al., 1988](#); [Jenike, 2001](#)). [Noshirvani and coworkers \(1991\)](#) suggest that male patients tend to have a longer duration of illness before they seek treatment, yet the ECA study ([Karno et al., 1988](#)) shows that subjects with OCD have rates of medical and mental health service use roughly equal to those of persons with other disorders.

## CLINICAL DESCRIPTION

The variety of obsessions and compulsions that can be expressed by the human mind and body is unlimited. The typical pattern is for the patient to experience both obsessions and compulsions. It has been presumed that few persons have only one or the other ([Riddle et al., 1990b](#); [Swedo et al., 1989a](#)) but, as noted earlier, having only obsessions may be more common ([Zohar et al., 1992](#)). At any moment, most patients experience multiple obsessions or compulsions; over time, changes in the objects and contents of symptoms develop ([Rettew et al., 1992](#)). Although the repertoire of obsessions can range widely, certain contents are reported more frequently, and development may influence content. Reports of adolescents suggest that the most common categories of obsessions are concern over dirt and germs, fears of an ill fate befalling loved ones, exactness or symmetry, and religious scrupulousness ([Swedo et al., 1989a](#)). Bodily functions, lucky numbers, sexual or aggressive preoccupations, and fear of harm to oneself are less common. In adults, the frequency of these categories is similar, except aggressive and sexual obsessions are more common ([Dowson, 1977](#); [Rachman and Hodgson, 1980](#); [Rasmussen and Tsuang, 1986](#)).

Although a compulsion can be created from any action, only a few are common. An adolescent clinical cohort ([Swedo et al., 1989a](#)) displayed (in descending order of frequency) cleaning rituals, repeating actions (doing and undoing), and checking rituals most commonly. Many fewer subjects reported rituals to protect themselves from illness or injury, ordering maneuvers, and counting behaviors. In adults, the most common compulsions are checking and cleaning ([Rachman and Hodgson, 1980](#); [Rasmussen and Tsuang, 1986](#); [Welner et al., 1976](#)). Slowness ([Rachman and Hodgson, 1980](#)), counting ([Rasmussen and Tsuang, 1986](#)), or doing things by numbers ([Welner et al., 1976](#)) each has been reported as third most common.

Several investigators ([Baer, 1994](#); [Leckman et al., 1997](#); [Summerfeldt et al., 1999](#)) suggest that obsessions and compulsions should no longer be viewed as separate entities. In what promises to be important contributions to OCD research, factor analyses of symptoms in independent studies point to a more meaningful, “multidimensional” way of clustering symptom patterns. Rather than just one homogeneous entity or as “two factors” (obsessions or compulsions), here are strong reasons to consider that OCD may be better viewed as composed of four subtypes. The most rigorous work has identified the following: (a) aggressive, sexual, religious, and somatic obsessions with checking compulsions; (b) symmetry obsessions with counting, arranging, ordering, and repeating compulsions; (c) contamination obsessions with cleaning and washing; and (d) hoarding obsessions with hoarding and collection compulsions ([Baer, 1994](#); [Leckman et al., 1997](#); [Summerfeldt et al., 1999](#)). These investigators propose that course, genetic risk, neuropathology, and treatment may be different among these subgroups and bear further scrutiny. For example, [Alsobrook and coworkers \(1999\)](#) find that a major gene locus model is more strongly supported in families in which the proband has symmetry and ordering symptoms than in probands with other factor symptom clusters. Nevertheless, a 5-HT reuptake inhibitor treatment study has failed to support differences in response among three of these four subtypes ([Mataix-Cols et al., 1999](#)). Several other caveats are suggested by [Summerfeldt and coworkers \(1999\)](#), including the possibility that “more-than-four” factor models may also be viable, that various items have been excluded in the analyses that have been performed to date, and that problems may occur with using “lifetime” ratings because these are subject to recall bias. Moreover, longitudinal studies demonstrate that symptoms



change over time; persons do not belong to one subgroup exclusively over the course of their illness ( [Rettew et al., 1992](#)).

Children with OCD are more selectively impaired than the general clinical population. Academic achievement and extracurricular functioning are often preserved, although the quality of peer relationships may be variable ( [Riddle et al., 1990b](#)). Studies with adults point to significant impairment in social and role function ( [Koran et al., 1996](#)). In the study by Koran and coworkers, moderate to severe illness was correlated in a linear way with social impairment. Clinical studies of children entering treatment programs have consistently demonstrated average intelligence quotients, although the selection bias of these cohorts must be considered in interpreting these data.

It is hard to convey how limiting OCD can be. On the surface, children and adolescents appear to function well and seem relatively well adapted to their lives. However, severe symptoms often envelop the patient and his or her family completely ( [Koran et al., 1996](#)). It is common to learn of washing rituals that consume 4 hours of scrubbing daily, dissolve an entire bar of soap each session, leave the patient's hands worn raw and macerated, and raise monthly water bills dramatically. Counting or ordering compulsions can waste half a day and can lead to complete obstruction. Rituals repetitively executed from night to early morning may curtail sleep to a few hours. Checking or cleansing rituals can produce physical injury such as skin lacerations, ulcers, and chemical burns.

The family's reaction to the patient's symptoms is crucial. Several common response patterns may produce delays in evaluation and treatment of childhood OCD. Although patients are embarrassed and secretive about the content of, and the limitations imposed by, their symptoms, serious impairments rarely elude family members. Parents may delay obtaining treatment as a result of a false hope that symptoms will extinguish if everyone acquiesces and aids in performing the activities. This kind of family assistance does not relieve the child's anxiety. Many parents have difficulty extricating themselves from aiding in rituals. Children may extort, implore, or otherwise compel their parents to collaborate; the patient and parent become pathologically entwined in rituals. Parents may not understand that their child suffers from a serious disorder despite adequate development in other domains. In addition, the child's claims that the thoughts or acts are ridiculous or unnecessary may instill false security in parents who think that "it's just a phase." Parents with subclinical obsession- or compulsion-like behaviors may be unable to recognize symptoms in their child and may unwittingly minimize the child's impairment. Reassurance from clinicians and pediatricians who are unfamiliar with the disorder may result in mistaking severe symptoms for "normal" reactions. It is a frightening and painful moment when parents recognize that their child is ill and has lost control of his or her thoughts and actions.

## ETIOLOGY AND PATHOGENESIS

### Genetic Studies

There is ever-stronger evidence that genetic transmission confers vulnerability to OCD in some persons. Elevated concordance rates are observed among monozygotic compared with dizygotic twins ( [Carey and Gottesman, 1981](#); [Rasmussen and Tsuang, 1986](#)), and higher rates for OCD are seen among first-degree relatives of clinically ascertained patients ( [Nestadt et al., 2000a, 2000b](#); [Nestadt et al., 2000b](#); [Pauls et al., 1995](#)).

The initial family studies did not find elevated rates of OCD in first-degree relatives ( [Black et al., 1992](#); [Hoover and Insel, 1984](#); [McKeon and Murray, 1987](#)). However, after the ECA study, methods changed, and rates began to climb. [Lenane and coworkers \(1990\)](#) report that 30% of 46 adolescent probands had a first-degree relative with OCD. OCD was reported in 17% of parents and the age-corrected rate for siblings was 35%. [Riddle and coworkers \(1990b\)](#) report that 10% of parents from a cohort of 21 clinic patients with OCD were diagnosed with OCD. Twenty-five percent had subthreshold symptoms of obsessions or compulsions. In contrast to studies that relied on questionnaires or telephone inquiry, [Pauls and coworkers \(1995\)](#) directly examined relatives of 100 persons with OCD and of a control group of 33 psychiatrically unaffected persons. The rate of DSM-III-R defined OCD among relatives of patients with OCD was 10% versus 2% of the unaffected probands' relatives. There were also significant differences in rates of obsessive-compulsive traits (or subclinical OCD), with 8% of OCD probands' relatives affected and 2% of the relatives of those in the control group. Combining the subclinical and clinical threshold groups, 18% of OCD probands' relatives were affected compared with 4% of the control relatives. Like Pauls and coworkers, [Nestadt and coworkers \(2000b\)](#) blindly evaluated 343 relatives of 80 adults with OCD and obtained a control sample of 300 relatives of 73 persons ascertained by random number dialing. Patients with OCD were obtained from clinic rosters. Most family members were interviewed directly, and all family members were required to participate for a family to be included. Using a threshold of "definite" DSM-IV criteria, 11.7% of relatives of persons with OCD were affected compared with 2.7% of control relatives. When "probable and definite" OCD were included, then the rates climbed to 16% versus 6%. An additional finding of [Pauls and coworkers \(1995\)](#) and [Nestadt and coworkers \(2000a\)](#) was that earlier age of onset was associated with greater "familiarity" (greater likelihood of OCD among relatives). [Nestadt and coworkers \(2000a\)](#) performed an elaborate study employing segregation analysis of 80 families and a control group. The model of an autosomal dominant pattern of transmission with familial residual effects provided the best fit to the data.

Reports of elevated rates of OCD among patients with Tourette's syndrome ( [Pauls et al., 1986](#)) and of tics and a family history of tics among OCD probands ( [Grados et al., 2001](#); [Green and Pittman, 1986](#); [Leonard et al., 1992](#)) suggest that some cases of OCD may arise from the same genetic origin as Tourette's syndrome ( [Pauls and Leckman, 1986](#)). Reevaluation of the National Institute of Mental Health cohort and their relatives in a 2- to 7-year follow-up lends support to this hypothesis ( [Leonard et al., 1993](#)). Fifty-seven percent of 54 probands had a lifetime history of tics, 15% met criteria for Tourette's syndrome, and 22% had chronic multiple tics. Among their relatives, 14% had lifetime diagnoses of tics. Using a control group, [Grados and coworkers \(2001\)](#) have found that tics and OCD are significantly more likely in relatives of probands ascertained for OCD. Even more provocative are findings that "any tic disorder" is equally likely in relatives of probands with OCD whether the proband has a lifetime history of tics or not. An additional finding is that those relatives with tics and OCD have an earlier age of onset when compared with those relatives with only OCD.

### Neuropsychological Processes

Neuropsychological investigations of OCD provide some support for frontal lobe dysfunction, although this has not been unequivocal. There appear to be significant problems in frontal lobe functions affecting visual-spatial integration, reasoning, and memory in studies performed on adults with chronic OCD ( [Hollander et al., 1990](#)). This does not hold true for adolescents or children, however. [Flor-Henry and coworkers \(1979\)](#) studied 11 subjects with adult-onset OCD by employing age- and sex-matched controls to find frontal deficits, especially in the dominant lobe. These investigators propose that associated dominant temporal and parietal dysfunction stem from the failure of frontal inhibitory responses. Others ( [Insel et al., 1983a](#)) could not confirm frontal lobe findings. [Behar and coworkers \(1984\)](#) found signs of immaturity among 16 adolescent subjects on two measures reflecting frontal lobe function. The performance of six subjects on another test suggested "neurodevelopmental immaturity." Neurologic soft signs, such as synkinesia, were seen in five of the seven adolescents. [Cox and coworkers \(1989\)](#) suggested deficits in regulatory functions localized to the frontal lobe, although they did not confirm dominance of the frontal hemisphere. Few measures supported right-left hemispheric differences. The group underscored that findings were independent of impairment from OCD, and this implies a stable deficit unrelated to severity. [Beers and coworkers \(1999\)](#) examined 21 drug-naive children with new-onset OCD using a neuropsychological battery and found no abnormalities.

### Structural and Functional Neuroimaging

#### STRUCTURAL NEUROIMAGING

##### Computed Tomography

Computed tomographic views of adolescents with OCD or patients with OCD whose onset was in adolescence suggest ventricular enlargement independent of sex, age, duration, and types of symptoms ( [Behar et al., 1984](#)). [Luxenberg and coworkers \(1988\)](#) employed quantitative scanning in male patients with adolescent-onset OCD and a never-ill control group. In the OCD cohort, decreased mean volumes were seen in caudate nuclei bilaterally. Values of normal control subjects and those with OCD overlapped considerably, but pooled mean differences were consistent with a hypothesis of basal ganglia changes in OCD.

##### Structural Magnetic Resonance Imaging

When Garber and coworkers compared magnetic resonance imaging scans of adults with OCD and healthy controls, abnormalities in frontal cortex, cingulate gyrus, and lenticular nuclei were seen ( [Garber et al., 1989](#)). Past medication treatment or family history did not significantly influence findings. Kellner and coworkers did not discover significant structural differences between patients with OCD and normal controls (1991), but [Calabrese and coworkers \(1993\)](#) reported increases in the size of the caudate nuclei. A subsequent study reported that left caudate nuclei volumes exceeded those on the right ( [Calabrese et al., 1993](#)). [Jenike and coworkers \(1996\)](#) studied 10 female patients with OCD and a control group by magnetic resonance imaging. Increased opercular and whole cortex volumes were noted as well as a decrease in total white matter. In a parallel study, Alward and coworkers (1996) failed to find any differences in 24 adults with OCD when compared with a control



group.

## FUNCTIONAL NEUROIMAGING

### Cerebral Blood Flow and Single-Photon Emission Tomography

[Zohar and coworkers \(1989\)](#) measured regional cerebral blood flow (rCBF) changes using single-photon emission tomography with xenon-133 inhalation. Subjects who were stressed specifically to increase anxiety displayed *decreased* temporal rCBF. This pattern was sustained as the stressful stimuli were changed to induce higher levels of anxiety.

There are no published studies of single-photon emission tomography in children with OCD, but two adult studies reported increases in medial-frontal rCBF ([Hoehn-Saric, et al., 1991](#); [Machlin et al., 1991](#)). Rubin and coworkers examined rCBF with xenon inhalation and technetium uptake (1992). Xenon studies did not reveal differences between adults with OCD and normal controls, but with technetium uptake, increased activity in the orbitofrontal cortex was observed with significantly decreased bilateral activity in the head of the caudate ([Rubin et al., 1992](#)), a phenomenon previously noted by others ([Machlin, et al., 1991](#)).

### Positron Emission Tomography

Positron emission tomography has emerged as one of the most powerful tools to extend our understanding of OCD. The first wave of studies examined regional glucose use in the brains of persons with OCD compared with controls ([Baxter et al., 1987, 1988](#); [Martinot, 1990](#); [Nordahl et al., 1989](#); [Perani, 1995](#); [Swedo et al., 1989](#)). Taken together, these studies report increased activity in orbital gyri and the caudate nuclei. There are differences among the studies regarding increased activity in the anterior cingulate gyrus. [Rauch and coworkers \(1994\)](#) scanned individuals with OCD while provoking them with a feared stimulus; increased activation in the right caudate, orbital, thalamic, and anterior cingulate gyrus was observed.

Initial comparisons of patients with OCD and depressed adults suggest bilateral increased glucose metabolism in the orbital gyri ([Baxter et al., 1987, 1988](#); [Nordahl et al., 1989](#)) and the head of the caudate nuclei ([Baxter et al., 1988](#)). Baxter and colleagues propose that the orbital gyri may be specific for tension and anxiety in OCD. [Swedo and coworkers \(1989b\)](#) scanned adults with both current OCD and documented adolescent onset; they found increased bilateral prefrontal and anterior cingulate gyri activity. Other observations of elevated caudate nuclei activity ([Baxter et al., 1988](#)) are not confirmed. Increased metabolism in premotor and midfrontal regions is reported in patients with obsessional slowness ([Sawle et al., 1991](#)).

Subsequent work has examined regional brain metabolic changes after behavioral or pharmacologic treatment. [Benkelfat and coworkers \(1990\)](#) scanned eight patients after clomipramine treatment and found that medication decreased left caudate and orbital frontal activity. [Baxter and coworkers \(1992\)](#) studied adults before and after treatment with fluoxetine or cognitive-behavioral therapy. Responders displayed decreased right caudate nucleus glucose metabolism; no changes appeared in treatment nonresponders and never-ill controls. Swedo and coworkers found that adults with childhood-onset OCD exhibit decreased fronto-orbital metabolism associated with medication-related or spontaneous improvement in OCD severity ratings ([Swedo et al., 1992](#)).

### Functional Magnetic Resonance Imaging

Breiter and coworkers used functional magnetic resonance imaging (1996) in 10 subjects who were studied during provocation of symptoms. Their results point to elevated activity in the medial orbitofrontal, lateral frontal, and anterior cingulate gyri and in the insular cortex, caudate nucleus, lenticular nuclei (putamen and globus pallidus), and amygdala.

### Functional Magnetic Resonance Spectroscopy

Magnetic resonance spectroscopy is a noninvasive technique for studying brain function using magnetic fields. It measures the effects on unbalanced elements of specific perturbations generated by a magnetic field. In particular, magnetic resonance spectroscopy measures the influence of tiny local magnetic forces exerted by nearby nuclei on elements subsequent to magnetic perturbation. It has been applied to learn the density and activity of glutamate and *N*-acetyl-aspartate (a measure of neuronal destruction or injury) in the brain. One study explored the density of *N*-acetyl-aspartate in 11 drug-naive pediatric subjects and reported decreased *N*-acetyl-aspartate levels in the medial thalamus ([Fitzgerald et al., 2000](#)). A subsequent study of the same 11 subjects found elevated glutamate levels in the caudate nucleus bilaterally ([Rosenberg et al., 2000](#)). Furthermore, after paroxetine treatment, levels of glutamate in the caudate nuclei were equivalent to those in normal controls ([Rosenberg et al., 2000](#)).

## SUMMARY OF STRUCTURAL AND FUNCTIONAL NEUROIMAGING

Taken together, the results of most structural studies suggest differences between patients with OCD and never-ill controls in frontal cortex and in caudate nuclei. These findings are further supported by more detailed, functional studies showing elevated activity in orbitofrontal, lateral frontal, and cingulate gyri and in the caudate and lenticular nuclei (putamen and globus pallidus). More recent work has focused on glutamate receptors in the caudate and decreased neuronal activity in medial thalamus. Drawing on other neuroanatomic studies and these findings, a broad consensus has emerged that functional defects in a corticostriothalamocortical circuit underlie OCD.

## Neurochemistry: Serotonin and Dopamine

### SEROTONIN

Several lines of evidence lend support to the importance of 5-HT in the pathophysiology of OCD. Agents that affect 5-HT reuptake inhibition (SRIs) have been the most effective medication treatments for persons with OCD. Decreased cerebrospinal fluid concentrations of 5-hydroxyindoleacetic acid, the primary metabolite of 5-HT in the central nervous system, have correlated with clinical response to SRIs. Substances that increase serotonergic transmission, such as *L*-tryptophan and lithium, also have been successful in augmenting serotonergic medication in unresponsive patients. In addition, drug-naive children with OCD treated with paroxetine, a highly selective SRI, showed reductions in caudate nucleus volumes after successful treatment ([Gilbert et al., 2000](#)). The decrement in caudate nucleus volume was correlated with clinical improvement ([Gilbert et al., 2000](#)).

However, studies of various indirect measures of 5-HT in patients with OCD have produced equivocal findings. Adolescents with OCD were found to have normal 5-HT platelet levels when compared with healthy volunteers ([Flament et al., 1987](#)), and adults with OCD showed tritiated imipramine binding and 5-HT uptake similar to those in normal control subjects ([Insel et al., 1985](#)). A cohort composed of adolescents and adults with OCD displayed a substantial decrease in tritiated imipramine binding but no differences in 5-HT uptake ([Weizman et al., 1986](#)). Moreover, when patients who are successfully treated with SRIs for depression or OCD undergo tryptophan depletion, depression reemerges in depressed patients, but obsessive-compulsive symptoms do not reappear in those with OCD ([Barr et al., 1994](#)).

"Pharmacologic probes" that are putatively specific for 5-HT, such as metachlorophenyl-piperazine (MCP), tryptophan, fenfluramine, and metergoline yield contradictory results ([McDougle, 1999](#)). MCP elevated temperature, obsessive-compulsive symptoms, and anxiety in one study ([Zohar and Insel, 1987](#)) and did not affect OCD symptoms or create physiologic responses in another ([Charney et al., 1988](#)). In yet another study ([Hollander et al., 1992](#)), oral MCP reportedly increased symptoms in 55% of subjects. Symptom exacerbation with MCP predicted nonresponse to a selective SRI ([Hollander et al., 1992](#)). MCP administered after 3 months of clomipramine treatment did not evoke acute elevations in obsessive-compulsive symptoms that initially were seen in the subjects with untreated OCD ([Zohar et al., 1988](#)). Metergoline, a specific 5-HT agonist, decreased symptoms only slightly ([Zohar and Insel, 1987](#)). Fenfluramine did not produce increased symptoms ([McDougle, 1999](#)). Unresponsiveness to tryptophan depletion, MCP, and metergoline administration implies that other transmitter systems are relevant in OCD ([Charney et al., 1988](#)).

### DOPAMINE

A contributing role also has been proposed for dopamine ([McDougle, 1999](#); [Rapoport and Wise, 1988](#)). There are several lines of support for this hypothesis. First, increased metabolism in basal ganglia regions, which are rich in dopaminergic neurons, is observed on positron emission tomography scanning in persons with OCD. Second, OCD symptoms are often observed in disorders affecting basal ganglia (e.g., Sydenham's chorea, exposure to toxic agents, trauma). Third, dopaminergic

blocking agents (such as pimozide, haloperidol, risperidone, and olanzapine) have been used with some success to augment treatment in patients who are unresponsive to SRIs ([McDougle et al., 1994](#)). There is also evidence that fluoxetine, paroxetine, and clomipramine exert dopaminergic and serotonergic activity ([Austin et al., 1991](#); [Baldessarini and Marsh, 1990](#)).

### Neuroendocrine Studies

When compared with a matched sample of normal controls and subjects with Tourette's syndrome, a cohort of adults with OCD, who did not have a personal or family history of tic disorders, displayed significantly elevated cerebrospinal fluid levels of oxytocin. This is all the more interesting in view of findings of low cerebrospinal fluid oxytocin in patients with Tourette's syndrome who exhibit OCD. [Leckman and coworkers \(1994\)](#) point out that cognitive and behavioral symptoms of OCD are similar to the behavioral effects of oxytocin (e.g. cognitive, grooming, affiliative, and reproductive behaviors) that have been observed in animals. However, a subsequent trial of intranasal oxytocin ([Epperson, 1996](#)) to treat OCD did not produce discernible effects. However, the investigators acknowledge that only small amounts of oxytocin cross the blood-brain barrier.

### Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections

It is now appreciated that some children develop obsessive-compulsive symptoms after streptococcal infection. Estimates are that this may be an important mechanism in 10% to 20% of children developing OCD. Typically, symptoms arise along with tics, and this phenomenon may be related to obsessive-compulsive symptoms seen in Sydenham's chorea ([Leonard et al., 1999](#); [Swedo et al., 1993](#)). Swedo and coworkers propose that this represents an autoimmune disorder caused by the cross-reaction of streptococcal bacteria and basal ganglia structures ([Swedo et al., 1998](#)). Several lines of evidence support this hypothesis. Scanning data suggest that increased basal ganglia volumes are associated with pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS), just as they are in OCD, tic disorders and Sydenham's Chorea ([Giedd et al., 2000](#)). [Hallett and coworkers \(2000\)](#) infused basal ganglia structures in rats with sera from boys who had both a PANDAS history and the presence of antineuronal antibodies or with sera from healthy boys without antineuronal antibodies. It appeared that the rats infused with sera from affected boys developed stereotyped behaviors, whereas those infused with normal sera did not. However, [Singer and coworkers \(1999\)](#) also looked at antineuronal antibodies and found them both in boys with Tourette's syndrome and in normal control subjects. Although patients with Tourette's syndrome had higher median levels, these investigators did not find any correlation among mean levels of antibodies, clinical severity, symptom types, history (age of onset or suddenness), or evidence of streptococcal infection ([Singer et al., 1999](#)). In addition, there was no correlation between symptom severity or duration and basal ganglia size in a study of children diagnosed with PANDAS ([Giedd et al., 2000](#)). Preliminary data support the efficacy of immunologically curbing treatments such as intravenous immunoglobulin or plasmapheresis ([Perlmutter, 1999](#)).

Several cautionary notes are in order. First, it is very common for children with tic disorders (and perhaps OCD) to report an abrupt onset and to experience exacerbation of symptoms with infection. [Singer and coworkers \(2000\)](#) report that more than 50% of patients with tic disorders report this finding, and 11% report exacerbation of symptoms within 6 months of streptococcal infection. Furthermore, it appears that an autoimmune mechanism is relevant only when the pattern of acute onset is observed. When [Peterson and coworkers \(2000\)](#) compared a cohort of children with "ordinary" OCD to normal controls, children with attention deficit hyperactivity disorder, and children with tic disorders, they found no correlation with antistreptolysin O, Anti-DNAase B, or basal ganglia volumes and OCD. Furthermore, it appears that treatments with immunoglobulin or plasmapheresis are not helpful for patients with OCD who do not exhibit the infection-related onset and exacerbation of PANDAS ([Nicolson et al., 2000](#)). Although an autoimmune mechanism would predict particularly high rates of illness among relatives of individuals with PANDAS, it is interesting that a family study did not show rates greater than those seen for all OCD ([Lougee et al., 2000](#)). In addition, the number of first-degree relatives with childhood-onset symptoms was the same as in studies of ordinary OCD probands and their families.

### Corticostriothalamocortical Circuit

There is a reinforcing relationship between learning about serious mental disorders and our understanding of the neural networks operating in the central nervous system. From the study of disorders we learn about the development, functional interconnections, and neurotransmitter circuitry of the central nervous system. In turn, this new knowledge about the brain further extends our understanding of the pathophysiology of brain disorders, our ability to treat symptoms, and our understanding of the relationships among disorders. OCD is a paradigm of this reverberating process. Once it was shown that medication could improve OCD ([Thoren et al., 1980](#)), the way was opened to identifying biological mechanisms that predispose to it, precipitate it, and maintain it. In turn, this has advanced our general understanding of neural networks.

The current understanding of OCD integrates neuropsychological, anatomic, neurochemical, and electrophysiologic findings ([Baxter, 1999](#); [Baxter et al., 1992](#); [Insel, 1992](#)). It posits the existence of somatotopically organized connections in distant brain structures arranged into corticostriothalamocortical circuits that subserve planning, execution, and termination of voluntary movements. In 1990, [Alexander and coworkers \(1990\)](#) proposed the existence of "basal ganglia-thalamocortical circuits." They suggested that a "lateral orbitofrontal circuit" projecting to ventromedial sectors of the caudate, the substantia nigra, and the globus pallidus may be impaired in OCD. There were doubts about how separate these circuits could be. Subsequent positron emission tomography findings led Baxter and coworkers ([1992, 1999](#)) to propose an "orbital-basal ganglia-thalamic circuit" similar to Alexander's.

This "basic model" ([Baxter, 1999](#)) puts forth that frontal cortical and limbic structures have excitatory effects on striatal structures through glutamatergic efferents. These striatal structures, including the caudate nucleus, putamen, nucleus accumbens, and olfactory tubercle, project to the internal and external globus pallidus. From the internal globus pallidus, inhibitory efferents using g-aminobutyric acid have tonic activity at the thalamus. Consequently, *increased* striatal activity *decreases* inhibitory activity (or creates disinhibition) at the thalamus. Increased activation of thalamic nuclei produces excitatory transmission to cortical structures and then movement.

However, this basic model has required modification in two ways. The first modification introduces separate *direct* and *indirect* pathways between the striatum and the thalamus. The direct pathway is so termed because efferents pass *directly* from the striatum to the thalamus, thus exerting a disinhibiting (activating) effect. Conversely, the *indirect pathway* sends various efferents to subthalamic nuclei that, in turn, send efferents to both the globus pallidus externa and the globus pallidus interna. The indirect pathway "moderates" activity in the thalamus ([Baxter, 1999](#)).

The second modification proposes two parallel systems from the striatum, one related to the dorsolateral region and the other the ventromedial region. Input to the ventromedial region is largely from limbic structures, whereas dorsolateral regions receive input from the dorsal cortex. From the striatum, both direct and indirect systems send information to the thalamus. Activation of the dorsolateral system can produce either disinhibition or inhibition of the dorsal thalamus through the direct or indirect system, respectively. Activation of the ventromedial pathway only results in inhibition of the dorsal thalamus ([Baxter, 1999](#); [Peterson et al., 1999](#)). Balance in the activity between the dorsolateral and ventromedial striatal systems is crucial in maintaining control. In OCD, increased tone in the pathway from limbic cortex to ventromedial striatum excessively activates the direct pathway, decreases inhibition, and produces symptoms (overactivation) ([Baxter, 1999](#)). With treatment using SRIs, afferents to the ventromedial pathway are attenuated at the level of the striatum; direct pathway tone decreases relative to the dorsolateral system, and equilibrium is restored. [Baxter \(1999\)](#) hypothesizes that behavioral treatment works by increasing tone in the dorsolateral system relative to the ventromedial system; increasing tone in dorsolateral system restores the balance.

This model is compatible with the variety of neurochemical findings. First, it accounts for the efficacy of SRIs. 5-HT selectively activates g-aminobutyric acid and reduces glutamatergic output from limbic efferents to the ventromedial caudate. Consequently, serotonergic augmentation substantially reduces activation in the ventromedial system.

The relationship between 5-HT and dopamine adds another important dimension. [Baxter \(1999\)](#) proposes that dopaminergic inputs distributed throughout the caudate nucleus have downstream effects on the thalamus. There is a gradient of D1 and D2 receptors across the caudate. Higher concentrations of D2 receptors are observed in the dorsolateral caudate and decrease as one moves toward the ventromedial region. Conversely, a lower concentration of D1 receptors in the dorsolateral caudate increases as one moves toward the ventromedial region. Blockade of D2 receptors would have a greater effect on dorsolateral circuitry and produce a decrease in both direct and indirect pathway activity. This too would also change the balance in activity between the dorsolateral and ventromedial regions.

### Psychoanalytic Theories

Psychoanalytic theories of OCD have not been subjected to rigorous, systematic scientific research. However, a rich anecdotal literature exists and is important for its historical value. In it, we can also see the seeds for later biological investigations. Freud observed obsessions and compulsions early in his investigations into the unconscious ([1953a](#)). Anna Freud proposed that the failure of two ego functions promoted obsessional neuroses. Fusion of sexual and aggressive drives from the id



ordinarily permits the infant to contain destructive, annihilating drives and to maintain attachment to the caretaker. Synthesis creates harmony out of “opposing strivings” before they reach consciousness (Freud, 1966). Her hypothesis is particularly interesting in light of the importance we now attach to the frontal lobe in inhibiting or modifying of sexual and aggressive impulses and our appreciation of impairment in frontal lobe function in OCD. Anna Freud offered that the consequence of these ego deficits was that patients with obsessional neuroses are unsuccessful in their mastering ambivalence, resolution of sexual and aggressive urges, and “intrasystemic contradictions” in the id, such as feelings of love–hate, masculinity–femininity, and so on (Freud, 1966). She also commented on the primacy of constitutional and environmental contributions. She also described characteristic defenses of undoing, intellectualization, reaction formation, and isolation that are so prominent in OCD.

## DIFFERENTIAL DIAGNOSIS

Table 68.2 offers a partial list of disorders in which obsessions or compulsions are seen. Although rare, some specific brain disorders (“organic brain syndromes”) display OCD characteristics (Drummond and Gravestock, 1988; Frye and Arnold, 1981; McKeon et al., 1984).

Anorexia nervosa
Body dysmorphic disorder
Delusional disorder (all types)
Depression
“Fear of AIDS”
Hypochondriasis
Obsessive-compulsive personality disorder
Organic mental disorder*
Panic disorder
Pervasive developmental disorder
Phobias
Posttraumatic stress disorder
Schizophrenia
Schizotypal personality
Somatization disorder
Somatiform disorders
Trichotillomania
Tourette’s syndrome

\*Specifically arising from central nervous system trauma, tumors, toxins.

**Table 68.2. Disorders Manifesting Obsessions or Compulsions**

The DSM-IV handles disorders comorbid with OCD by requiring that the content of the obsessions not be “restricted” to any coexistent disorder. Although this eliminates some difficulties, it creates others; sometimes it is not easy to discern how “related” or “unrelated” a symptom is. The DSM-IV combines a heterogeneous group of disorders and comorbid features (Leonard et al., 1993; Swedo et al., 1989a) under the term OCD on the basis of presence of a single symptom. Many disorders and comorbid symptoms of different origins are subsumed under this one label. Therefore, clinicians are obliged to conduct thorough diagnostic assessments to detect all the coexisting conditions that are active along with OCD (Towbin et al., 1987). Neglecting to do so leads one to miss findings that are relevant to treatment. Similarly, if research studies are to be generalizable and valid, investigators are obliged to provide comprehensive descriptions of their sample subjects (Towbin et al., 1987). Moreover, care should be taken not to equate *subclinical* obsessions and compulsions and OCD, especially in adolescents. Most subclinical phenomena appear to be stable features that do not interfere with development and functioning (Flament et al., 1988).

Overlapping symptoms sometimes create confusion between obsessive-compulsive personality disorder (OCPD) and OCD. Most patients with OCD do not exhibit OCPD, but some data suggest that OCPD may be more common among patients with OCD than in the general population, particularly among those who have hoarding symptoms (Frost et al., 2000). Although persons with OCPD may have rigid routines, needs for orderliness, hoarding behaviors, and indecisiveness, they usually do not experience these as ego-dystonic. Their compulsive behaviors do not cause them anxiety and, ordinarily, do not result in impairment. Impairment in OCPD is a product of patterns of beliefs or behaviors, not isolated symptoms. OCPD often undergoes phases of exacerbation and remission, and particular characteristics may escalate when a patient is stressed. Conversely, patients with OCD generally are not emotionally cold, unexpressive, stingy, or especially rigid about moral or ethical matters. Furthermore, hoarding, list making, and rigid scheduling behaviors are uncommon among compulsions reported by patients with OCD (Rasmussen and Tsuang, 1986). Adding to the complexity, Samuels and coworkers (2000) systematically assessed OCD probands and relatives against a control group. Compared with controls, OCD probands and their relatives showed elevated rates of OCPD. This may be hypothesized to reflect a “spectrum” of OCD to OCPD conditions, which display vertical transmission.

Differentiating psychosis from an overvalued idea in a patient with OCD can be vexing. Some patients with OCD precariously balance between delusional conviction and insight. At times, they may believe that they are right to engage in compulsive behaviors, but at other times they are aware that their urges are senseless (or excessive) and a product of their minds. The DSM-IV permits the diagnosis of OCD in the presence of when schizophrenia or delusional disorders. Comorbid syndromes like these can frustrate attempts to discover whether the patient possesses insight (the ability to understand that their obsessions originate in their minds and are not imposed by external sources). The absence of insight carries a poorer prognosis and should influence treatment strategies. Clinicians should carefully search for other comorbid diagnoses when patients have little or no insight.

*Fear of acquired immunodeficiency syndrome (AIDS)* consists of obsessional fears and preoccupations about contracting AIDS (Fisman and Walsh, 1994; Wagner and Sullivan, 1991). Although blood tests permit a positive diagnosis of AIDS, a clinician cannot give a diagnosis of hypochondriasis confidently to those with negative or equivocal serologic results. The long incubation period and high risks to adolescents who do not practice safe sex means that those with “fear of AIDS” constitute a group whose concerns can be neither completely refuted nor confirmed. Many cases straddle the boundary between subthreshold obsessions and OCD.

## EVALUATION

### Clinical Evaluation

The critical first step in the treatment of children with OCD is evaluation. Children often feel embarrassed about their symptoms and underreport impairment. Frequently, children fear that their symptoms are bizarre and “crazy.” They are most likely to describe symptoms to a clinician who resists confrontation, conveys acceptance, and respects privacy; it takes time for the patient to reveal his or her fears fully. Consequently, assessment usually cannot be completed in one meeting (American Academy of Child and Adolescent Psychiatry, 1998).

An adequate evaluation of OCD must sample multiple sources. It is desirable to gather history from the patient alone, from his or her parents alone, and from the family together. Helpful information can be obtained from teachers on academic performance, peer relationships, areas of impairment, and tasks presenting special challenges. Siblings may provide valuable information on family responses to the patient.

The objectives of individual meetings are to estimate the extent of impairment, the developmental level attained by the patient, and the associated diagnoses and symptoms that are hampering progress. The clinician needs to learn about the patient's strengths and weaknesses, fears and aspirations, achievements and disappointments. Impairment created by symptoms often permeates home, school, work, peer relations, and self-image. This dictates assessing the patient's adjustment in each of these domains. Although it may be tempting to use meetings with the patient to overpower denial, root out secrets, and unmask obsessions, fostering a relationship is crucial. It is a common mistake to become entangled in an effort to root out symptoms and conquer the patient's concealment.

A family may provide information on the extent to which symptoms interfere with the patient's life, but the family also may unwittingly perpetuate them. OCD is a familial disorder in several ways; it has immediate influences on a family's use of time, its emotions, and energies, and it is an enduring family feature genetically and psychologically. The aims of the family evaluation are to characterize the prevailing family dynamics and to identify the manner and extent of effect on the patient's family. This means learning the significance of the patient's symptoms to the parents, how the parents understand their child, and the family's responses to the patient's behaviors. These aims are most readily accomplished with direct observation and in interaction with the family. A critical question is whether concomitant family treatment may be needed to change patterns of communication, to alter affiliations, and to clarify sources of conflict. When parental psychopathology aggravates the patient's condition, parental treatment may be necessary.

Meeting with parents alone provides time to educate them and to learn sensitive information from them. Information about the parents and their marriage, parental disappointment, concern, and frustration about their child or each other, and potentially confidential information about family history or about their own symptoms

should be obtained without the patient. A second major aim of the meeting with parents alone is to teach and reassure them about their child's illness. They may fear that their child is psychotic or untreatable, feel guilty that they have caused their child's condition, or fear that the clinician is going to blame them for their child's difficulties or compete with them for their child's affection. Meetings are opportunities to reassure and solidify a collaborative relationship with parents.

Creating an arena where symptoms and stresses can be discussed and monitored may not be conducive to achieving a thorough overview of all the domains of strength and difficulty. Standardized instruments are more effective for general assessment and screening. Standardized instruments may not convey the quality of difficulties or the impact of symptoms on the patient and others, but they do review certain behaviors or domains. They also permit the clinician to assess severity in comparison with clinical populations. Two instruments have been used with children and may assist a general clinician in this way: the self-report LOI ([Berg et al., 1986](#); [Cooper, 1970](#)) and the clinician-rated Children's Yale–Brown Obsessive Compulsive Scale (CY-BOCS) ([Goodman et al., 1989](#); [Scahill et al., 1997](#)).

### Laboratory Studies

There are no pathognomonic laboratory findings in OCD. Appropriate laboratory evaluation follows from the findings of the history, physical, mental status, and psychological examination. An electrocardiogram, complete blood count with differential, and baseline blood chemistries, including electrolytes, as well as liver function tests, blood urea nitrogen, and creatinine may be necessary before commencing somatic treatment. Measures of serum copper for Wilson's disease are unnecessary in the absence of psychotic symptoms or physical findings of tics or chorea. Computed tomography or magnetic resonance scanning is warranted only when focal neurologic findings are discovered. Electroencephalography is indicated only when other features suggest a seizure disorder.

Psychological tests can provide a detailed picture of intellectual function, severity of acute stressors, and characteristic defensive structures in patients with OCD. Standardized intelligence tests, such as the Wechsler Intelligence Scales for Children, third edition or the Differential Abilities Scale for younger children, are appropriate. The role of projective tests, such as the Rorschach, Thematic Apperception Test, or Draw-a-Person Test to identify sources of stress is more equivocal. The Adolescent Multiphasic Personality Inventory may be useful in identifying current characteristic defense patterns.

Severity measures of obsessions and compulsions are discussed below. In addition, the parents-on-child version of the Child Behavior Checklist ([Achenbach and Edelbrock, 1983](#)) or the Behavior Assessment System for Children can help to discover other maladaptive behaviors.

## TREATMENT

Various treatments have been applied to children with OCD, although only behavioral approaches and medications have been systematically studied. Adjunctive family treatment and individual psychotherapy are often necessary. There is a role for inpatient hospitalization under some circumstances.

### Family Support and Illness Education

Whichever modalities will be used, the clinician's relationship with the patient is paramount. First, most children with OCD are apprehensive and secretive. They are anxious about treatment and their symptoms. The relationship with the clinician can reassure and absorb much of the anxiety in a way that promotes discussion and treatment. Second, many patients are uneasy discussing their thoughts or rituals because the content is scatological or sexual. Patients must feel that the clinician will understand the distress they experience and can be trusted with the unacceptable content of their symptoms. Third, when treatments require a lengthy trial period or cause increasing anxiety, the relationship with the clinician must be sturdy if treatment is to be sustained. Fourth, because treatment usually does not produce a cure, most patients have a chronic course. The relationship established with a clinician may be long and must be capable of sustaining both parties over years.

After the diagnosis is established, it is critical to sit with the patient and his or her parents to review the causes, nature, and course of illness. The clinician should explain the relevance of comorbid features for interventions and prognosis. This may need repetition at other times during the course of treatment. It is helpful to the family and patient to understand the likely chronic nature of OCD, the objectives of treatment to reduce the interference that symptoms impose on the patient's life, and the primacy of supporting optimal development. It may be unrealistic to expect that symptoms will disappear. Furthermore, it is important to assist the family to learn how they can best support the patient and discover what supports they need to sustain them for the long term. At the same time, clinicians have every reason to be encouraging and optimistic that symptoms can be reduced.

### Behavioral Therapy

The consensus is that behavioral treatments, particularly cognitive–behavioral therapy, are the first line of intervention for children and adolescents when symptoms are mild to moderate ([March et al., 1997](#)). Behavioral treatments have been systematically investigated for adults ([Foa et al., 1985](#); [Marks et al., 1980](#)) and children ([de Haan et al., 1998](#); [March, 1995](#); [March et al., 1994](#)) with OCD. There are many reports of single case studies without control groups ([Wolff and Wolff, 1991](#)). Behavioral techniques such as self-observation, extinction, operant conditioning, and modeling have been used with adolescents ([Wolff and Wolff, 1991](#)). The preliminary studies suffered from design flaws such as those that plagued the early pharmacologic research ([Towbin et al., 1987](#)). The combination of *flooding and response prevention* appears to produce favorable results with adults and adolescents who suffer from compulsions ([Steketee et al., 1982](#)). This method uses direct and imaginary exposure to the feared object or event, followed by thwarting any opportunity to engage in symptom reactions. A patient with contamination fears about garbage would thrust his or her hand into a filled garbage can and be restricted from washing for a period of hours. Reported rates of improvement are near 90% in achieving moderate to complete improvement ([Foa et al., 1985](#)) and correlate with neuroanatomic changes ([Baxter et al., 1992](#)). *Graduated exposure with response prevention* is a modification of this technique, which is less threatening to children and adolescents ([March et al., 1994](#); [Wolff and Wolff, 1991](#)). [March and coworkers \(1994\)](#) employed a cognitive–behavioral method with exposure and response prevention to treat 15 children, of whom nine (60%) achieved at least a 50% reduction in symptoms that was sustained for 18 months. De Haan and coworkers found exposure with response prevention to be as effective as clomipramine treatment (1998). Despite many design shortcomings, evidence suggests that behavioral interventions can be useful in the treatment of OCD ([Rapaport and Inhoff-Germaine, 2000](#)). Clinicians who are trained and experienced in applying these techniques achieve the best results.

### Pharmacologic Treatment

#### SINGLE-DRUG TREATMENT

Treatment with a single agent has been shown to be effective in nearly 50% of patients with OCD. Early systematic investigations of medication were plagued by problems in diagnosis and measurement that made it difficult to generalize and interpret the findings ([Towbin et al., 1987](#)). Today, it is widely known that various SRIs appear to be useful in the treatment of OCD. Although the claims for efficacy are ubiquitous in advertisements and media, they may mislead patients to conclude that everyone with OCD will be healed by medication. It is helpful to inform patients at the outset that reliable studies have shown that 40% to 50% of drug-naïve patients experience a reduction of 25% to 40% severity in symptoms. This is important when obtaining consent before treatment and in helping patients understand why they may continue to experience symptoms after a course of treatment.

The choice of agents should take into account not only OCD, but also whether other psychopathology is present. The choice of medication for OCD should consider the presence of coexisting panic disorder, psychotic or schizotypal features, depression, or Tourette's syndrome.

To determine whether a patient has responded to a medication, it is crucial that sufficient doses be given for a sufficient duration. Most studies suggest that an adequate trial has been offered when the patient receives either the maximum allowable dose or the maximum dose that a patient can tolerate for no less than 12 weeks. Gradual dose reductions are necessary to avert withdrawal reactions when discontinuing those medications with shorter half-lives such as fluvoxamine, paroxetine, and sertraline.

The most thoroughly studied medications in the treatment of OCD are potent SRIs, which also affect other neurotransmitter systems. Blinded, placebo-controlled trials in children have been conducted with clomipramine ([DeVeugh-Geiss et al., 1992](#); [Flament et al., 1985](#); [Insel et al., 1983a](#)), fluoxetine ([Fontaine and Chouinard, 1986](#); [Riddle et al., 1990a](#)), fluvoxamine ([Price et al., 1987](#), [Riddle et al., 1996](#); [Riddle et al., 2001](#)), and sertraline ([Alderman et al., 1998](#); [Chouinard et al., 1990](#); [March et al., 1998](#)). Open trials with paroxetine ([Rosenberg et al., 1999](#)) and with citalopram ([Pallanti et al., 1999](#); [Thomsen, 1997](#)) have also been reported. With the advent of new U.S. federal government rules requiring safety and efficacy studies in children, clinicians can look forward to seeing more of these agents approved for use in children. There have been few “head-to-head” comparisons of these agents, and so there is little to guide the clinician in making first choices among these agents. [Mataix-Cols and coworkers \(1999\)](#) attempted to predict the outcome to placebo or active agents by stratifying the cohort according to factor-analyzed subgroups (see earlier). Their results suggest that the hoarding group is less likely than other subtypes to respond to SRIs. The other factor groups are not different



from one another in their responsiveness to SRIs.

In adolescent patients, clomipramine has been studied most ([DeVeugh-Geiss et al., 1992](#)). Initial studies reported an average 46% symptom reduction. Response rates were noted in 74% of patients ([Flament et al., 1985](#)). These results correlate with the findings from adult trials, in which an average 30% reduction occurred ([Insel et al., 1983b](#)). The improvement occurred in a broad variety of patients in both studies and was independent of symptom type, age of onset, or response to previous medications. [Flament and coworkers \(1987\)](#) find that response correlates with platelet 5-HT concentration and monoamine oxidase activity; lower 5-HT concentration is associated with greater symptom severity, and high 5-HT concentrations appear to predict clinical response to clomipramine. The specificity of serotonergic effects of clomipramine in the treatment of OCD symptoms is suggested by two studies. In a double-blind, placebo-controlled investigation of desipramine versus clomipramine ([Leonard et al., 1989](#)), desipramine was no more effective than placebo in reducing OCD symptoms in adolescents. Later, a double-blind substitution study ([Leonard et al., 1991](#)) suggested substantially higher relapse rates in patients taking desipramine compared with clomipramine.

Side effects of clomipramine can be problematic. Anticholinergic side effects, including dizziness, xerostomia, blurred vision, postural hypotension, tachycardia, sedation, and constipation can occur and can generate noncompliance. Side effects are less likely when one starts with very low doses and gradually increases the medication until symptoms decline. The maximum recommended dose is 5 mg/kg per day or 250 mg per day. One should obtain electrocardiograms and liver function studies at 3-month intervals for the first year of treatment and then at 6-month intervals.

In adults, fewer side effects are reported with fluoxetine than with clomipramine ([Pigott et al., 1990](#)). Double-blind, placebo-controlled trials with children suggest that fluoxetine is effective in controlling OCD symptoms ([Riddle et al., 1992](#)). Doses beginning with 5 mg per day and increasing gradually to a maximum of 60 mg per day are most commonly used. Of the reported side effects, the most frequent are agitation, insomnia, anorexia, dizziness, xerostomia, and increased anxiety. Fluoxetine also may cause a disturbing akathisia. Concerns about suicidal ideation and aggression have also been raised ([King et al., 1991](#); [Riddle et al., 1991](#)).

Sertraline was reported to be safe and useful in an open trial ([Alderman et al., 1998](#)) and then superior to placebo in a multisite, double-blind, placebo-controlled trial of 187 patients ([March et al., 1998](#)). Doses up to 200 mg per day were provided by a flexible strategy. Using a definition of 25% or greater reduction in symptoms, 42% of patients taking sertraline were judged to be "responders." The response rate on placebo was 26%. Thirteen percent of patients taking medication discontinued treatment subsequent to the trial because of side effects. The most common ill effects were insomnia, nausea, agitation, and tremor. There were no effects on vital signs or cardiac function.

Fluvoxamine has a single-ring structure. Side effects resemble those of fluoxetine and include nausea, lethargy, and insomnia ([Riddle et al., 2001](#)). Doses beginning at 25 mg per day and increasing gradually up to a maximum of 5 mg/kg per day or 300 mg per day have been employed ([Apter et al., 1994](#); [Price et al., 1987](#)). [Apter and coworkers \(1994\)](#) conducted an open trial in 14 adolescents. [Riddle and coworkers \(2001\)](#) performed a multisite, double-blind, placebo-controlled trial of fluvoxamine employing doses up to 200 mg per day in 136 children and adolescents with OCD; active treatment produced a mean 25% (or greater) decline in CY-BOCS scores over 10 weeks. Overall, 42% of patients were deemed responders.

#### AUGMENTATION STRATEGIES

It can be readily seen that between 40% and 50% of persons with OCD without associated diagnoses may not respond to adequate trials of SRIs. The response to one SRI agent does not predict the response to another, and side effects of one agent may not predict side effects of another. For this reason, it is important to offer adequate doses for a sufficient period of at least two agents before moving on to augmentation strategies. When one is combining agents with selective SRIs, particular attention should be directed to the metabolic pathways of the agents employed. Drug-drug interactions based on inhibition of the cytochrome P-450 system can lead to toxicity and side effects. Before one adds any prescription medication to a selective SRI agent, there are excellent and reliable website resources that can be consulted, such as the one maintained by David Flockhart, M.D., Ph.D., at Georgetown University in Washington, DC ([flockhad@gusun.georgetown.edu](mailto:flockhad@gusun.georgetown.edu)).

Although the use of polypharmacy is generally to be avoided, when adequate trials of two agents fail, an augmentation strategy may be necessary. [McDougle and coworkers \(1994\)](#) find that patients with Tourette's syndrome, tics, or a family history of tics who are refractory to single-drug SRI treatment may benefit from the addition of dopaminergic blocking agents, such as haloperidol or pimozide. In addition, a few SRI-refractory patients with OCD who did not have tics or a family history of tics also improved with addition of dopaminergic blocking agents. This work was extended to risperidone in blinded, placebo-controlled trials ([Fitzgerald et al., 1999](#), [McDougle et al., 2000](#)) and olanzapine in open trials ([Weiss et al., 1999](#)). This step was conceptually important, too. Dopaminergic inhibition in the corticostriatohalamocortical circuit may be critical to successful 5-HT facilitation.

There are only scanty data on adding lithium ([Rasmussen, 1984](#)) or triiodothyronine. Buspirone can increase serotonergic activity in conjunction with SRIs, but there was no evidence of benefit using buspirone augmentation in OCD ([Grady et al., 1993](#); [Markovitz et al., 1990](#)). Small-scale studies suggest that addition of clomipramine to a selective SRI may be warranted ([Figueroa et al., 1998](#)). Care must be taken to monitor electrocardiographic changes and cardiovascular side effects closely. Clonazepam has been useful to add for high levels of comorbid anxiety or panic ([March and Leonard, 1996](#)), but sedation and memory problems may be encountered. Serious toxicity results from taking fluoxetine with L-tryptophan, and this combination is discouraged ([Steiner and Fontaine, 1986](#)).

#### Psychodynamic Psychotherapy

Psychodynamic psychotherapy may be indicated when children have conflicts, associated with their obsessions or compulsions, that interfere with their optimal development. The use of psychodynamic psychotherapy for patients with OCD does not mean that a clinician has determined that the symptoms have a psychodynamic origin. Psychodynamic treatment has a role in the treatment of reactions or conflicts that accompany OCD. Examples of these concerns may include insecurities related to family dysfunction or divorce, damage to self-esteem, inappropriate expectations, perfectionistic strivings that do not respond to pharmacologic or behavioral treatment, regulation of sexual or aggressive impulses, and the impact of a potentially chronic illness during the developmental years. Moreover, patients may adopt defenses that obstruct their recovery. Psychotherapy geared to diminishing the impact of these impediments can reduce the stress experienced by patients and can promote their treatment.

#### Partial Hospital and Inpatient Treatment

In most cases, it is unnecessary to hospitalize patients to evaluate their symptoms thoroughly. However, patients and their families do encounter crises. Examples are when symptoms spiral completely out of everyone's control, the family's capacity to support the patient is thoroughly depleted, symptoms are dangerous, or a course of adequate treatment fails. Although these circumstances can pressure clinicians to initiate additional interventions quickly, it should be remembered that, usually, crises emerge from chronic strains that accumulate over time. Initiating treatments without a sufficient understanding of these strains may undermine subsequent therapy.

In a crisis, the patient may benefit from partial (or day-treatment) hospitalization and, in the event this does not prove sufficiently helpful, inpatient treatment. These are constructive alternatives to precipitous changes in outpatient treatment. Partial or inpatient hospitalization can reduce the burden on parents to manage and contain uncontrollable symptoms and can reduce the impossible demands on the patient to improve instantly. It reduces the risk of intervening improperly. The primary objectives of hospitalization are to provide rapid, objective assessment of the severity of the patient's impairment outside the home, to facilitate simultaneous initiation of psychological, family, and pharmacologic treatments, and to diminish symptoms by reducing stresses and anxiety.

#### OUTCOME AND FOLLOW-UP DATA

The outcome of an episode of OCD can range from complete, permanent remission to relentless decline. Points along this continuum include complete remission with discrete recurrent episodes, partial remission (chronic low to moderate symptoms), and partial remission punctuated by severe flare-ups ([Goodwin et al., 1969](#)). Studies of patients who are self-referred for treatment do not provide conclusions about outcome that are generalizable to other patient groups. [Rasmussen and Tsuang \(1986\)](#) suggest that, in adults, "continuous" illness with fluctuating severity is most common (84%), and a deteriorating course is next most common (15%). Others ([Rachman and Hodgson, 1980](#)) report 2-year spontaneous remission rates of 65%. [Mawson and coworkers \(1982\)](#) followed their treatment cohort of 40 patients over 2 years and discovered greater than 80% improvement on all measures for the 37 subjects available. However, when hospitalized patients are sampled, the rates of those who are rated "greatly improved" declines to 30% ([Goodwin et al., 1969](#); [Welner et al., 1976](#)).

It has been challenging to obtain reliable longitudinal clinical data. This gap shrank with the study of [Skoog and Skoog \(1999\)](#) on the natural history of 251 adult patients over 30 years. In this sample, 29% had their onset of OCD before age 20, 40% between ages 20 and 29 years, and 32% after age 30. This finding

corresponds to [Black's \(1974\)](#) report of 40% with onset before age 20, and an average age of onset in the early 20s. The ECA data also reflect this; the mean onset was 20.9 to 25.4 years of age ([Karno et al., 1988](#)). Departing from reports of others ([Rachman, 1985](#)), Skoog and Skoog reported that the most common course of illness was intermittent (i.e., remission of symptoms between episodes) (56%), although chronic illness (unremitting, constant severity) was common (27%), as was an episodic course (single episode lasting 5 years or less) (17%).

There is one prospective study of a nonclinical adolescent cohort. [Berg and coworkers \(1989\)](#) resampled a public high school adolescent cohort of 46 subjects 2 years after they were identified with either OCD (n = 16) or "subclinical" obsessions or compulsions (n = 10). Initially, of the students with elevated LOI scores, only one had sought treatment. Two findings emerged at follow-up. First, those with "subclinical" obsessions and compulsions did not worsen; only one of 10 developed OCD. Second, only five of 16 (31%) initially diagnosed with OCD met criteria 2 years later. Furthermore, the mean LOI interference scores of those diagnosed OCD diminished by 30%, beneath the cutoff for clinically significant impairment. The authors suggest that this could reflect actual improvement or methodologic unreliability. The most likely predictors of an OCD diagnosis after 2 years were previous diagnosis of OCD and the presence of another psychiatric diagnosis with OC features. This finding supports the concept of OCD as a heterogeneous disorder with waxing and waning symptoms. The 69% recovery rate approaches the spontaneous adult remission rate proposed by [Rachman and Hodgson \(1980\)](#) and [Goodwin and others \(1969\)](#).

In a prospective study of an adolescent clinical cohort, [Swedo and coworkers \(1989a\)](#) evaluated 27 adolescents who participated in clinical trials of clomipramine at the National Institute of Mental Health. Two to 5 years after successful clinical trials, 25 subjects (92%) continued to have depression or anxiety, despite improvement in OCD. [Bolton and coworkers \(1983\)](#) reviewed the outcomes of 15 adolescent inpatients and outpatients 9 to 48 months after family and behavioral treatment. A "good" response to treatment occurred in 66%. [Leonard and coworkers \(1993\)](#) reviewed records of 54 adolescents at 3.5 years (range, 2 to 7 years) after treatment. Although 94% had some symptoms, 57% did not meet criteria for OCD; only 19% were worse than on initial evaluation. Seventy percent remained on medication. Poor response to medication, presence of tics, and parental psychopathology predicted a poorer outcome ([Leonard et al., 1992](#)).

## FUTURE RESEARCH

OCD research reflects the exciting possibilities in this new millennium. Already we have come a great distance. We now know the prevalence of this disorder, have learned the limitations of screening instruments for ascertaining internalizing disorders, have demarcated a boundary between ordinary defenses and pathology, and have discovered potentially meaningful subtypes. Newer imaging techniques have illuminated structural relationships and have linked neurochemical and anatomic findings in meaningful ways. Family study methods have provided important clues to patterns of transmission, and genetic linkage studies are under way.

OCD continues to provide opportunities to discover answers to basic, far-reaching questions. Among these are the basic relationships between genetic endowment and environmental experience ([Hyman, 2000](#)). Purer agonists and antagonists that act at specific subtype sites may uncover more details of 5-HT physiology, relationships among brain structures, and complex frontal, basal ganglia, and thalamic pathways. The lines of inquiry in genetics are already charted. Products of these genes can reveal the molecular biology of OCD and can provide clues about the relationships among anatomy, physiology, and psyche. While learning about these genetic sites, risk and protective factors contributing to severity and outcome can be studied and understood.

We continue to be optimistic about clinical care. Interest in OCD has produced well-informed clinicians with greater diagnostic and treatment skills. Self-help organizations, such as the OC Foundation (P.O. Box 9573, New Haven, CT 06535), have informed the public and are lessening the isolation and embarrassment of many persons with OCD. As organizations and clinicians educate the public, persons who fear treatment or who have been disappointed in previous efforts are now receiving treatment and achieving some relief. The use of specific behavioral techniques, behaviorally and dynamically informed family treatments, psychotherapy, and medications offer improvement for a majority of seriously ill patients. Nevertheless, we cannot sit back now. Our pharmacologic treatments, even with augmentation, are still not sufficiently effective for up to 40% of OCD sufferers, particularly those with prominent hoarding symptoms. It is apparent that many persons with OCD continue to suffer in secret. Epidemiologic studies remind us that most persons with OCD have not sought consultation or treatment. There are too few clinicians schooled in the specific techniques of cognitive-behavioral treatment, and there is a tendency to neglect this intervention. Continued vigorous effort to inform the public and to assist primary care clinicians in recognition of OCD is still needed. As we learn more about neural circuitry and brain chemistry, there is a realistic basis to be hopeful that new and better treatments will become available in the next 5 years. Moreover, preventive measures can reduce the risk of developing OCD and severity of symptoms once genetic factors are known.

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## 69 SOMATOFORM DISORDER

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Physical symptoms and complaints are exceptionally common in children and adolescents ([Campo and Fritsch, 1994](#); [Fritz et al., 1997](#)), with *somatoform disorders* representing the severe end on a continuum in which medically unexplained physical symptoms are associated with significant distress and functional impairment. The common feature of somatoform disorders is the presence of physical symptoms suggesting an underlying medical condition, but a general medical condition either is not identified or does not fully account for the symptoms or the degree of functional impairment ([American Psychiatric Association, 1994](#)). Psychiatrists are most likely to be involved in the less common, more extreme cases; most children who present with distressing physical symptoms are seen by primary care physicians, who manage them with varying degrees of enthusiasm and success. The term *somatization* has been used descriptively to refer to the experience of physical symptoms when medical evaluation reveals no explanatory disease or pathophysiologic process sufficient to explain the symptoms or their impact on the individual ([Kellner, 1986](#); [Lipowski, 1988](#)). Lipowski's definition of somatization is generically useful: "the tendency to experience and communicate somatic distress and symptoms unaccounted for by pathologic findings, to attribute them to physical illness, and to seek medical help for them" ([Lipowski, 1988](#)). In addition to its descriptive uses, the term has also been employed to refer to a presumed psychological mechanism of symptom production, as well as the operative term in the categorical diagnosis of *somatization disorder*, a relatively specific diagnostic entity characterized by multiple medically unexplained physical symptoms across numerous different organ systems ([American Psychiatric Association, 1994](#)).

The diagnostic criteria for the somatoform disorders are established for adults and are applied to children for lack of a child-specific research base and a developmentally appropriate alternative system. Available research has not applied the categories of somatoform disorder to children and adolescents, but instead has focused on the presenting, medically unexplained symptoms ([Campo and Fritsch, 1994](#)). Although there is reason to suspect a continuity between pediatric somatization and adult somatoform disorders, definitive longitudinal studies have not been performed. However, data do suggest that medically unexplained physical symptoms such as recurrent abdominal pain (RAP) in childhood are predictive of adult emotional disorders ([Campo et al., 2001](#); [Hotopf et al., 1998](#)).

This chapter is based directly on published reviews ([Campo and Fritsch, 1994](#); [Fritz et al., 1997](#)) and chapters ([Campo and Garber, 1998](#); [Campo and Reich, 1999](#)), and it synthesizes the available literature addressing somatization in children and adolescents as categorized within the fourth edition of *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) ([American Psychiatric Association, 1994](#)) as somatoform disorders, such as somatization disorder, conversion disorder, pain disorder, hypochondriasis, and body dysmorphic disorder (BDD), as well as important related conditions including vocal cord dysfunction (VCD), reflex sympathetic dystrophy, and functional RAP. [Campo and Fritsch \(1994\)](#) and [Garralda \(1992\)](#) reviewed the current literature in depth regarding somatization in children and adolescents. The term *somatization* is employed in this chapter descriptively because the categorical diagnosis of somatization disorder as defined by the DSM-IV provides diagnostic criteria more appropriately used for the adult population than for children and adolescents. Because the pediatric literature is sparse, adult studies are cited when relevant. A generic model addressing management is then presented based on a collective review of treatment approaches employed across specific disorders characterized by pediatric somatization.

### NOSOLOGY

Seven specific somatoform disorders are described in the DSM-IV: somatization disorder; undifferentiated somatoform disorder; conversion disorder; pain disorder; hypochondriasis; BDD; and somatoform disorder, not otherwise specified ([American Psychiatric Association, 1994](#)). *Somatoform disorders* are defined by the presence of physical symptoms that suggest a physical disorder but are not fully explained by the presence of a general medical condition, the direct effects of a substance, or another mental disorder ([American Psychiatric Association, 1994](#)); the symptoms must cause distress or functional impairment and should not appear to be voluntarily or intentionally produced. The diagnosis of somatoform disorder can be quite subjective, because the diagnostician must make a judgment about whether a particular physical symptom is caused by a physical disease or disorder. Furthermore, the examiner must also infer the patient's sense of control over the production of the symptom. Pediatric somatoform disorders are distinguished from *factitious disorders* and *malingering*, in which physical symptoms are voluntarily fabricated, feigned, or intentionally produced ([American Psychiatric Association, 1994](#)). Another distinction is made from *psychological factors affecting medical condition*, in which psychological factors have an adverse effect on a general medical condition ([American Psychiatric Association, 1994](#)). Physical symptoms are also included in some of the criteria used to diagnose specific anxiety and depressive disorders. Different diagnosticians may conceptualize particular symptom constellations such as irritable bowel syndrome, chronic fatigue, or fibromyalgia differently, with some considering such syndromes representative of a disease process and other considering the associated physical symptoms medically unexplained. Diagnosis may thus be influenced by clinician training and experience, as well as by the site of initial presentation ([Barsky and Borus, 1999](#)), findings suggesting that the diagnosis of somatoform disorder in children and adolescents is likely to be relatively unreliable in its current form. Unfortunately, studies addressing the reliability or validity of the diagnosis of somatoform disorder *per se* in children and adolescents have not been performed. It has also been argued that several of the somatoform disorders are best conceptualized as personality disorders ([Bass and Murphy, 1995](#)). Character traits are viewed as evolving rather than firmly established in childhood, so many child and adolescent psychiatrists are reluctant to diagnose personality disorders or to label presumed enduring patterns of health-related behaviors such as described in the somatoform disorders.

### Somatization Disorder

The diagnosis of *somatization disorder* is rooted historically in early diagnostic conceptualizations of "Briquet's syndrome" or "hysteria" and refers to a recurrent disorder beginning before the age of 30 years and characterized by multiple and diverse somatic complaints associated with medical help seeking or significant functional impairment. The French physician Briquet described patients with multiple medically unexplained physical symptoms and suggested that most of these patients developed symptoms and associated disability before the age of 20 years, with early onset associated with an especially poor prognosis ([Mai and Merskey, 1980](#)).

The diagnostic criteria for somatization disorder are quite specific, requiring a history of pain in at least four different body sites, at least two gastrointestinal symptoms, one sexual or reproductive symptom, and one pseudoneurologic symptom other than pain ([American Psychiatric Association, 1994](#)). The criteria are based on the work of psychiatric researchers in the Midwest, and earlier editions of the DSM employed elaborate symptom counts from an extensive list of physical symptoms ([Cloninger, 1994](#)). Current DSM-IV criteria are an empirically based simplification aiming to address the core features of the disorder. Given the requirement for at least one sexual or reproductive symptom, the diagnosis is difficult to make in children, but children meeting diagnostic criteria for earlier formulations of the diagnosis have been reported ([Kriechman, 1987](#); [Livingston and Martin-Cannici, 1985](#)).

### EPIDEMIOLOGY AND PREVALENCE

Although the actual diagnosis of somatization disorder is rare, recurrent complaints of somatic symptoms are quite common in the pediatric population. Children and adolescents meeting the diagnostic criteria set forth in the third revised edition of the DSM (such as DSM-III-R) have been identified ([Kriechman, 1987](#); [Livingston and Martin-Cannici, 1985](#)), but the use of such strict diagnostic criteria probably limited the prevalence of the diagnosis. In a general population survey, [Offord et al.](#)

(1987) found recurrent distressing somatic symptoms to be present in 11% of girls and 4% of boys ages 12 to 16 years. Garber et al. (1991) evaluated somatic symptoms in 540 school-age children and reported that children endorse various somatic complaints that may be symptoms of somatization disorder. Headaches, fatigue, sore muscles, abdominal distress, back pain, and blurred vision are commonly reported symptoms across ages and sexes, with approximately 1% of children and adolescents endorsing the threshold 13 symptoms required for the DSM-III-R diagnosis of somatization disorder. No pediatric studies have been reported with the DSM-IV criteria, which require at least four pain symptoms, two gastrointestinal symptoms, one sexual symptom, and one pseudoneurologic symptom to be present. In summary, the rarity of the diagnosis of somatization disorder in children and adolescents probably reflects developmentally inappropriate criteria more than the possibility that the disorder arises *de novo* only in adulthood.

Age and gender are important variables in pediatric somatization. Somatization is more likely in older children and in adolescents ( Campo et al., 1999), but methodologically sound longitudinal studies are lacking ( Campo and Fritsch, 1994). Girls may also be more consistent in somatic symptom reporting than boys (Walker and Greene, 1991a, 1991b). Low socioeconomic status and low levels of parental education have been associated with pediatric somatization ( Aro et al., 1987; Campo et al., 1999; Steinhausen et al., 1989), but the impact of social and cultural factors needs to be examined more critically.

#### DEVELOPMENTAL CONSIDERATIONS

Somatic symptoms and expression of pain in children appear to follow a developmental sequence. Prepubertal children may experience affective distress as somatic sensations. RAP (see later) and headaches appear to be the most prominent physical complaints in the prepubertal child ( Belmaker et al., 1985; Faulk and Nicol, 1986; Garber et al., 1990). Headache and RAP are frequently reported painful somatic symptoms, with 10% to 30% of school-age children and adolescents reporting symptoms at least weekly (Garber et al., 1991; Larson, 1991; Tamminen et al., 1991). Limb pain, aching muscles, fatigue, and neurologic symptoms increase with age (Walker and Greene, 1989; Walker and Greene, 1991a). Polysymptomatic presentations may be more common with increasing age (Offord et al., 1987). Conversion symptoms are quite rare before the age of 6 years (Grattan-Smith et al., 1988; Lehmkuhl et al., 1989; Leslie, 1988; Volkmar et al., 1984), but they become somewhat more common in later childhood and adolescence (Stefansson et al., 1976). Pseudoneurologic or conversion symptoms are more prevalent in girls across all ages (Goodyer and Mitchell, 1989), but complaints of recurrent pain occur equally in boys and girls until late childhood and puberty, after which female symptom reporting predominates (Garber et al., 1991; Oster, 1972; Walker and Greene, 1991b). Bass and Murphy (1995) propose that somatization may reflect temperamental or personality traits, given its persistent course, long duration, and early age of onset.

#### GENETIC AND FAMILY FACTORS

Few studies are available exploring the genetic contributions to the development of somatization disorder. Wender and Klein (1981) note that antisocial personality disorder, somatization disorder, attention deficit hyperactivity disorder, and alcoholism cluster in families more than expected by chance. A twin study by Torgersen (1986) explored the links of somatoform disorders in monozygotic and dizygotic same-sex twins born between 1910 and 1955 in Sweden. Twelve subjects were found to meet the criteria for somatization disorder. Of the 12, no monozygotic or dizygotic co-twins were diagnosed with somatization disorder. Both monozygotic and dizygotic co-twins were diagnosed with conversion disorder, pain disorder, generalized anxiety disorder, obsessive-compulsive disorder, and depression. Only three of the 12 co-twins were without concomitant psychiatric disorder. Somatizing children have been found to share similar physical symptoms with family members (Garber et al., 1990; Kriechman, 1987; Walker and Greene, 1989; Walker and Greene, 1991b). In addition, anxiety and depression have been found to be more common in the families of somatizing children. Parents of children with RAP report significantly more psychiatric symptoms than parents of well controls. The presence of family members with chronic physical illness may be associated with increased somatic symptoms in the children (Wasserman et al., 1988; Zuckerman et al., 1987). A pilot study involving family members of subjects with somatization disorder revealed that family members had more illnesses, used illness for stress reduction, reported more substance abuse and legal difficulties, and appeared more dysfunctional than the control families ( deGruy et al., 1989).

#### PSYCHOSOCIAL CONTRIBUTORS TO DEVELOPMENT OF SOMATIZATION

Sexual abuse experienced in childhood has been found to be associated with somatization disorder in adulthood ( Kinzl et al., 1995), and it is suggested to lead to an increase report of subjective physical complaints in children and adolescents ( Friedrich and Schafer, 1995). Adolescents with histories of physical and sexual abuse have been found to score higher on measures of somatization than adolescents without histories of abuse ( Atlas et al., 1995). A pattern of adult chronic somatization has been associated with lack of parental care coupled with a history of childhood illness ( Craig et al., 1993; Craig et al., 1994).

#### Undifferentiated Somatoform Disorder

Children and adolescents with multiple somatic complaints across numerous different body locations insufficient to justify a diagnosis of somatization disorder are more likely to meet diagnostic criteria for *undifferentiated somatoform disorder*, a somatoform disorder characterized by the presence of one or more physical complaints (e.g., individual symptoms of fatigue, urinary, or gastrointestinal distress) lasting at least 6 months ( American Psychiatric Association, 1994). *Neurasthenia* is a diagnosis made in Europe, but not included in the DSM-IV; the disorder has a long tradition in Western medicine and is characterized by persistent and troubling complaints of fatigue after mental effort or minimal physical effort, as well as at least two symptoms from a list that includes muscular aches and pains, dizziness, headache, sleep disturbance, inability to relax, irritability, and dyspepsia ( Wessely, 1990). Children with so-called neurasthenic symptoms of sufficient duration would be diagnosed with undifferentiated somatoform disorder.

#### Conversion Disorder

*Conversion disorder* is the somatoform diagnosis made when the clinician is confronted by one or more deficits or symptoms affecting voluntary motor or sensory function that suggest a neurologic or other general medical condition, and psychological factors are judged to be associated with the symptoms or deficits ( American Psychiatric Association, 1994). The presence of psychological factors is inferred from an association of the symptom with a significant psychological stressor such as family conflict, bereavement, or psychological trauma. Presenting symptoms classically resemble neurologic dysfunction (paralysis, paresis, anesthesia, paresthesia), follow the psychological stressor by hours to weeks, and may cause more distress among parents or physicians than within the patient (*la belle indifférence*). Symptoms frequently reported in children and adolescents include pseudoseizures, apparent paresis, paresthesia, and gait disturbances ( Grattan-Smith et al., 1988; Leslie, 1988; Spierings et al., 1990; Thomson and Sills, 1988). Symptoms are usually self-limited but may be associated with chronic sequelae such as contractures or iatrogenic injury (Fritz et al., 1997). There are four subtypes of conversion disorder in the DSM-IV, based on whether the symptoms presented are primarily motor, sensory, nonepileptic seizures, or mixed.

#### PREVALENCE AND EPIDEMIOLOGY

Because of the need both to specify a psychological association and to exclude medical or cultural causations, conversion disorder cannot be accurately diagnosed using only interview-based epidemiologic methods. Pseudoneurologic symptoms, or symptoms suggestive of a neurologic disorder in the absence of demonstrable neurologic disease, nevertheless appear to be unusual in community samples of children and adolescents in most modern Western cultures ( Garber et al., 1991; Rutter et al., 1970; Stefansson et al., 1976). One retrospective chart-review study of an Iowa medical population identified conversion disorder in as few as 11 patients between 9 and 20 years old out of 220,306 patients of all ages seen during a 2-year period ( Tomasson et al., 1991). Conversion disorder does appear to be more common among girls than boys across all age groups (Spierings et al., 1990; Steinhausen et al., 1989). Conversion disorder seems to become increasingly common in tertiary referral centers and pediatric neurology services, where nonepileptic seizures, faints, falls, and abnormalities of gait or sensation are the most commonly reported symptoms (Goodyer and Mitchell, 1989; Grattan-Smith et al., 1988; Leslie, 1988; Maloney, 1980; Spierings et al., 1990; Steinhausen et al., 1989; Volkmar et al., 1984). Higher prevalences have also been reported in some non-Western clinical settings ( Chandrasekaran et al., 1994).

#### DEVELOPMENTAL CONSIDERATIONS

Conversion disorder is quite unusual in very young children, in whom the diagnosis should provoke considerable skepticism unless there has been an exceptionally careful medical workup. Preschool-age children may at times limp or refuse to walk after a minor injury for a few hours or days, and the symptom may elicit increased attention on the part of parents and other caretakers, but it is often difficult to determine the degree to which such behavior may be associated with stress or psychological factors. Conversion disorder, although still relatively rare, becomes more common with increasing age into later childhood and adolescence ( Campo and Fritsch, 1994). *La belle indifférence*, an apparent lack of concern by the patient for his or her primary symptom, is not invariably seen among children and adolescents and is reported in as few as 8% of cases (Spierings et al., 1990).

The eventual association of conversion disorder with a diagnosable physical disease or injury is variable ( Grattan-Smith et al., 1988; Spierings et al., 1990).



Psychiatric comorbidity in children and adolescents with conversion disorder is poorly studied. A study by [Bowman and Markand \(1996\)](#) of adults diagnosed with pseudoseizures used the Structured Clinical Interview for DSM-III-R and found high rates of psychiatric comorbidity, particularly with other dissociative disorders (91%) and affective disorders (64%). In a retrospective chart-review study of patients of all ages, [Tomasson et al. \(1991\)](#) found significantly higher rates of major depressive disorder, panic disorder, and substance abuse in adults diagnosed with somatization disorder than in those with conversion disorder.

The course of conversion disorder is thought to be brief, and most cases reported resolve within 3 months of diagnosis ([Leslie, 1988](#)). The time to diagnosis is variable and ranges from weeks to a year, or more, during which time the child may be subjected to numerous diagnostic tests and unsuccessful medical interventions. Recurrence of symptoms is thought to be exceptional and may foreshadow the emergence of a polysymptomatic somatization disorder.

#### GENETIC AND FAMILY FACTORS

There is little in the way of firm evidence of a genetic contribution to the development of conversion disorder *per se*. However, family factors are thought to play a prominent role in the expression of the illness and the persistence of symptoms. Conversion disorder may present with symptoms that mimic those of a close friend or relative, a finding reported in one-fourth to one-half of children in two case series ([Grattan-Smith et al., 1988](#); [Spierings et al., 1990](#)). The relationship between conversion disorder and parental psychopathology is poorly delineated; however, [Grattan-Smith et al. \(1988\)](#) identified two broad patterns of disturbance among families of children with conversion disorder: anxious families preoccupied with disease and disorganized and chaotic families. [Turgay \(1990\)](#) suggests that poor family functioning and denial may prolong time to recovery, but such hypotheses remain untested.

#### NONEPILEPTIC SEIZURES AND PSEUDOSEIZURES

The most commonly reported conversion symptom in the recent child and adolescent psychiatric literature is the *nonepileptic seizure* or *pseudoseizure*, an event that resembles an epileptic seizure in the absence of electroencephalographic abnormalities or characteristic epileptic clinical course. Case series of conversion disorder previously cited report frequencies of pseudoseizure in clinical samples of conversion disorder between 15% and 50%. Although epilepsy may be difficult to distinguish from pseudoseizures, clinical distinctions have been proposed and reviewed by [Stores \(1999\)](#). More technologically sophisticated methods to distinguish seizures from pseudoseizures include videoelectroencephalography ([Cohen et al., 1992](#); [Duchowny et al., 1988](#)) and measuring of "postictal" elevations in serum prolactin levels ([Fisher et al., 1991](#)), although the latter technique may be of questionable utility. However, obtaining a "normal" electroencephalographic recording does not exclude the presence of a seizure disorder, and documentation of a seizure disorder does not exclude the possibility of a pseudoseizure ([Lancman et al., 1994](#)).

#### Pain Disorder

*Pain disorder* is diagnosed when pain in one or more anatomic sites of sufficient severity to warrant clinical attention and to cause significant distress or functional impairment is the focus of clinical attention ([American Psychiatric Association, 1994](#)). The three subtypes of pain disorder are as follows: *pain disorder associated with psychological factors*, in which psychological factors are judged to play the predominant role in the causation or persistence of the pain; *pain disorder associated with both psychological factors and a general medical condition*, in which psychological factors and a general medical condition are judged to interact significantly in the development or maintenance of pain; and *pain disorder associated with a general medical condition*, in which psychological factors appear to play no more than a minimal role. The third subtype is not considered a mental disorder and is coded on Axis III. The pain disorder diagnoses are considered *acute* if less than 6 months in duration and *chronic* when lasting 6 months or more. Recurrent complaints of pain have been associated with an increased risk of psychopathology, functional impairment, family difficulties, and higher health and mental health service use in a large sample of children evaluated in the primary care setting ([Campo et al., 1999](#)). The cause of pain, affective suffering, disability, and handicap are all components of pain and are best evaluated independently given the complexity of their associations. For example, such an approach allows the child with pain driven by an "organic" cause and with an associated psychologically determined handicap to be recognized and prescribed comprehensive treatment.

Even though pain is a universal human experience, it has proven surprisingly hard to define. The International Association for the Study of Pain's definition is now widely accepted: "pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage." The definition highlights the complexity, the psychological aspects, and the subjective nature of pain. Pain must always be assessed through self-report, because no direct or objective measurement technique exists. Thus, dealing with pain in infants and young children is especially problematic because of their limited self-reporting abilities. The task of determining when a pain syndrome warrants diagnosis as a psychiatric disorder can be somewhat daunting, given the potential for subjectivity inherent in such judgments. A developmental perspective is essential. Pain responses in infants younger than 3 months appear reflexive, with negative affect more consistently accompanying pain after 3 months of age. More observable fear and avoidance of pain-provoking situations and common words for pain ("booboo") develop in infants 6 to 18 months of age; children 18 months and older begin to localize pain, to use the word "hurt," and to recognize pain in others ([McGrath and McAlpine, 1993](#)). Preschoolers exhibit coping strategies, such as seeking hugs or using distraction, to alleviate pain. However, their preoperational cognitive abilities lead to magical thoughts about pain and prohibit understanding a painful procedure as being potentially beneficial. School-age children can clearly specify levels of pain intensity and can link psychological feelings to pain. With formal operations comes an increasingly complex and abstract idea of pain, its causes, and its course. Clinical studies show that the pain threshold increases with age, and younger children are more sensitive to pain from medical procedures than children who are more than 7 years old ([Fradet et al., 1990](#)). Cultural differences in pain expression and pain behavior have been reported in the past, but the existing literature lacks studies that do not confound ethnicity, socioeconomic status, or acculturation levels. Although cultural factors need further investigation, several studies find similar pain responses in Hispanic and white children ([Pfefferbaum et al., 1990](#)).

The diagnostic process for somatoform disorders frequently entails the quantification of children's pain. Because direct, physiologic measures of pain are lacking, four alternative approaches to assessing the response to pain are employed. With infants, associated indicators of distress are useful, either behavioral (body movement, facial expression) or physiologic (heart rate, respiration). Observational scales, completed by either parents or professionals, can be reliable measures of overt distress ([Manne et al., 1990](#)). Direct scaling techniques, in which children pick the face that matches how they feel out of a graduated series, allow pain to be systematically rated and compared over time by children as young as 5 years of age ([Bieri et al., 1990](#)). Finally, pain questionnaires have been developed for children of school age and older to quantify pain ([Varni et al., 1987](#)). Self-report of pain experiences is highly desirable whenever possible, especially when parental objectivity may be compromised. Adolescents may hide their pain from parents or, alternatively, may exaggerate it in their parents' presence; in neither case does parental report provide accurate information. The repeated use of a scale or questionnaire is frequently useful to judge the effectiveness of interventions over time.

Family influences on pain are not well documented, although clinical wisdom and some evidence suggest that disability (reduction of activity) and handicap (social role impairment) associated with pain may aggregate in families ([Palermo, 2000](#)). [Osborne, Hatcher, and Richtsmeier \(1989\)](#), although not specifically using the DSM-IV categorization, report that children whose pain is unexplained and, presumably, more psychologically determined, have more family members as "pain models" than children whose pain is related to a purely organic cause. Adolescents with pain syndromes leading to school absences may experience more maternal reinforcement of illness behavior than matched controls who have pain but who attend school ([Dunn-Geier et al., 1986](#)). Overall, there is scant empirical evidence regarding the impact of parental pain on children, the determinants of handicap when pain exists, and the potential mechanisms of transmission of pain experiences within families.

#### RECURRENT ABDOMINAL PAIN

Recurrent abdominal pain (RAP) has been defined as at least three episodes of abdominal pain occurring over a period of at least 3 months that are severe enough to affect the activities of the child ([Apley and Naish, 1958](#); [Scharff, 1997](#)). The condition is common and affects 10% to 25% of school-age children and adolescents ([Alfven, 1993](#); [Faul and Nicol, 1986](#); [Garber et al., 1991](#); [Hyams et al., 1996](#); [Oster, 1972](#); [Zuckerman et al., 1987](#)). Approximately 2% to 4% of pediatric office visits may be consequent to abdominal pain ([Starfield et al., 1984](#)). RAP is more common with increasing age into adolescence ([Hyams et al., 1996](#)) and in girls ([Alfven, 1993](#); [Eminson et al., 1996](#)). The gender ratio is equal in early childhood ([Faul and Nicol, 1986](#); [Zuckerman et al., 1987](#)), but girls predominate in later childhood and adolescence ([Mortimer et al., 1993](#)). Most cases of RAP are medically unexplained, particularly in the absence of clues such as weight loss, bleeding, fever, other systemic symptoms, or laboratory abnormalities ([Boyle, 1997](#)). Some have nevertheless speculated that so-called "functional" RAP may result from gastroesophageal reflux, gastritis, small bowel dysmotility, or carbohydrate malabsorption ([Murphy, 1993](#)). RAP is most often considered to be a functional gastrointestinal disorder in the absence of explanatory structural or biochemical abnormalities ([Drossman et al., 1990](#)). Many patients meet criteria for irritable bowel syndrome, which is characterized by RAP or discomfort in which the symptoms are relieved by defecation or are associated with changes in stool frequency or consistency.

RAP is commonly comorbid with symptoms of anxiety and depression and with other somatic symptoms such as headache ([Abu-Arafeh and Russell, 1995](#); [Egger et al., 1999](#); [Garber et al., 1990](#); [Hodges et al., 1985](#); [Mortimer et al., 1993](#); [Walker et al., 1993](#); [Wasserman et al., 1988](#); [Zuckerman et al., 1987](#)). RAP is associated with functional impairment such as school absenteeism ([Garber et al., 1990](#); [Hyams et al., 1996](#); [Walker et al., 1993](#)), as well as a greater risk of unnecessary medical

investigations and procedures. [Walker et al. \(1993\)](#) compared children with RAP to controls with respect to life events, emotional and somatic complaints, family illness behavior, and functional disability; children with RAP had significantly more emotional and somatic complaints, and their families rewarded illness behavior more than families of healthy controls, but no differences were found in rates of negative life events or in levels of family functioning. RAP may thus be associated with a family model of illness behavior and may develop in a climate in which somatic and emotional distress is more likely to be experienced and expressed by both parents and children.

Early follow-up studies reported persistence of abdominal pain into adulthood for one-third to one-half of affected children ( [Apley and Hale, 1973](#); [Christensen and Mortensen, 1975](#); [Stickler and Murphy, 1979](#)). Children who complain of stomachaches at the age of 4 years are three times as likely to have similar complaints on follow-up at age 10 than peers ( [Borge et al., 1994](#)). More recent studies suggest that childhood RAP is predictive of emotional disorder in adulthood ( [Campo et al., 2001](#); [Hotopf et al., 1998](#)). Adult studies have suggested that visceral hypersensitivity may be a component of functional abdominal pain, as evidenced by reports of increased awareness of balloon distention in all segments of the gastrointestinal tract compared with normal controls ( [Zigheboim and Talley, 1993](#)). This visceral hypersensitivity may be related to the cognitive distortions previously described as somatosensory amplification by [Barsky et al. \(1988\)](#).

## REFLEX SYMPATHETIC DYSTROPHY

Although more commonly seen among adults, complex regional pain syndrome—reflex sympathetic dystrophy may also occur in children and adolescents. Typically, the condition presents with chronic, painful swelling in an extremity, decreased skin temperature, cyanosis, delayed capillary refill, and limitation of functioning. In adults, complex regional pain syndrome—reflex sympathetic dystrophy commonly follows injury of the involved extremity; however, in children and adolescents, the history of prior injury is less consistent. Moreover, in contrast to adults with this condition, children show decreased uptake during radionuclide imaging in the affected extremity ( [Goldsmith et al., 1989](#)).

The pathophysiology of complex regional pain syndrome—reflex sympathetic dystrophy is still undetermined but may be related either to dysregulation of the sympathetic nervous system or to an inflammatory response to injury. Psychological mediators have been proposed and outlined in case series (Sherry and Weisman, 1989; [Silber and Majd, 1988](#)), but the lack of adequate control groups and the potential for investigator biases methodologically limit these studies. In general, there appears to be an association of reflex sympathetic dystrophy with psychosocial stressors; however, it remains possible that findings of “parental enmeshment” may be a consequence of the effects of chronic pain on family functioning rather than a causative factor.

## Hypochondriasis

*Hypochondriasis* refers to the persistent, preoccupied fear that one has a serious disease. This fear is likely based on misinterpretation of the threat associated with one or multiple physical symptoms, it persists despite appropriate medical workup and reassurance, and it must be present for at least 6 months ( [American Psychiatric Association, 1994](#)). Hypochondriasis is associated with dissatisfaction regarding medical care, “doctor shopping,” deteriorating interpersonal relationships, and the risk of iatrogenic complication from excessive or repeated diagnostic procedures in adults ( [Barsky, 1992](#)). Hypochondriasis can be an independent disorder (primary hypochondriasis) or part of another underlying psychiatric disorder (secondary hypochondriasis). The adolescent who is mistakenly convinced that she is pregnant or the medical student who fears developing a recently studied illness would not be diagnosed with hypochondriasis because of the transient nature of their reactions.

Considerable debate and some research deal with the relationship between hypochondriasis and other psychiatric disorders. Hypochondriasis and obsessive-compulsive disorder often share intense and disabling fears of illness, injury, or contamination, and the lifetime prevalence of obsessive-compulsive disorder in a series of hypochondriacal patients was four times higher (8% versus 2%) than in a comparison group ( [Barsky, 1992](#)). However, in contrast to those with hypochondriasis, patients with obsessive-compulsive disorder view their fears as abnormal, attempt to suppress them, and avoid publicizing their symptoms, which are frequently seen as shameful. Depression and hypochondriasis may overlap, especially when the morbid ideation of depression takes the form of disease phobias.

Little is known and less is reported in the literature about the occurrence of hypochondriasis in children or adolescents ( [Campo and Fritsch, 1994](#)). Although it is reasonable to assume that the disorder would be more common in pediatric than psychiatric settings (similar to the predominance of adult hypochondriasis presenting in medical settings), no data exist regarding the epidemiology in younger age groups. The need for children and adolescents to involve their parents in seeking help for medical concerns may contribute to the rarity with which hypochondriasis is reported in youth. Adolescents' bodily focus means that specific disease fears (of acquired immunodeficiency syndrome or cancer, for example) are not uncommon, although usually at a subsyndromal level. In hypochondriasis, the patient's disease conviction is not of delusional intensity, as in delusional disorder, somatic type. Hypochondriasis is also not diagnosed when the belief or preoccupation is limited to an imagined defect in appearance.

## Body Dysmorphic Disorder

Body dysmorphic disorder (BDD) is defined in the DSM-IV as a preoccupation with an imagined or slight defect in physical appearance causing clinically significant distress or impairment in functioning ( [American Psychiatric Association, 1994](#)). BDD has been described as a disorder in which the sufferer experiences shame and the need for secrecy; thus, it may be missed unless clinicians ask directly about symptoms related to BDD. Parents of children with BDD may seek evaluation after witnessing excessive mirror checking, grooming, and reassurance seeking in their child. From a developmental perspective, preoccupation with appearance can be common during adolescence, but the adolescent with BDD exhibits either clinically significant distress or impairment in functioning. People with BDD often undergo costly and potentially dangerous cosmetic surgeries and dermatologic treatments.

[Phillips et al. \(1995a\)](#) report a virtual absence of psychiatric literature on BDD in children and adolescents despite preliminary evidence suggesting that the onset of BDD occurs in adolescence. To date, no epidemiologic data are available for BDD in children and adolescents. The literature focuses on case reports ( [Albertini et al., 1996](#); [El-khatib and Dickey, 1995a](#); [Phillips et al., 1994a](#); [Sondheimer, 1988](#)), all cases resulting in excessive preoccupation with perceived defects and impairment in functioning. A series of patients with DSM-IV–defined BDD reported a mean age of onset of 16.9±6.9 years ( [Phillips et al., 1995b](#)). The neurobiology of BDD is only now beginning to be studied. Evidence from demographics, features, phenomenology, course, and treatment response suggest the BDD may be related to obsessive-compulsive disorder and the pathophysiology may involve serotonin ( [Phillips, 1996](#)), as suggested by a report by [Albertini et al. \(1996\)](#) of a 6-year-old boy meeting diagnostic criteria for BDD who responded to serotonergic reuptake blockade.

## Somatoform Disorder, Not Otherwise Specified

*Somatoform disorder, not otherwise specified* is diagnosed when symptoms consistent with a somatoform disorder are present, but criteria for a specific disorder are not met ( [American Psychiatric Association, 1994](#)). Examples include unexplained physical symptoms such as fatigue or hypochondriacal concerns that are of less than 6 months' duration.

## VOCAL CORD DYSFUNCTION

Vocal cord dysfunction (VCD) is an often-unrecognized disorder in which spasm of the vocal cords leads to narrowing of the glottis and results in symptoms that mimic acute asthma. Typically, there is a history of asthma unresponsive to very aggressive medical management, including multiple inhaled medications, systemic steroids, and inpatient hospitalization with endotracheal intubation. VCD is differentiated from asthma by the absence of nocturnal symptoms, localization of wheezing to the upper chest and throat, normal blood gases despite extreme symptoms, and significant adduction of the vocal cords when visualized on laryngoscopy ( [Brugman and Newman, 1993](#); [Goldman and Muers, 1991](#)). The prevalence of VCD in children's hospitals is unknown, in part because of clinicians' lack of awareness of the disorder ( [McQuaid et al., 1997](#)). Psychiatrists and psychologists become involved as consultants, often because of suspicion that the symptoms are factitious or intentionally produced (neither is true of VCD). Overt, serious psychopathology is often not identified in patients with VCD or their families, although stress or trauma may be important in the evolution of the disorder. Asthma medications are unhelpful, but speech therapy to reduce tension in the extrinsic laryngeal musculature combined with other psychosocial treatments can be helpful. Psychiatric input regarding the diagnosis, treatment planning, and communication with the family can be important in helping the child return to normal functioning.

## MANAGEMENT

### Assessment

Assessment provides the foundation for treatment and is a particularly critical factor in determining success or failure with an individual patient and family. Both the



content and the process of the assessment are critically important. Most, if not all, children referred for psychiatric consultation for presumed somatization have been evaluated by a primary care physician or even a subspecialist. It is nevertheless the consultant's responsibility to ensure that sufficient medical assessment has been performed, including a careful physical examination. There is no lack of anecdotal and published reports of patients initially believed to suffer from somatoform complaints who were subsequently identified with a physical disease sufficient to explain the presenting symptoms ([Rivinus et al., 1975](#)). The finding that available case series suggest that serious missed disease is relatively uncommon in both presumed conversion disorder and in patients with recurrent pain ([Maisami and Freeman, 1987](#); [Spierings et al., 1990](#); [Volkmar et al., 1984](#); [Walker et al., 1993](#)) should provide little comfort in the individual case unless competent medical assessment has been performed.

The clinician thus should be open to initiating additional medical evaluation if the initial assessment raises concerns about undiagnosed physical disease or if the clinical picture has changed. Previous medical and psychiatric records should be carefully reviewed. Inconsistencies or apparent fabrications in the history can be a clue to the uncommon, but real possibility of malingering, factitious disorder, or factitious disorder by proxy. Medical or psychiatric records hand carried by a parent should provoke suspicion, because record tampering has been reported in some cases of factitious disorder by proxy ([Schreier and Libow, 1993](#)).

Children with unexplained symptoms and their parents often feel misunderstood by their doctors and the professionals involved in their care. They are prone to feel dismissed when a psychiatric referral is suggested and become concerned that the complaints have not and will not be taken seriously. Also of concern is the perhaps more common problem of excessive and inappropriate medical investigations and evaluations performed because of professional anxiety or discomfort associated with the perceived stigma of somatization and psychiatric disorder. Sadly, concerns related to the stigma can serve to motivate patients, families, and clinicians to pursue potentially dangerous and unnecessary medical investigations and treatments. Nonpsychiatric physicians are also likely to be more comfortable with the assessment of physical disease than with the diagnosis and treatment of psychiatric disorder. The clinician must be alert to the powerful influence of anxiety, not only for the patient and family, but also for the clinician. The anxiety experienced by patients and family members in relation to the uncertainties of dealing with subjectively real and frightening physical symptoms can become contagious and may distract otherwise thoughtful professionals from usual sound clinical practice. Unnecessary medical tests and treatments are not only potentially dangerous from a physical perspective, but they may also represent a metacommunication to the family and patient that the clinician expects to find physical disease. This may perpetuate somatization by giving the impression that the clinician fears that a serious physical disease has been missed and can make verbal efforts to reassure the patient and family appear incongruous or deceptive ([Goodyer and Taylor, 1985](#); [Grattan-Smith et al., 1988](#)).

The presence of a physical disease in the child or a family member does not definitively exclude the possibility of pediatric somatization. Chronic physical disease may increase the risk of experiencing symptoms consistent with somatization ([Kellner, 1986](#); [Livingston, 1993](#); [Pilowsky et al., 1982](#)), and unexplained physical symptoms and disability may develop after an acute illness or accident ([Carek and Santos, 1984](#); [Leslie, 1988](#)). Similarly, children with medically unexplained physical symptoms commonly have family members with a history of physical illness. Experience with physical illness may influence vulnerable persons by introducing them to the potential benefits associated with the sick role.

The diagnosis of a somatoform disorder should not just involve the exclusion of serious physical disease, but ideally should be based on certain positive findings that support the diagnosis ([Campo and Garber, 1998](#); [Campo and Reich, 1999](#); [Goodyer and Taylor, 1985](#)). Numerous "clues" to the identification of somatization and the diagnosis of a somatoform disorder have been identified ([Campo and Garber, 1998](#); [Friedman, 1973](#); [Goodyer and Taylor, 1985](#)), including the following: (a) temporal contiguity of the symptom with significant psychosocial stress; (b) a prior history of somatization; (c) social or familial reinforcement of the symptom; (d) a model for the symptom within the family or immediate environment; (e) comorbid psychiatric disorder; (f) apparent communicative or symbolic meaning of the symptom; (g) violation of known anatomic or physiologic patterns by the symptom; and (h) responsiveness of the symptom to placebo, suggestion, or psychological treatment. These clues are clearly less than definitive, with a constellation of clues taken together being most persuasive if not convincing. Clearly, virtually all of the clues mentioned here could be found in association with physical disease. Because somatization is often associated with elevated rates of psychopathology in the identified patient and the family, careful psychiatric assessment is required ([Campo and Garber, 1998](#); [Campo and Reich, 1999](#)). Anxiety symptoms and school refusal are common in children with recurrent pain ([Garber et al., 1990](#); [Hodges et al., 1985](#); [Walker et al., 1993](#); [Wasserman et al., 1988](#)), and depressive symptoms are also commonly associated with pediatric somatization ([Garber et al., 1991](#); [Kashani et al., 1982](#); [Kowal and Pritchard, 1990](#); [Larson, 1991](#)).

Multiple sources of information such as parents, health care providers, other relevant professionals, and the school are key to a comprehensive evaluation. The school is an especially valuable source of objective information. It is exceptionally important to identify any learning skills deficits, because a learning disorder can provide motivation for school avoidance. Physical symptoms may also be employed as "self-handicapping strategies," particularly in perfectionistic and socially anxious children, by providing an ostensible excuse for why a particular child may not be performing up to his or her own expectations or the expectations of others ([Walker et al., 1995](#)).

Careful assessment of the social and family environment is similarly critical. A family history of psychiatric disorder, somatization, or physical illness and disability is important to recognize because of observations that there is often a familial "model" for a given child's symptoms and complaints. Pediatric somatization is commonly associated with separation fears, a sense of parental "overprotection" ([Robinson et al., 1990](#)), and the perception within the family that the child is especially "vulnerable" from a physical or emotional perspective ([Green and Solnit, 1964](#)). The child may have been the result of prolonged efforts for the couple to conceive, or there may be a prior history of trauma for the family associated with medical care, such as the loss of a relative after physicians mistakenly provided reassurance. Inquiring about previous experiences in the health care system is often productive. Other areas relevant to the assessment include possible marital conflict and parent-child relational problems. For example, a focus on the child's physical symptoms may allow the family to preserve its current level of function and may help the parent avoid serious marital conflict by diverting attention to the child's symptoms ([Mullins and Olson, 1990](#)). Other negative life events such as the loss of death of a family member or other close relation may be of critical importance ([Aro et al., 1989](#); [Livingston, 1993](#); [Scaloubaca et al., 1988](#)). Perhaps the most critical environmental issue to consider is that of maltreatment. Although maltreatment can be relevant to the presentation of any child with somatization or emotional and behavioral problems in general, it is particularly important to consider in children with conversion disorder, genitourinary complaints, and chronic, polysymptomatic somatization ([Klevan and DeJong, 1990](#); [Livingston et al., 1988](#); [Rimza et al., 1988](#)).

## Treatment

Treatment needs to be individualized to address the specific problems of a particular child, but therapeutic approaches to the various somatoform disorders share certain common features. A thoughtful generic approach is described by [Garralda \(1999\)](#). Especially with chronic somatoform problems, the child and adolescent psychiatrist will often function as a consultant to the primary care physician who largely manages the case. Somatizing patients and their families think and speak in terms of physical illness, medical problems, and somatic dysfunction and thus resist psychiatric referral, either actively or passively. The psychiatric consultant and primary care physician must coordinate their planning closely if the physician-patient alliance is to be maintained and "doctor shopping" is to be avoided. Working with somatizing patients and their parents can be frustrating for the pediatrician, who experiences critical and dissatisfied responses to conservative management or side effects and new symptoms when an aggressive approach is taken. Comorbid psychiatric disorders are common in somatizing patients and should be diagnosed and treated appropriately. Psychotherapy is often difficult to apply directly with somatizing patients and their families because it may initially seem irrelevant to them. Nonetheless, various types of psychotherapy have been reported as beneficial for some patients, always with the caveats of respecting the patient's somatic language and avoiding premature confrontation that inevitably leads to a sense of misunderstanding. Family cognitive-behavioral therapy for RAP ([Sanders et al., 1989, 1994](#)) has demonstrated higher rates of elimination of pain, lower levels of relapse at 6- and 12-month follow-up, and greater function than with the usual pediatric treatment without psychologic intervention.

## NATURAL HISTORY AND COURSE

As noted previously, current practice is handicapped by the lack of methodologically sound longitudinal studies of groups of children and adolescents with medically unexplained physical symptoms. Furthermore, most available studies have focused only on the presence or absence of the presenting symptom as the sole outcome measure, despite its being clear that other outcomes such as functional status or psychiatric status may be relevant. For example, earlier studies suggest that 25% to 50% of children and adolescents with RAP experience some abdominal pain in adulthood ([Apley and Hale, 1972](#); [Christensen and Mortensen, 1975](#)). More recent follow-up studies report significantly higher levels of abdominal discomfort, other somatic symptoms, internalizing psychiatric symptoms, and functional disability in former RAP patients than in a comparison group ([Walker et al., 1995](#); [Walker et al., 1998](#)), and some studies suggest that pediatric RAP is a better predictor of emotional disorder than physical distress in adulthood ([Campo et al., 2001](#); [Hotopf et al., 1998](#)). Significant physical improvement or complete recovery is reported in 50% to 100% of children and adolescents with conversion disorder in terms of the presenting symptom alone ([Goodyer and Mitchell, 1989](#); [Grattan-Smith et al., 1988](#); [Leslie, 1988](#); [Maisami and Freeman, 1987](#); [Spierings et al., 1990](#)), but data regarding other outcomes are lacking.

## DIAGNOSIS AND ESTABLISHING A FOUNDATION FOR TREATMENT



The diagnostic impression should be discussed directly in the context of a review of the clinician's understanding of the symptoms presented, the time course, the child's temperament, and family environment. Historical details should be clarified, and a shared consensus of the child's presentation should be arrived at together. This establishes the precedent for a partnership in treatment. When a definitive diagnosis of somatoform disorder cannot be made, it is important to acknowledge diagnostic uncertainty. After the diagnosis has been established and reviewed with the patient and family, additional diagnostic evaluation should be avoided in the absence of new clinical information, a change in the clinical picture, or evidence that intervention cannot proceed without the reassurance provided by an additional, relatively low-risk investigation.

Psychoeducation is of value in managing somatoform disorders, and it is a relatively low-cost first-line intervention. Education can be directed at understanding and adhering to a treatment regimen, clarifying when to worry about symptoms and when not to worry, enhancing communication with treating professionals, and using problem-solving coping techniques. Education of the patient and family helps to challenge stigma and to develop hope and positive expectations for recovery. Given the pervasive nature of stigma, it is especially important to educate referring physicians about the dangers of communicating any sense of embarrassment about the diagnosis of somatoform disorder or other psychiatric disorder, because this can contribute to treatment resistance and a wish to perpetuate the search for unrecognized physical disease. Discussing the relationship between mind and body and the false dichotomy implied by separate systems of care of "physical" and "mental" disorders can be useful. Patients and families need to understand that the problem presented by the child is recognized as real and is familiar to experienced clinicians. Explaining the physical symptoms as simple reactions to psychosocial adversity or "stress" may prove unproductive, particularly when clear-cut stressors are not evident or are not appreciated by the patient or family. In addition, a preoccupation with "stress" may inadvertently encourage the patient to avoid uncomfortable and distressing situations, and this is essentially counter to the rehabilitative approach emphasized later.

The importance of a therapeutic partnership or alliance among the child, the family, and the involved professionals must be emphasized, and the roles and responsibilities of each should be carefully defined. The value of close communication and working together to solve the problem should also be discussed directly. The importance of close communication, honesty, and a foundation of trust make the use of placebo or other sham interventions generally unacceptable. From a practical perspective, if suggestion or placebo proves unsuccessful, the clinician then is forced to consider new deceptions or to return to prior efforts to convince the patient that serious physical disease is absent and that symptom removal is not really necessary for a return to function. A simple consultation letter from a consulting psychiatrist to the primary care physician significantly improved the management of adults with chronic, disabling somatization and reduced associated health care expenditures (Smith et al., 1986). The clinician can serve as a bridge to help bring all the involved parties together in a productive alliance to help the child. Medical care is best consolidated by a single physician. Regularly scheduled medical visits can reassure the patient and family that their concerns have not been dismissed. The primary physician may serve as a powerful attachment figure for families when rejection sensitivity and fears of abandonment are prominent. Regularly scheduled visits allow the patient and family to see the physician without the requirement that the child be sick. It is often useful to help define what constitutes a legitimate, medically excused school absence for both the family and the school and to clarify who will be the physician responsible for legitimizing medical excuses. The patient will have to understand that absence from school without the approval of the collaborative treatment team and an appropriate medical excuse will be viewed as truancy, and the school will take the appropriate action. With such a treatment plan, the cooperation of the school can benefit the treatment effort. Once it is clear that any school excuses must come from the primary physician alone, the tendency to "doctor shop," particularly for medical excuses, will be curtailed.

#### REASSURANCE

Reassurance that serious physical disease is unlikely is important for most children and their families (Goodyer and Mitchell, 1989; Grattan-Smith et al., 1988; Maisami and Freeman, 1987; Schulman, 1988). The presenting symptoms should be demystified and helped to appear less threatening by uncoupling the perceived distress from concerns about actual tissue damage. Excessive reassurance can be problematic when obsessional illness worries are present and should be avoided in such circumstances after initial reassurance (Warwick and Salkovskis, 1988). Illness worry can then be framed as a problem to be solved by the child and family.

#### COGNITIVE-BEHAVIORAL INTERVENTIONS

Such interventions have been demonstrated to be successful as components of multimodal interventions for medically unexplained RAP in school-age children (Sanders et al., 1989; Sanders et al., 1994). Cognitive coping skill training, an emphasis on return to usual function, and cognitive-behavioral family interventions have all been employed as core components of treatment programs that also include behavioral interventions and the use of self-management strategies such as relaxation training. Although sample sizes are relatively small, results are encouraging, with significant improvements noted in the treated groups. Cognitive-behavioral therapy combined with serotonin reuptake inhibitors has been reported as helpful for children and adolescents with BDD (Albertini et al., 1996; El-Khatib and Dickey, 1995; Phillips et al., 1995a; Sondheimer, 1988). Supportive psychotherapy (Warwick, 1992), group therapy (Stern and Fernandez, 1991), and individual psychotherapy (Sharpe et al., 1992) have been applied successfully with somatizing adults, although data are lacking regarding the efficacy of these approaches in children or adolescents.

The cognitive-behavioral principle most relevant in the treatment of pediatric somatization appears to be that of taking a *rehabilitative approach*, which encourages the patient to return to usual activities and responsibilities regardless of whether symptoms persist (Dubowitz and Hersov, 1976; Leslie, 1988; Maisami and Freeman, 1987; Schulman, 1988). Such an approach helps patients and families to view the patient's symptoms and the associated disability as challenges to be overcome and shifts emphasis away from the importance of finding a "cure" to that of coping. The *avoidance of homebound schooling* is a corollary of the rehabilitative approach. The patient is treated as an active agent with the strength and wherewithal to live a productive life in spite of the acknowledged subjective distress. Improvement becomes a personal success and an accomplishment. For example, physical therapy has been used in conversion disorder (Leslie, 1988; Maisami and Freeman, 1987). Active coping has been found to be superior to passivity, which has been associated with greater symptoms and disability (Walker et al., 1997). Parents and family members must be helped to understand that it is not cruel or punitive to expect active coping, but actually both kind and therapeutic, given what is known. High expectations communicate parental and professional belief in the fundamental soundness of the child and in the child's ability to overcome adversity now and in the future. This approach directly challenges any long-standing misperceptions that the child is somehow vulnerable or weaker than other children and emphasizes strength and adaptability rather than sickness.

*Behavioral intervention* is a core component of most treatment strategies. Although there are no controlled trials of behavior therapy for pediatric somatization, case reports highlight potential benefits (Campo and Fritsch, 1994; Fritz et al., 1997). Most have emphasized *positive reinforcement* of healthy behaviors and *extinction* or *withdrawal of reinforcement* for symptoms and sick role behaviors (Klonoff and Moore, 1986; Mizes, 1985). Time out from reinforcement has also been applied (Miller and Kratochwill, 1979), but punitive approaches should be avoided. *Negative reinforcement* produces an increase in the frequency of a desired response by removing an aversive event immediately after the desired response has been performed and has been employed by lifting the restrictions theoretically imposed by illness once functional improvement has been demonstrated (Campo and Negrini, 2000; Leslie, 1988). For example, discharge from the hospital may be allowed only when the patient evidences sufficient physical improvement (Leslie, 1988), or persistent bed rest may be imposed with removal contingent on the patient's returning to premorbid function and responsibilities. Similarly, the threat of inpatient psychiatric hospitalization can be removed contingent on the child's maintaining at least minimally acceptable function (e.g., returning to school).

*Self-management interventions* have been reported to be successful in the management of recurrent pain, with specific techniques including *self-monitoring*, *relaxation training* (Masek et al., 1984), *hypnosis* (Elkins and Carter, 1986), and *biofeedback* (Klonoff and Moore, 1986; Mizes, 1985). Relaxation techniques and hypnosis have been reported as helpful in treating headaches, VCD, conversion disorder, and pain syndromes (Brugman and Newman, 1993; Manne et al., 1990). Self-management strategies are likely best employed as part of a multimodal approach (Sanders et al., 1989). Evidence indicates that these techniques are associated with symptomatic relief and by their very nature encourage active coping. Interpersonal and expressive psychotherapies may be useful, particularly in the presence of psychological trauma, but they have not been systematically evaluated.

#### FAMILY AND GROUP INTERVENTIONS

The use of family therapy has been promoted (Mullins and Olson, 1990), but not well studied. Sanders et al. (1994) report on a successful cognitive-behavioral family intervention. Because children presenting with medically unexplained physical symptoms are often considered health impaired or sickly by parents, family health beliefs and any encouragement of illness behavior must be addressed with the goal of reducing anxiety about illness within the family system. Group psychotherapy in the treatment of pediatric somatization has not been evaluated.

#### PSYCHOPHARMACOLOGIC INTERVENTIONS

There have been no systematic studies of psychoactive medications in pediatric somatization (Campo and Garber, 1998). Psychopharmacologic interventions are



nevertheless worthy of consideration in the treatment of persistent medically unexplained pain, gastrointestinal symptoms, or fatigue, particularly in the presence of psychiatric comorbidity or when psychotherapeutic interventions have not been entirely successful. Antidepressant and anxiolytic medications have been shown to reduce somatic symptoms in depressed and anxious adults (Simon et al., 1998). Antidepressant medications appear to be of significantly greater benefit than placebo in the treatment of adult somatoform pain disorders (Fishbain et al., 1998) and medically unexplained physical symptoms in general (O'Malley et al., 1999). Even without other Axis I disorders identified, there is preliminary evidence of the usefulness of selective serotonin reuptake inhibitors in treating adults with BDD or hypochondriasis when obsessive-compulsive aspects are prominent (Kellner, 1992; Phillips, 1996). Comparable work is not available to date for children and adolescents. Pain disorder concomitant with a medical problem improves when analgesics are employed optimally; narcotic adjuvants (nonanalgesics that help relieve pain) may be recommended when narcotics are ineffective or when their side effects are a problem. Tricyclic antidepressants, anticonvulsants such as carbamazepine (Tegretol) or phenytoin (Dilantin), and stimulants such as methylphenidate (Ritalin) or dextroamphetamine (Dexedrine) are demonstrated effective narcotic adjuvants (Lynn, 1990). In some patients who experience physical symptoms predominantly associated with emotional arousal and anxiety, a short course of a benzodiazepine can provide symptomatic relief and can help to reassure the patient and family that emotional distress is operative (Campo and Garber, 1998). As with other interventions, a successful response to treatment can help to reassure the patient and family that the original somatoform diagnosis was correct and can thus allow additional treatment to proceed.

## SUMMARY

Somatization and the epidemiology, diagnosis, and treatment of somatoform disorders constitute one of the remaining frontiers in child and adolescent psychiatry. Definitional ambiguity, theoretical complexity, lack of seminal studies in any domain, and overlapping boundaries with pediatrics have resulted in an area that is poorly mapped and rather forbidding to child mental health clinicians and researchers. Knowledge about somatoform disorders in the young is, thus, at a stage comparable to that of childhood depression in the mid-1980s. Somatoform disorders are conceptually linked to the mind-body split that has plagued Western medicine, and their management highlights the problems associated with parallel systems of care for mental and physical disorders. New ways of conceptualizing the problems posed by patients with physical symptoms that defy traditional medical explanations may prove to be important in the future. Epidemiologic studies are needed comparing numbers, frequency rates, and severity of somatic symptoms in community, pediatric, and psychiatric populations, to provide the empirical base for modified diagnostic criteria. Longitudinal studies of children with somatic symptoms are needed to determine the relationship between childhood presentations and adult disorders. Efforts to explore the nature of comorbidity between medically unexplained physical symptoms and emotional disorders, including from a neurobiological perspective, are likely to be important in the future. Empirical data on treatment efficacy are scant, pointing to the need for creative approaches and systematic follow-up investigations. Changes in medical economics and recognition of the major impact of pediatric somatization on health and mental health service use make it likely that the next 10 years will see rapid progress in our understanding of somatoform disorders in children and adolescents. Important advances such as real inculcation of the biopsychosocial model in medical training, the growing influence of physicians whose backgrounds integrate psychiatry and pediatrics, and the perspective of developmental psychopathology make it quite possible that the next 10 years will see an explosion in empirical knowledge and clinical wisdom regarding somatization in children and adolescents.

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## 70 DISSOCIATIVE IDENTITY DISORDER

Catherine A. Yeager, M.A., and Dorothy Otnow Lewis, M.D.

### Definition

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There is a certain historical irony in the fact that as psychoanalysis waxed, the diagnosis of *multiple personality disorder*, now called *dissociative identity disorder* (DID), waned, because to observe the phenomenon of DID is to see the unconscious played out before one's very eyes. DID is unbelievable to those who have never seen it. Perhaps it would be more accurate to say that DID is unbelievable to those who have not recognized it, because many, if not most, clinicians have seen it in their child, adolescent, and adult patients but have failed to recognize it. The signs, symptoms, and behaviors that together constitute the disorder are characteristics that we have been taught to associate with other syndromes; hence patients manifesting them are usually given other diagnoses. In the case of DID, it would be safe to say that, for the most part, seeing has been disbelieving.

How can a person have two, three, four, even more different states of consciousness, each relatively independent of the others and often unaware of their existence? Impossible? And yet, the expanding clinical literature attests to the improved ability of clinicians to recognize the syndrome, at least in adults. Our current understanding of the etiology, neurobiology, and treatment of pathologic dissociation supports the validity of DID as an indisputable diagnostic entity. However, for reasons that are elucidated in this chapter, children suffering from DID often go unrecognized, misdiagnosed, and improperly treated.

### DEFINITION

*Dissociation* is defined as the separation of thoughts, feelings, and experiences that are normally integrated into consciousness and memory ( [Maldonado and Spiegel, 1998](#); [Putnam, 1996a](#); [van der Kolk et al., 1996](#)). Absorption, absent-mindedness, and fantasy-proneness are examples of normal dissociative processes. Depersonalization, derealization, somatization, and psychogenic amnesia are examples of dissociative processes that emerge in the presence of trauma and its aftermath. Dissociation is thought to be an adaptational defense against overwhelming physical or emotional trauma. Dissociation enables the person to separate himself or herself cognitively and emotionally from the immediate environment (derealization) and from the body (depersonalization), to deal with unendurable stress. In the aftermath of trauma, psychogenic amnesia enables the victim to maintain self-control through detachment from the experience altogether ( [Maldonado and Spiegel, 1998](#)). Dissociation has been reported to occur frequently during combat, rape, motor vehicle or airplane accidents, and natural disasters such as earthquakes and fires ( [McFarlane and de Girolamo, 1996](#)).

In some cases, the dissociative, anxiety, and amnesic symptoms become so severe and persistent that the victim develops posttraumatic stress disorder (PTSD) or a dissociative disorder, such as dissociative amnesia, dissociative fugue, or dissociative disorder, not otherwise specified (DDNOS). On the continuum of dissociation, its most extreme, pathologic form is DID, which is the focus of this chapter. [Table 70.1](#) describes the manifestations of DID.

- 
- A. The presence of two or more personality states each with its own relatively enduring pattern of perceiving, relating to, and thinking about the environment and self
  - B. At least two of these identities or personality states recurrently taking control of the person's behavior
  - C. Inability to recall important personal information that is too extensive to be explained by ordinary forgetfulness
  - D. Not related to the direct effects of a substance or a general medical condition; note: in children, symptoms not attributable to imaginary playmates or other fantasy play
- 

Adapted from American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. Washington, DC: American Psychiatric Association, 1994, with permission.

**Table 70.1. DSM-IV Diagnostic Criteria for Dissociative Identity Disorder**

As described in this chapter, childhood manifestations of DID may not be as distinct as the adult manifestations of the disorder. Movement between alternate identity states is frequently subtle and fluid. Thus, identity state switches can be difficult to detect. To enable clinicians to recognize pathologic dissociation in their young patients, [Peterson and Putnam \(1994\)](#) developed a set of diagnostic criteria, resembling those of the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV) ([American Psychiatric Association, 1994](#)), that reflect childhood DID ([Table 70.2](#)).

- A. A substance of at least 3 months duration during which either one or two of the following are present:
1. recurrent amnesic periods or missing blocks of time
  2. frequent fugitive states or appearing to be in a state or in another world
- B. Increasing, major fluctuations in behavior that include at least two of the following:
1. dramatic fluctuations in school or work performance and behavior
  2. variations in apparent social, cognitive, or physical abilities
  3. sudden, recurrent shifts in identifiable interests
  4. changes in language, accent, and voice tone
  5. frequent changes in preferences for clothes, food, toys, games, etc.
- C. At least three of the following:
1. refers to self in third person or uses another name to refer to self or parts of self
  2. has vivid imaginary companions
  3. frequently observes observed behavior
  4. exhibits frequent inappropriate sexual behaviors or is sexually precocious
  5. has intermittent depression
  6. has auditory hallucinations from inside the head
  7. has frequent sleep problems
  8. exhibits unprovoked aggressive (anger) and violent behaviors
  9. exhibits other psychotic behaviors
- D. Does not meet the criteria for the adult form of dissociative identity disorder
- Adapted from Peterson, C. Putnam, F.W. Preliminary results of the final test of proposed criteria for dissociative disorder of childhood. *Dissociation* 7 (2): 200, 1996, with permission.

**Table 70.2. Proposed Diagnostic Criteria for Dissociative Disorder of Childhood**

## HISTORY OF DISSOCIATIVE IDENTITY DISORDER IN CHILDREN

The first case of what we would now designate childhood DID was reported by Despine in 1840. Despine described Estelle, an 11-year-old girl who experienced visions, went into trancelike states, was comforted by the voices of angels, and found herself unable to walk. Through the use of hypnosis, or what then was called animal magnetism, Despine was able to bring to light the existence in Estelle of an alternate personality state whose likes and dislikes differed from those of Estelle; while in this state, Estelle was able to walk. By means of hypnosis, and in a relatively brief period, Despine was able to integrate the two identity states and thereby enable his patient to walk once more ([Despine, 1840](#)).

For almost a century and a half, Estelle remained the only reported case of the diagnosis and treatment of DID in a child. Then, in 1979, in a course given under the auspices of the American Psychiatric Association, Kluff described the diagnosis and treatment of an 8-year-old boy who suffered from multiple personality disorder ([Kluff, 1985](#)). Since then, more and more clinicians have reported cases of DID in children and adolescents ([Lewis and Yeager, 1996](#)). Since the early 1990s, we have had the opportunity to evaluate more than 100 children and adolescents with signs and symptoms meeting the criteria for a diagnosis of DID or DDNOS. Four of these children were between 3½ and 4½ years of age at the time of their referrals to the Bellevue Dissociative Disorders Clinic in New York. Thus, gradually, a diagnosis that in the 1980s was practically unknown to child psychiatrists and psychologists has become a recognized entity, having specific, objectively identifiable characteristics as well as specific, effective treatment strategies.

## EPIDEMIOLOGY

Multinational epidemiologic studies ([Mulder et al., 1998](#); [Ross, 1991](#); [Ross et al., 1990a](#); [Vanderlinden et al., 1991](#); [Vanderlinden et al., 1996](#)) indicate that dissociative disorders are not as rare as previously assumed, but they appear to affect between 1% and 6% of adults in the general population, a prevalence rate considerably higher than that of schizophrenia and bipolar mood disorder ([Blazer, 2000](#); [Norquist and Narrow, 2000](#)). The prevalence of dissociative disorders in clinical populations of adults is exceptionally high. Several studies have found that up to 12% of North American adults admitted to general psychiatric inpatient settings meet criteria for DID ([Horen et al., 1995](#); [Latz et al., 1995](#); [Rifkin et al., 1998](#); [Saxe et al., 1993](#)). Studies from the Netherlands ([Friedl and Draijer, 2000](#)) and Turkey (Tutkin et al., 1998) have reported that between 2% and 5% of general psychiatric inpatients meet criteria for DID. In adults, certain psychiatric conditions, particularly substance abuse disorders and eating disorders, have an especially high rate of comorbidity with dissociative disorders ([Berger et al., 1994](#); [Kronson et al., 1990](#); [McCallum et al., 1992](#)).

The prevalence of dissociative disorders in the general population of children is currently unknown. However, findings from a small study we conducted in 1992 (unpublished) to evaluate the validity of a new semistructured interview protocol, the *Bellevue Dissociative Disorders Interview for Children* ([Lewis, 1996](#)), may begin to shed some light on the prevalence of dissociative disorders among ordinary children. Using the new interview protocol, we interviewed a group of 46 “supernormal” children (i.e., children from suburban, largely professional families who had no known histories of harsh corporal punishment, much less abuse) and a group of 24 emotionally disturbed, severely abused children in a residential treatment center. Of the emotionally disturbed children, 10 had severe dissociative symptoms. Of these, seven (six boys and one girl) met DSM-III and DSM-IV criteria for DID. Much to our surprise, of the normal children, one child (2%), a 10-year-old girl, met both DSM-III and DSM-IV criteria for DID.

Several studies of clinical samples of children and adolescents have found high rates of dissociative symptoms. For example, a study of severely abused and neglected child psychiatric inpatients ([Waterbury, 1991](#)) reported that 74% of patients admitted over a 1-year period had dissociative disorders; 23% of them met criteria for DID. [Hornstein and Tyson \(1991\)](#) reported that 5% of children admitted to a general psychiatric inpatient unit over a year's time were diagnosed with a dissociative disorder, including 3% with DID. [Ross \(1996\)](#) reported that 44% of adolescents undergoing evaluations at a general psychiatric facility had some type of dissociative disorder, including 16% with DID. A study of 64 juvenile delinquents ([Carrion and Steiner, 2000](#)) reported that 28% met criteria for a dissociative disorder, including 20% with DDNOS. Apparently, no subject in this study met criteria for full-blown DID. Our especially high rate of DID in the residential treatment sample as compared with the psychiatric hospital samples of other researchers reflects, we believe, the fact that all of our subjects, unlike hospital patients, had histories of extreme physical and sexual abuse and neglect. Clearly, pathologic dissociation is much more common among troubled children and adolescents than has hitherto been appreciated. Therefore, assessment for the presence of dissociative symptoms is essential in the psychiatric evaluations of all children and adolescents.

Estimates of the ratio of adult female to male cases have ranged from 2:1 to 14:1 ([Putnam et al., 1986](#); [Ross et al., 1989a](#); [Stern, 1984](#)). However, these figures are probably skewed because psychiatrically disturbed, aggressive men are more likely than their female counterparts to be channeled to the criminal justice system, and the nature of their psychopathology is less likely to be recognized. Less is known about gender differences in child and adolescent clinical populations. Some authors have hypothesized that the female:male ratio may be equal ([Waterbury, 1991](#)). Indeed, in the Carrion and Steiner study of juvenile delinquents (2000), equal numbers of boys and girls were found to suffer from dissociative disorders. Other investigators suspect that the prevalence of dissociative disorders is higher in girls because girls are more likely to have been abused sexually than boys ([Hornstein, 2000](#)). In our experience, similar numbers of girls and boys with pathologic dissociation are referred to our clinic.

## ETIOLOGY

Derealization, depersonalization, time distortion, alterations in cognition, memory, and somatic sensations, and emotional numbing are common dissociative responses to natural and human-made disasters, physical and sexual victimization, and the witnessing of horrifying events such as torture and homicide ([Classen et al., 1993](#); [Griffin et al., 1997](#); [Shalev et al., 1996](#)). These symptoms reflect a disconnection with the surrounding environment and a lack of integration of trauma with autobiographical memory and self-identity. Dissociation occurs peritraumatically (i.e., at the time of trauma), as well as posttraumatically as a long-term sequelae of traumatic exposure ([Bremner et al., 1992](#); [Ursano et al., 1999](#)). In fact, the severity of peritraumatic dissociation is predictive of who will develop PTSD ([Shalev et al., 1996](#)).

Whereas adults and adolescents may develop PTSD with accompanying dissociative symptoms in response to overwhelming trauma, young children are thought to be at high risk of developing much more complex, pervasive, biopsychological reactions. Children have less mature coping mechanisms with which to tolerate extreme stress; they also have a greater innate capacity to use trance and fantasy to separate mentally from traumatic experiences ([Putnam, 1996b](#); [Pynoos et al., 1996](#); [Roth et al., 1997](#)). In young children, when dissociative mechanisms are repeatedly called into play, the normal development of a coherent, unified sense of self goes awry.

### Physical and Sexual Abuse

When children are the victims of early, ongoing, extreme *physical, sexual, or psychological abuse*, they cannot endure it. Their developmental immaturity, their small size, and their limited strength preclude physical escape. Unlike adults, they cannot rely on the body's fight-or-flight response to propel them to safety. However, it would seem that children's intrinsic abilities to lose themselves in fantasy and role-play have special survival value ([Putnam, 1996b](#)). When they are repeatedly subjected to intolerable pain, children dissociate; that is, they spontaneously remove themselves mentally from their surroundings and in so doing carry themselves to safety. To them, the terrifying event is not even happening; or it is happening to someone else. At these times, sensations, perceptions, thoughts, emotions, and



behaviors become dissociated from consciousness and may later remain unavailable to normal recall.

The exact timing, quality, intensity, and duration of abuse required to engender DID is not known and, perhaps, cannot be ascertained empirically. The very function of dissociation is to block painful experiences from consciousness; the victim therefore would be a poor historian. Sometimes abuse begins so early in life that the capacity to think in terms of words or time is not yet established developmentally. In our study of 12 murderers with DID ( [Lewis et al., 1997](#) ), seven subjects had only fragmentary memories of their histories of extraordinary abuse, whereas four others had no memory of it whatsoever. Old records, interviews with relatives and childhood friends, and scars consistent with floggings and cigarette burns attested to the abuse our subjects could not recall. These kinds of data are especially important because even the experiences that patients with DID recount or seem to relive while they are in dissociated states can be unreliable ( [Yeager and Lewis, 1997](#) ).

An as yet unanswered question is whether physical abuse or sexual abuse has a greater propensity to engender pathologic dissociation, especially DID, in the young child. Although the general consensus is that sexual abuse is the most potent form of trauma giving rise to DID, some studies have indicated that there may be a closer link between physical abuse and pathologic dissociation than has hitherto been appreciated ( [Berger et al., 1994](#); [Carlson et al., 1998](#); [Chu and Dill, 1990](#); [Mulder et al., 1998](#); [Rhue et al., 1995](#) ). The kinds of environments that permit or invite chronic sexual abuse also facilitate other kinds of maltreatment. Although a dose–response relationship between forms and intensities of maltreatment and the development of DID may exist ( [Roth et al., 1997](#) ), it would be difficult to quantify. We have found that children with DID are those who have been placed on heated burners, tied to blisteringly hot radiators, bound hand and foot, brutally beaten, hanged in closets by their wrists, and repeatedly raped and sodomized with all manner of objects. They are the children who have been prostituted, the ones forced to star in pornographic films, and those tortured in bizarre family rituals. They have experienced unspeakable horrors—physical, sexual, and psychological—over extended periods of time.

Retrospective reconstructions by adult patients with DID of the timing of abuse, based on the age at which they recall the first emergence of alternate identity states, place the onset of the disorder well before 12 years of age and usually between the ages of 4 and 6 years ( [Ogawa et al., 1997](#); [Putnam, 1986](#) ). Our own work at the Dissociative Disorders Clinic at Bellevue Hospital indicates that, in most cases, abuse and the dissociative symptoms to which it gives rise begin much earlier than 12 years of age. We have evaluated children 3½ and 4½ years of age with the signs and symptoms of DID well established and with histories suggesting that their disorders have been active for at least a year before clinic referral. Our clinical data indicate that the kind of abuse that generates DID often begins in the very first months and years of life.

A major impediment to the accurate documentation of abuse in children with DID stems from the problem that the very persons on whom clinicians rely to provide a history (i.e., parents) are themselves perpetrators of maltreatment and are therefore unlikely to disclose it. In addition, many perpetrators were themselves victims, dissociate, and consequently are unaware of their own actions. In a remarkable article on the parenting capabilities of 75 women suffering from multiple personality disorder, [Kluft \(1987a\)](#) reported that 16% of the women in his sample were extremely abusive toward their children. In our own study of 11 children with DID or DDNOS ( [Yeager and Lewis, 1996](#) ), parents of nine children exhibited pathologic dissociation. In only one case was the parent aware that she suffered from DID. She was also the only parent to admit to physically abusing her son while she was in an alternate personality state. The other dissociative parents were consciously unaware of how they or others had mistreated their children. In fact, when one of the mothers in this study was in a violent, dissociated state, she asked us to call protective services before she killed her child. We called. The following week, she had no memory of her prior request and berated us for interfering. Only three of these nine families were known to child protective services at the time of our evaluations. This demonstrates how effectively dissociative families are able to keep secret extraordinary abuse.

### **Neglect**

Severe *neglect* is frequently overlooked as a cause of pathologic dissociation. We have evaluated dissociative children who were abandoned by their caregivers to live like animals. Often, they not only had to fend for themselves, but they also had to protect and provide sustenance for younger siblings. It is not known whether severe neglect alone can cause DID because neglect and abuse tend to go together. For example, [Hornstein and Putnam \(1992\)](#) report that 80% of their sample of child and adolescent dissociaters had documented histories of neglect as well as histories of physical and sexual abuse. Findings from a prospective longitudinal study by [Ogawa and colleagues \(1997\)](#) of children living in poverty indicate that neglect during infancy played a significant role in the development of dissociative symptoms during the preschool and elementary school years. Still these children were also subjected to other forms of trauma during the preschool and elementary period.

### **Disorganized Infant Attachment Patterns and Dissociation**

Neglect and maltreatment in infancy have been associated with the development of disorganized–disoriented patterns of attachment to the primary caregiver ( [Carlson et al., 1989](#); [Main and Hesse, 1990](#) ). Maltreated infants may display various peculiar behaviors in the presence of abusive or dissociative parents. Main and colleagues ( [Main and Morgan, 1996](#); [Main and Solomon, 1990](#) ) note that these disorganized–disoriented infants experience apprehension and fear when they interact with the parent. Torn between approaching and fleeing, they “freeze” and go into trancelike states. These behaviors appear to be brief episodes of dissociation ( [Liotti, 1992](#); [Main and Morgan, 1996](#) ). [Main and Hesse \(1990\)](#) postulate that disorganized–disoriented attachment behavior occurs when the child is forced to interact with a parent who is both protector and abuser. Liotti suggests that the need to adapt to such unpredictable parenting engenders dissociative patterns of interaction and, ultimately, may lead to DID ( [Liotti, 1992, 1999](#) ). [Ogawa and colleagues \(1997\)](#), in a prospective, longitudinal study, demonstrate that infants with disorganized attachment patterns, subjected to ongoing abuse during childhood, are more likely than their more securely attached peers to suffer from dissociative pathology in adolescence and early adulthood.

In summary, research to date suggests that extremely aberrant, unpredictable, and abusive parenting, especially during the earliest years of life when the formation of a secure attachment is so important, is at the core of severe dissociative pathology in childhood. Whether all severely maltreated children would develop dissociative disorders is not known. Some infants may be inherently more vulnerable than others to the effects of extreme abuse and neglect.

### **Individual Differences in the Capacity to Dissociate**

Children as a group are able to dissociate more readily than adolescents or adults ( [Putnam, 1996b](#) ). Young children routinely assume different roles and identities, become absorbed in pretend play, and generate imaginary friends and enemies to play out their fantasies. They also exhibit rapid shifts in demeanor, readily fall into brief, trancelike states, demonstrate amnesia for behaviors and events, and engage in denial as a psychological defense more readily than their elementary school-age peers ( [Cramer, 1991](#) ). The capacity to dissociate ordinarily wanes as children reach their teenage and young adult years ( [Ogawa et al., 1997](#); [Ray, 1996](#); [Ross et al., 1989b](#); [Waller et al., 1996](#) ). However, there is good evidence from life span research that recurrent, overwhelming traumas occurring early in life, when dissociative capacity is high, are especially likely to engender dissociative disorders ( [Sanberg and Lynn, 1992](#) ).

Studies of healthy adults, using the Dissociative Experiences Scale (DES), show that people vary in their capacities to dissociate, to disengage from immediate surroundings, and to become absorbed in reverie ( [Bernstein and Putnam, 1986](#); [Ray et al., 1992](#); [Ross et al., 1991](#) ). Whether such individual differences influence the development of pathologic dissociation is unclear. The only study published to date using methods of genetic analysis to investigate genetic contributions to the expression of pathologic dissociation found no evidence of a heritability factor in a sample of 280 monozygotic and dizygotic twin pairs ( [Waller and Ross, 1997](#) ). Rather, one-third to one-half of the variance was attributed to the shared social (abusive) environment, whereas the remainder of the variance was attributed to nonshared environmental factors. Because severe abuse tends to perpetuate itself from generation to generation, so, too, does dissociation. As such, evidence of intergenerational dissociative disorders does not necessarily bespeak genetic heritability ( [Yeager and Lewis, 1996](#) ).

Accumulating evidence suggests that pathologic dissociation is distinctly and categorically different from the normal dissociative continuum. Waller and colleagues, using DES data obtained from normal and clinical adolescent and adult samples, have distinguished two distinct groups or classes of persons, those who experience a range of ordinary dissociative phenomena (e.g., fantasy-proneness and absorption) and those who experience pathologic dissociation (e.g., amnesias, derealization, identity alterations) ( [Waller and Ross, 1997](#); [Waller et al., 1996](#) ). Our own studies comparing the imaginary companions of abused, dissociative children and normal children suggest that these differences in the nature of fantasy life exist early in life ( [Trujillo et al., 1996](#) ). Pathologic dissociation is rare in most other serious neuropsychiatric disorders (e.g., schizophrenia, mood disorders).

### **Hypnotizability and Dissociation**

Pathologic dissociation has been likened by some to self-hypnosis gone awry ( [Maldonado and Spiegel, 1998](#) ). However, whether dissociation and hypnosis are

similar mental processes is currently a subject of debate. Like dissociative capacity, *hypnotizability* varies on an individual basis and throughout the life span; it reaches its height during childhood and declines thereafter ( [Morgan and Hilgard, 1979](#)). Early investigations of the relationship between trauma and hypnotizability suggest that harsh punishment in childhood is associated with increased hypnotizability scores in adulthood ( [Hilgard, 1979](#)). In contrast, several more recent studies on the subject do not support these earlier findings (DiTommaso and Routh, 1993; [Nash et al., 1993](#); [Rhue et al., 1990](#)). In a review of research on the relationship between dissociation and hypnotizability, [Putnam and Carlson \(1998\)](#) conclude that hypnotizability and the tendency to dissociate appear to be weakly correlated. Although the relationship between hypnotizability and dissociation remains uncertain, children and adolescents with severe dissociative disorders look clinically as if they can self-hypnotize and enter into trancelike states more easily than their healthy peers.

### Models of Dissociative Identity Disorder

Numerous different models have been proposed to explain the hallmark of DID, alternate identity states. These range from the supernatural (e.g., demonic possession) to sociologic (e.g., taking on different identities to sidestep societal controls on behavior) to malingering for secondary gain ( [Kluft, 1996](#)). [Putnam \(1996b\)](#) hypothesizes that during the first months and years of life, children lack an integrated personality structure. The infant's responsivity to the environment is organized as a series of discrete behavior states that evolve naturally into a unified sense of self. He suggests that infants who are repeatedly and unpredictably traumatized may fail to integrate these behavioral states and, as a result, develop a discontinuous sense of self-identity. This hypothesis is based on state-dependent learning models. In these models, alternate identity states are thought to evolve when experiences and behaviors occurring in one state of consciousness are bound together as memories disconnected from usual conscious awareness, and they are accessible only in the original psychophysiologic state. Similarly, neural network models ( [Damasio, 1999](#); [Li and Spiegel, 1992](#)) hypothesize that alternate identity states are related to the creation and reinforcement of particular neural networks. These parallel networks are thought to give rise to multiple discontinuous representations of self and multiple autobiographical memory maps. In the absence of further research, the foregoing models remain speculative.

### NEUROBIOLOGY OF TRAUMATIC STRESS AND DISSOCIATION

To date, little research exists on the neurobiology of dissociative disorders. However, we do know that dissociation occurs peritraumatically as well as posttraumatically ( [Bremner et al., 1992](#); [Marmar et al., 1994](#); [Ursano et al., 1999](#)). Therefore, it makes sense to review the studies that have been done on the neurobiology of trauma. The following section reviews briefly the newer research on the neurobiological underpinnings of dissociative states and the neurobiological effects of repeated stress (e.g., child abuse and neglect) on the developing brain.

#### Neural Pathways Mediating the Fear Response

Ordinarily, incoming sensory input is registered by the thalamus and is relayed to the limbic and cortical areas of the brain involved in arousal, attention, emotion, learning, and memory. The thalamus, in turn, receives information from these brain regions and creates a feedback network that modulates an organism's responsiveness to environmental stimuli ( [McCormick, 1992](#); [Turner and Herkenham, 1991](#)). When incoming stimuli are perceived as threatening, however, the amygdala, the limbic structure responsible for initiating the fear response, sounds a "central alarm" ( [Goleman, 2000](#)) through the thalamus and triggers a cascade of stress-related neurochemicals to focus attention, to heighten vigilance, to slow the perception of time, and to stimulate recall of similarly threatening experiences so the organism can determine whether to fight or flee ( [DeWied and Croiset, 1991](#)).

The prefrontal cortex, the seat of executive and metacognitive functions, is believed to be responsible for our ability to distinguish between external and internal models of the world ( [Knight et al., 1995](#)). When a stressful event becomes completely overwhelming, the thalamic–limbic–cortical feedback network is thought to break down, thus causing distortions in both sensory and perceptual processes. Animal research has shown that during exposure to intense stress, extremely high levels of glutamate, dopamine, and norepinephrine are released in the prefrontal cortex to produce a functional lesion, that is, thus effectively shutting down executive function and working memory ( [Arnsten, 1999](#)). Several groups of researchers have hypothesized that, when the prefrontal cortex is unable to integrate diverse sensory, perceptual, cognitive, emotional, and psychomotor processes, a person's distinction between self and surroundings becomes impaired, and this results in dissociation ( [Allen et al., 1999](#); [Chambers et al., 1999](#); [Guralnik et al., 2000](#); [Newport and Nemeroff, 2000](#)). Functional neuroimaging studies of adults with PTSD seem to support this hypothesis. Samples of victims of combat distress, as well as victims of childhood abuse, have demonstrated decreased activity in the prefrontal and parietal cortices and increased activation in the limbic system while they are reliving traumatic experiences ( [Bremner et al., 1999a](#); [Bremner et al., 1999b](#); [Liberzon et al., 1999](#); [Rausch et al., 1996](#); [Shin et al., 1999](#)).

The hippocampus and its surrounding structures, that region of the brain vital for encoding new information as well as for retrieving memories, also seem to be essential for our cohesive sense of self. Electrical stimulation of the hippocampus has been shown to cause depersonalization, derealization, odd visceral sensations, time and space disorientation, and auditory and visual hallucinations ( [Halgren et al., 1978](#)). Furthermore, hippocampal function is exceptionally vulnerable to the effects of acute stress. High levels of endogenous opioids, norepinephrine or epinephrine and corticosteroids, neurochemicals released in response to acute fear or distress, have been found to impair information encoding and retention ( [Lupien et al., 1999](#); [McGaugh, 1995](#); [Newcomer et al., 1999](#)). Chronically high levels of circulating corticotropin releasing hormone in particular appears to be neurotoxic to the hippocampus ( [Brunson et al., 2001](#); [Kaufman and Charney, 2001](#)). High concentrations of glutamate and low concentrations of neurotrophins (essential for neurogenesis) also are thought to contribute to hippocampal atrophy. Hippocampal volume loss has been demonstrated in both combat veterans and sexually abused adults with PTSD ( [Bremner et al., 1995](#); [Bremner et al., 1997](#); [Gurvits et al., 1996](#); [Stein et al., 1997](#)). What is more, these studies report that greater hippocampal atrophy is associated with greater explicit memory deficits ( [Bremner et al., 1995](#); [Bremner et al., 1997](#); [Gurvits et al., 1996](#)) and is also predictive of dissociative symptoms ( [Stein et al., 1997](#)).

#### Conditioning of the Fear Response: A Habit of Mind

Significant prior exposure to excessive stress-related neurochemicals seems to create heightened neurosensitivity not only to extreme stress, but also to benign events. The amygdala, the structure that initiates the fear response, also encodes the emotional component of memories. Together, the amygdala and the hippocampus are responsible for producing what is called the *conditioned fear response* ( [LeDoux, 1993](#)). The conditioned fear response, a form of implicit (i.e., outside of conscious awareness) memory, develops from repeated pairings of painful stimuli with neutral stimuli, such as a sound, a smell, or an environmental context. Eventually, the neutral stimulus alone is able to elicit fear. [Post and colleagues \(1998\)](#) postulate that repeated stimulation of the amygdala by recurrent external and internal (e.g., flashbacks) exposure to trauma may induce seizurelike kindling. At the same time, the anterior cingulate cortex, implicated in extinguishing the fear response, fails to turn it off ( [Hamner et al., 1999](#)). Consequently, the fear response begins to occur autonomously, without any cueing from the external environment or internal world. This hypothesis is supported clinically by the observation that dissociative children and adults often switch reflexively into another identity state the instant the brain registers a word or a sound or a sight that is associated with past traumas. It is also supported empirically by functional neuroimaging studies that demonstrate anterior cingulate hypometabolism in both children and adults with PTSD ( [Bremner et al., 1999a](#); [De Bellis et al., 2000](#); [Shin et al., 1999](#)).

#### Neurobiological Effects of Child Abuse and Neglect

Children are especially vulnerable to the effects of traumatic stress. Unlike the mature brain, the developing brain can suffer profound, potentially enduring neurophysiologic impairment as a result of overwhelming stress, especially when such events occur during critical periods of neural development. Poverty, prior exposure to traumatic stressors, and caregiver emotional instability make the child especially susceptible to the effects of trauma ( [Pfefferbaum, 1997](#)). Evidence is emerging that suggests child maltreatment in particular may cause long-term neurobiological dysfunction. For example, chronic abuse and neglect have been associated with hypothalamic–pituitary–adrenal axis dysregulation in children ( [De Bellis and Putnam, 1994](#); [De Bellis et al., 1994](#); [De Bellis et al., 1999a](#); [Kaufman et al., 1997](#); [Perry, 1994](#); [Putnam and Trickett, 1997](#)).

Severe abuse has electrophysiologic and neuroanatomic sequelae as well. Ito and colleagues report abnormal electroencephalographic findings in the left hemispheres of abused children ( [Ito et al., 1993](#); [Ito et al., 1998](#)). In their magnetic resonance imaging study of maltreated children with PTSD and matched controls, [De Bellis and colleagues \(1999b\)](#) found that, compared with healthy children, abused children had significantly smaller intracranial and cerebral volumes, with enlarged ventricles and excessive cortical cerebrospinal fluid. The extent of neurologic abnormalities correlates with onset and duration of abuse. The possible role of these kinds of neuroanatomic and neurophysiologic changes in severely dissociative children remains to be explored.

#### Psychopharmacologic Analogs of Dissociation

Certain kinds of drugs cause dissociative states in humans. Ketamine, an *N*-methyl-D-aspartate antagonist, is thought to cause dopamine dysregulation, alter



glutamate availability, and thus disrupt cortical–subcortical pathways ( [Chambers et al., 1999](#)). Clinically, it produces dissociative states, cognitive dysfunction, and memory impairment in healthy persons ([Krystal et al., 1994](#); [Zukin, 2000](#)). Subanesthetic doses cause depersonalization, derealization, hallucinations, and distorted sensory, time, body, and identity perceptions ([Curran and Morgan, 2000](#); [Hansen et al., 1988](#); [Jansen, 1990](#); [Krystal et al., 1994](#); [Malhotra et al., 1996](#)). Animal research shows that the long-term use of high doses of ketamine destroys temporal and parietal neurons as well as neurons in the posterior cingulate gyrus, amygdala, and hippocampus ([Wozniak et al., 1993](#); [Zukin, 2000](#)).

Another drug, yohimbine, an  $\alpha_2$ -adrenergic receptor antagonist that indirectly increases availability of brain norepinephrine ( [Starke et al., 1975](#)), has been reported to induce panic attacks, flashbacks, and dissociative symptoms in patients with PTSD but not in healthy controls ( [Southwick et al., 1993](#); [Southwick et al., 1999](#)). These findings are consistent with the hypothesis that, over time, in chronically traumatized persons, the threshold for activation of the fear response is lowered, triggering posttraumatic and dissociative responses to even minor stressors. The foregoing findings provide indirect evidence of possible neurobiological mechanisms involved in pathologic dissociation.

### Neurophysiology and Alternate Personality States

We do not know what happens neurophysiologically when a person with DID switches from one personality state to another. Adult patients frequently manifest staring episodes, stuttering, twitches, eye rolls, and other facial or body movements during transitions between states. Movements appear to be involuntary and outside the patient's awareness. Clinically, these phenomena are observed more rarely in children with DID than in adults.

Studies of the neurophysiologic differences among alternate personality states have been conducted on adults. They include testing of the following: differences in autonomic responsivity such as galvanic skin response ( [Brende, 1984](#); [Larmore et al., 1977](#); [Ludwig et al., 1972](#); [Putnam et al., 1989](#)), as well as heart rate and respiration ([Bahnon and Smith, 1975](#)); differences in electromyography, ([Larmore et al., 1977](#)), electroencephalographic changes ([Cocores et al., 1984](#); [Coons et al., 1982](#); [Larmore et al., 1977](#); [Ludwig et al., 1972](#); [Thigpen and Cleckley, 1954](#)), evoked potentials ([Braun, 1983](#); [Larmore et al., 1977](#); [Ludwig et al., 1972](#); [Putnam, 1984](#)), and regional cerebral blood flow ( [de Vito et al., 1985](#); [Lefkof et al., 1984](#); [Mathew et al., 1985](#); [Saxe et al., 1992](#)); and differences in cognitive functioning ([Armstrong and Loewenstein, 1990](#); [Coons and Sterne, 1986](#); [Erickson and Rappaport, 1980](#); [Lovitt and Lefkof, 1985](#); [Ludwig et al., 1972](#); [Silberman et al., 1988](#); [Wagner et al., 1983](#)). Data from these kinds of studies, even when viewed with caution because of their small or single case sample size, do suggest that physiologic changes occur in different personality states, and some of these changes cannot be feigned by method actors or by subjects pretending to suffer from DID.

### CLINICAL PICTURE

It is harder to diagnose DID in children than in adults. The switches from one personality state to another may be fleeting and may go unnoticed. The characteristics of alternate personality states are often less fixed and their differences are less pronounced, so when changes from one identity state to another occur, they may be attributed simply to mood swings. Symptoms and behaviors characteristic of the disorder also mimic numerous other more commonly recognized psychiatric disorders. Many of the signs, symptoms, and behaviors of the illness (e.g., communicating with vivid imaginary companions, behaving like an animal, changing voice and demeanor, having peculiar beliefs or convictions) are easily dismissed as child's play. Further complicating recognition of the disorder is the finding that the abuse history is almost invariably concealed by both children and caregivers. However, the major reason for the failure to recognize DID is that many clinicians question its very existence, and one cannot see what one does not believe exists.

Assuming now a willingness to entertain the diagnosis, what will the clinician encounter? The most common sign in children suffering DID is a tendency for the child to fall into trancelike states and to become oblivious to the environment. Before witnessing these behaviors in the office, the clinician hears complaints from teachers or caretakers that the child "spaces out" and fails to respond to questions or instructions. This particular symptom is often so prominent that it leads clinicians to wonder whether the child suffers from a seizure disorder. At the Bellevue Dissociative Disorders Clinic, we evaluated a 10-year-old child whose lapses were so severe, beginning at age 7 years, that at another clinic she was diagnosed with narcolepsy and was treated with stimulants. Usually, however, lapses or trances are less obviously pathologic and are dismissed as manifestations of boredom or daydreaming.

Although these trancelike states may appear to others to be random, they seem to occur in response to stimuli that have special, idiosyncratic meanings to the child. A teacher raises her voice and momentarily sounds like an abuser; a phrase in a book awakens a painful memory, and the child suddenly dissociates. Often a word or a nudge on the playground acts as a sudden reminder of past horrors. In a blink, the child unconsciously switches into another personality state to cope with the potential threat. At such times, the ordinarily docile child may curse or may even assault the perceived enemy. Later on, the child is oblivious to what transpired. Adults witnessing these sudden behavioral changes often describe the child as "not himself" or even as "possessed."

Another hallmark of DID is amnesia for behaviors and events. These amnesias are often spotty. Children may recall certain happy events, such as visits to the park with a parent, but are amnesic for abusive experiences. Dissociative children sometimes recall abusive events, but they misidentify perpetrators. Unaware of their amnesias, they deny any loss of memory. Several of the children we evaluated at Bellevue complained spontaneously of not knowing what was real and what was a dream. Many suffered the nightmares, flashbacks, and episodes of apparent sleepwalking typical of patients with PTSD.

Trances, lapses, amnesias, and personality state switches can wreak havoc on academic performance. When children are "spaced out," in alternate states of consciousness, they are oblivious to what is being taught. Subsequently, perplexed by unfamiliar homework, these children accuse teachers of never having taught the material. These children may study for a test while in one state, take the test in a different state, and consequently draw a complete blank. Scores on intelligence tests and achievement tests are often erratic, causing perfectly intelligent, capable, but dissociative children to be labeled learning disabled or even retarded. Homework papers and other assignments may vary from messy and indecipherable to neat and flawless. Occasionally, a neat, bright, well-organized aspect of the child always performs impeccably at school while dissociative episodes occur primarily in other settings. Contrary to the folklore that insists that persons with DID are exceptionally bright, we have evaluated several dissociative children, adolescents, and adults who were of borderline intelligence or severely learning disabled.

The same lapses and switches that often compromise academic performance give rise to severe behavioral problems in school settings. Several children whom we have evaluated have described imaginary friends who entertain them and tell them jokes at times of stress. Reviews of the school records of these children reveal comments such as "laughs inappropriately in class." Sometimes these imaginary friends give unfortunate advice. For example, one 9-year-old girl who was forever in trouble for "having a fresh mouth," would hear a voice in her head whisper, "Say 'dumb' to the teacher."

Alternate personality states may appear briefly to take objects, break things, or shout insults or curses. They may disappear as quickly as they arrived, leaving the bewildered child to deal with the consequences of his or her acts. Sometimes children suffering from DID find objects in their rooms or in their pockets and have no idea how they got there. In one state, they may assault a peer and, in another state, deny it. When the evidence against them is strong and the denials are vociferous, such children are often called pathologic liars.

As is evident from the foregoing description, children suffering from DID are changeable. One moment they may be sweet and agreeable; the next they are having a tantrum or assaulting a playmate. They may start the day upbeat and end it withdrawn and depressed, even suicidal. If their behavioral and academic problems have not already earned for them a disruptive disorder diagnosis, then these fluctuations may cause them to receive the diagnosis of a mood disorder. As mentioned earlier, sometimes they do indeed suffer from such a disorder as well as from DID.

One of the most common symptoms of DID is auditory hallucinations. Dissociative children hear the voices of imaginary friends or alternate personality states speak to them and to each other. They also receive instructions from them. Often they talk aloud to these voices. Caretakers, listening in on such conversations, sometimes think the child is playing with a friend, so different are the voices overheard. Several children and adolescents whom we have evaluated have also experienced vivid, grotesque, visual hallucinations of blood and body parts, apparent flashbacks to overwhelming experiences. Sometimes children report a hierarchy of imaginary companions that has the quality of a fixed delusion. As children report their experiences, they often become confused, contradictory, and momentarily inarticulate. They stutter; they pause and look upward, as if listening to instructions, before resuming speech. The combination of command hallucinations, voices speaking to each other, delusional beliefs, illogical thought patterns, and ostensible thought blocking sometimes leads to a diagnosis of schizophrenia. However, this cluster of signs and symptoms is far more common in patients with DID than in schizophrenic patients ([Kluft, 1987b](#); [Ross et al., 1990b](#)).

Thus, children with DID have a plethora of signs, symptoms, and behaviors suggestive of many different kinds of medical and psychiatric disorders. If clinic and emergency room or hospital records can be obtained, they often document numerous doctor visits and a multiplicity of diagnostic procedures ranging from barium enemas to magnetic resonance imaging scans and electroencephalograms. Depending on the personality state of the child at the time of a psychiatric evaluation, he or she may be treated with stimulant medication, antidepressants, antipsychotics, antiepileptics, antacids, stool softeners, enemas, or all of these. When none of

these interventions has lasting benefit (although at first any one of them may seem to help), such children are dismissed either as having a disruptive disorder or as malingering.

**Nature of Alternate Personality States in Children and Adolescents**

The trances, the amnesias, and all the behaviors that follow from them are like the dust storm underneath which hides the child's alternate personality states. A principle to keep in mind is that each identity state has a function. Without it, the child on some level feels vulnerable. Therefore, all identity states are important, and the memories, thoughts, and feelings of each must be understood and respected.

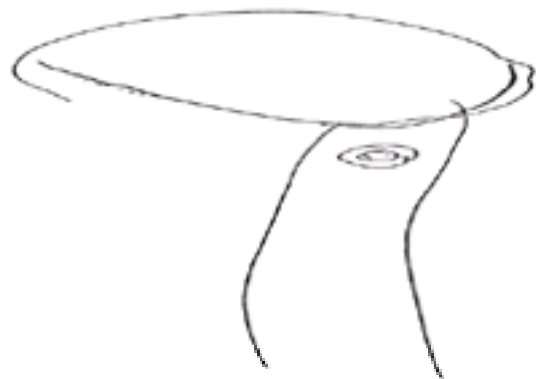
The different personality states of children with DID are remarkably similar to those of adults, and they serve very similar functions. Together, their major function is to protect the child from experiencing physical and emotional trauma. By their "taking over" at the time abuse is occurring, memories of maltreatment are kept out of conscious awareness. Sometimes alternate personality states function as violent protectors, attacking both real and imagined abusers. Those alternate identity states that retain memories of past horror are typically the most aggressive and dangerous. In fact, it is common for an alternate personality state to take on the characteristics, even the name, of an abuser.

Closely related to the aggressive personality states are seductive, sexually precocious identity states. They serve several functions. In such states, children act as if they enjoy and even invite the sexual activity to which they are being subjected. Sometimes they perform explicit sexual acts that the child was forced to perform for the abuser or for the camera of the abuser. We have seen a 4½-year-old child who was used in pornographic films scuffle with a male anatomically correct doll, stomp on its genitals, fall on top of it, twist its arms while holding it between her legs and, in a voice strangely similar to the child's mother's voice, call it a bastard. Shortly thereafter, when the doll was placed out of reach, the child lay back, knees to her chest and panties exposed, and beckoned toward the doll, purring, "I need him!"

Not all identity states are aggressive or sexually inappropriate. There are almost invariably benign identities that are also protective. Sometimes they are the first to come into being, emerging at a time of loneliness and vulnerability. These are characters who comfort and amuse. Some are "friends," even "twins." Others are like fairy godmothers. Some are infants. We have treated several young children who had 1- or 2-year old personality states.

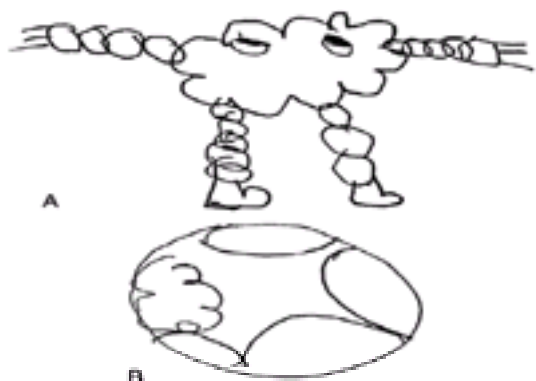
Is there a "host" personality? Therapists tend to consider the "host" the first personality state they meet, the one who first walks into the office. Therapists tend to assume the existence of a solid, core personality from which other personality states diverge. However, if maltreatment begins very early, during infancy, a core sense of self may never develop. The child's personality is undeveloped, fragmented, and changeable. There is no unified core personality structure.

Children who suffer from DID usually have no idea of the nature of their disorder. They know only that they have imaginary friends who reside in their heads, or in other parts of their bodies, or who come to them from afar, and who both help and torment them. [Figure 70.1](#) illustrates a self-portrait, by a 6-year-old boy, showing an alternate personality state who, as he described him, "lives in my heart."



**Figure 70.1.** Self-portrait of a 6-year-old boy with dissociative identity disorder/multiple personality disorder, depicting the place where his alternate personality lives—"in my heart."

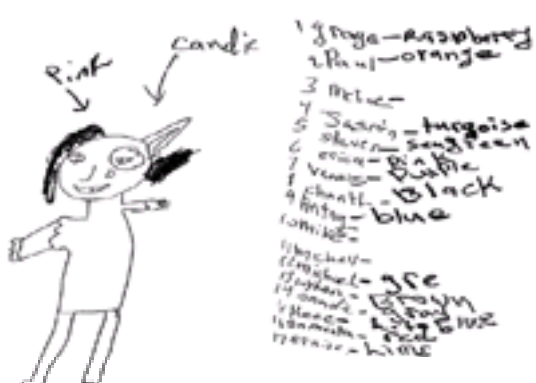
[Figure 70.2A](#) shows the drawing an 8-year-old boy did of his alternate personality state, "Joe," who, he explained, lived in a scalloped area of his brain ( [Fig. 70.2B](#)). This boy drew other compartments in his brain; he volunteered that others lived there, but he did not know who they were.



**Figure 70.2.** A: Picture drawn by an 8-year-old boy with dissociative identity disorder/multiple personality disorder of his alternate personality, "Joe." B: Picture drawn by the same boy, depicting the place in his brain where "Joe" lives.

Individual experiences and cultural practices influence the nature of alternate personality states. Strong defenders are often identified as the latest superhero or television character. Angry, aggressive identity states are identified by some children as a devil; benign personalities sometimes receive names like Angel. Many dissociative children name their inner friends after cousins or schoolmates, thus making it hard for therapists to know whom the child is describing.

Animal personality states are especially characteristic of young children. [Figure 70.3](#) was drawn by a 10-year-old child with DID. Listed are her numerous alternate personality states. The most powerful is a bull, depicted here as occupying half her body.





**Figure 70.3.** Picture, drawn by a 10-year-old girl, depicting a powerful alternate personality, a bull, as taking over half of her body. Also listed are her numerous other alternate personalities.

In our experience, animal personality states have tended to be protectors. For example, one 4½-year-old severely abused boy, when threatened, became a tiger, growled, and bared his teeth. On one occasion, he actually bit his foster mother. Another 8-year-old boy, who was referred to our clinic with numerous dissociative symptoms, had a dog as an imaginary companion. We did not realize that the dog took control of the boy's behavior until we reviewed videotapes of a therapy session; the microphone picked up a menacing growl that was inaudible to the therapists during the interview.

### Imaginary Companions Versus Alternate Personality States

Many normal children have imaginary companions or special toys that seem real to them. By the time a psychologically healthy child reaches the age of 6 or 7 years, most imaginary companions are long gone. Thus, the existence of an imaginary companion much beyond 8 years of age could be cause for concern. However, even 4- and 5-year-olds can usually distinguish between what is real and what is a product of their imaginations.

In a study we conducted on the nature of imaginary friends in dissociative children and in normal school children ( [Trujillo et al., 1996](#) ), we found that although similar proportions of children reported having had imaginary friends, by 10 years of age the normal children had all dispensed with their fictional playmates; many of the dissociative children, conversely, reported that their imaginary friends were still active parts of their lives. The functions of the imaginary friends also differed between groups. All the imaginary friends of the normal children were benign, played games with them, and offered good advice. The imaginary friends of the dissociative children, however, were not always benevolent. Often they were menacing and provocative. How the imaginary companions of young dissociative children evolve into alternate identity states remains to be understood psychologically and neurophysiologically.

### Other Signs and Symptoms

Many of the indicators of DID in children are also characteristic of psychologically healthy children (e.g., imaginary companions, spacing out, or day dreaming) and others are seen various other diagnoses (e.g., conduct disorder, attention deficit hyperactivity disorder). Several authors have created lists of the most prevalent signs, symptoms, and behaviors of childhood DID ( [Fagan and McMahon, 1984](#); [Hornstein and Putnam, 1992](#); [Kluft, 1985](#); [Peterson, 1990](#); [Putnam, 1993](#) ). These include trancelike states, amnesias, fluctuations in mood, fluctuations in abilities, disavowed behaviors (e.g., lying or stealing and denying having done so), marked behavioral changes (e.g., from aggressive to overly good), rapid regressions in behavior, evidence of active imaginary companions, auditory hallucinations, flashbacks, apparent sleepwalking, amnesia for having been abused, hysterical symptoms, fluctuating physical complaints, and referring to the self in the third person. Refractoriness to various forms of psychotherapy and medication directed at treating other diagnoses is also frequently part of the history. We would add to this list difficulty in distinguishing fantasy from reality (e.g., believing certain experiences were dreamed), acting like an animal (e.g., making animal sounds; moving in animal-like ways), using different names at different times, and having different writing and drawing styles. The clinician must keep in mind that any one of the foregoing characteristics may be seen in normal children and in children with disorders other than DID. There is no fixed number of signs and symptoms that unequivocally indicates the existence of DID. However, unlike other diagnoses with coherent sets of symptoms (e.g., depression), the extraordinary number of diverse signs, symptoms, and behaviors (e.g., auditory hallucinations, amnesias, trance states, mood swings) should alert the clinician to the possibility of a dissociative disorder.

### Psychosomatic Symptoms and Phenomena

Children who suffer from DID tend to complain of the very same kinds of physical symptoms as do their adult counterparts.

#### HEADACHES

A history of frequent, sometimes severe *headaches* is common. At least some of these headaches occur in response to conflicts among different personality states. For example, a 9-year-old child with a history of sexual abuse experienced severe headaches when the characters in her head argued over whether or not to visit the former abuser.

#### SEIZURES AND NARCOLEPSY

*Seizurelike symptoms* or *pseudoseizures* also may be part of the picture in children suffering from DID. We have evaluated several children and adults with seizurelike symptoms who underwent extensive neurologic evaluations before referral. We have evaluated three children with DID whose symptoms led previous doctors to suspect (and in one case treat) narcolepsy. In all these cases, the children suddenly seemed to fall asleep for no apparent reason. These "attacks" occurred in response to perceived threats in the child's environment.

#### ANESTHESIAS AND SCARRING

Children and adolescents with DID often report being able to block out physical pain. Often, they are amnesic for events that resulted in severe *scarring*. Proof of abuse can often be found on the child's back, arms, legs, and even on genitalia. Dissociative children often also have scars from self-inflicted cuts or burns for which they are amnesic. Sometimes one dissociated aspect of the child whispers "Cut yourself" or "Jump in front of a car."

#### PARALYSES

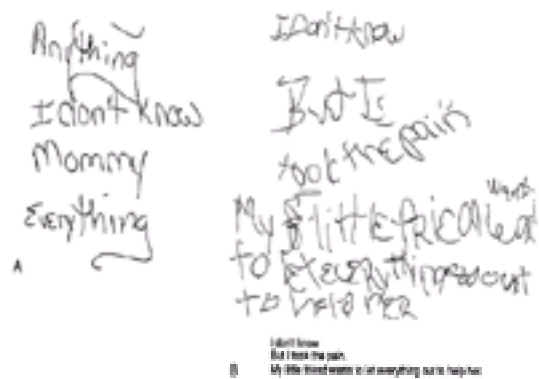
Conversion symptoms, including apparent *paralyses*, have also been observed in children with DID. One 4½-year-old, severely traumatized boy claimed at times to be unable to walk. During therapy, it was learned that the boy had an infant personality who could not yet walk. Suggestions that the "baby" would mature and be able to walk cured the problem.

#### VISUAL, AUDITORY, AND SPEECH ALTERATIONS

The kinds of differences in *visual acuity and hearing* reported to exist among the so-called "alters" of adult patients may well exist in children but go unrecognized. At the Bellevue clinic, although we do not test the visual acuity of different personality states, we occasionally observe a slight strabismus in a child while in one state, but not in others. It is also common in children to have one or more personality states that cannot or will not speak. Sometimes a personality state is reported to be too young to speak. Some children, while in particular states, are selectively mute. However, many children, while in these states, write to the therapist. Selectively mute personality states are often very concrete. Abusers have threatened them if they tell what happened to them, but writing is somehow permitted. Penmanship differs markedly in different states. In one case, an adolescent boy had an alternate personality state that spoke with an Irish brogue.

#### HANDEDNESS AND WRITING

Adults with DID have been known to change *handedness* while they are in different identity states ( [Putnam, et al. 1986](#) ). We have treated several children who spontaneously changed writing hands after switching into different identity states. [Figure 70.4](#) illustrates the right- and left-handed writings of a 13-year-old girl with DID. Handwriting styles change not only with handedness but also according to the developmental stage of the alternate personality state.



**Figure 70.4.** A: Writing sample of a 13-year-old, right-handed female patient with dissociative identity disorder/multiple personality disorder. B: Writing of left-handed alternate personality of the same 13-year-old girl.

## GASTROINTESTINAL AND GENITOURINARY PROBLEMS

Boys and girls with DID often have histories of a variety of *gastrointestinal and genitourinary complaints*. Some of these complaints (e.g., painful defecation) are the direct result of sexual abuse. We have found a history of constipation and encopresis to be especially common among sexually traumatized, dissociative boys (Morrow et al., 1997). Unfortunately, during medical workups, the examinations, tests, and treatments (e.g., digital rectal examinations, barium enemas, suppositories) further traumatize these already abused children. Such children often have nausea and vague abdominal pains for which no medical cause can be found. For example, the female personality state of an adolescent boy in our clinic experienced episodic gagging when reliving fellatio by an older male relative. Another adolescent girl could not tolerate using toothpaste because it reminded her of the semen she had been forced to swallow.

## DIAGNOSTIC EVALUATION

As is true of most other psychiatric diagnoses, there are no specific physiologic tests for DID. Careful, comprehensive clinical interviews, not only with the child, but more importantly with knowledgeable adults in the child's life, are the only accurate means of diagnosing the disorder.

### Screening Instruments and Interview Protocols for Dissociative Disorders

Several screening instruments have been created to identify the signs, symptoms, and behaviors indicative of pathologic dissociation in children and adolescents (Fagan and McMahon, 1984; Kluff, 1985; Peterson, 1990; Putnam et al., 1993; Reagor et al., 1992). These instruments are completed by caregivers and other adults who know the child well. The Child Dissociative Checklist by Frank Putnam and colleagues (1993), a 20-item behavioral screening tool, is the most widely used checklist in circulation. The checklist reportedly has good test-retest reliability and satisfactory concurrent and discriminant validity (Putnam and Peterson, 1994; Wherry et al., 1994). Conversely, when we have asked two different family members to fill out the Child Dissociative Checklist for the same child, we have found striking differences between the two reports.

Two self-report screening instruments are also available. The Children's Perceptual Alteration Scale (Evers-Szostak and Sanders, 1992) is a 28-item measure completed by children between the ages of 8 and 12 years. The Adolescent Dissociative Experiences Scale (A-DES) (Armstrong et al., 1997) is a 30-item screening instrument designed especially for adolescents between the ages of 12 and 18 years. Initial psychometric data on the A-DES have demonstrated good validity and reliability (Armstrong et al., 1997; Smith and Carlson, 1996).

High scores alone on any of these instruments are not necessarily diagnostic of a dissociative disorder; a comprehensive diagnostic interview is the only accurate means of making the diagnosis. One such interview protocol, the Structured Clinical Interview for DSM-IV Dissociative Disorders—Revised (Steinberg, 1994), originally designed for adults, is reportedly useful for adolescents as young as 14 years of age (Steinberg, 1996; Steinberg and Steinberg, 1995). It assesses the presence and severity of five core dissociative symptoms (i.e., amnesia, depersonalization, derealization, identity confusion, identity alteration). However, because the protocol does not inquire about issues other than pathologic dissociation, it must be used in conjunction with a more comprehensive diagnostic interview protocol, such as the *Diagnostic Interview for Children—Revised* (Shaffer et al., 1993).

We at the Bellevue clinic created a semistructured diagnostic interview for both children and adolescents, the *Bellevue Dissociative Disorders Interview for Children* (Lewis, 1996). The protocol not only taps signs, symptoms, and behaviors indicative of pathologic dissociation, but it also inquires about medical and neurologic symptoms, mood dysregulation, auditory and visual hallucinations, aggression, posttraumatic stress symptoms, experiences of physical and sexual abuse, neglect, and intrafamily violence, and parental dissociative symptoms. Because the protocol was designed to permit an experienced interviewer to inquire about topics in a flexible order, it has been especially useful with very disturbed, anxious children and adolescents who are unable to cooperate with the demands of a highly structured protocol. To date, the protocol has been pilot tested on a group of severely abused children who were in residential treatment and on a sample of ordinary fifth grade school children. It distinguishes well between the groups and has good interrater reliability regarding the presence or absence of specific dissociative symptoms. Test-retest reliability has not yet been tested.

### Clinical Interview

The goals of the diagnostic interview are (a) to learn how the child experiences his or her inner world, (b) to identify the nature of personality states, (c) to learn when these states first appeared, (d) to understand their functions, (e) to explore their memories of past events, and, most important, (f) to determine whether the child is safe from further abuse. The child who continues to be abused will not improve with psychotherapy, no matter how talented the therapist, because the child must continue to dissociate to survive.

In addition to assessing for signs, symptoms, and behaviors indicative of pathologic dissociation (as described earlier), a thorough evaluation for DID should cover questions regarding history of caretaking, forms of parental and caretaker discipline, parental dissociative behaviors, sexual experiences including pornography and prostitution, various medical questions dealing with gastrointestinal, genitourinary, and neurologic symptoms, accidents, loss of consciousness, and the origin of scars. The *Bellevue Dissociative Disorders Interview for Children* (Lewis, 1990) addresses these important topics.

Dissociative children and adolescents often do not know they suffer from a psychiatric condition, or, if they are aware that something is wrong with their minds, they tenaciously guard their secret. We have found that asking about the child's imagination is an acceptable way of beginning the clinical interview; it is a nonthreatening topic for the child and can produce a wealth of important information for the clinician. Questions about favorite television shows and movies can elicit information about intense absorption in fantasy, as well as about trances and time loss. Questions about special toys and make-believe friends often reveal a complex inner world to which the child escapes to block out painful or frightening events. This line of questioning enables the child to talk about imaginary characters who seem real and who talk to the child and to each other. Questions about what "they" do for the child often bring forth descriptions of how they take over when the child is in a tight spot. Some children switch spontaneously into other identity states in response to the clinician's asking, "Would it be okay for me to talk with [a particular character]?"

A word of caution regarding the discovery of alternate personality states: until the time the child is evaluated, the very existence of these states has been a secret kept from the rest of the world. Alternate personality states in children as well as adults perceive this secrecy as vital for survival. The clinician's discovery of the existence of these personality states is often viewed by "them" as betrayal, and "they" may retaliate (e.g., tell the child to harm himself). After the child reveals his or her secret inner life, it is not unusual for self-mutilation or even a suicide attempt to occur. For this reason, after almost every session, and especially after the first session in which one learns of alternate identity states, it is prudent to end the session by expressing gratitude and admiration for all aspects of the child and by asking for a promise from all not to hurt themselves, each other, or anyone else.

It is vital that the child undergo a physical examination at some point during the evaluation process. The purpose of the of the physical examination is threefold: (a) to obtain a detailed medical history; (b) to assess the nature of specific physical symptoms such as seizurelike episodes, headaches, trancelike states, and stomach pains; and (c) to document evidence of physical or sexual abuse if it has occurred. Children with a suspected dissociative disorder should not be referred to a



pediatrician for simply a routine examination. The pediatrician must be informed of the clinician's suspicions that the child may have been abused, and care must be taken to avoid retraumatizing the child during the examination. An essential part of this physical examination is an examination of the skin for scars. Dissociative children usually carry the signs of physical abuse and self-mutilation all over their bodies, especially on their heads, arms, legs, and backs. The most severely abused children are often consciously unaware of particular scars and their origins. Sometimes just pointing out scars and inquiring about them causes the child to dissociate, so traumatic are the memories associated with them.

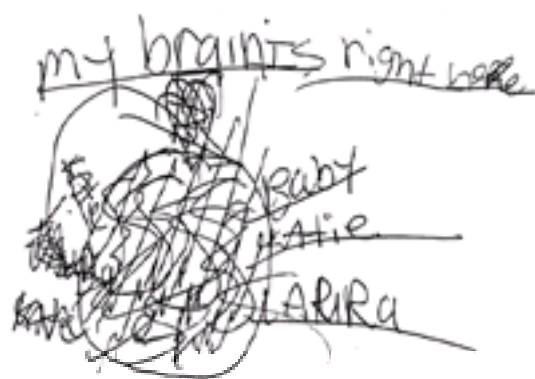
### Use of Drawings to Aid in Diagnosis

Asking the child or adolescent to draw can be a useful adjunct to the clinical interview. Such drawings can range from age-appropriate to infantile and primitive, again, depending on which personality state the child is in while performing the task. Children with DID may make self-portraits that are divided or split, or they may draw multiple heads or many appendages. In addition, patients with personality states of the opposite sex sometimes draw sexually ambiguous figures. The drawings in [Fig. 70.5](#) were done by Karen, a young adolescent girl. The first set of drawings ([Fig. 70.5A](#)) was done in her usual personality state in response to the requests: "Draw a Person. . . . Now draw the opposite sex." The second set ([Fig. 70.5B](#)) was done while she was in a younger personality state in response to the same requests. Note the immaturity of the drawing as well as the sexual confusion (i.e., the boy figure, on the left, is wearing a dress, and the girl figure, on the right, is wearing slacks).

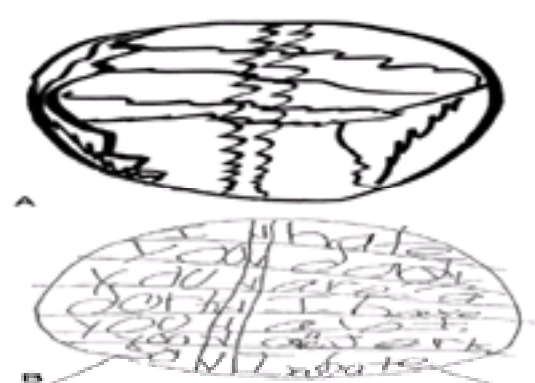


**Figure 70.5.** Drawings of a girl and boy made by a 13-year-old girl with dissociative identity disorder/multiple personality disorder. *B*: Drawings made by an infantile alternate personality of this same adolescent girl.

We have also asked children to draw a picture of their brain. Children with DID often imagine their brains to be compartmentalized, housing different identity states. [Figure 70.6](#) shows a picture of her brain, drawn by a 9-year-old girl with DID. Note how she depicts different characters living in different sections of her brain. [Figure 70.7A](#) shows an 8-year-old boy's picture of his brain. [Figure 70.7B](#) shows a picture of his brain drawn by the same child approximately 30 minutes later in response to the very same request by a different clinician. At the time of the second drawing, this child was in an altered state and had no memory of having drawn the first picture.



**Figure 70.6.** A drawing by a 9-year-old girl depicting her brain. Sections are designated for several alternate identity states.



**Figure 70.7.** A: Picture, drawn by an 8-year-old boy with dissociative identity disorder/multiple personality disorder, depicting his brain. *B*: Picture, drawn minutes later by the alternate personality of the same 8-year-old boy, depicting his brain.

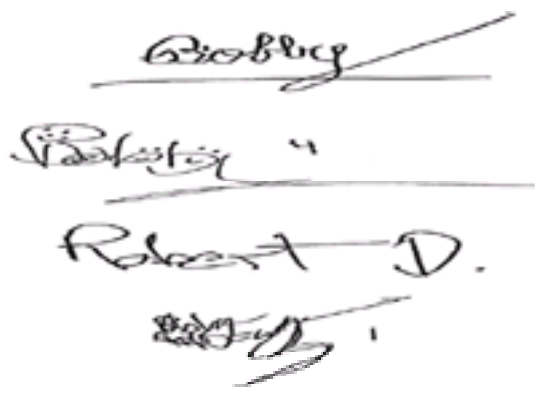
The simplest task of all is to ask the child to write his or her name while in different identity states. Not only does handwriting vary, but also the very ability to write at all may differ from state to state. Some characters may simply make an "X" or a special sign because they are illiterate; others may have an immature way of lettering, characteristic of a small child; and others may be too young to write at all. The way in which the child grasps the writing instrument also tells a lot about the age of that particular personality state.

### Use of Schoolwork and Journals to Aid in Diagnosis

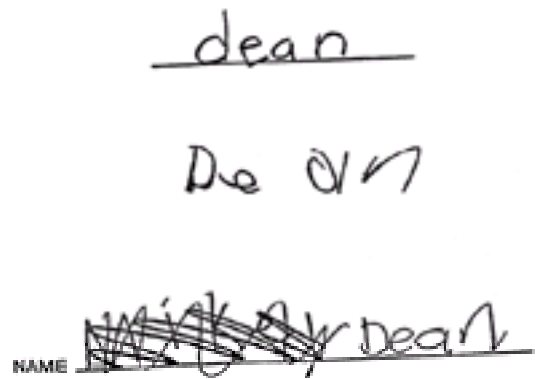
A dissociative child's handwritten and drawn materials can provide excellent corroborative evidence of DID. In contrast, the writings produced in the course of an evaluation may be influenced by the examiner and may become the focus of suspicion by DID skeptics. Therefore, it is especially helpful diagnostically to find graphic evidence of DID from writings and drawings made before the evaluation. Homework and classroom exercises are invaluable sources of documentation. A careful examination of all available written work not only can confirm the diagnosis, but also can furnish information regarding the names, identities, and functions of alternate personality states. Sometimes the content of these extraordinary caches can even document the nature of the abuse endured by a child. The following figures come from homework papers, classroom exercises, and journals of children with DID.

[Figure 70.8](#) illustrates four different signatures found in the homework of a 12-year-old boy. They illustrate the different developmental levels of several of his personality states. [Figure 70.9](#) illustrates the age-appropriate signature found at the top of the worksheets of an 8-year-old boy with DID; below it is the signature of an immature personality state, and below that is the crossed-out name of a personality state (Mikey), followed by the child's given name. [Figure 70.10](#) illustrates the

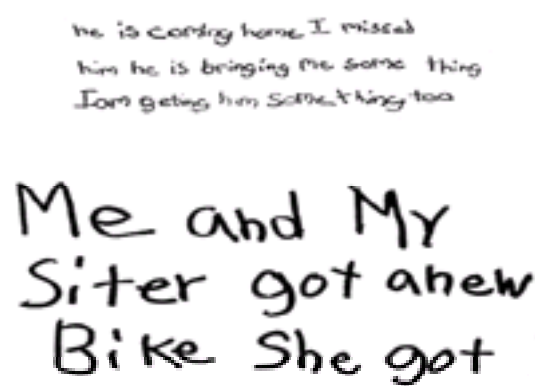
markedly different handwritings found in the school journal of an 8-year-old boy with DID. This right-handed child had several alternate personality states, some of similar age, some several years younger, and one a left-handed baby. [Figure 70.11](#) and [Figure 70.12](#) were found in the schoolwork of two school-age boys with DID who had been sexually abused or used in pornography. These kinds of writings and drawings provide some of the most objective, untainted evidence of the phenomenon of DID in children.



**Figure 70.8.** Four different signatures found in the schoolwork of a 12-year-old boy with dissociative identity disorder/multiple personality disorder.



**Figure 70.9.** Three different signatures found in the schoolwork of an 8-year-old boy with dissociative identity disorder/multiple personality disorder: the first, his usual signature; the second, the signature of a younger personality; the third, the signature of a personality named Mikey, hastily scribbled out and replaced.



**Figure 70.10.** Different penmanship and spelling ability from the school journal of an 8-year-old boy with dissociative identity disorder/multiple personality disorder.



**Figure 70.11.** Drawing found in a notebook of a sexually abused boy with dissociative identity disorder/multiple personality disorder.



**Figure 70.12.** Drawing found among the papers of an 8-year-old boy who was used in pornography.

## DIFFERENTIAL DIAGNOSIS

The signs, symptoms, and behaviors of children and adolescents with DID mimic a multiplicity of other, more commonly made diagnoses. These include attention deficit hyperactivity disorder, complex partial seizures, mood disorders, schizophrenic disorders, borderline states, and conduct disorder. [Table 70.3A](#), [Table 70.3B](#),



[Table 70.3C](#), [Table 70.3D](#), [Table 70.3E](#) and [Table 70.3F](#) lists many of the signs, symptoms, and behaviors common to DID and to the disorders for which DID is most frequently mistaken.

- 
1. Hearing voices
  2. Command hallucinations
  3. Hearing voices talking to each other
  4. Feeling controlled from outside
  5. Having a hierarchy of "imaginary" companions
  6. Apparent illogical thought processes
- 

**Table 70.3.A. Signs and Symptoms of DID/MPD That Cause It to Be Confused with Schizophrenia**

- 
1. Dreamlike states
  2. Blackouts/loss of time
  3. Impaired memory for acts, violent and otherwise
  4. Appearing as though in a trance
  5. Out-of-body experiences
- 

**Table 70.3.B. Signs and Symptoms of DID/MPD That Cause It to Be Confused with Psychomotor Seizures**

- 
1. Restlessness and difficulty concentrating
  2. Fluctuating academic abilities (e.g. math, spelling, language skills)
  3. Tantrums
  4. Problems following instructions
  5. Impaired memory for learned material
  6. Changes in maturity of handwriting
- 

**Table 70.3.C. Signs and Symptoms of DID/MPD That Cause It to Be Confused with Hyperactivity or Attention Deficit Disorder**

- 
1. Fights with peers and authority figures
  2. Episodic violence
  3. Apparent lying
  4. Apparent stealing
  5. Inappropriate sexual behaviors
  6. Running away
  7. Use of aliases
  8. Apparent lack of feelings or guilt when describing violent acts
- 

**Table 70.3.D. Signs and Symptoms of DID/MPD That Cause It to Be Confused with Conduct Disorder or Antisocial Personality**

- 
1. Mood swings
  2. Episodic rages
  3. Suicidal behaviors
  4. Long periods without sleep
  5. Inappropriate sexual behaviors/hypersexuality
  6. Grandiosity
  7. Self-mutilation
- 

**Table 70.3.E. Signs and Symptoms of DID/MPD That Cause It to Be Confused with Manic-Depressive Disorder**

- 
1. Psychosomatic pain
  2. Mood swings
  3. Binge eating
  4. Suicidal behaviors/self-mutilating behaviors
  5. Episodic rages
  6. Unstable interpersonal relationships/poor social adaptation/impulsivity
  7. Episodic psychotic symptoms
- 

DID/MPD: dissociative identity disorder/multiple personality disorder.

**Table 70.3.F. Signs and Symptoms of DID/MPD That Cause It to Be Confused with Borderline Personality Disorder**

We found that among a group of severely abused, dissociative children in residential treatment, the most common diagnosis they had received before admission was attention deficit hyperactivity disorder. This diagnosis tended to be made because of their frantic, hyperactive behaviors and their apparent distractibility. Unlike most other children with attention deficit hyperactivity disorder, however, these children periodically experienced auditory hallucinations, retreated into trancelike states, and were amnesic for discrete periods of time. Their responses to stimulant medication were also equivocal.

The trancelike states and memory lapses of dissociative children often cause them to be diagnosed as having seizure disorders. These episodes tend to occur in the presence of others, at times of stress, and usually are not accompanied by the loss of urine or feces. Unlike epileptic attacks, during these spells, dissociative children can hear and respond to the therapist's suggestions (e.g., "I want everyone to calm down. Everyone is safe. Now, when I count to 3 you will wake up and feel fine.").

Children with DID exhibit rapidly changing moods as they switch from one personality state to the next. The changes are sometimes so striking that they appear to be evidence of a bipolar mood disorder. Certain personality states "keep the memories" and are always sad. Others are jokesters. However, in bipolar illness, moods are usually of longer duration, and switches from sad to happy are not instantaneous. Unlike children with mood disorders, children with DID have memory lapses, trancelike episodes, and other signs and symptoms of dissociation. That is not to say bipolar mood disorders cannot be concomitant with DID.

Almost all children who suffer from DID experience auditory hallucinations, and, when these are revealed, a misdiagnosis of schizophrenia is often made. Children with DID, like their adult counterparts ([Kluft, 1987b](#); [Ross et al., 1990b](#)), have numerous first-rank symptoms (e.g., command hallucinations, voices arguing in their heads, feeling controlled from outside). Periodically, as they switch from one personality state to another, their thought processes may become confused, vague, and illogical. Blocking also occurs when one personality state tries to silence another. However, unlike children diagnosed with schizophrenia, children with DID usually relate to others warmly and with a normal range of affect. Their episodic amnesias, trances, and changes in voice and demeanor also distinguish them from children with the kinds of psychoses traditionally designated as schizophrenic.

Children who suffer from DID often behave in dangerous, aggressive ways. They run away. They take things, and they deny behaviors that they cannot recall. Sometimes they are cruel to peers and animals, and they appear cold and unfeeling. These behaviors often result in a misdiagnosis of conduct disorder. Again, the amnesias, trances, and auditory hallucinations of DID distinguish it from conduct disorder.

A word should be said regarding the diagnosis of borderline personality disorder. Whether this diagnosis is appropriate for children may be debatable, but the diagnosis tends to be made. The psychosomatic pain, the mood swings, the impulsivity, the unstable interpersonal relationships, the self-mutilation, and the episodic psychotic symptoms characteristic of adult patients who are called "borderline" are also characteristic of persons with DID. We suggest that many children and adults called "borderline" actually suffer from severe dissociative disorders, including DID. Therefore, the very consideration of such a diagnosis in a youngster should alert the clinician to the strong likelihood that he or she is dealing with a severely dissociative child.

Because of the tendency of DID to mimic other conditions, there is no single, easy way of making the diagnosis of DID in children. The multiplicity of symptoms, the fluctuating moods and behaviors, and the invariable history of amnesic states or well-documented behaviors for which the subject denies responsibility, as well as a history of having received several different diagnoses in the past, alert the clinician to the possibility of DID. Another clue to the diagnosis is that children with DID have often received different medications (e.g., stimulants for attentional problems, antipsychotic medications for hallucinations, antiepileptic medication for trances). However, the attentional problems of DID do not respond to stimulants, the hallucinations are untouched by antipsychotics, and the trances continue in spite of antiepileptic medications. A history of abuse cannot be relied on to make the diagnosis because the child and caretaker are often amnesic for the abuse or protective of each other, and they are reluctant to tell what they know.

This confusing conglomeration of neuropsychiatric phenomena should in and of itself indicate to the clinician the need for the kind of diagnostic evaluation described.

## TREATMENT

Clinicians who have treated adults and children suffering from DID have reported that progress toward integration is easier and faster with children than with adults ([Hornstein and Tyson, 1991](#); [Kluft, 1996](#)). In children, alternate personality states tend to be less fixed and less invested in separateness. However, we have found that this greater flexibility or malleability in children is often counterbalanced by the problem that children are often still in the care of extremely disturbed families whose child-rearing practices engendered the disorder in the first place. In these cases, we have found that children are unable to make much, if any, progress until their parent's psychopathology is addressed. As [Hornstein and Tyson \(1991\)](#) suggest, when a child with DID fails to make progress or regresses to more seriously disturbed behaviors, it is likely that events in the home are reigniting feelings of endangerment. Often siblings and parent figures must be assessed and even engaged in treatment if the child is to feel secure at home. It is not unusual to discover that older siblings have continued to intimidate or abuse a child after parental abuse has subsided.

We have found improvement to be fastest in cases in which formerly severely abused children have been placed in excellent foster or adoptive homes or when the abuser has been removed from the home. When a child is in a secure home, we often include the parent or surrogate parent in therapy sessions. In so doing, we teach parents to recognize the phenomena characteristic of DID and to deal fairly and lovingly with the child in all of his or her personality states.

Once a diagnosis has been made and a therapeutic alliance is created, the tasks of learning about the memories and experiences of each personality state, working through trauma, and sharing knowledge among personality states are similar to those of working with adult patients. Care must be taken with children to show empathy and respect for each personality state while encouraging all aspects to work together. Usually the child's most problematic behaviors result from the actions of one or more aggressive protector personality states. We have found it useful to recognize the power of these angry entities, to interpret their original need to be strong to protect the child, and to suggest that their powers could better be used in new, helpful ways, such as in sports. One rather skinny 10-year-old boy with whom we used this strategy became his football team's most valuable player.

We have found that, over time, as children feel increasingly safe, and as the needs, fears, and functions of different personality states are addressed, their distinct characteristics begin to blur, and they become so similar to each other and to the child as to be almost indistinguishable. Unfortunately, we have also found that months of progress can be undone when a child who seems to have recovered is forced once again into the company of a former abuser.

### Role of Pharmacotherapy

Until recently, prevailing opinion was that dissociative disorders were not amenable to psychopharmacologic treatment. Indeed, such psychotic symptoms as auditory and visual hallucinations, delusions of being two or more separate individuals, and feelings of being controlled by outside forces that are typical of DID are not touched by neuroleptic medications. However, when DID is viewed as chronic, complex PTSD with episodic panic attacks, then medication can play a very important role in alleviating the kinds of posttraumatic intrusions (e.g., flashbacks), hyperarousal symptoms, and panic episodes that trigger switches into alternate personality states. The tricyclic drugs amitriptyline (Elavil) and imipramine (Tofranil) and the selective serotonin reuptake inhibitors fluoxetine (Prozac) and sertraline (Zoloft) have successfully controlled core PTSD symptoms in adults. Anticonvulsant agents such as carbamazepine (Tegretol) and divalproex (Depakote) also have shown promise



in reducing hyperarousal, flashbacks, and sleep disturbance in adults ( [Amaya-Jackson, 2000](#); [Brady et al., 2000](#); [Krystal et al., 1998](#)). Clinically, we have found that when panic attacks diminish, dissociative youngsters switch personalities less frequently, become calmer and less aggressive, think more clearly, and thus are able to make progress in psychotherapy. Of course, if the child is diagnosed with a comorbid psychiatric illness such as depression or bipolar mood disorder, it goes without saying that the child first must be stabilized pharmacologically before the problem of dissociation can be addressed.

In the coming years, as we gain a greater understanding of the neurobiology of dissociation, new pharmacologic approaches targeting specific neurotransmitter systems that mediate dissociative symptoms may be found. Already, [Chambers and colleagues \(1999\)](#) have proposed for further study drugs that limit glutamate release and enhance hyporesponsive g-aminobutyric acid-ergic and endogenous opiate systems.

## RESEARCH DIRECTIONS

The 1990s have seen a shift from the study of DID to the study of PTSD. Although PTSD research has done much to legitimize dissociation as a normal response to extreme physical and psychological stress, such research has been unable to shed much light on the development of DID. Clearly, DID in children continues to provide fertile ground for psychodynamic and psychophysiological exploration. The autonomic, electroencephalographic, and immunologic differences among alternate personality states make the disorder especially useful for the study of mind-body interactions. The finding that it is common for several members of the same family to suffer from the disorder makes it a promising research area for the study of the intergenerational transmission of symptoms and behaviors. The relationship of early, ongoing trauma to the development of DID makes the illness especially relevant to studies of child abuse and of normal and pathologic child development. The timing, nature, and duration of abusive experiences sufficient to engender the phenomenon remain to be understood. Above a certain age, are human beings invulnerable to the disorder? Will the disorder always respond to appropriate therapy, or, after a certain period, is integration impossible? Will the disorder ever resolve over time on its own if a traumatized, dissociative child is given proper nurturing? Longitudinal and cross-sectional studies of children and adolescents suffering from pathologic dissociation should begin to answer these kinds of questions.

From the point of view of child psychiatry and of the welfare of children, the most important questions must concern the early identification of the signs, symptoms, and behaviors associated with the development of DID. What are the features of an incipient dissociative disorder in infancy? What does the disorder look like in childhood? Adolescence? The answers to these kinds of questions will lead to a better understanding of the incidence, prevalence, and early manifestations of the disorder and, hence, will have implications for prevention as well as treatment.

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# 71 SLEEP DISTURBANCES AND DISORDERS

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From the day of a child's birth, his or her sleep patterns are a matter of primary concern to the parents, second only to feeding and excretion patterns. Because in the early weeks and months of life, feeding, excretion, and sleeping frequently alternate with periods of crying, often interrupting parental sleep cycles, it is only natural for parents to be concerned if their child's sleep is irregular or frequently interrupted or if the infant is slow to sleep throughout the night. Parental concern about sleeping habits is established very early in a child's life. Thus, it is logical that sleep disturbances and disorders will come to the attention of professionals working with children throughout the childhood years.

In the psychiatric assessment, diagnosis, and treatment of children and adolescents, a history of variations in the usual patterns of the sleep–wake cycle is often the key to a more complete understanding of the presenting disorder. Such alterations may be either transitory phenomena or manifestations of normal phases of development. However, they may also appear as signs or symptoms of specific sleep disorders or of certain other mental, emotional, and physical disorders of childhood. It is therefore essential to have a full understanding of sleep, sleep patterns, and all their various manifestations to diagnose and manage these disorders appropriately.

For our purposes, we define childhood as the period extending from birth to the completion of adolescence or approximately 18 years of age. Although sleep disturbances may be transitory and relatively benign, sleep disorders, in general, are characterized by greater severity, duration, and degree to which they significantly disrupt or interfere with the child's daily functioning. Thus, the disruptions of sleep that we cover are those of more than occasional duration and more than minimal severity. They include specific sleep disorders that often are related to emotional or physical alterations associated with specific phases of development.

## SLEEP–WAKE PATTERNS IN CHILDREN

### Development of Sleep–Wakefulness Cycle

The newborn infant alternates between sleep and wakefulness every 3 or 4 hours, with awakenings usually related to hunger. The periods of wakefulness during the day gradually lengthen, the number of naps dwindles, and nighttime sleep periods grow longer. A definite diurnal cycle is established at 5 weeks in the majority of infants ([Kleitman, 1963](#)). Sleeping through the night, or “settling,” is found in 70% of infants by 3 months of age ([Moore and Ucko, 1957](#)). However, it takes about 5 years from birth for a child to develop the single night–day cycle that is retained throughout the balance of childhood and adulthood.

Newborn children sleep about 16 hours a day, and, by the 16th week of life, an extended, uninterrupted period of nocturnal sleep may reach 8.5 hours ([Kleitman and Englemann, 1953](#); [Parmelee et al., 1964](#)). Sleep requirements diminish to 12 hours plus a 1-hour nap at 2 to 3 years of age; in studies of normal preschool children, duration of sleep at 2 years was about 10 hours ([Kohler et al., 1968](#)). Requirements for sleep further decrease to about 9 hours at 8 to 12 years of age ([Ross et al., 1968](#)), whereas it appears that adolescents probably need more sleep than prepubertally. Nocturnal sleep time stabilizes at about 8 hours in young adulthood and remains there through midlife ([Feinberg and Carlson, 1968](#)).

The development of the diurnal cycle is most likely related to maturation of the central nervous system (CNS) because the ability to sustain prolonged periods of sleep and wakefulness appears to depend on an increasingly higher level of organization of the CNS. Infants who have suffered some type of prenatal injury or trauma such as anoxia fail to develop until a later age than normal the ability to sustain sleep without interruption ([Preston, 1945](#)).

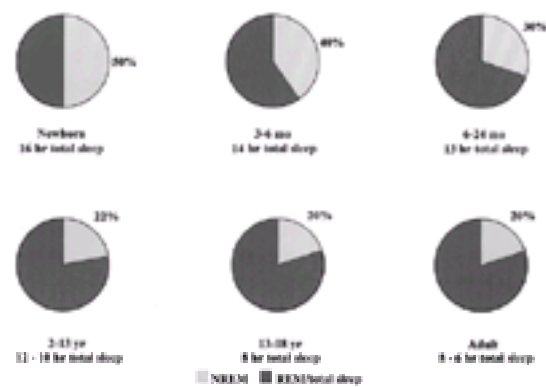
Learning and conditioning are also involved, in terms of the child's response to the family's patterns of sleep–wake behavior ([Anders and Weinstein, 1972](#); [Parmelee et al., 1964](#)). From a practical standpoint, the sleep of the infant gradually becomes similar to that of the parents and is therefore less disturbing to parents; as this occurs, the infant is able to sleep for more prolonged periods and predominantly at night ([Kleitman, 1963](#)).

### Sleep Stage Patterns

Early sleep laboratory studies by [Aserinsky and Kleitman \(1953\)](#) focused on the assessment of sleep in children, first with regard to slow eye movements and, later on, rapid eye movement (REM) sleep. Subsequently, [Rechtschaffen and Kales \(1968\)](#) published the first manual standardizing terminology, techniques, and scoring systems of adult stages of sleep. This was followed by a similar manual that standardized terminology, techniques, and criteria in newborn infants ([Anders et al., 1971](#)).

Three basic sleep states occur in infants: active-REM sleep and quiet–non-REM (NREM) sleep—which are considered precursors of REM and NREM sleep—and indeterminate sleep ([Anders et al., 1971](#)). At term, the approximate percentages of the sleep phases are as follows: active-REM sleep, 50%; quiet-NREM sleep, 35%; and indeterminate sleep, 15% ([Parmelee et al., 1967](#)). In the infant, frequent sucking movements are observed during REM sleep, as well as fine twitches, grimaces, tremors, and smiles. Indeterminate sleep, which is poorly organized, is derived from the concept of transitional sleep. It is considered an immature state that is characteristic of premature infants but is also present in full-term infants and infants with various abnormalities ([Anders et al., 1971](#)).

The proportion of the various sleep phases in the newborn also changes considerably within a short period. The percentage of REM sleep decreases progressively after birth to a level of 20% to 25% in childhood and continues a more gradual decline in the adult years ([Feinberg and Carlson, 1968](#); [Roffwarg et al., 1966](#)). Because premature and full-term infants spend approximately two-thirds of the day asleep, the total REM time for this group is considerably higher than the total REM time for any other age group, in whom the total sleep time is invariably less. By about 3 months of age, the electroencephalographic (EEG) patterns of quiet sleep can be divided into various stages of NREM sleep. Combined stages 3 and 4 sleep (slow-wave sleep) are longest in childhood, about 20% to 30% of total sleep time, decreasing to 10% to 20% in young adults ([Feinberg and Carlson, 1968](#); [Roffwarg et al., 1966](#)) ([Fig. 71.1](#)).



**Figure 71.1.** Changing sleep structure and duration with age. (Data from Roffwarg H, Muzio I, Dement W: Ontogenetic development of the human sleep-dream cycle. *Science* 152:604–619, 1966.)

This age-related shift in the proportion of NREM to REM sleep is thought to indicate maturation of the CNS. REM sleep, which is characterized by irregular respiration and heart rate, is considered an archisleep or primitive sleep. NREM sleep, a more highly controlled state, with more regular respiration and heart rate, is regulated by higher cerebral centers, including the cortex.

The temporal sequences of REM and NREM sleep within a sleep cycle also change with age. Newborn infants frequently initiate sleep with a period of REM sleep and have relatively equal amounts of REM sleep in the first and second halves of a sleep period (Roffwarg et al., 1966). In contrast, adults begin sleep with about 70 to 100 minutes of NREM sleep and have a much larger proportion of REM sleep in the last third of the night's sleep (Feinberg and Carlson, 1968).

## CLINICAL SLEEP EVALUATION

### History

Before any formal sleep laboratory evaluation is conducted, an adequate sleep–wakefulness history is essential for the accurate evaluation and proper management of any sleep disorder of infancy, childhood, or adolescence (A. Kales et al., 1980d). A description of the disorder and its frequency, severity, development, and associated circumstances should be obtained, with a complete, 24-hour profile of active and quiescent periods. The history should include a description of the chronologic development of sleep patterns and any reports of difficulties with sleep as well as difficulties with feeding and toilet training. The pattern and level of achievement of all developmental milestones should be noted. The history also should ascertain the presence or absence of prenatal infections or illnesses of the mother, prematurity, trauma in relation to the birth process, or history of fetal distress. In addition, the occurrence of infections or injuries during infancy and childhood must be determined.

Further, in assessment of infants for sleep problems, addressing maternal limit setting, anger, and parenting doubts is essential (Morrell, 1999). Temperamentally intense, negative emotions may be significantly associated with sleep disturbances (Owens–Stively et al., 1997). Maternal sleep patterns, distress, and depression both before and after pregnancy may also be factors (Armstrong et al., 1998).

The child's psychological adjustment should be thoroughly evaluated (A. Kales et al., 1980d), including assessment of the maturity, competency, and emotional health of the parents and other caretakers. Changes in residence or sleeping environment are noted; for example, sleep disturbance may appear among children who are moved frequently from one home to another.

### Differential Diagnosis

In young children, it is important to differentiate between sleep difficulty that is secondary to immaturity of the CNS or other organic factors and that caused by a disturbance in the parent–child relationship. If this distinction is not made, the parent may assume responsibility for the disturbance even when he or she is not at fault and, through guilt and anxiety, superimpose psychological difficulties on the original problem.

It is also important to determine whether the sleep problem has been present since birth or early infancy or whether it appeared after the normal development of sleep patterns and behavior. The infant or young child is more likely to present with a sleep disturbance that has existed since birth or early infancy. In these cases, the physician is primarily concerned with the possibility of physiologic immaturity or organic conditions, such as prematurity, birth trauma, infection, thyroid dysfunction, or other physical illness. After such developmental and organic factors have been ruled out, the mother–child relationship must be evaluated to determine whether it is the primary factor causing the disorder or whether it is contributing to another, underlying disorder. It is important to differentiate between sleep difficulty caused by separation anxiety and chronic resistance to going to sleep (Ferber, 1989; Spock, 1957). Sleep disturbances associated with separation anxiety usually occur in the second or third year and are associated with the symptom of fear, whereas marked resistance to going to sleep usually occurs in the first year.

In children more than 3 years of age, it is more likely that the sleep problem has been acquired. Here, the physician assesses psychological, environmental, or organic causes, as well as developmental milestones that can account for the temporal development of the sleep disturbance. In children of this age and older, psychiatric disorders that may have a sleep problem as a symptom must be considered.

In the 3- to 5-year-old child with sleep difficulties, the factors mentioned earlier are evaluated, as well as the child's relationship with peers and his or her adjustment to nursery school or other preschool activities. In the child who is more than 5 years old, general daytime behavior and adjustment are thoroughly assessed, including evaluations of the child's adjustment at school, relationship with peers, and general social adaptation.

In a community-based survey of school children and their parents (Blader et al., 1997), bedtime resistance predominated. This was associated with falling asleep away from bed and inconsistent bedtime. Insomnia, night waking, and anxiety were also noted. Decreased verbal creativity and abstract thinking occur after decreased sleep (Randazzo et al., 1998), as does daytime sleepiness (Ishihara, 1999). Too early school starting time (Epstein et al., 1998) and bedtime television watching (Owens et al., 1999) also affect daytime behavior. Treatment lies in child–parent training (Mindell, 1999).

Adolescents in our society are currently obtaining less sleep than at previous times in history, as well as less sleep than in other cultures. Carskadon and colleagues (1998) at Brown University in Providence, Rhode Island, performed extensive research documenting the degree of the problem, as well as its various causes. Increased need for sleep, circadian changes associated with a pattern of decreasing total sleep time and delaying the timing of sleep secondary to school schedules, extracurricular activities, part-time employment, and decreased parental involvement in setting bedtimes are the main underpinnings of adolescents' sleepiness. Thus, a detailed assessment of an adolescent sleep schedule and daytime routine and activities during the weekdays and weekends is of great importance.

Furthermore, when an adolescent presents with a sleep difficulty, a thorough assessment of the patient's adjustment in school, peer relationships, and relationships with parents or parental figures is indicated, especially in terms of issues of dependence and independence, response to authority, and need to rebel. The physician also determines whether there is a history of delinquency or other types of acting-out behavior. Psychiatric disorders, such as depression, must also be ruled out (Wolfson and Carskadon, 1998).

## SLEEP DISORDERS

Although the categories and criteria set forth in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) (American Psychiatric Association, 1994) are more appropriate for classifying sleep disorders in adults than in children, in this chapter we follow the DSM-IV nomenclature and nosology as follows. Sleep disorders may be divided into four major groups: the *dyssomnias*, which show disturbance in the duration, type, and pattern of sleep; the *parasomnias*, which consist of experiences a child may have during sleep that are directly related to the sleep process and sleep stages; sleep disturbances that frequently accompany the common major child *psychiatric disorders* seen in clinical practice; and sleep disorders resulting from *general medical conditions or substance abuse*



(Anders and Eiben, 1997).

## Dyssomnias

### PRIMARY INSOMNIA AND CIRCADIAN RHYTHM DISORDERS

Unlike adults or adolescents, younger children and, of course, infants are less likely than their parents to complain about their sleeplessness. *Insomnia* in children may best be defined as observable prolonged or abnormal sleeplessness. The DSM-IV characterizes insomnia as a problem in initiating sleep, maintaining it, or not feeling rested after an apparently normal nocturnal sleep period (nonrestorative sleep). Insomnia in children as well as adults may be related to other mental disorders, which we discuss later in this chapter.

In one epidemiologic study, 14% of 3-year-old children were found to have problems with persistent waking at night, and 12% had difficulty in falling asleep. In 8-year-old children, 12% had trouble falling asleep, and 3% were troubled by waking at night ( Richman et al., 1982).

Sleep difficulty in children tends to have causes that are different from those in adults ( Ferber, 1989; J. Kales et al., 1984). The problem, even in younger children who are less than 2 years of age, does not seem to be neurodevelopmental ( Anders and Keener, 1985; Anders et al., 1980). Rather, it seems to be related more to interactional patterns between parents and their children and to parental reaction to short-term transitory behavior problems that have become more chronic. In a study of 23 infants 6 months of age, Keener et al. (1988) found that differences in waking at night varied according to whether the infant cried for the parents on awakening or simply self-soothed and went back to sleep. Infants fed to sleep were more likely to cry on awakening and to seek parental attention. The more a parent becomes overinvolved in the child's sleep, the more likely it is that a sleep problem, including insomnia, will develop and persist ( Ferber, 1989). Anders et al. (1992), using all-night time-lapse video recordings of 21 infants at 3 weeks, 3 months, and 8 months of age, found that, by 3 months of age, infants who were awake when put in their cribs at bedtime were more likely to return to sleep on their own when they awoke later in the night than those who were put in their cribs asleep. At 8 months of age, "problem sleepers" were all boys who were put in their cribs when they were already asleep. No other sleep factors were predictors. In a similar study, Minde et al. (1993) found, in 12- to 36-month-old children, that good sleepers woke as frequently as poor sleepers, but the latter had more behavior problems, more difficult temperament, and adverse medical histories. Comparing the mothers of 20 sleep-disordered toddlers with 21 mothers of toddlers without sleep disorders, Benoit et al. (1992) found that 100% of the former mothers were insecure with respect to attachment, as compared with only 57% of the controls. Changing patterns of feeding also may frequently affect sleep patterns in infants ( Ferber, 1989). In contrast, preschool and school-age children have few sleep difficulties, except as they relate to lack of limit setting or bedtime struggles, in which the child wishes to remain awake and active in play or with parents. It is recommended that excessive napping or sleeping during the day be restricted in children, particularly those of school age, to prevent interference with the normal length or onset of nocturnal sleep ( Ferber, 1989).

Fears of nightmares may also occur, giving rise to insomnia problems (J. Kales et al., 1984). In a comprehensive review, King et al. (1992) addresses the issue of children's nighttime fears, their variation according to level of cognitive development, their causes, and successful treatment approaches without medication. Infants fear direct environmental stimuli; preschoolers, imaginary creatures; and school children, representations of real sources of anxiety in their environment, such as the possibility of health problems, physical injury, or school failure. With regard to treatment of night fears, *emotive imagery*, in which children are trained to imagine a superhero who helps them to be brave in the face of the fears, has been most successful. Parents have been successfully taught to help their own children with this method, strengthened by positive reinforcement. In a related study, Connell et al. (1987) reported on six children, three boys and three girls, between the ages of 10 and 12 years who developed severe phobic anxiety at bedtime subsequent to exposure to the death of a relative or close friend. Psychotherapy with them focused on their confused concepts of the relationship between sleep and death. Pollock (1994), in a 5-year prospective study, also reported that emotional distress, as well as chronic medical conditions, commonly caused night waking at age 5, but less frequently by age 10.

Because insomnia in children seems to be more of a psychosocial problem based on the relationship between parent and child, treatment is best addressed primarily through psychological or behavioral means. It appears that sleeping alone through the night without parental intervention is a learned process. In helping the parents to facilitate this process, a few principles are critical in a successful behavioral intervention: first is reducing the amount of parental involvement at sleep onset, second is consistency; and last is the need to deal effectively with child or parental anxiety ( Dahl, 1998). Richman et al. (1985) found that behavioral methods of treatment improved bedtime disturbances and night waking (childhood equivalents of adult insomnia) in 35 children aged 1 to 5 years, whereas treatment with medication (trimeprazine) of 22 children with severe waking problems showed no permanent effect on sleep patterns ( Richman, 1985).

Minde et al. (1994), reporting on a study of the effects on daytime mother-child interactions of a treatment program for severely sleep-disturbed children aged 12 to 36 months, found that relatively simple interventions, such as having the father put the child to bed instead of the mother, helped to improve the mother-child relationship, as well as alleviating the sleep problem.

Adolescents seem to have an increased need to sleep during the daytime. In one study, ( Price et al., 1978), 13% of adolescents were classified as having difficulty sleeping through the night on a regular basis. Similar findings have been reported for an adolescent psychiatric outpatient population ( Monroe and Marks, 1977). Morrison et al. (1992), reporting on 943 adolescents from the general population, found that 10% reported difficulty falling asleep. In addition, whether male or female, those who had sleep problems showed more DSM-III disorders, namely anxiety, depression, inattention, and conduct disorder. Carskadon (1990) reported accident proneness, daytime sleepiness, and behavior and mood problems in adolescents who had insufficient sleep. Based on a large survey of more than 1,000 metropolitan households (Bixler et al., 1979), only 11% of adults with chronic insomnia reported the onset of their disorder in adolescence (between the ages of 11 and 20 years), a finding suggesting that chronic insomnia is relatively uncommon in this age group. However, because insomnia in the adult population is frequently associated with depressive or dysthymic disorders, appropriate diagnosis and clinical treatment of those conditions in adolescents with antidepressant medication and psychotherapy may serve to relieve symptoms of insomnia (see later).

In adolescents, insomnia unrelated to other primary psychiatric disorders may also appear as *delayed sleep phase syndrome* (DSPS) (Carskadon et al., 1998; Ferber and Boyle, 1983; Thorpy et al., 1988). DSPS consists of a chronic involuntary delay in falling asleep at night when desired and an inability to awaken at an appropriately scheduled time in the morning. In the sleep disorders clinic population, it is often associated with major depression and is more resistant to treatment than other sleep disorders (Regestein and Monk, 1995). Multiple and varied treatments of DSPS in adolescents include behavioral approaches ( Cashman and McCann, 1988), sleep phase advance techniques ( Thorpy et al., 1988), light therapy, and antidepressants. Moreover, high doses of methylcobalamin (vitamin B<sub>12</sub>) (Ohta et al., 1991) or melatonin (Okawa et al., 1998b) have been reported to foster the establishment of an appropriate 24-hour sleep pattern, resulting in improved school performance, mood, and behavior.

More recently, in case reports or noncontrolled studies, investigators have suggested that the hormone melatonin may be useful for the treatment of sleep-wake cycle disorders in children and adolescents ( Ellis et al., 1996; Jan et al., 1999; Okawa et al., 1998a). Although the potential usefulness of melatonin in circadian rhythm sleep disorders warrants further investigation ( Okawa et al., 1998a), melatonin appears to be of no use in persistent insomnia ( Ellis et al., 1996). In addition, dosage levels are uncertain, and long-term side effects are unknown.

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#### CASE ILLUSTRATION

A 14-year-old girl presented with the primary complaint of difficulty falling asleep at night; she remained awake 1 to 2 hours after going to bed, a finding suggesting delayed sleep phase syndrome. Psychotherapy revealed an obsessional preoccupation with the need for a full night's sleep. This disturbance eventually resolved in the course of psychotherapy without the use of medication, and she returned to her normal sleep pattern.

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### PRIMARY HYPERSOMNIA (EXCESSIVE DAYTIME SLEEPINESS)

*Hypersomnia*, or excessive daytime sleepiness, affects about five in 100 people in the general population, with a slight male predominance ( Bixler et al., 1979). In children, it tends to appear more often as they approach or enter adolescence. Carskadon and Dement (1987), Carskadon (1990), Carskadon et al. (1998), and Wolfson and Carskadon (1998) describe the evolution of daytime sleepiness in adolescents. In several studies of children over a decade who maintained a stable 10-hour sleep time, these investigators assessed whether the need for sleep actually gradually decreased as youngsters entered adolescence. The study indicated that sleep needs did not change significantly during adolescence. Total sleep appeared to remain at a stable level when the opportunity existed to have a continuous 10 hours of sleep. There were some differences between boys and girls. The only changes noted during the adolescent years were that the combined duration of

slow-wave sleep stages relative to other stages tended to decline.

Although primary or CNS hypersomnia is thought to be a relatively rare disorder, the diagnosis should be considered in children and adolescents who complain of excessive daytime sleepiness. In a large series published by [Roth \(1980\)](#), about 50% of patients with idiopathic hypersomnia had the onset of their symptoms before the age of 20 years. This disorder is characterized by excessive daytime sleepiness, prolonged and deep sleep, and morning sleep drunkenness or grogginess. Usually, it is diagnosed several years after the onset of the symptoms.

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#### CASE ILLUSTRATION

A 15-year-old male student was reported to be regularly falling asleep in class, including once when standing in line. His history was positive for major depressive disorder and chronic dysthymia, whereas in the sleep laboratory, he demonstrated a nighttime sleep latency of 25 minutes and rapid eye movement (REM) latency of 102 minutes. During the first of two naps, the patient demonstrated a sleep latency of 4 minutes and an REM latency of 53 minutes. During the second nap, the patient demonstrated a sleep latency of 0 minutes and an REM latency of 51 minutes. There was no evidence of sleep apnea. Treatment with modafinil, 100 mg every morning, resulted in remission of these symptoms attributed to primary (idiopathic) hypersomnia.

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#### KLEINE-LEVIN SYNDROME

A rarer form of periodic hypersomnia, usually evidenced in adolescents, is the *Kleine-Levin syndrome* ([Billiard, 1989](#)). This disorder is a periodic condition characterized by episodes of continuous daytime hypersomnolence associated with hyperphagia, irritability, aggression, sexual disinhibition, and mental disturbances, with a slight male predominance. The initial onset of this disorder is often related to an ill-defined flulike condition. Diffuse paroxysmal slowing is found in the EEG. Treatment of this disorder has included amphetamines and lithium carbonate, but a definitive cure has not been achieved.

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#### CASE ILLUSTRATION

A 16-year-old boy experienced the onset of hypersomnolence during holiday home visits from a residential school. These visits were characterized by excessive holiday food intake, followed by a 1- to 2-week period of hypersomnolence, and a drunkenlike condition in the daytime. Medical workup, which was negative for other abnormal findings, confirmed the diagnosis of Kleine-Levin syndrome. The onset had been related to a flulike illness approximately 1 year previously.

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#### NARCOLEPSY

*Narcolepsy* is a disorder consisting of excessive somnolence in the nonsleep or daytime hours ([Guilleminault et al., 1976](#); [A. Kales et al., 1982](#); [Roth, 1980](#)). The general population prevalence among both adults and children is less than one in 1,000 ([Roth, 1980](#)). The age of onset of narcolepsy tends to be late adolescence or young adulthood, although rarely its onset may be traced during the first decade of life.

Children with narcolepsy have excessive daytime sleepiness, with irresistible sleep attacks usually occurring in conjunction with one or more of three auxiliary symptoms: cataplexy, sleep paralysis, and hypnagogic hallucinations ([Guilleminault and Pelayo, 1998b](#); [Guilleminault et al., 1974](#); [Guilleminault et al., 1976](#); [A. Kales et al., 1982](#); [Roth, 1980](#)). The sleep attacks may last from a few seconds to 30 minutes and may be precipitated by sedentary, monotonous activity, such as watching television, reading, or sitting in class. Narcolepsy in children with hypersomnia, particularly in early stages, is not always accompanied by cataplexy, sleep paralysis, or hypnagogic hallucinations, so it may be underdiagnosed ([Kotagal, 1996](#)).

*Cataplexy*, present in about 60% to 80% of patients with narcolepsy ([Hishikawa and Kaneko, 1965](#); [Roth, 1980](#)), is a brief (lasting a few seconds to about 2 minutes), sudden, complete or partial loss of muscle control that, in its severe form, may cause the person to collapse while remaining conscious. This loss of muscle tone, which may include facial and neck muscles and slurring of speech ([Anic-Labat et al., 1999](#)), frequently occurs in relation to strong emotional experiences such as laughter, surprise, or anger. In patients who have cataplexy, REM periods usually occur at or shortly after sleep onset ([Hishikawa and Kaneko, 1965](#); [A. Kales et al., 1982](#); [Roth, 1980](#)), rather than preceded by the 70 to 90 minutes of non-REM sleep seen in normal controls.

With *sleep paralysis*, patients have a temporary loss of muscle tone and a resulting inability to move, most often occurring on awakening. *Hypnagogic hallucinations* are vivid hallucinatory perceptions (usually visual or auditory) that occur even in normal children at sleep onset.

Sleep attacks and certain manifestations of the auxiliary symptoms of narcolepsy appear to be closely related to the neurophysiologic mechanisms of REM sleep ([Broughton, 1971](#); [Hishikawa and Kaneko, 1965](#); [Rechtschaffen and Dement, 1969](#)). Two lower brainstem centers discharge together during REM sleep. The nucleus reticularis pontis caudalis stimulates an ascending activating system and produces EEG patterns of arousal, REM bursts, autonomic irregularity, myoclonic twitches, and pontogeniculoccipital spikes. Conversely, the locus ceruleus triggers a descending inhibitory system and causes areflexia and loss of muscle tone ([Broughton, 1971](#)).

Sleep attacks may involve a dysfunction of the activating part of the reticular formation in addition to a dysfunction of REM mechanisms ([Broughton, 1971](#); [Roth, 1980](#)). The intense muscle atonia of cataplexy and sleep paralysis is probably more directly related to stimulation of the descending inhibitory pathway, whereas hypnagogic hallucinations can be viewed as dreamlike experiences accompanying REM sleep.

Hereditary factors are clearly involved in the development of narcolepsy; 10% to 50% of patients have a first-degree relative with either narcolepsy or idiopathic hypersomnia ([Broughton, 1971](#); [Hishikawa and Kaneko, 1965](#); [Guilleminault et al., 1974](#); [A. Kales et al., 1982](#); [Kessler et al., 1974](#); [Nevsimalova-Bruhova and Roth, 1972](#); [Rechtschaffen and Dement, 1969](#)). Genetic studies have revealed human leukocyte antigen markers in most narcoleptic patients and in almost 100% of patients with cataplexy ([Langdon et al., 1984](#); [Mignot, 1998](#)). The pattern must be multifactorial, however, because monozygotic twins have a high rate of discordance ([Honda and Matsuki, 1990](#)). In addition, both clinical and polygraphic characteristics of the disorder, such as disturbed nocturnal sleep and abnormal timing of REM sleep, suggest the presence of a chronobiological abnormality ([Leckman and Gershon, 1976](#)). More recent studies have shown that somnolence-inducing cytokines are elevated in narcolepsy ([Vgontzas et al., 1997](#)), and canine narcolepsy is caused by a mutation in the orexin receptor 2 gene ([Lin et al., 1999](#)). Narcolepsy is unrelated to epilepsy; their respective symptoms are easily distinguishable ([Guilleminault et al., 1976](#); [A. Kales et al., 1982](#); [Roth, 1980](#)).

Attention deficit disorder (ADD) in children may include symptoms of inattention, impairment of learning, and hyperactivity, all of which may be symptoms of childhood disorders of excessive daytime sleepiness, including narcolepsy. The presence of daytime sleepiness, sleep attacks, or even cataplexy would suggest the presence of narcolepsy.

Treatment of narcolepsy has consisted primarily of therapeutic naps and the use of stimulant substances, such as methylphenidate or pemoline ([A. Kales et al., 1987b](#)) or more recently modafinil ([Billiard et al., 1991](#); [Boivin et al., 1993](#)). Tricyclic medications and serotonin reuptake inhibitors may also be considered when cataplexy is a problem, although the former are used with caution in children because of their anticholinergic side effects and association with cardiac conduction defects.

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#### CASE ILLUSTRATION

A 17-year-old boy evaluated in a sleep disorders clinic complained, "I've been sleepy and tired since seventh grade." He reported feeling sleepy and fatigued during the day, falling asleep in classes, and taking a 3- to 4-hour nap at home after school. After getting a driver's license, he could drive no longer than an hour before stopping because of irresistible sleepiness. During the past 12 months, he had also been experiencing sudden buckling of the knees, dropping of the jaw, slurred speech, and, on one occasion in the car with his father, sudden complete paralysis and inability to talk. In the sleep laboratory, he presented no evidence of sleep apnea but, on 3 successive nights, demonstrated decreased sleep latencies and sleep-onset REM. During his nap studies, sleep latency was 3 minutes and 5 minutes and REM latency 30 minutes and 8 minutes, respectively. He was diagnosed as having narcolepsy with cataplexy. The treatment recommended included methylphenidate a stimulant medication for sleepiness, imipramine for cataplexy, and appropriate driving precautions.



## BREATHING-RELATED SLEEP DISORDER (SLEEP APNEA)

*Sleep-disordered breathing* and *sleep apnea* based on polysomnographic studies have been reported in infants as well as children ( [Guilleminault and Pelayo, 1998a](#); [McNamara and Sullivan, 1996](#); [McNamara and Sullivan, 1998](#)). These disorders are serious and may produce symptoms ranging from failure to thrive, behavioral problems, and enuresis to disruptions in sleep (increased wake and stage 1 sleep and reduced REM sleep).

Breathing-related sleep disorder (sleep apnea) is a condition characterized by intermittent periods of cessation of breathing during sleep ( [Block, 1980](#); [Guilleminault, 1987](#); [Guilleminault and Dement, 1978](#); [A. Kales et al., 1985](#); [A. Kales et al., 1987b](#); [Lugaresi et al., 1978](#); [Sadoul and Lugaresi, 1972](#)). These periods last more than 10 seconds and are often followed by loud and prolonged snoring sounds. Two general types of sleep apnea are described, obstructive and central. The former is characterized by peripheral obstruction of the airway, whereas the latter is characterized by the absence of respiratory drive, most likely secondary to CNS dysfunction. Some cases of sudden infant death syndrome have been thought to be caused by central sleep apnea ( [Guilleminault, 1987](#); [Steinschneider, 1975](#)).

Significant sleep and breathing disorders (sleep apneas) have been reported in 0.7% of 4- to 5-year-old children ( [Ali et al., 1993](#)). Habitual snoring (12.1%) has been associated with daytime sleepiness, restless sleep, and hyperactivity. Obstructive sleep apnea–hypoventilation syndrome is described as negatively affecting school performance in young children ( [Gozal, 1998](#); [Owens et al., 1998](#); [Rosen, 1999](#)).

In young children, the signs and symptoms of breathing-related sleep disorders, which primarily consist of the obstructive sleep apnea syndrome, are more subtle than those in adults, and the diagnosis is more difficult to establish. In children younger than 5 years old, nighttime manifestations, such as observed apnea, labored breathing, or restless sleep, are more often the presenting symptoms. In older children, the presenting complaint is often the presence of daytime symptoms, such as sleepiness, attention and learning difficulties, and morning headaches. Other signs and symptoms associated with breathing-related sleep disorders in children include brief arousals, nocturnal enuresis, and daytime mouth breathing.

In children, obstructive apnea may frequently be seen secondary to enlarged tonsils ( [Guilleminault, 1987](#)). Other less frequent anatomic causes include mandibular malformation, micrognathia, acromegaly, and glottal web ( [A. Kales et al., 1987b](#)). A strong positive correlation exists between sleep apnea and obesity and between sleep apnea and certain endocrinopathies, such as hypothyroidism ( [Guilleminault, 1987](#)). In addition, children with Down's syndrome are at risk of obstructive sleep apnea, with a prevalence of 31% to 72% ( [Marcus and Loughlin, 1996](#)).

When sleep apnea syndrome is suspected, parents should be questioned about whether the child exhibits loud snoring, heavy breathing, or interrupted nocturnal breathing ( [Block, 1980](#); [Guilleminault, 1987](#); [Guilleminault and Dement, 1978](#); [A. Kales et al., 1987b](#); [Lugaresi et al., 1978](#); [Sadoul and Lugaresi, 1972](#)). A thorough physical examination must be undertaken, including the careful inspection of the upper airway, neck, and jaw. In children, polysomnography in association with direct observation and recording of the child's breathing pattern is useful in confirming the diagnosis. There are no established criteria for obstructive sleep apnea syndrome in children, but common sleep laboratory findings include partial obstructive hypopneas, snoring, and labored breathing, with its associated brief semiarousals or awakenings.

Severity, as determined by the sleep laboratory indices and level of daytime functioning, should be considered when deciding on the treatment for a child with sleep apnea ( [Block, 1980](#); [Guilleminault and Dement, 1978](#); [A. Kales et al., 1987b](#); [Lugaresi et al., 1978](#); [Sadoul and Lugaresi, 1972](#)). When the origin is secondary to tonsillar or adenoidal hypertrophy, tonsillectomy and adenoidectomy are often curative. In other cases, the treatment options are not always as convenient or successful. Weight reduction can be helpful in some patients in whom the condition is secondary to obesity ( [Lugaresi et al., 1978](#); [Sadoul and Lugaresi, 1972](#)). Weight control in children with obstructive sleep apnea and morbid obesity may provide a partial cure, but adenoidectomy and tonsillectomy may result in marked improvement in the presence of retained obesity ( [Kudoh and Sanai, 1996](#)). Continuous positive airway pressure is used in those children who do not exhibit any adenotonsillar hypertrophy or who do not respond to adenotonsillectomy ( [Marcus and Loughlin, 1996](#)). In the past, in some cases, tracheostomy was used to relieve the symptoms and possible long-term adverse effects of severe sleep apnea ( [Handford et al., 1984](#)).

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### CASE ILLUSTRATION

An 8-year-old boy with classical hemophilia A (factor VIII deficiency <1%) was referred to a sleep research and treatment center after a period of stuporous hypersomnia. His history revealed daytime sleep attacks and dull and uncoordinated behavior on arising in the morning. He was also reported to be falling asleep frequently in the classroom and experiencing declining academic ability. Physical examination revealed hypertrophy of the tonsils and cardiac enlargement with evidence of left-sided heart failure. Because of the potential seriousness of the condition, sleep laboratory studies were conducted. These revealed the presence of severe obstructive sleep apnea, with more than 500 events recorded in an 8-hour period, each lasting 20 to 40 seconds. During apneic periods, minimum oxygen saturation was 50% or less. Because the patient did not show improvement after tonsillectomy, a permanent tracheostomy was undertaken, to place an open tracheostomy tube during sleeping ( [Handford et al., 1984](#)). Five-year follow-up revealed that significant improvement resulted from all initial measures. At age 18, however, death occurred secondary to pulmonary complications of human immunodeficiency virus (HIV) infection.

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## Parasomnias

### SLEEPWALKING AND SLEEP TERROR DISORDERS

*Sleepwalking* (somnambulism) and *sleep terrors* (formerly called night terrors) are much more common in children than in adults ( [Jacobson et al., 1969](#); [A. Kales et al., 1980c](#); [A. Kales et al., 1987a](#); [J. Kales et al., 1980](#)). About 15% of children are estimated to have had at least one sleepwalking episode ( [Jacobson et al., 1969](#)), compared with 2.5% of the general adult population ( [Bixler et al., 1979](#)). Sleep terrors are reported as a problem for 1% to 3% of all children and are considered to be disorders of impaired arousal ( [Broughton, 1968](#)). These conditions usually begin in late childhood or early adolescence and generally cease by late adolescence ( [A. Kales et al., 1980b](#); [A. Kales et al., 1987a](#); [J. Kales et al., 1980](#)). In a sample of 50 sleepwalkers, only three began sleepwalking after age 18 ( [A. Kales et al., 1980a](#)).

A sleepwalking episode usually lasts less than 20 minutes. During this period, patients generally have blank expressions, behave as though they are indifferent to the environment, and show low levels of awareness and reactivity. After awakening, they usually do not remember the events that occurred during the episode ( [Broughton, 1968](#); [Jacobson et al., 1969](#)).

The sleep terror episode has the additional and often dramatic characteristics of extreme vocalization and motility, excessive autonomic discharge, and intense terror or panic ( [Broughton, 1968](#); [J. Kales et al., 1980](#)). These episodes generally occur within a more restricted range than do sleepwalking episodes; for example, fewer patients with sleep terrors report leaving their homes during the event ( [J. Kales et al., 1980](#)). Sleepwalking and sleep terrors are believed to share a common neurophysiologic substrate and thus share many clinical and physiologic similarities ( [A. Kales et al., 1987a](#)), as summarized in [Table 71.1](#).

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Episodes early in night  
Confusion on waking and minimal recall of event  
High risk of injury  
Family history of sleepwalking or night terrors usually present  
Onset usually in childhood or early adolescence  
Most often outgrown by late adolescence  
Psychopathology suspected if onset is in adulthood

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Adapted from [Vignatilis AN, Kales A. Sleep and its disorders. Annu Rev Med 50:397-400, 1999](#), with permission.

**Table 71.1. Differentiation of Night Terrors and Nightmares**

Genetic ( [A. Kales et al., 1980a](#)), developmental ( [A. Kales et al., 1980c](#)), organic ( [J. Kales et al., 1979](#)), and psychological factors ( [A. Kales et al., 1980b](#); [A. Kales et](#)

al., 1987a; J. Kales et al., 1980) have been identified as causes of both sleepwalking and sleep terrors. In a study of a large sample of patients, a family history of either or both conditions was present in 80% of probands with sleepwalking episodes and in 96% of those with sleep terrors ( J. Kales et al., 1979). The frequent onset of these disorders in childhood, with termination by late adolescence, strongly suggests the role of maturational factors ( A. Kales et al., 1980c; A. Kales et al., 1987a; J. Kales et al., 1980). In addition, in a group of children with somnambulism, an “immaturity factor” (sudden, rhythmic, high-voltage, slow activity) has been identified in the EEG pattern during sleep (Jacobson et al., 1969). In a few cases, febrile episodes have been noted to precede the onset of somnambulism (J. Kales et al., 1979).

Psychological factors are not usually prominent in children who develop these disorders early in childhood ( A. Kales et al., 1980c; A. Kales et al., 1987a; J. Kales et al., 1980). However, sleep terrors, as well as other sleep disturbances, such as inability to fall asleep and nightmares, have been described as symptoms of posttraumatic stress disorder in children (Benedek, 1985). Children who develop these disorders and continue to have them as adults are much more likely to show significant psychopathology when they are evaluated as adults. Sleep terrors need to be differentiated from temporal lobe epilepsy, which rarely occurs only at night (J. Kales et al., 1980). EEG studies confirm that sleepwalking and sleep terrors are rarely manifestations of seizure activity ( Soldatos et al., 1980; Tassinari et al., 1972).

The most important consideration in managing children and adolescents with sleepwalking or night terror episodes is protection from injury ( A. Kales et al., 1980c; A. Kales et al., 1987a; J. Kales et al., 1980). Attempts to interrupt sleepwalking and night terror episodes should be avoided because intervention often confuses and frightens the child even more. Specific safety precautions may include special latches for outside doors and bedroom windows and sleeping accommodations on the ground floor. An important aspect of management consists of counseling and reassuring the parents that children with these disorders will usually outgrow the conditions by late adolescence, if not sooner.

With regard to more specific treatment, Lask (1988) reported the successful elimination of sleep terrors and sleepwalking by behavioral alteration of the sleep pattern, that is, charting the approximate time of the episodes each night and then waking the child 10 to 15 minutes before (when autonomic arousal is observed). Relief of the disorders occurred within a week, both initially and when there was recurrence. Psychotropic drugs (e.g., diazepam, imipramine) for controlling sleepwalking and night terrors should be used cautiously in children and only if the disorders are frequent, intense, and very disruptive ( Lask, 1988).

#### CASE ILLUSTRATION

A 16-year-old boy reported, “They tell me I get up at night and walk around.” His parents reported that, since the age of 5 years, he would abruptly awaken during the first third of the night frightened, disoriented, somewhat unresponsive, and screaming. These episodes seemed to cluster around stressful events in his life and would occur every 3 to 4 months. In addition, in the past year he had a dramatic episode in which, while sleeping at a friend’s house, he arose while asleep, left the house, walked through several backyards to his own home, retrieved the house key from the garage, entered the house, and returned to his own bed for the rest of the night without awakening. No such activity was recorded during 3 nights in the sleep laboratory, but, based on history, the diagnoses of sleep terror disorder and sleepwalking disorder were made. Management with diazepam at bedtime and bedroom security measures were recommended.

#### NIGHTMARE DISORDER

The problem of chronic *nightmares* usually begins early in life; the onset of the condition for about half of adult patients with nightmares is before the age of 10 years (A. Kales et al., 1980b). In a sample of 900 schoolchildren aged 6 to 12 years, 22% reported having nightmares ( Vela-Bueno et al., 1985).

Nightmares are most often associated with fears of attack, falling, or death, and, in many patients, the nightly themes recur ( A. Kales et al., 1980b; A. Kales et al., 1987a). Nightmares occur during periods of REM sleep and thus are most likely to occur late in the night, when REM sleep periods increase in duration ( A. Kales et al., 1980b). The characteristics of nightmares easily differentiate them from the more dramatic sleep terrors ( Broughton, 1968; A. Kales et al., 1980b; A. Kales et al., 1987) (Table 71.2), which occur during slow-wave sleep early in the night and generally are not well remembered, if at all. An REM-sleep behavior disorder has been described that is associated with dream-enacting behavior, often resulting in violent acts or injuries. However, this disorder is more prevalent in advanced age and is often associated with neurologic disorders ( Schenck and Mahowald, 1996).

Characteristic	Night Terrors	Nightmares
Vocalization	Intense	Limited
Autonomic activity	Marked increase	Slight increase
Arousal	Difficult	Easy
Motility	Marked	Limited
Recall	Minimal	Vivid
Sleep stage	Non-rapid eye movement sleep	Rapid eye movement sleep

Adapted from Kales A, Soldatos CR, Kales JD: Sleep disorders: insomnia, sleepwalking, night terrors, nightmares, and enuresis. *Ann Intern Med* 106:582-592, 1987, with permission.

**Table 71.2. Differentiation of Night Terrors and Nightmares**

When nightmares occur in childhood, they are most often related to specific developmental phases ( A. Kales et al., 1980a; A. Kales et al., 1987a; Mack, 1965). They are especially frequent during the preschool and early school years because a child between the ages of 3 and 8 may at times be unable to distinguish among reality, fantasy, and dream content. Because the child has an active fantasy life during this developmental period, fears of imaginary figures and misperceptions of shadows and objects frequently trouble the child while he or she is preparing to go to sleep, thereby predisposing the child to fears during the night. Nightmares in children have also been associated with febrile illness ( Karacan et al., 1968). When they occur frequently in late childhood and adolescence, nightmares are more suggestive of psychopathologic causes (A. Kales et al., 1980b).

The family of a child who has nightmares needs to know and be reassured that children frequently experience nightmares as part of normal development ( A. Kales et al., 1980b; A. Kales et al., 1987a). Otherwise, they may treat the child as though he or she were psychologically disturbed. In addition, minimizing the child’s exposure to potentially traumatic experiences, such as terrifying movies and television programs or frightening bedtime stories, can be helpful ( A. Kales et al., 1987a). In general, pharmacotherapy is not indicated for this disorder.

#### CASE ILLUSTRATION

A 4-year-old girl complained of frequent nightmares of monsters. One especially vivid dream was that her two younger sisters were killed by a monster while she survived. This dream was particularly disturbing to her and resulted in her crying and going to her parents’ bedroom. Historically, there was a detailed history of sibling rivalry problems, as well as parental discord and a tendency on the part of the parents to favor the two younger siblings over this child. She was also having serious behavior problems. A brief focused course of therapy for the child and for the parents proved helpful for relief of the nightmare symptom.

#### ENURESIS

*Enuresis* is defined as bedwetting that occurs after bladder control should have been achieved, usually between the ages of 2 and 3 years ( A. Kales et al., 1987a). This condition is among the most distressing of childhood sleep disorders for both child and parents, and its impact frequently persists long after the disorder has ceased. Between the ages of 3 and 12 years, about 10% to 15% of children still wet the bed at night ( Essen and Peckham, 1976). The prevalence decreases with age, reaching about 3% at age 12 (A. Kales et al., 1987a).

This sleep disorder exists in two forms: primary (persistent) and secondary (acquired or regressed) enuresis ( A. Kales et al., 1987a). In *primary enuresis*, children



have never been able to stop bedwetting for more than a month. Often, they have a family history positive for the disorder and usually have a small functional bladder capacity (A. Kales et al., 1987a; Starfield, 1967). Patients with *secondary enuresis* may be dry for months or years before bedwetting begins once again. Primary enuresis is by far the most prevalent of the two types, and both forms of the disorder are more prevalent in boys (Rutter et al., 1973).

Sleep laboratory studies have shown that enuresis is related more to time of night than to sleep stages (A. Kales et al., 1977; Mikkelsen et al., 1980). A common misconception is that enuresis occurs primarily during REM sleep. Two-thirds of all enuretic episodes occur during the first third of the night, predominantly during non-REM sleep (A. Kales et al., 1977; Nevéus et al., 1999). If the child is not awakened and given a change of clothes after an episode of bedwetting, the sensation of wetness may later be incorporated into a dream about bedwetting during the next REM period (Pierce et al., 1961). Nevéus et al. (1999) comment that enuretic children with a high arousal threshold may sleep more deeply than other children.

Genetic and maturational factors underlie primary enuresis, with the major problem being a smaller than normal functional bladder capacity (Starfield, 1967). Anatomic abnormalities of the genitourinary system are causative in only a few patients with enuresis (Starfield, 1972). Although general population surveys have shown an association between psychiatric disorders and enuresis (Rutter et al., 1973), most children with primary enuresis are actually free from any problems (other than bedwetting) affecting their behavior. Thus, in children with primary enuresis, psychopathologic disorders, when they are present, are predominantly the effects rather than the causes of their enuresis. Yeung et al. (1999), reporting on children with persistent nocturnal enuresis studied with a combination of cystometry (bladder studies) and polysomnography, found combination patterns of sleep disturbances and bladder dysfunction in these children.

In secondary enuresis, psychological factors are usually the etiologic agents (A. Kales et al., 1987a), for example, a significant event that disrupts the child's life, such as the birth of a new sibling or the parent's separation or divorce. However, bedwetting may also be a symptom of diabetes mellitus, diabetes insipidus, nocturnal epilepsy, severe mental retardation, or neurologic disorders.

Determining whether a child has primary or secondary enuresis is critical to the effective management of the patient (A. Kales et al., 1987a). Therefore, evaluation of the patient with enuresis should include a thorough history, with specific attention to the development and clinical course of bedwetting, life events at the onset of bedwetting, and family history of this disorder (A. Kales et al., 1985d). General growth and development, as well as parental attitudes and expectations regarding toilet training, are evaluated.

Physical examination and urinalysis are also required for a complete assessment. Only 1% to 4% of children with enuresis are found to have genitourinary abnormalities (Starfield, 1972). To determine the baseline functional bladder capacity, the physician should have the child refrain from voiding as long as possible and then measure the volume of urine voided (Starfield, 1967).

The physiologic, psychological, and behavioral principles (Doleys, 1977) in the management of primary enuresis are listed in Table 71.3 (Chapter 56). Given the psychological difficulties that typically underlie secondary enuresis, psychotherapeutic management is usually indicated (A. Kales et al., 1987a). Such an approach may range from parental education to psychodynamic treatment for the child and family. In taking this approach, it is important to explore the specific life-stress events usually found to have precipitated the onset of the enuresis. In young adults with enuresis unrelated to organic factors, psychotherapy is indicated.

1. Physiologic	Decreased functional bladder capacity is often present. Bladder training exercises are indicated to increase capacity and sphincter control. Child drinks increased amounts of water and waterfalls diverting urination for gradually increasing time periods; child is trained to stop and start urination. Most children eventually outgrow disorder as bladder capacity increases.
2. Psychological	The case of a child with enuresis is frequently mishandled. Guilt, shame, and anxiety are often superimposed on the problem. Parental counseling and reassurance are critical. Psychotherapy may be indicated.
3. Behavioral conditioning	Bed and pad are placed in the bed at night. Bell or buzzer awakens the child after first drops of urine. Complications include anxiety and skin irritation. Initial success rate is 75%, but there is a high relapse rate.
4. Pharmacologic	Imipramine markedly decreases frequency of urination. Difficulty with urination may be a side effect. A high relapse rate occurs after withdrawal of drug therapy. Treatment recommended mainly for older children with persistent enuresis. Desmopressin (DDAVP) may be a more reliable replacement.

Adapted from Kiese A, Speltz M, Jones K, et al. Sleep disorders (nocturnal, sleepwalking, night terrors, nightmares, and enuresis). Am J Orthopsychiatry 1994;64:100-106, with permission.

**Table 71.3. Principles in the Management of Primary Enuresis**

In the pharmacologic management of enuresis, imipramine has long been the drug of choice (A. Kales et al., 1977; A. Kales et al., 1987a; Werry et al., 1975). However, its use should usually be limited to older children and adolescents. The dosage approved by the U.S. Food and Drug Administration (FDA) is 1 to 2.5 mg/kg of body weight, or 25 to 75 mg daily in an average child. Fritz et al. (1994) confirm that imipramine is efficacious in 73% of reported cases at a dosage level of 2.5 mg/kg; side effects are rare but include cardiac conduction changes. Responses improve with increased measured serum levels, but these levels vary at least 700% between subjects at equal dosage levels, a finding indicating limited usefulness overall in determining therapeutic serum levels. Because of the potential for cardiotoxicity, a baseline electrocardiogram should be obtained, and higher doses should be avoided.

The antidiuretic hormone desmopressin (DDAVP) has also been employed with success in enuretic children. It is described as being more expensive but clinically safer than imipramine and as having fewer side effects (Shaffer, 1994).

#### CASE ILLUSTRATION

A 9-year-old boy, who was placed at the age of 6 years in a residential school because his unmarried mother was unable to care for him, experienced nightly enuresis. Further evaluation revealed a history of grossly inadequate mothering before and continuing maternal rejection after placement. The boy responded to weekly play therapy and a course of 50 mg of imipramine at night with a marked decrease in enuretic episodes. However, there were periodic recurrences whenever his mother demonstrated unpredictability and open rejection with regard to holiday home visits.

### Sleep Disorders Related to Another Mental Disorder

Children with *psychiatric disorders* appear to suffer more frequently from sleep disorders. Reporting on the comparison of a university psychiatric outpatient clinic population of 150 children and adolescents with a nonclinical sample of 309 subjects, Simonds and Parraga (1984) found that a significantly higher prevalence of restless sleep, limb movements, nightmares, night terrors, reluctance to go to sleep, needing to sleep with others, bedtime rituals, fears of dying, and daytime overactivity distinguished the clinical from the nonclinical population. Certain characteristic sleep problems were also associated with specific psychiatric disorders and are described in this section.

#### PERVASIVE DEVELOPMENTAL DISORDER (AUTISTIC DISORDER)

Sleep disturbances are frequently observed in children with *pervasive developmental disorder (autistic disorder)* (Hosino et al., 1984; Richdale, 1999; Segawa, 1982). However, because of their frequent perseverant motor activity, these children are very difficult to assess in the sleep laboratory. In general, children with pervasive developmental disorder or autism tend to continue their restlessness or stereotyped activities into the usual hours of sleep; they often have difficulty in falling asleep and show nocturnal or early-morning awakening patterns.

Sleep patterns of 75 autistic children showed that 65% had sleep disturbance beginning very early in life (Hosino et al., 1984; Patzold et al., 1998). The form that the disturbance takes can be difficulty in both falling asleep and remaining asleep. This study suggests that the sleep disturbance of these children is closely related to the severity of their disorder and their overall prognosis. Rutter (1968) reported that 43% of nonverbal autistic children showed problems with sleep, as compared with 30% of verbal autistic children. Environmental factors are also found to contribute to sleep disturbance, which is decreased by means of psychotherapy or play

therapy ([Segawa, 1982](#)).

In contrast, [Hering et al. \(1999\)](#), comparing actigraphy with parental questionnaires in 22 autistic children, find sleep patterns of autistic children like those of psychologically normal children, with the exception of early morning arousal time. When sleep studies are restricted to questionnaires or sleep diaries ([Patzold et al., 1998](#); [Taira et al., 1998](#)), parents of autistic children report sleep difficulties more frequently.

[Richdale \(1999\)](#), in a comprehensive literature review, reports the success of behavioral interventions in the sleep problems of autistic children. The use of melatonin and chronotherapy is also reported as potentially useful in autistic children ([Jan and O'Donnell, 1996](#); [Jan et al., 1994](#)), although further studies are needed.

*Rett's syndrome*, an autisticlike neurologic disorder appearing primarily in girls, is also characterized by changes in normal sleep patterns ([Glaze et al., 1987](#)). REM sleep is decreased in percentage. Respiratory patterns are abnormal during waking hours, characterized by disorganized breathing and compensatory hyperpnea, but nocturnal respirations are usually normal.

#### ATTENTION DEFICIT HYPERACTIVITY DISORDER AND CONDUCT DISORDER

Clinical and parental reports on children with *attention deficit hyperactivity disorder* (ADHD) indicate that certain alterations in sleep patterns are associated with this common childhood psychiatric disorder ([Busby et al., 1981](#); [Day and Abmayr, 1998](#); [Kaplan et al., 1987](#); [Khan, 1982](#); [Stein 1999, 2000](#)). Difficulty in falling asleep and tiredness on awakening are the most common reported complaints ([Trommer, 1988](#)). However, objective studies fail to show consistently that children with ADHD have longer sleep latencies or decreased total sleep time ([Corkum et al., 1998](#)).

[Ramos-Platon et al. \(1990\)](#) report a large number of nocturnal awakenings and a high increase of slow-wave sleep percentage in ADD, and patients with ADD with hyperactivity and ADD without hyperactivity show distinct hypnographic correlates, findings indicating that they may be different clinical states. These findings support the hypothesis that ADHD is related to deficient control of the arousal level, either hypoarousal or hyperarousal, perhaps at the level of the reticular formation. Hyperarousal may occur in situations with new stimuli, and hypoarousal may occur in situations that require sustained attention ([Douglas, 1984](#)).

Studies of these children at home also suggest a significant increase in body movements during sleep ([Porrino et al., 1983](#)). Comparing children in psychiatric clinical settings with those in nonpsychiatric nonclinical settings, [Simonds and Parraga \(1984\)](#) found that children with ADHD have significantly more snoring, head banging, restless sleep, and nighttime awakening problems. [Palm et al. \(1992\)](#) found three children with ADHD in a series of 10 to have short daytime sleep latencies suggestive of daytime hypersomnolence. [Ring et al. \(1998\)](#) compared medicated children with ADHD with their siblings using a sleep questionnaire and found initial and middle insomnia reported in those with ADHD. [Corkum et al. \(1998, 1999\)](#), also using sleep questionnaires, described dyssomnias, parasomnias, and involuntary movements in children with ADHD. Only involuntary movements seemed directly related to ADHD, combined type. Looking at the reverse situation in one small study, [Picchetti et al. \(1998\)](#) found two-thirds of the children with ADHD studied to have periodic limb movement disorder. More recently, [Chervin and Archbold \(2001\)](#) reported a significant association between periodic leg movements and hyperactivity in 113 children evaluated for sleep-disordered breathing.

Based on their review of the literature on sleep problems in children with ADHD, various investigators ([Barkley et al., 1990](#); [Busby and Pivik, 1985](#); [Dahl and Puig-Antich, 1990](#); [Dahl et al., 1991](#); [Greenhill et al., 1983](#); [Hunt et al., 1985](#), and [Wilens et al., 1994](#)) concluded that difficulty in falling asleep and shorter sleep duration—often a stimulant-induced insomnia in these medicated children—constitutes a problem calling for additional treatment. Based on their clinical experience, these investigators advised the use of clonidine at bedtime in 4- to 7-year-old children with ADHD, at a dosage of 0.05 mg, titrated upward to as high as 0.4 mg, to improve sleep. Although not FDA approved for use in children, clonidine has been demonstrated to be effective as an alternative to stimulant medication in ADHD when insomnia is a problem ([Hunt et al., 1985](#); [Prince et al., 1996](#)).

In a comprehensive review of the literature, [Stein and Pao \(2000\)](#) conclude that children with ADHD who are not treated with medication do not commonly show disrupted sleep, and increased sleep latency with methylphenidate (Ritalin) is to be tolerated. These investigators also caution that sleep deprivation itself may replicate the symptoms of ADHD, a finding calling for further study.

*Conduct disorder*, which widely overlaps with ADHD because of the secondary behavioral effects of the chronic inability to focus attention and to control behaviors, has shown a similarity to ADHD in terms of increased slow-wave sleep EEG activity ([Coble et al., 1984](#)). This finding further substantiates the close relationship between the two disorders that is observed clinically.

#### DEVELOPMENTAL LEARNING DISORDER

In a controlled study of sleep patterns of 24 8- to 10-year-old boys with developmental reading disorder ([Mercier et al., 1993](#)), findings included significantly more stage 4 sleep, less REM sleep, a longer REM onset latency, and an extended initial non-REM (NREM) cycle. Such factors could conceivably impair information processing and could contribute to the cognitive deficits seen in these children. In children with severe *learning disabilities*, severe sleep problems, and strong daytime challenging behaviors, Wiggs and Stores found that a behavioral program led to mothers' reporting improvement in their own as well as their children's sleep patterns and an increased sleep duration in the children ([Wiggs and Stores, 1998](#); [Wiggs and Stores, 1999](#)).

#### DEPRESSIVE DISORDER

Because sleep disturbance is one of the primary vegetative symptoms reported in *major depression*, it could be expected that depressed children may have abnormal sleep patterns. [Hawkins et al. \(1985\)](#) report extended sleep in depressed patients aged 17 to 25 years. Compared with age-matched control subjects, these depressed patients have almost double the length of extended sleep when they are allowed to sleep as long as they want.

A group of depressed children 9 to 14 years of age showed decreased REM latencies ([Emslie et al., 1987](#)), a finding that is in agreement with some previous data by [Lahmeyer et al. \(1983\)](#). However, [Puig-Antich \(1987\)](#) found the opposite and conclude that depressed children seem to differ from depressed adults because of developmental factors. This suggests that REM sleep disturbance may not be a reliable criterion for the diagnosis of depressive disorder in prepubertal children. However, further work by [Emslie et al. \(1990\)](#), who compared 25 hospitalized, depressed prepubertal children with 20 age-matched healthy controls, and by [Kutcher et al. \(1992\)](#), who compared 23 teenagers with major depression with matched controls, again showed reduced REM sleep latencies in both age groups. The depressed children and adolescents also had an increase in sleep latency and an increase in REM sleep, but without stage 4 differences.

Like 30% to 70% of depressed adults, experimentally sleep-deprived, depressed adolescents show a significant decrease in depression severity, with a persistence of 1 day after recovery sleep. These findings suggest a common pathophysiology in depressed adolescents and adults and a relationship between depression and sleep regulation ([Naylor et al., 1993](#)). [Dahl et al. \(1992\)](#) show a blunting of sleep growth hormone beginning with sleep onset in suicidal adolescents with major depressive disorder. This finding is in contrast to adolescents with major depressive disorder who are nonsuicidal and to prepubertal subjects with major depressive disorder, who show an increase in sleep growth hormone. This finding, in addition to other sleep-onset changes, may be a combined result of depression and developmental changes in adolescence and is comparable to the blunting of growth hormone stimulated by sleep in recurrently depressed adults. The possible significance of this finding is that major depressive disorder occurring in adolescence may be related to sleep dysregulation and age-related hormonal changes, combined. [Kallepalli et al. \(1997\)](#) compared the relief of insomnia in 20 inpatient adolescents (aged 13 to 17 years) with depression by using the antidepressant medications trazodone and fluoxetine. Both drugs resolved insomnia by 11 days; the median resolution was twice as fast with trazodone (2.5 versus 5.1 days).

#### BIPOLAR DISORDER

Although the symptoms of young children who are not yet adolescents with *bipolar disorder* may be atypical compared with those of adolescents or adults, sleep disturbances are frequently associated with the disorder in children. As in adults or adolescents, decreased sleep duration or early wake-onset time may predict manic or hypomanic behavior the next day ([Leibenluft et al., 1996](#); [Reite, 1998](#)). Clinically, as with bipolar adults, once patients are taking a mood stabilizer, an atypical neuroleptic agent may be used to assist sleep onset in children with bipolar disorder.

#### GENERALIZED ANXIETY DISORDER AND POSTTRAUMATIC STRESS DISORDER

The sleep disturbances in *generalized anxiety disorder* (formerly overanxious disorder) and *posttraumatic stress disorder* may be fairly specific ([van der Kolk, 1987](#)). They are often characterized by nightmares relating to specific traumatic events or experiences, that is, traumatic, physically injuring episodes, such as falling off



one's bicycle or being in an automobile accident. Depending on their intensity, such traumatic events may result in recurrent nightmares depicting the episode. In addition, patients may have difficulty in getting to sleep as a result of these same psychological factors. Significant sleep disruption in children may also follow involvement in a major disaster ([Benedek, 1985](#); [Dollinger, 1986](#)).

Some evidence has been reported in the stress literature that corticosteroid receptors are increased in the hippocampal area of the brain subsequent to traumatic stress in experimental animals ([van der Kolk, 1987](#)). The hippocampal area has often been associated with dream activity. However, there are no sleep laboratory studies confirming such findings in children.

Treatment considerations suggest that a combination of psychotherapy, behavioral therapy, and anxiety-reducing medications, such as benzodiazepines, is useful in preventing or alleviating this condition. This approach appears more helpful if treatment is administered at the time of the traumatic event or shortly thereafter ([Dollinger, 1986](#)).

#### PSYCHOTIC DISORDER

Clinically, children and adolescents with *psychotic disorders* may complain of sleep disturbance secondary to nocturnal hallucinations. [Krishnamoorthy and King \(1998\)](#) report a small study including children with psychosis not otherwise specified and schizophrenia whose sleep was improved when they used the atypical neuroleptic agent olanzapine for their psychosis.

#### PERSONALITY DISORDER

Although not described as a specific symptom associated with *personality disorder*, sleep disturbances may be reported by these patients. [Dagan et al. \(1998\)](#) reported that 10 of 63 hospitalized adolescents with personality disorders suffered from delayed sleep phase syndrome (DSPS). These investigators hypothesize that the morning sleepiness associated with this disorder may contribute to the development of Axis II disorders.

#### NEUROLOGIC DISORDERS AND MENTAL RETARDATION

Pediatric neurologists commonly treat children with specific *neurologic disorders* who also have signs and symptoms of sleep disturbance ([Scher, 1998](#)). These disorders include epilepsy, benign neonatal myoclonus, infantile spasms, static encephalopathies, cerebral palsy, familial dysautonomia, muscular dystrophy, and structural lesions such as myelodysplasia.

Patients with Tourette's disorder have very disturbed sleep patterns ([Glaze et al., 1983](#); [Hashimoto et al., 1981](#); [Mendelson et al., 1980](#); [Scher, 1998](#)). One study found an increased percentage of stages 3 and 4 sleep, a decreased percentage of REM sleep, and an increased number of awakenings during a nocturnal session ([Glaze et al., 1983](#)). Sudden, intense arousal was also seen in stage 4 sleep, including combativeness. In addition, Tourette's disorder is frequently associated with other sleep disorders, including enuresis and sleepwalking. [Sverd and Montero \(1993\)](#) reported the presence of obstructive sleep apnea and sudden infant death syndrome in the pedigrees of families with Tourette's disorder. Dopaminergic mechanisms are cited in the pathogenesis of this illness ([Glaze et al., 1983](#)). Treatment with a dopamine-blocking agent, such as haloperidol or pimozide, results in some improvement in the sleep patterns of these patients.

Sleep disturbances are also commonly reported in *brain-injured and mentally retarded children* ([Lancioni et al., 1999](#)). *Circadian rhythm sleep disorder* (formerly sleep-wake schedule disorder), in which a person's circadian sleep-wake pattern varies significantly from the sleep-wake schedule of the person's environment, has been reported to follow a closed head injury, a finding suggesting that the insomnia that follows head injuries may represent such a disorder ([Patten and Lauderdale, 1992](#)). Irregular sleep-wake rhythms appear in patients with Down's syndrome ([Levanon et al., 1999](#)) and severe mental retardation, as well as in autistic behavior with retardation and Rett's syndrome ([Okawa and Sasaki, 1987](#); [Segawa, 1982](#)). *Prader-Willi syndrome*, characterized by hypotonia, mental retardation, obesity, hypogonadism, and hyperphagia, also includes excessive daytime somnolence and sleep-onset REM periods, disturbances that are mostly independent of obstructive sleep apnea or obesity ([Okawa and Sasaki, 1987](#); [Vela-Bueno et al., 1984](#); [Vgontzas et al., 1995](#); [Vgontzas et al., 1996a](#); [Vgontzas et al., 1996b](#)). Other disorders of retardation also show sleep changes, findings indicating that malfunctions in sleep patterns may be characteristic of brain dysfunction in general.

In a review article on sleep studies in mentally handicapped children, [Stores \(1992\)](#) reports sleep spindle abnormalities in stage 2 NREM sleep and increases in stage 4 NREM sleep. In children with tuberous sclerosis and mental handicap, more than 90% had sleep disturbances, even more so when seizures were present. Melatonin has been reported to improve sleep in this disorder ([O'Callaghan et al., 1999](#)).

In *epilepsy*, severe sleep problems are also seen ([Beran et al., 1999](#); [Quine, 1991](#)), with marked shifting of sleep stages, apart from nocturnal seizures or medication effects ([Quine, 1991](#)). In a study of 200 children with severe mental handicap, 33% who had sleep problems had epilepsy, whereas only 13% of those without these problems had epilepsy. Quine believes that this finding strongly suggests a neurologic component in sleep disorders and is indicative of the wide prevalence of sleep problems in epilepsy.

#### General Medical Disorders and Substance Abuse

Many *general medical disorders* may produce either temporary or more prolonged disruption of normal sleep patterns in children. An example of the latter is found in pediatric HIV infection ([Franck et al., 1999](#)), in which children studied had significantly more postsleep-onset wake time, more frequent awakenings, and daytime naps and tiredness. In chronic fatigue syndrome, adolescents also were found to have more frequent and longer than normal awakenings ([Stores, 1997](#); [Stores et al., 1998](#)).

Adolescents using alcohol, tobacco, and coffee showed a significant relationship between sleep habits, *substance use*, and perceived daytime tiredness ([Tynjala, 1997](#)). Illegal substance use affects sleep patterns, depending on the specific substance, from the insomnia of cocaine and amphetamine abuse to the hypersomnolence of the opiates.

#### GENERAL RESEARCH DIRECTIONS

Despite the tremendous expansion of our knowledge in adult sleep disorders, our understanding of sleep disorders in children is in its infancy. Current data are limited or inconsistent. Sleep research in children should include studies on the epidemiology, etiology, and treatment of primary sleep disorders in children and on sleep problems accompanying specific psychiatric disorders and mental retardation syndromes. Data-based comparisons of behavioral therapies such as bedtime fading and response extinction, chronotherapy, and psychopharmacologic treatment interventions are sorely needed. These data would permit generalizations about specific characteristics and effective treatments of sleep disorders of children. In addition, in these studies, the various stages of the metabolic development of children's brains should be accounted for because changes in brain function, relative to the maturation of neurotransmitter systems occurring during childhood, may influence the nature of sleep dysfunction associated with psychiatric disorders ([Puig-Antich, 1986](#)).

In the 1990s, there was a dramatic increase in the use of multiple psychopharmacologic agents in children ([Campbell and Cueva, 1995a](#) and [b](#)) which continues in the present. Controlled, primarily polysomnographic studies to measure their effects on child sleep patterns, and changes in daytime behavior, will improve our knowledge of the usefulness of medication in the treatment of sleep disorders in children.

Finally, technical improvements in sleep laboratory and home-based polysomnographic assessment of children, including telemetry and computerized methods, will also enhance the ability of clinical investigators to gather the data necessary to understand these disorders in children better. As a result of these changes, clinicians will be able to determine more accurately the diagnostic criteria most appropriate for children with sleep disorders and the most effective treatments for these disorders.

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## 72 BORDERLINE DISORDERS IN CHILDREN AND ADOLESCENTS

Melvin Lewis, M.D., and Fred R. Volkmar, M.D.

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### HISTORICAL NOTE

Early definitions of “borderline” features were largely drawn from inferences based on psychoanalytic theory and psychodynamic descriptions ( [Bemporad et al., 1982](#); [Ekstein and Wallerstein, 1956](#); [Frijling–Schreuder, 1969](#); [Kernberg, 1975](#); [Pine, 1974, 1983](#); [Robson, 1983](#)). From the side of early theory and clinical observations at that time, some clinicians postulated a condition in which functioning impairment was less severe than that observed in childhood psychosis but more severe than in neurosis, as though there were a continuum from neurosis to psychosis with borderline somewhere in between ( [Mahler, 1971](#)). [Ekstein and Wallerstein \(1956\)](#), for example, suggested that in this borderline state constant fluctuation occurred between neurotic and psychotic modes of functioning.

In many respects, these early descriptions influenced the description of *borderline personality disorder* in adults ([Kernberg, 1975](#)). Among adults, the term *borderline* also has a similarly complicated history, with the observation of borderline functioning on projective testing, of intense transference–countertransference reactions, and a tendency for patients to regress markedly when faced with perceived abandonment. There was also a putative possible relationship with schizophrenia.

This earlier emphasis on theoretical speculation was not tested with empirical studies, and this continues to be limiting in this area. Meanwhile, the utility of a continuum concept of schizophrenia in children was subsequently questioned as the validity of well-defined conditions such as autism came to be established ([Volkmar, 1996](#)). Petti and Vela, in their 1990 review of the existing extensive child literature in this area, dryly note that “the amount of space required to present a topic area is inversely proportional to the degree of scientific certainty associated with it” and conclude more seriously that “there appear to be a number of borderline disorders with different etiologies, phenomenology, associated features, and required treatments” ( [Petti and Vela, 1990](#)).

Although the condition is firmly recognized in adults, many questions regarding the validity of borderline conditions in childhood remain to be addressed. Indeed, some investigators propose an alternative concept of multiple complex developmental disorder (MCDD) and eschew the term *borderline* altogether ( [Towbin et al., 1993](#)).

### DEFINITION

A *personality trait* may be defined as an enduring pattern of perceiving, relating to, and thinking about one's environment and oneself ( [Lewis and Volkmar, 1990](#)). This is a dimensional concept, which in its extreme form may be recognized as pathologic, although not necessarily fulfilling the criteria for a disorder. A personality trait becomes a *personality disorder* (i.e., a disease) when the trait not only is severe, but also becomes inflexible and maladaptive and causes either distress or impairment in social or occupational functions. Although it was once fashionable to question the validity and stability of personality traits over the course of development, several studies have suggested that some such continuities do exist; for example, uncontrolled 3-year-old children tend, as adults, to be more impulsive and antisocial, whereas inhibited ones tend to become depressed ( [Caspi, 2000](#)).

The fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) ([American Psychiatric Association 1994](#)) codes adult personality disorders on Axis II. Clinically, personality disorders in adults appear to be divided among three broad clusters ( [Table 72.1](#)).

Cluster A	Cluster B	Cluster C
Paranoid	Antisocial	Avoidant
Schizoid	Borderline	Dependent
Schizotypal	Histrionic	Obsessive-compulsive
	Narcissistic	Not otherwise specified

Adapted from American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. (DSM-IV) Washington, DC, American Psychiatric Association, 1994, with permission.

**Table 72.1. DSM-IV Clusters of Adult Personality Disorders**

There is no single or specific description of borderline disorders in children or adolescents. However, children and adolescents less than 18 years of age who seem persistently, and over a long time, to have the essential characteristic features, modified by developmental factors, of any of the personality disorders described for adults may be diagnosed in the same way as adults.

In contrast to the DSM-IV, the tenth edition of the *International Classification of Diseases* (ICD-10) ([World Health Organization, 1992](#)) classifies abnormalities of personality in adults into personality disorders, personality trait accentuation, and enduring personality change, and it also has a separate category for organic personality disorder, but it does not include schizotypal, borderline, or narcissistic personality disorder under personality disorders. Under the heading “Disorders of Adult Personality,” the ICD-10 does include a description of borderline personality disorder (F60.31) as a subcategory of emotionally unstable personality disorder ([World Health Organization, 1992](#)). Among the particular additional characteristics noted are the presence of a disturbance of self-image, aims, and internal preferences (including sexual) and chronic feelings of emptiness. “Intense and unstable relationships may cause repeated emotional crises and may be associated with excessive efforts to avoid abandonment and a series of suicidal threats or acts of self harm” ( [World Health Organization, 1992](#)).

### CHILD CLASSIFICATIONS

The diagnosis of personality disorders, including borderline personality disorder, in children has been in doubt in part because it appears to be based on the assumption that whereas personality disorders may be appropriately diagnosed in adulthood when the constellation of traits subsumed under the term “personality” are presumed to be relatively stable, similar stability has not been documented in children. However, as noted earlier, a body of work does suggest the stability of

some personality traits in children (Caspi, 2000), although empirical data supporting the stability of personality disorders are still lacking.

In one study, Guzder et al. (1999), in a chart review of 98 latency-age children in day treatment, find that those who had “borderline pathology” are significantly more likely to have histories of sexual abuse, severe neglect, and parental substance abuse or criminality. These investigators also note that these findings parallel findings of studies in adults with borderline personality disorder; that is, these adults similarly report high frequencies of childhood sexual abuse, physical abuse, and neglect (Paris, 1994). Guzder et al. also hypothesize a continuity between children with borderline pathology and adults with personality disorders, this time on the basis of similarities in risk factors that may indicate a common origin and may therefore give some support to the concept of continuity between children and adults. Similarly, in a study using a family study method, Johnson et al. (1995) find adolescents with a borderline personality disorder and further find that these adolescents have first-degree adult relatives with increased prevalence of borderline personality disorders.

Follow-back studies of adults with borderline personality disorder are complicated in that childhood precursors of the condition may include a range of difficulties, such as problems in modulation of affect, attention, or excessive shyness and insecurity. Again, the evidence regarding the existence of a similar borderline personality disorder in childhood is not a simple matter because children with such problems may exhibit many different outcomes, including depression and schizophrenia.

The DSM-IV definition of *borderline personality disorder* (Table 72.2) does presume to be applicable at least to adolescents, by citing as an example dropping out of school just before graduation (as an expression of undermining oneself) (American Psychiatric Association, 1994). One method by which the DSM-IV criteria for borderline personality, at least in adults and adolescents, may be measured is the *Personality Disorder Examination (PDE) Manual* (Loranger et al., 1988).

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A pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

1. Frantic efforts to avoid real or imagined abandonment. Note: Do not include suicidal or self-mutilating behavior covered in criterion 5.
2. A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation.
3. Identity disturbance: markedly and persistently unstable self-image or sense of self.
4. Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating). Note: Do not include suicidal or self-mutilating behavior covered in criterion 5.
5. Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior.
6. Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days).
7. Chronic feelings of emptiness.
8. Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights).
9. Transient, stress-related paranoid ideation or severe dissociative symptoms.

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From American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. (2000). Washington, DC: American Psychiatric Association, 1994, with permission.

**Table 72.2. DSM-IV Diagnostic Criteria for Borderline Personality Disorder**

## PREVALENCE

Borderline personality disorder in adults is said to be diagnosed predominantly (about 75%) in women (American Psychiatric Association, 1994). Again, there is no specific mention of children. Given the uncertain validity of the concepts and the poorly established diagnostic criteria and outcomes, true prevalence figures are not available for children. Among adults, borderline personality disorders are estimated at 2% of the general population and constitute 30% to 60% of all personality disorders.

## CLINICAL FEATURES

Clinically, several features commonly seen in children (Table 72.3) have been suggested to constitute the diagnosis of borderline personality disorder. There may be rapid, unpredictable (so-called “predictably unpredictable”) regression in thinking, reality testing, and affective control. Extreme vulnerability to stress may lead to sudden suicidal or homicidal behavior or to psychotic manifestations, including hallucinations. Such children may reintegrate quickly once the stress stops, for example, when they are placed in a safe, structured, stress-free environment, such as a hospital. The child may be chronically regressed. Severe regression on separation from the parent (who often also has a borderline personality condition) and reintegration when reunited with the parent may occur, as though the child can function only within the symbiotic relationship with the parent. In theory, there seems to be a failure of individuation and of object constancy. A generalized restriction in the development in relationships, affective range, cognitive functions, and language may occur. Retreat into a preoccupation with fantasy and emotional withdrawal from relationships may occur, leading to a schizoidlike clinical picture. The child may be flooded and overwhelmed with rage and violent fantasies that threaten the child’s self-control and produce extreme anxiety. Symptoms such as rage, temper outbursts, violent fantasies, regression, and impulsivity may all occur, sometimes mimicking aspects of attention deficit hyperactivity disorder (ADHD) or anxiety disorders with depression (Table 72.4).

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Rapid regression in thinking, reality testing, and affective control  
 Extreme vulnerability to stress, with psychotic decompensation  
 Chronically regressed state  
 Severe separation anxiety and regression  
 Generalized restricted development (e.g., in relationships, affect, cognition, and language)  
 Schizoid retreat into preoccupation with fantasy life and withdrawal from relationships  
 Overwhelming rage and violent fantasies with extreme anxiety and loss of control

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**Table 72.3. Clinical Features of Borderline Personality Disorder**

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Attention deficit hyperactivity disorder  
 Anxiety disorders  
 Major depressive disorder

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**Table 72.4. Comorbid Disorders (Symptom Clusters) in Borderline Conditions in Childhood**

## COMORBIDITY



Such associated symptom clusters may be conceptualized as *comorbidity*, or the total clinical picture may be characteristic of borderline personality disorder in children, comparable perhaps to the way in which the term *conduct disorder* is sometimes used as an all-inclusive term that covers a wide range of behaviors that may otherwise be viewed as other associated conditions.

The issue of comorbidity is complex (Chapter 39), given the sometimes very different approaches used in the DSM-IV and ICD-10. As it happens, few conditions in child and adolescent psychiatry are mutually exclusive (in the way that, say, autism and Down's syndrome do not commonly coexist in the same child). On the contrary, children diagnosed with borderline personality disorder may well have such associated DSM-IV diagnoses as ADHD, anxiety disorder, major depressive disorder, and posttraumatic stress disorder. In any event, these other conditions present a need for a careful differential diagnosis.

Hypotheses to account for these coexistent diagnoses include the following:

1. The child with a borderline personality disorder may be particularly vulnerable to the other diagnoses listed earlier.
2. The borderline condition may itself be an atypical variant of one or more of these other disorders.
3. A more fundamental disorder may be the basic factor common to all the concomitant diagnoses.
4. True comorbidity, that is, the concomitance of one or more of the conditions by chance alone, may account for the multiple diagnoses.
5. The appearance of comorbidity or coexistence may be an artifact of our classification systems (DSM-IV and ICD-10), which are currently based largely on symptom lists and not on mechanisms, etiology, or natural history of the alleged disorder.
6. The reported apparent coexistence in some instances may result from flawed research design or other factors and may ultimately not be significant.

Whatever the explanation, clinicians report that they are not surprised to find that a child or adolescent with a borderline personality disorder has a history of having been labeled with several other diagnoses over the course of the illness.

In general, the behavior of a child or adolescent with a presumed borderline personality disorder is characterized by unpredictable moods and a pervasive, fluctuating, and unstable level of organization in multiple functions in such major areas as impulse control, affect modulation, attention, cognition, and relationships. Relationships are immature, often at a need-fulfilling stage, with a great deal of ambivalence and dependency and a minimal capacity for empathy. The child may have paranoid rages and may regress to the point of uncontrolled temper tantrums and loss of reality testing. A fluctuating thought disorder is often present, accompanied by a disorder of mood (especially depression), of behavior (which may be disorganized, aggressive, withdrawn, or bizarre), or of perception (with delusions and hallucinations). The anxiety level in these children is often quite high and is poorly defended against, resulting in an interference with relationships and thinking. Compensatory omnipotent fantasies are often quite prominent. Unfortunately, the striking lack of empathy in the child may lead to repetitious reinforcement of failure and rejection in almost every social interaction in which the child is involved and a consequent increase in the child's depression, despair, and anxiety.

## COURSE AND PROGNOSIS

Regrettably, most outcome studies use variable definitions and are poorly controlled. In essence, there are still no satisfactory systematic studies on the course of children diagnosed as having a borderline personality disorder (Petti and Vela, 1990). Several clinically interesting treatment and follow-up case studies have been reported (Kestenbaum, 1983; Petti and Unis, 1981).

## ETIOLOGY AND PATHOGENESIS

As has been true for borderline personality disorder in adults, various potential etiologic mechanisms have been identified. Given the early interest in psychodynamic theory and treatment of these conditions, many theories invoke various experiential factors. However, a growing body of work suggests the potential importance of biological vulnerability, which may, itself, shape the course of development and personality. For example, Weiss and colleagues (1996) report that children of mothers with borderline personality disorders are themselves at significantly increased risk of various forms of psychopathology. One could postulate that vulnerabilities in impulse control or processing experience may negatively affect social interactions, and this, in turn, may adversely influence the development of self-concept and affective development (Marohn, 1990). Another possibility is that not only does the child have multiple comorbidities (however defined), but also the comorbidities themselves may have multiple causes. Such causal pathways may include those shown in Table 72.5. Thus, a working model for understanding the pathogenesis of this disorder (or perhaps cluster of disorders) may be a complex interactive model involving polygenic, organic, cognitive, intrapsychic and interpersonal, and socioenvironmental factors.

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Genetic loading (Andriuloni, 1990)
Organic factors (Bemporad et al., 1982)
Disturbances in the development of relationships (Pine, 1982)
Serious disturbances in family functions and systems (Combrink-Graham, 1988)
Major parental psychopathology and major external stresses, especially abuse (Pine, 1988)

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**Table 72.5. Causal Pathways in Borderline Personality Disorder**

### Biological Factors

Personality styles (or temperament) in general are derived in part from genetic factors (Torgesen and Kringlen, 1978). In addition, several disorders embedded in or comorbid with borderline personality disorder have been shown to have some genetic components. Such disorders may include antisocial personality disorder (Schulsinger, 1972) and ADHD (Cantwell, 1976; Zametkin and Rapoport, 1987). The heritability of certain aspects of personality and of certain major psychopathologic disorders may be in the range of 30% (Plomin, 1990).

There is no established pathognomonic organic finding in children who have a borderline condition. However, several reports support the presence of some form of organicity (Aarkrog, 1981; Bemporad, 1982), and one report suggests a disturbance of the enzyme dopamine b-hydroxylase (Rogeness et al., 1984, 1986). More research is required to confirm these reports.

### Psychological Trauma and Environmental Factors

The psychoanalytic theories of Kernberg (1975) and Kohut (1971) regarding borderline disorder are essentially " sleeper " theories. They imply a delayed effect of early psychological trauma during the separation-individuation phase, because the effects are not readily apparent at the time of the trauma but appear later, as a result, it is hypothesized, of an induced or heightened vulnerability to subsequent stresses. In adults with borderline personality disorders, various studies, mostly relying on retrospection, suggest that difficulties in early parent-child relationships (e.g., as a result of psychological problems in the parents, separation from the parents, or loss of the parent) and sexual and physical abuse are strongly related to the disorder (Kernberg, 1975). Although data in children are quite limited, one study (Bradley, 1979) notes that children and adolescents with borderline personality are significantly more likely to have experienced early separation from the mother.

The generally retrospective nature of these data limit their validity. Such experiences undoubtedly are major risk factors for difficulties, and the specific issue of why one person goes on to develop a presumed borderline personality while another does not remains to be addressed, although, of course, it is possible that some

interaction of experiential and biological factors may occur at just this point.

## DIFFERENTIAL DIAGNOSIS

The hypothesis of multiple comorbidities mentioned earlier often translates clinically into a difficult differential diagnosis ( [Table 72.6](#)). Many of the children labeled borderline may also have symptoms of ADHD, major depressive disorder, conduct disorder, somatoform disorder, dissociative disorder, posttraumatic stress disorder, cyclothymic disorder, schizophrenia, anxiety disorders, and partial complex seizure-type disorders.

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Attention deficit hyperactivity disorder  
Major depressive disorder  
Conduct disorder  
Somatization disorder  
Schizophrenia  
Partial complex seizures  
Posttraumatic stress disorder

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**Table 72.6. Differential Diagnosis in Borderline Conditions in Children**

In some instances, a child who presents with the clinical picture labeled borderline personality disorder (an Axis II diagnosis) may be suffering from a peculiar form of dissociative disorder, specifically dissociative identity disorder (an Axis I diagnosis, not commonly diagnosed in children). Both disorders appear to have a similar origin, including severe sexual abuse during childhood. In addition, the symptoms of the two conditions are often similar, including some form of thought disorder, and the course is often chronic and difficult, with suicide attempts not infrequent.

The diagnostic distinction between borderline personality disorder and dissociative identity disorder is important because the treatments are different. In particular, in the treatment of children with dissociative phenomena, including dissociative identity disorder ( [Chapter 70](#)), an effort must be made to help the child to become conscious of dissociative defenses, such as splitting and regression, which appear to take place as a way of dealing with the terror of the sexual abuse, and the of consequent seemingly inexplicable episodes of rage and violence that sometimes occur and that the child does not appear to remember.

A history of trauma, often sexual abuse, is relatively common in patients with borderline personality disorder, particularly if the sexual abuse is recurrent. It is clear that severe stress can produce phenomena suggestive of psychosis, such as hallucinations, in some persons ( [Spivak et al., 1989](#)). In addition, hallucinatory phenomena can be part of the intrusive “flashback” experienced in posttraumatic stress disorder. Usually, such hallucinations capture some aspect of the traumatic event; they may be accompanied by feelings of depersonalization or anxiety ( [Mueser and Butler, 1987](#); [Waldfoegel and Mueser, 1988](#)).

The overinclusiveness of the diagnosis can be problematic. Thus, its use may carry two burdens. First, once the label is given, no further diagnostic studies may be done, and a basis for treatment planning may accordingly be lost. Second, the treatment may be half-hearted because the prognosis of such a personality disorder is generally thought to be guarded at best.

Because eventual adult outcomes are unclear ( [Rutter, 1988](#)), outcome for children with borderline personality disorder may similarly be unclear and may eventually turn out to represent an intermediate cluster of behaviors in the development of a child in whom multiple causes and possible multiple comorbidities are present. For this reason, one should not easily settle for the diagnosis of borderline personality disorder in children. Perhaps the concept of a “multiple complex developmental disorder” ( [Towbin et al., 1993](#)), together with a carefully documented dimensional account, may eventually have more appeal.

## TREATMENT

The recognition of symptom clusters of other treatable conditions, including bipolar illness, ADHD, or complex partial seizure disorder, is very important because specific targeted treatment, including psychopharmacotherapy, for these better-defined conditions may then be offered. Unfortunately, well-controlled treatment response studies for the basic borderline personality disorder are not available at present. In one study ( [Bentivegna et al., 1985](#)), only half the treated children improved, and in those who did improve, residential treatment was often requested, and the treatment was long term. In any event, the fluctuating range of etiologic factors and of clinical features, along with the paucity of well-designed natural history and treatment outcome studies, leads to the present need to use multimodal treatment approaches ( [Table 72.7](#)).

---

Individual therapy  
Parental therapy  
Family therapy  
Behavioral therapy  
Pharmacotherapy  
Residential treatment  
Day (partial) hospitalization  
Hospital treatment  
Milieu therapy

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**Table 72.7. Multimodal Treatments of Borderline Personality Disorder**

Such treatments may include family therapy, individual therapy, parent therapy, pharmacotherapy, hospital and milieu therapy (residential treatment, day treatment, or both), and behavioral therapy. At present, data are insufficient to rely solely on any one of these approaches; all may be necessary when clinically indicated. Controlled follow-up studies are not currently available.

One major consideration in the intensive psychotherapy of children presumed to have a borderline personality disorder, especially those children who are in the context of residential treatment, is the development of *reality testing*. Reality testing helps the child to recognize and understand his or her reactions to various events within current life experiences, such as disappointments when parents fail to show up, provocations from peers, and imposition of controls by staff. Precipitating events, fantasies, and warded-off affects are defined for the child. In this way, the child’s regressive behavior is made more understandable not only to the child, but also to the therapist, the staff, and the parents.

Reality testing often requires the close day-to-day contact of residential treatment to understand the meaning of the child’s behavior. Reality testing clarifies not only what is real and what is fantasy, but also which thoughts, fantasies, and ideas belong to the child and which belong to the therapist, parent, or child care staff member. Further, when the child demonstrates an essentially correct perception of his or her parents, this should be confirmed for the child.

Because of the instability of object representation, especially when the other person is not present, psychodynamic interpretation of preconscious or unconscious



affects, fears, or wishes is often limited in its effectiveness, at least during the early phases of treatment. In some instances, it seems as though the child can barely hold onto the concepts being conveyed in the verbal interpretation and only for the time it takes to say them; once the verbal interpretation is completed, the child seems to lose touch with the words and is at the mercy of other demands, usually primitive inner demands. Because interpretation is sometimes limited in its effectiveness in this way, "working through" is also difficult in these children (see [Chapter 79](#)).

The phenomenon of *delayed object constancy* may provide a key to an important therapeutic agent, perhaps even a key agent: the need for the reliable presence of a stable person in the child's life. Thus, the single most important component of the treatment of these children may be the availability of a consistent, stable relationship. This finding reflects in part a metapsychological view of the development of these children, in which early instinctual needs are still experienced, unstable early defenses (such as projection and identification) and impaired reality testing prevail, and primary process is clinically evident. The child manages to make the therapist feel a regressive pull toward similar chaotic and archaic levels of functioning. Whether the child does this intentionally as part of the child's desire to project his or her own feelings of impotence onto the therapist, thus leaving the child to feel temporarily more omnipotent and the therapist to feel acute discomfort, or whether the effect on the therapist is simply a product of being in the presence of such a child, is not always clear. However, the therapist can use the reaction evoked to gain some understanding of what the child may be experiencing.

Essentially, the therapist conveys to the child, mostly through the *metaphor of play*, his or her understanding of what the child appears to be experiencing, consciously or preconsciously, in the here and now. Boundaries are defined: What is fantasy? What is actuality? The therapist then offers an explanation to the child that is simple and convincing, again, most often through the metaphor of play. The purpose of using the metaphor of play is to enable the child to maintain the buffer of distance and not be overwhelmed by anxiety.

At the same time, the therapist also begins to suggest other, more effective and realistic ways of obtaining gratification and dealing with staff or parents. This, too, is frequently done through play, but eventually it can be done in a straightforward, almost didactic way.

The therapist as well as the staff must maintain controls during the treatment, to limit regressive, sexualized, and aggressive behavior to manageable levels and to avoid the excessive guilt and anxiety that is so quickly mobilized in the child who feels that controls are slipping. Similarly, when the child begins to act out aggressive impulses toward the therapist, this, too, must first be controlled before it is explored and interpreted. The acting out may begin as "accidentally" throwing some of the therapist's desk belongings on the floor, then kicking them. This may escalate to breaking the various objects or throwing them out the window. It is essential to prevent this kind of escalation by imposing controls early. Because children who are regressed or regressing feel acutely uncomfortable, limits must be set and regression halted to prevent any further increase in anxiety. Separations, for example, may precipitate intense feelings of abandonment, accompanied by characteristic regressive behaviors, which, in turn, need supportive but firm controls.

The high level of anxiety is not the only complication in the treatment of these children. Depression is often present, too. The sense of despair, hopelessness, and helplessness and, at times, the frightening anger need to be acknowledged, clarified, and understood. Usually, this comes at a later stage in the treatment, when the child feels more secure in his or her relationship with the therapist, has a better defined sense of self, and is not in dread of being overwhelmed by inner feelings. Often a child who is ready to look at his or her depression is also ready to hear and comprehend a reconstruction that will help the child to make some sense and give some order and meaning to his or her state of feelings and turmoil.

In any case, it is almost always useful, whether or not in the context of exploring depression, to provide the child with a meaningful history of his or her life, a history that will help the child to understand why he or she had, or has, to feel and behave in certain ways. In some cases, it may be helpful to depict this pictorially, in a concrete way, with pictures and drawings, with the child participating in the drawing and labeling process.

Concomitant work with the parents is essential. Sometimes the parent is idealized by the child, even the parent who repeatedly fails the child. Sometimes the parent has an ambivalent, or unrealistic, expectation of the child. In some instances, the parent must be helped to enable the child to live in another setting when that is in the best interests of the child.

The child may present to the staff a distorted picture of the parent as part of the child's unconscious attempts to ward off anxiety through splitting and projection. Sometimes the child will use the therapist and other staff members as convenient scapegoats in expressing rage at a parent.

The regressive pull and the reactions evoked in the therapist who treats a borderline child are almost pathognomonic. Anxiety and defensiveness in the presence of a child who is almost incomprehensible or who is threatening to lose control constitute a common experience. Rescue fantasies, unrealistic goals, and despair may also occur. The therapist often needs emotional support during this work with the child.

The loose use of the term *borderline* attached to a child is misleading because it is used as an adjective or label, and not a diagnosis, for what are after all a varied collection of symptoms that together may suggest the existence of serious and multiple, pervasive, and unstable affective, behavioral, and cognitive regulatory dysfunctions, none of which may have been fully validated. Further, this clinical picture may be further qualified by specific dimensional descriptions of the prominent clinical features in a given child, features that may be used as a basis for formulating and monitoring a multimodal treatment plan for that child. Finally, adolescents who have unstable relationships, identity disturbances, chronic feelings of emptiness, inability to relate appropriately, and especially such behavioral elements as impulsivity, inappropriate anger, and almost frantic efforts to avoid abandonment, may need hospitalization ([Sarrislow et al., 2000](#)).

## DIRECTIONS FOR RESEARCH

The problem of the lack of definitive scientifically valid research must be addressed. Even the question of whether or not a personality disorder is a mental disorder is still unsettled ([Kendell, 2002](#)). In some ways, attempts at closure on the diagnosis of borderline personality disorder in children are premature. Before closure, careful longitudinal studies of the developmental pathways ("chain of operations") and individual symptoms or clusters of symptoms, family studies, and both short-term and long-term treatment outcome studies are needed. There are indications that current studies are moving in these directions. For example, [Ad-Dab'bagh and Greenfield \(2001\)](#) have performed an extensive review of several electronic databases in relation to the term "borderline" and subsequently proposed a process for arriving at a new nomenclature to be called "multiple complex developmental disorder" (MCDD) for children who have the symptoms currently being subsumed under the label "borderline." Perhaps such a change may occur in time for DSM-V.

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### CASE ILLUSTRATION

Timmy, a 9-year-old child in residential treatment, generally did fairly well in the on-grounds school. One week, he became obsessed with fantasies about airplane crashes, boats being torpedoed, and people getting burned or drowned. At the same time, he began to smash toy cars and trucks and developed a sleep disorder. The treatment team finally discovered that a few days earlier he had overheard his parents discussing a vacation they were going to take, and he was very resentful that they were not taking him. He was almost panicked at the thought that his hostility toward them for that, and for other things, may somehow endanger them. The "unpacking" of these fantasies and the terror that they evoked were made possible by a process of interdisciplinary interstaff collaboration, whereby little bits of information were gathered from the school, child care staff, and therapists and pieced together at a combined staff conference.

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# 73 ALCOHOL AND DRUG ABUSE IN CHILDREN AND ADOLESCENTS

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Alcohol and drug abuse are of epidemic proportion in our adolescent population and is a major public health problem. Although most adolescents (85%) at least experiment with alcohol and drugs without serious consequences, substance abuse may have devastating effects on children and adolescents. Accidents, suicides, and homicides account for more than 80% of the deaths in the teenage years, and alcohol and drugs are involved in at least half of these tragedies ( [Soderstrom et al., 1993](#)). Substance abuse is a major or complicating factor in at least 40% of teenagers treated in day and residential state programs ( [Gruenbaum et al., 1991](#)) and in two-thirds of teenagers detained in juvenile delinquent facilities. Although the number of teens abusing illicit drugs and alcohol has declined slightly from 11.4% in 1997 to 9% in 1999, data from the Substance Abuse and Mental Health Services Administration (2000) indicate that those who are abusing substances are experiencing more drug-related problems. These data indicate that all mental health clinicians involved with teenagers need to be knowledgeable of the assessment and treatment of adolescent substance abuse.

Adolescent substance abuse qualitatively differs from adult substance abuse in certain important aspects. These include the following:

1. *Developmental level:* The biopsychosocial level of development must be considered. The early adolescents' diminished ability to delay gratification and to plan for the future and pubertal physical and drive intensity changes make normal early adolescents very different from late teens or young adults. Evaluation and treatment planning need to recognize these different physical, emotional, and cognitive immaturities.
2. *Use of multiple drugs:* Adolescents who progress to substance abuse or dependency disorder, as defined by the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* ( [American Psychiatric Association, 1994](#)), tend to use multiple drugs. Drug-abusing adolescents move from cigarettes and beer or wine to other forms of alcohol and then to marijuana. If the abuse progresses, this stage is followed in sequence by problem drinking, use of downers or uppers, crystal methamphetamine, cocaine, and heroin. Adolescents frequently continue to use the drugs they previously used in the sequence. Adolescents usually have a drug of choice but also intermittently abuse those drugs used earlier.
3. *Use of inhalants:* Inhalant abuse, which includes inhalation of gasoline, glue, freon, and butane, has a peak incidence in preadolescence and early adolescence and is almost nonexistent in adult populations.
4. *Use of "club" drugs:* The club drugs became increasingly popular in the 1990s and are primarily used by older adolescents and young adults. These drugs include ecstasy (methylenedioxymethamphetamine or MDMA), g-hydroxybutyrate (GHB), and ketamine ("special K"). These drugs are rarely used by older adults.
5. *Presence of comorbid disorders:* Comorbid disorders, such as attention deficit hyperactivity disorder (ADHD), conduct disorder, affective disorders, and anxiety disorders, exist in 40% to 90% of adolescents with a substance abuse or dependency disorder ( [Jaffe, 1996](#)). Some studies show that comorbidity is more common in adolescents than in adults ( [Kandel et al., 1999](#)). Comorbid depressive disorders in adolescents with substance use disorder (SUD) tend not to resolve after a few weeks of abstinence, whereas in adults, the comorbid depressive disorders usually resolve ( [Bukstein et al., 1992](#); [DiMileo, 1989](#)).

In the following sections of this chapter, the epidemiology, risk factors, clinical issues of drugs of abuse, assessment, treatment, comorbid conditions, and prevention of child and adolescent alcohol and drug abuse are addressed.

## EPIDEMIOLOGY

From 1996 to 2001, illicit drug use reached levels not seen since the previous usage peaks in the late 1970s and early 1980s. Recent drug use has not yet climbed to the heights of the 1970s and 1980s, when marijuana was used by one of two high school seniors in 1979 as compared with one in four in 1996. Still illicit drug use "within the last month" among 12- to 17-year-olds doubled from 1992 to 0.9% in 1995.

Inhalants were the fourth most commonly used drug among students of high school age. Inhalant use was almost twice as high among eighth graders (6% cited use during the last month) than among 10th or 12th graders (3% cited use in the last month). Twenty-one percent of eighth graders had tried inhalants at least once, likely because of the availability of these drugs ( [National Institute on Drug Abuse, 1996](#)). The highest annual use rate of inhalants was reached in 1993 at 7%, among high school seniors. This represents the highest rate seen since 1975, when usage rates climbed to 7.2%.

Usage of lysergic acid diethylamide (LSD) and phencyclidine (PCP) approached its late 1970s and early 1980s peak in 1996. LSD use among high school seniors

increased from a low of 8.3% in 1989 to 12.6%, and hallucinogen use rose to 14% for lifetime prevalence. Use of marijuana, the most common illicit drug used among youth, has continued to expand since 1992. Lifetime annual and current use of marijuana has doubled in eighth graders since 1992, with 11% reporting use in the last month and 5% reporting daily use. Although marijuana use has fallen among older teens (10.7% of high school seniors admitted using the drug in 1978 compared with 2.0% in 1991), it is troubling that younger teens are now experimenting with the drug. The statistics are particularly worrisome because research has shown that earlier use indicates a greater risk for later development of substance abuse and dependency.

In 1996, 66% of high school seniors reported lifetime use of any illicit drug, and more than 85% of all adolescents had tried alcohol and drugs by the time they finished high school (Johnston, 1996). Eighty-five percent of high school seniors surveyed that same year had some experience with alcohol, and 31% had drunk five or more drinks on at least one occasion during the past month. Two percent of high school seniors reported trying heroin. By 1996, heroin use had doubled its 1991 levels. Any reports of heroin use are significant because the potency of heroin on the streets in the 1990s is much greater than that available in the late 1970s and early 1980s.

Through 1999, use of some illicit substances decreased. A significant drop occurred in the use of crack cocaine among eighth and 10th graders. The use of crystal methamphetamine or "ice" declined to its lowest level in 5 years in 1999. A continuing decline in cigarette smoking among younger teens was also documented. Club drugs such as flunitrazepam (Rohypnol) began to be included in surveys in 1996. A small, significant decline occurred in 1999 among eighth graders to an annual prevalence rate of 0.5%. However, use of another popular club drug, MDMA (ecstasy), began to rise in older teens. In 1999, 4.4% of 10th graders reported use of MDMA during the prior 12 months, (up from 3.3% in 1998) and 5.6% of the 12th graders (up from 3.6% in 1998). No data have been collected on another club drug, GHB. Accidental deaths have been attributed to use of this drug.

Among younger teens, eighth and ninth graders, anabolic steroid use rose in 1999. Use by eighth graders rose from 1.6% in 1998 to 2.5% in 1999; for 10th graders, it rose from 1.9% to 2.8% during the same period (Johnston, 1996; Johnston, 1999; National Institute on Drug Abuse, 1996).

## RISK AND PROTECTIVE FACTORS

Clearly, many risk or protective factors lead to substance abuse or dependence. Field professionals have begun to recognize that these factors can be classified into three types: markers (known as surface indicators), modifiers (which either augment or modify), and mediators (or causal mechanisms) (Pandina, 1996).

Cicchetti and Rogosch (1999) divide markers further into two types: fixed (e.g., sex or premature birth) and variable (e.g., exposure to peer use). However, it is possible for some variable factors to become causal factors. For instance, if a variable factor is intervened on and the intervention changes what would have otherwise been a negative outcome, the variable factor will also become a causal risk factor (Cicchetti and Rogosch, 1999).

No one risk factor leads to substance abuse. In fact, the more risk factors someone has, the greater the risk is of developing the disorder. If changing a variable factor such as a parent's favorable attitude toward drinking, for example, did not prevent the child from using alcohol, then other risk factors may have influenced this outcome (e.g., the child has a conduct disorder). Different combinations of risk factors can lead to different potentials for negative outcomes, based on the strength and nature of the individual risk factors. Some protective factors, however, may cushion the effect of the risk factor and may modify the severity of or prevent a negative outcome. Investigators have begun to identify an interaction between risk and protective factors (Johnson et al., 1992; Newcomb, 1995).

The age at which a risk factor has its greatest influence is also important to consider. Newcomb's work has begun to enlighten some of us in the field about how specific risk factors have a greater influence at certain developmental stages than others. He divides risk factors into four domains: cultural and societal, interpersonal, psychobehavioral, and biogenetic (Table 73.1). According to Newcomb, cultural and societal elements seem to influence risk factors throughout all developmental stages.

**Table 73.1. Domains of Factors Associated with Drug Use**

Interpersonal risk factors clearly delineate those factors with the most influence during childhood and adolescence. Research examples of interpersonal risk factors expected to demonstrate the most influence during childhood include the following: the effects of parental modeling (Johnson and Leff, 1999; Kandel et al., 1978); parents' belief in the harmlessness of substances (Kandel et al., 1978); parental monitoring (Chilcoat and Anthony, 1996; Duncan et al., 1998; Molina et al., 1994); family abuse (Bennett and Kempfer, 1994); family disruption, negative communication patterns, and lack of anger control in families of substance abusers (Baumrind, 1983; Reilly, 1979); and lack of closeness and involvement with children's activities, maternal passivity, and low academic aspirations (Brook et al., 1980).

The impact of parental monitoring in initiating alcohol, tobacco, or other drug use has been found to have the greatest risk for children younger than 11 years old. A higher risk of initiating marijuana, cocaine, and inhalant drug use occurs in children who are seldom monitored during middle childhood (Chilcoat and Anthony, 1996). Not only may many of these risk factors increase the probability of initiating illicit substance use, but also combining multiple factors may influence continued use. For instance, modeling by smoking parents influences children in initiating smoking, but, when coupled with poor school adjustment, low levels of parental monitoring, easy access to cigarettes, and other risk factors, early initiators are more likely to continue smoking (Jackson et al., 1998). The effect of family abuse increases the risk of substance use not only in families with that history, but also in families without substance abuse (Bennett and Kempfer, 1994).

In considering interpersonal risk factors that are more powerful during adolescence, peer influence plays an important role. Peer attitudes about use of substances predict initiation of alcohol and other substance use (Bauman and Ennett, 1994; Kandel et al., 1978). In fact, strong peer attachment rather than parental attachment has been shown to be more influential during adolescence in predicting susceptibility to substance abuse (Brook, 1980; Kandel et al., 1978). Peer influence also plays a strong role in predicting relapse. Ninety percent of teens who have a relapse do so because of peer pressure (Brown, 1993).

The third category of risk factors established by Newcomb—psychobehavioral—can influence drug use during childhood and adolescence, as well as after adolescence and throughout life. Age is a risk factor likely to influence the onset of substance use during childhood and adolescence. Youngsters who begin drinking at an early age, at 11 to 12 years, have a higher percentage of probability of meeting the criteria, set forth by the third revised edition of the DSM (DSM-III-R), for substance abuse (13.5%) and substance dependence (15.9%), compared with those who begin drinking at age 13 or 14 years (13.7% and 9.0%, respectively). Those who drink at age 19 or 20 years of age have rates of 2% and 1%. Importantly, rapid progression of alcohol and drug disorders often occurs in persons with an earlier age of onset and frequency, not duration, of use (DeWitt et al., 2000; Kandel et al., 1992). Those persons with an earlier onset have a shorter time span from first exposure to dependence than do groups with adult-onset disorders (Clark et al., 1998). The age of onset of heavy drinking also predicts alcohol-related problems (Lee and DeClemente, 1985). An early age of onset also is associated with higher risks of the use of other substances. Adults with adolescent-onset substance abuse have higher lifetime rates of cannabis and hallucinogen use disorders, shorter times between the development of their first and second dependence diagnosis, and higher rates of disruptive behaviors and major depression (Clark et al., 1998).

Academic failure and low commitment to school are other psychobehavioral influences on drug use during childhood and adolescence. Beyond early onset of use, poor academic achievement, poor social skills and competence, learning problems and poor self-esteem are found to be related to drug abuse (Scheier et al., 1999; Stacy et al., 1993). Although many early-onset adolescent substance abusers may meet criteria to diagnose dependence—and the effect of this abuse can have



devastating results on their development—many do not continue to meet these criteria during young adulthood ( [Hasin et al., 1990](#)). Much research is needed to determine which psychobehavioral factors are more likely to lead to continued abuse and dependence in adulthood. Newcomb's work does show that young adults who continue to be problem drinkers are likely to have been rebellious, nonconforming, and deviant during high school ( [Newcomb, 1997](#)). For those who do not continue to abuse alcohol, it has been suggested that they display an increasing responsibility and maturation after the age of 24 years ( [Donovan and Jessor, 1983](#)).

As noted, persons with continued symptoms of rebelliousness and antisocial behavior are more likely to abuse substances in adulthood. However, other factors such as temperament are important. Both [Cloninger \(1987\)](#) and [Babor \(1992\)](#) identified personality traits consistent with those who have poorer prognoses. Persons with Cloninger's type 2 and Babor's type B alcoholism share common characteristics such as early onset of spontaneous alcohol-seeking behavior, diagnosis during adolescence, and rapid course of onset; they are thought to have genetic precursors that put them at risk to develop substance abuse, and they have severe symptoms of deviant behavior including fighting and arrests when drinking, as well as greater psychological vulnerability. Persons with these types of temperaments also share other characteristics found under Newcomb's psychobehavioral factors that lead to continued substance use in adulthood: high novelty seeking, low harm avoidance, and low reward dependence. Patients with Cloninger's type 1 and Babor's type A alcoholism share opposite characteristics of those cited earlier and are unlikely to continue substance abuse into adulthood.

Psychiatric disorders are also classified under Newcomb's psychobehavioral risk factors that continue to influence substance abuse throughout life. Lewinsohn identifies risk factors common to SUD and the specific comorbid diagnosis of major depression disorder. Risk factors specific to SUD are tobacco use, academic difficulties, and a past episode of SUD. Risk factors specific to major depression disorder are stress, emotional reliance, physical symptoms and disease, a history of a suicide attempt, and a history of depression or anxiety. Risk factors common to both disorders are current symptoms of depression, internalizing and externalizing behavior problems, poor coping skills, interpersonal conflict with parents, and dissatisfaction with grades ( [Lewinsohn et al., 1995](#)). Research pertaining to psychiatric disorders found under Newcomb's psychobehavioral risk factors are discussed in the section of this chapter on comorbidity. Postpsychobehavioral factors are not explored because they are more pertinent to young adulthood than to childhood or adolescence.

The final domain set forth by Newcomb that contributes to risk factors related to drug use is biogenetic. Inherited susceptibility often plays a role once a person begins to use substances, usually during adolescence. Psychophysiological vulnerability to drug use usually occurs after a person is persistently exposed to substances and would typically have an effect much later in adulthood. An example could be cirrhosis of the liver resulting from chronic alcoholism. Inherited susceptibility can be explained by the neurobiological research done by Nestler ( [1994, 1995](#)). Those who have a greater genetic predisposition to develop substance abuse may be more susceptible to the influence by alcohol on gene expression. Consistent exposure of the ventral tegmentum and nucleus accumbens to alcohol and other drugs may form permanent changes in the second messenger system inside the cell. This may influence the turning on of genes, which may, in turn, influence the drive mechanism to use substance.

The dopamine D2 receptor found on chromosome 11 ( [Blum et al., 1990](#)) and allele A1 was thought to be associated with alcoholism. However, A1 is also associated with Tourette's syndrome, ADHD, and autism. Persons with variants of three different genes for dopamine seem to have a higher number of symptoms associated with ADHD. Persons with two variants have fewer symptoms and those with one variant have even fewer symptoms of ADHD. These findings may help to support a reward deficiency syndrome thought to be associated with addictive, impulsive, and compulsive behavior and personality disorders ( [Blum et al., 1996](#)).

Repeated self-administration of cocaine appears to be associated with the D1 receptor. Three dopamine receptors—D1, D2, and D3—are involved in cocaine-seeking behaviors. Activating the D1 receptor can suppress cocaine seeking in drug-experienced animals, whereas activating D2 can trigger cocaine seeking ( [Self et al., 1996](#)). Adoption studies have shown an increased risk of alcoholism of adopted-away children of persons with alcoholism ( [Goodwin et al., 1974](#)) and an increased risk for substance abuse other than alcohol in studies of adopted-away children ( [Cadoret et al., 1986](#)). However, alcohol use by adoptive parents did not increase risk of alcohol abuse in adoptive children ( [Cloninger et al., 1985](#)).

In the cases mentioned here, greater genetic susceptibility seems to be a stronger predictor of risk of substance abuse rather than exposure to adoptive parents who use substances. However, both genetic and environmental influences may be correlated to substance use initiation, whereas progression to substance abuse and dependence may be more strongly related to genetic factors alone. In adoption studies conducted by Kendler and Prescott ( [1998a,b](#)), 485 monozygotic and 335 dizygotic female twins demonstrated that cannabis use was influenced by genetic and familial environmental factors, whereas cannabis abuse and dependence were solely related to genetic factors. This was also true for cocaine use versus abuse and dependence. [Schukit \(1999\)](#) showed greater tolerance in children of parents with alcoholism. In his study, children of parents with alcoholism had to use greater proportions of alcohol before the reflex response to a stimulus was delayed to the same degree as the degree found in responses of children of parents who did not have alcoholism. In children of nonalcoholic parents, the reflex response to a stimulus was delayed to the same degree on lower proportions of alcohol. This diminished response to alcohol was also measured by subjective feelings, levels of body sway, electrophysiologic functioning, and change in three hormones ( [Schukit, 1999](#)).

Biological susceptibility may also influence the early onset of substance abuse. [Buydens-Branchey et al. \(1989b\)](#) report that persons with early-onset substance abuse have a low ratio of plasma tryptophan, a finding that suggests a decline in serotonin. The teens in this study were incarcerated for violent crimes and had a severe course. Nicotine self-administration may be explained by the nicotine receptor. Mice lacking the  $\beta_2$  subunit of the nicotine receptor (one of the 10 proteins making up the nicotine receptor) fail to self-administer nicotine ( [Picciotto et al., 1998](#)).

It seems that protective factors tend to have an extreme influence on the prevention of SUDs. In a study of resilience in the sons of fathers with alcoholism, more good life events and an internal locus of control seemed to cushion the development of SUD ( [Springer and Gastfriend, 1995](#)). Because protective factors tend to cushion risk factors, finding interventions that would turn risk factors into protective factors would be useful in treating adolescents and in reducing continued use and progression of SUDs. This conversion of risk factors into protective factors is even more important when one considers that an accumulation of risk factors appears to have a more powerful effect than an accumulation of protective factors. Examples of protective factors include a stable environment, a high degree of motivation, a strong parent-child bond, consistent parental supervision and discipline, bonding to prosocial institutions, association with peers who hold conventional attitudes and consistent, and community-wide messages against drug use ( [Glantz and Sloboda, 1998](#)).

## DRUGS OF ABUSE: CLINICAL ISSUES

### Alcohol

Alcohol continues to be the most commonly used psychoactive substance. About one-third of high school seniors have had a binge (five or more drinks) episode in the past month. The blood alcohol level (BAL) is calculated by the milligram of alcohol per 100 mL of blood. In many states, the BAL of illegal driving while intoxicated is at or above 0.10%, whereas other states use 0.08%. The psychological and behavioral effects are related to the BAL. In nontolerant adolescents, impaired judgment occurs at a BAL of 0.06%, impaired muscle coordination at 0.08%, impaired reaction time at 0.10%, impaired balance at 0.15% and confusion or unconsciousness at 0.30% ( [Dimeff et al., 1999](#)). Adolescents need to learn that excessive intake can cause death, which may occur at 0.40% to 0.50%.

Drinking alcohol causes a biphasic response; an initial rising BAL is associated with arousal, excitement, and increased confidence. This is always followed by falling BALs, which are associated with fatigue and dysphoria. Tolerance to alcohol decreases the initial stimulating effects and increases the secondary depressant effects ( [Dimeff et al., 1999](#)). Pathologic intoxication occurs when, after drinking alcohol, the adolescent suddenly becomes violent and aggressive, in contrast to his or her usual temperament. Sedation with benzodiazepines may be needed for behavior control.

Adolescents may develop nausea, vomiting, peptic ulcer disease, and gastrointestinal hemorrhaging. Alcohol withdrawal may yield the following symptoms: nausea and vomiting, tremors, increased heart rate, elevated blood pressure, sweating, anxiety, irritability, and seizures. Adolescents who drink heavily may develop a life-threatening withdrawal syndrome with fever and convulsions, although delirium tremors are not seen. Detoxification is indicated with a stabilizing dose of benzodiazepine followed by tapering doses. Adult studies have shown that symptom-triggered dosing using a withdrawal rating scale such as the Clinical Institute Withdrawal Assessment reduces the total dose, shortens the length of stay, and maintains patient comfort and safety ( [Gastfriend et al., 1998](#)).

### Marijuana

Marijuana is the most commonly abused illegal drug. The principal psychoactive agent is D-9-tetrahydrocannabinol, which is lipid soluble. The mean elimination half-life is 4 days. Acute physiologic effects include increased heart rate, conjunctival injection, photophobia, dry mouth, tremor, and bronchodilation. Acute psychological effects include euphoria or apathy, sensation of slowed time and intensified perception, and often increased appetite. For adolescents, the most dangerous effects are in the areas of cognition and psychomotor performance. [Schwartz et al. \(1989\)](#) describe the short-term memory loss in marijuana abusers that

persists for 6 weeks after last use of the drug. This loss, combined with decreased attention, may markedly impair the adolescent's ability to learn and to do schoolwork. A double-blind study of experienced pilots ([Leirer et al., 1991](#)) using a flying simulator demonstrated that a single marijuana cigarette impaired complex visual-motor functioning up to 24 hours after smoking. Of significant concern is the issue that six of the seven pilots were not aware that they were impaired. This finding may explain why so many adolescents believe they are better at driving cars when they are high on marijuana. There is extensive evidence linking fatal accidents to cannabis use ([Nahas and Latour, 1992](#)). Long-term daily marijuana users often fulfill DSM-IV criteria for substance dependency disorder. Abrupt cessation after long-term heavy use has been reported to result in a withdrawal syndrome characterized by insomnia, irritability, restlessness, drug craving, depression, body aches, and general malaise ([Duffy and Milin, 1996](#)). Marijuana use may precipitate anxiety, panic, and, rarely, psychosis. Development of toxic delirium suggests that the marijuana is adulterated with PCP.

### **Hallucinogens**

LSD showed a resurgence of use by adolescents during the 1990s. Whereas the sugar cube dosage of the 1960s contained 150 to 250 µg of LSD, the 1-inch square "hit" blotter paper dosage of the 1990s contained 50 to 80 µg. Adolescents often take two or more hits at a time. Physical effects of a "trip" include dilated pupils, increased body temperature, increased heart rate and blood pressure, decreased appetite, sleeplessness, dry mouth, and tremors. Psychological effects vary greatly and may include altered states of perceptions of reality, distorted feelings and experiences of time and space, and impaired perceptual motor performance. Visual hallucinations may occur. "Bad trips" involve experiences of anxiety, panic, and paranoia. Flashbacks in which the adolescent reexperiences an LSD trip, although he or she has not recently ingested any LSD, are quite frequent, especially in heavy, chronic users. Psychotic disorders and affective and anxiety disorders have developed after LSD experiences.

Milder hallucinogens used by adolescents include mescaline, from the peyote cactus, and psilocybin, which comes from mushrooms. Jimson weed, whose seeds when chewed may produce a hallucinatory experience, contains atropine and scopolamine and may produce an anticholinergic syndrome.

### **Dissociative Anesthetics**

PCP was developed as an anesthetic but was discontinued because patients became agitated and delusional. PCP is illegally manufactured and is sold for a low price. It can be ingested, snorted, smoked, or injected intravenously. As a powder, it can be dusted on tobacco or marijuana, and adolescents may smoke it without knowing that it is present. Physical and psychological effects are extremely variable. Feelings of detachment and derealization are common. PCP may produce euphoria, dysphoria, paranoia, perceptual distortion, psychosis, aggressive and violent behavior, depression, seizures, coma, and even death. Neurologic signs include horizontal and vertical nystagmus, ataxia, dysarthria, seizures, and, at high doses, open-eyed coma. Hypertension, flushing, and sweating may also occur. Effects and presence in the urine may last for several days. Confusion with disorientation, bizarre behavior with delusions, and agitation with violence are possible ([Brown and Coupey, 1993](#)).

Ketamine is an animal anesthetic that may be snorted, smoked, or injected intramuscularly. Large doses may produce dissociation and derealization, but high doses may also produce delusions, amnesia, poor motor functions, and increased blood pressure. Death from respiratory failure may also occur. Use of ketamine is mainly by older adolescents and by young adults as a club or "rave" drug.

The other club or rave drugs include stimulants and depressants. MDMA or ecstasy is the most common club drug because of its combined stimulant and hallucinogenic effects. Decreases in heart rate, blood pressure, and body temperature may occur. An increased sense of alertness may contribute to dancing for hours, and this has led to dehydration, heart and kidney failure, and even death. Acute effects include anxiety, jaw clenching, and the positive experience of feeling a special connection to others. Higher doses may produce panic and paranoia. MDMA is the most recent example of a drug that was at first publicized as having very positive effects on enhancing psychotherapy and promoting love, but then continued experiences yielded evidence of brain damage, memory loss, and death.

GHB as a club drug is used for its intoxicating sedative effects, which can last up to 4 hours. The euphoria and sedation may be accompanied by nausea, vomiting, dizziness, and decreased respiration. GHB has been associated with overdoses, date rape, coma, and death.

Flunitrazepam, a sedative benzodiazepine that is odorless and tasteless, produces anterograde amnesia. These properties have resulted in the use of this drug for date rape and sexual assault. In addition to slurred speech and a staggering gait, flunitrazepam may cause decreased blood pressure, visual disturbances, confusion, gastrointestinal disturbances, and urinary retention.

### **Depressants**

These drugs include barbiturates, benzodiazepines, and specific sleeping pills such as methaqualone. These drugs are abused for their sedative and anxiety-relieving effects. Although rare in adolescents, physical dependence can occur, resulting in life-threatening withdrawal symptoms. Restlessness, postural hypotension, seizures, and death may ensue if detoxification is not done. Because of the long half-life of some benzodiazepines, such as diazepam (Valium), withdrawal symptoms including seizures may not occur for 7 to 10 days after use. Management of physical dependence involves gradual tapering of the dose or substitution of a same-class long-acting agent and slow tapering of the substitute ([Gastfriend et al., 1998](#)).

### **Stimulants**

Ephedrine and pseudoephedrine are mild stimulants that adolescents often obtain from convenience stores. These legal over-the-counter preparations are mixed with guaifenesin and have labels such as Mini-Thins or Magnum 357s. These names are related to their appetite suppression and exhilarating effects.

Amphetamines, which include dextroamphetamine (Dexedrine), biphphetamine, are often intermittently used by adolescents to lose weight, stay awake, or feel excited. Amphetamines may be taken orally or injected intravenously. The "rush" of intravenous use is often followed by the "crash" of dysphoria and irritability. Increasing doses of amphetamines may result in teeth grinding, repetitive picking of the skin of the face and extremities, and perseverative speech and behavior. Suspiciousness and paranoia also often develop. Withdrawal symptoms in long-term users of high doses results in dysphoria, fatigue, apathy, and somnolence lasting for several days.

Methamphetamine is a synthetic stimulant closely related to amphetamines, but its stimulating effects are stronger and last longer. Methamphetamine may be smoked, snorted, injected, or ingested by mouth. Low doses can produce agitation, decreased appetite, increased respiration, hyperthermia, and euphoria. Higher repeated doses may produce insomnia, confusion, tremors, aggressiveness, and paranoia. Cardiovascular collapse, strokes, and convulsions may result and may cause death.

Cocaine is usually snorted and is quite expensive, whereas the freebase form, a small rock called crack, is smoked and costs significantly less. It takes 8 seconds after smoking crack for the euphoria to develop, which lasts for 5 to 10 minutes. Dysphoria and paranoia may rapidly follow the brief euphoric state. Intranasal cocaine use produces intense euphoria with increased energy, dilated pupils, decreased appetite, tachycardia, and hypertension. Tolerance rapidly develops. Cocaine users tend to take repeated doses over periods of hours or days, which are commonly referred to as "runs." Depression and exhaustion follow the "runs." Heavy use of cocaine frequently causes paranoia and psychosis. Often, repeated use never achieves the initial euphoria of the first high. Cocaine produces the most intense cravings between binges, and chronic abusers describe an irresistible compulsion to use the drug again regardless of negative consequences. Certain medications used to treat cocaine withdrawal or postwithdrawal anhedonia have been studied (e.g., dopamine agonists such as bromocriptine or amantadine, antidepressants, antipsychotics and anticonvulsants), but results of controlled adult trials have not been positive ([Gastfriend et al., 1998](#)). Acute intoxication can produce delirium, confusion, paranoia, hypertension, tachycardia, seizures, cardiac arrhythmia, and death. Sudden death may occur with the first use. This apparently happened to Len Bias on June 19, 1986, when he died of a cocaine overdose after he signed a multimillion dollar professional basketball contract ([DuPont, 1997](#)). Ulcerated nasal mucous membranes and nasal septum collapse may occur with prolonged cocaine snorting.

### **Opiates: Heroin (Diacetylmorphine Hydrochloride)**

Opiates include opium, opium derivatives, and other synthetic substitutes. These substitutes include codeine, fentanyl, morphine, meperidine (Demerol), and oxycodone. Medical uses are for pain relief, cough suppression, and relief of diarrhea. Opiates may be swallowed, snorted, smoked, or injected. Heroin is the usual drug of choice for opiate addicts because it has a very short duration of action, which gives it a quick peak effect with intense euphoria. Onset of the euphoria is usually within 30 minutes after snorting, 15 minutes after subcutaneous injection, and 16 seconds after intravenous injection. Heroin use by American high school students was higher in 1997 than in any year between 1990 and 1996. The reason for this increase appears to be that middle-class white adolescents were initiating



heroin use by the intranasal route ([Schwartz, 1998](#)). An increase in emergency room visits and heroin overdose-related deaths in adolescents occurred.

Acute physiologic effects include drowsiness, pinpoint pupils (miosis), reduced cough reflex, and constipation. Higher doses may produce nausea, vomiting, and respiratory depression. Acute opiate intoxication or overdose produces miosis and respiratory and central nervous system depression. Associated hypothermia, hypotension, and bradycardia may also occur. Overdoses can occur when the use of opiates is combined with alcohol or sedatives. Overdoses also occur when potent heroin is used unexpectedly. In the emergency room, a diagnostic trial of low-dose naloxone (Narcan) at 0.1 to 0.2 mg is given intravenously to suspected addicts for heroin overdose ([Schwartz, 1998](#)). The target symptom for the naloxone administration is reversal of the respiratory depression produced by the heroin overdose.

Heroin produces rapid tolerance, so increased doses are needed to give the same euphoric effect. Physical dependence evolves, and a characteristic withdrawal syndrome develops when heroin use is terminated. Acute withdrawal symptoms begin within 8 hours, peak at 48 to 72 hours, and decrease over the next 4 to 7 days. Withdrawal symptoms include agitation, piloerection, tachycardia, mild hypertension, and pupillary mydriasis. Sweating, vomiting, diarrhea, and urinary frequency also occur. At 8 to 12 hours after termination of heroin use, anxiety is high, and tremors, shakes, muscle cramps, and joint pain develop. Although there is much discomfort, the withdrawal state is not life threatening. These symptoms are usually treated with clonidine or methadone ([Brown and Coupey, 1993](#)).

We had a teenage patient who developed an opiate withdrawal syndrome from the opium he had sprinkled on his daily marijuana joints and blunts. Clonidine was used to alleviate the withdrawal symptoms. Adolescents who use drugs intravenously (opiates or amphetamines) are at increased risk of human immunodeficiency virus infection and hepatitis B and C. These infections may result from shared needles or unprotected sexual intercourse.

## Inhalants

Inhalants are breathable chemical vapors that produce mind-altering effects. The abuse of inhalants is highest in the early adolescent years. Inhalants include industrial solvents (gasoline, paint thinner), office supply solvents (correction fluid), household aerosol propellant, household or commercial product gases (butane, freon), and medical anesthetic gases (nitrous oxide). Inhalants may produce excitation followed by depression, confusion, and disorientation. Inhalant use may produce severe irreversible consequences, which include hearing loss, peripheral neuropathy, central nervous system brain damage, and liver, kidney, and bone marrow deterioration. Sudden sniffing death syndrome occurs when the adolescent "huffing" an inhalant is suddenly surprised by another person and dies of cardiovascular collapse. It is hypothesized that the inhalant sensitizes the heart muscle to ventricular fibrillation.

## ASSESSMENT

Evaluation of the child and adolescent presenting with a possible substance abuse problem involves a full biopsychosocial psychiatric evaluation (described in other chapters), as well as specific evaluation of the substance abuse and possible consequences. In addition, any adolescent being evaluated for emotional, cognitive, or behavioral problems needs to also be evaluated for possible abuse of alcohol and drugs. In this section, the focus is on the specific evaluations of the substance abuse. The adolescent should be interviewed alone and with his or her family.

### Interview

In evaluating the teenager, the clinician should be knowledgeable about the drugs teenagers use and the types of effects that may occur. The clinician may quickly obtain an education about the drugs and the latest "druggie language" by simply asking teenagers what their peers use and abuse and what names are used. The clinician's personal experience with alcohol and drugs should not be revealed except in exceptional circumstances. An attitude of concern, interest, and seriousness, mixed with a little humor, is often best.

Substance-abusing youth often distort, minimize, and deny the existence or extent of their drug abuse and other problems. Therefore, information from parents, school personnel, and legal authorities is essential. Other family members, especially siblings, as well as concerned peers, are important sources of information. The degree of difficulty in interviewing the possibly substance-abusing adolescent will vary with the motivation and openness of the adolescent. The drug-abusing adolescent who knows that he or she has a problem and who comes willingly for help, although rare, does exist. It is much more common for the adolescent to appear because someone else (e.g., parent, judge, principal) requests the evaluation and the adolescent begrudgingly submits to the interview. This situation calls for the clinician's highest level of skill and patience. Beginning with a nonconfrontational approach is usually most helpful to engage the adolescent in the interview process. The first issue is to clarify the adolescent's understanding of why he or she is at the interview. The television character Colombo portrays how one may slowly investigate the presenting problem by asking clarifying questions with the clinician taking a position of confused ignorance. Current life circumstances, including home, family, and school relationships and level of functioning, are then explored. Peer relationships, leisure activities, employment, and self-perception are carefully reviewed. It is important to evaluate accomplishments and strengths as well as weaknesses and problems.

Finally, one explores the adolescent's use of drugs and alcohol. One helpful approach described by [Anglin \(1987\)](#) uses an ordered discussion of the substances the teenager ingests. The clinician asks questions first about dietary patterns (e.g., snack food, whether breakfast is skipped) and then questions about prescribed medications and compliance with these. Next are over-the-counter medications (including antihistamines to sedate and caffeine or ephedrine pills to stimulate) and finally substance abuse. Here questions move from tobacco to alcohol to marijuana, sedatives and stimulants, hallucinogens, cocaine, and opiates. Use of inhalants, which peaks in early adolescence, is also explored. [Bukstein \(1995\)](#) describes that evaluation of substance use behavior involves inquiring into (a) patterns of use (onset, how often, agent and route), (b) negative consequences, (c) context of use (time, place, peers), and (d) control (attempts to decrease, use more than planned). It is extremely important to explore high-risk behavior engaged in by the adolescent under the influence of alcohol and drugs. This includes driving under the influence of alcohol, marijuana, or LSD or being in a car in which the driver is high or intoxicated. Other high-risk behaviors, such as unprotected sex or becoming intoxicated on substances (e.g., alcohol or ecstasy) in a social setting in which the adolescent could be beaten or raped, are extremely important to identify because of their life-threatening nature.

Evaluating negative consequences involves exploration with the adolescent and family members about how the adolescent functions at school, at work, within the family and peer group, and at recreational activities. Other family members may be abusing substances, and it is essential to identify those members. School failure is one of the most common signs of substance use or abuse. Marijuana use impairs concentration and short-term memory and leads to schoolwork decline. Direct objective information from the school is helpful. A change in peer group or a loss of interest in previous activities, such as music or athletics, often results from drug involvement. Legal issues such as arrests and involvement with the juvenile justice system must also be ascertained.

[Halikas \(1990\)](#), in a study of adolescents and delinquency, developed specific key questions to examine patterns of use and abuse in teenagers. Questions related to alcohol are the following:

1. Do you drink on school grounds?
2. When you are truant, do you ever go drinking?
3. Do you miss school because of drinking or being hung over?

Halikas found that a positive answer to one of these questions correlated highly with severe alcohol problems. Three key questions related to drug use are as follows:

1. Do you mix alcohol and drugs?
2. Is your family concerned about your drug use?
3. When you are truant, do you get high on drugs?

A positive answer to one of these questions correlated highly with severe drug problems. [Bergmann et al. \(1995\)](#) developed specific key questions that also correlate significantly with alcohol and drug problems. These questions are as follows:

1. Do you prefer to go to places where drugs and alcohol are available?
2. Do you ever drink or use drugs more than you planned?
3. Does it take more alcohol or drugs to get you high than it used to?

## Confidentiality

Confidentiality holds with regard to what is discussed with the teenager, except it must be broken if there are concerns of safety. This means not just issues of suicide and homicide, but also if the teenager is engaging in sufficiently high-risk behavior. If the teenager is driving while intoxicated or tripping on LSD, confidentiality must be broken and the parents informed. This issue calls for the considered judgment of the physician.

A useful intervention technique to handle the issue of confidentiality during an initial assessment is the following: After interviewing the teenager, the clinician asks the parents to join the interview and asks the teenager to talk about some of his or her high-risk behavior in the presence of the parents. In this situation, the teenager reveals the life-threatening, high-risk behavior directly to the parents, so the clinician does not have to break confidentiality. In many situations, if the clinician informs the parents about the teenager's alcohol and drug abuse, the parents, because of their own denial, may be confrontational. Asking the teenager to reveal directly to the parents what has been discussed allows the clinician to maintain an alliance with the parents, so the clinician and the parents can respond to the high-risk drug-related behavior the teenager has revealed.

## Standardized Assessments

While the CAGE screening questions have not been demonstrated to be useful for adolescents, some screening and evaluation instruments have been specifically developed to identify adolescent substance abuse. The CRAFFT questions developed by [Knight et al. \(1999\)](#) have been studied in medical office screening. Two or more "yes" answers suggest a serious problem with alcohol or drugs, and referral for further evaluation is indicated. The CRAFFT questions are as follows: C: Have you ever ridden in a car driven by someone (including yourself) who was "high" or had been using alcohol or drugs? R: Do you ever use alcohol or drugs to relax, feel better about yourself, or fit in? A: Do you ever use alcohol or drugs while you are alone? F: Do you ever forget things you did while using alcohol or drugs? F: Do your family or friends ever tell you that you should cut down on your drinking or drug use? T: Have you ever gotten into trouble while you were using alcohol or drugs?

Two self-administered initial screening instruments yield scores indicating possible problems in 10 domains. This approach is important because substance abusing adolescents have problems in many areas of their life. The Problem Oriented Screening Instrument for Teenagers (POSIT) developed by Elizabeth Rahdert, Ph.D., at the National Institute on Drug Abuse (NIDA), includes 139 true-or-false questions that can be self-administered in 20 to 30 minutes. The ten domains are substance use or abuse, physical health status, vocational status, mental health status, family relationships, peer relationships, educational status, social skills, leisure and recreation, and aggressive behavior or delinquency. As a screening instrument, domains identified as possible problem areas are to be further, rigorously evaluated. The POSIT is available at no charge from the NIDA.

The Drug Use Screening Inventory ([Tarter, 1990](#)) consists of 149 self-report yes-or-no questions that take 20 to 30 minutes to answer. As with other screening instruments, the clinician must decide whether the answers are honest and valid. The 10 domains are substance abuse, behavior patterns, health status, psychiatric disorder, social competence, family system, school performance or adjustment, work adjustment, peer relationships, and recreation. Converting the scores into absolute and relative problem densities yields bar graphs showing which problem areas need more comprehensive evaluations and specific targeted treatment interventions.

Winters ([Center for Substance Abuse Treatment, 1999](#)) has developed three adolescent instruments. The Personal Experience Screening Questionnaire is a 38-item self-report questionnaire that is administered in 10 minutes. Problem severity and drug use history, along with validity scales for lying, are measured by this initial screening instrument. The Personal Experience Inventory is a more extensive 300-item self-report inventory that usually takes 45 minutes to complete. The Problem Severity Scales measure the onset, nature, degree, and duration of chemical involvement. The Psychological Scales identify the personal risk factors that may precipitate or sustain substance abuse. Validity scales estimate honesty of report. Five Problem Screens address eating disorders, sexual abuse, physical abuse, family chemical dependency, and suicide potential. The third instrument is the Adolescent Diagnostic Interview, a structured interview that evaluates the following: the presence of DSM-III-R diagnosis of psychoactive SUD (PSUD), school and interpersonal functioning, psychosocial stressors, and a rating of orientation and memory. The Personal Experience Inventory and the Adolescent Diagnostic Interview may be computer scored.

Another important assessment instrument in development is the Teen Addiction Severity Index ([Kaminer et al., 1991](#)). This is a modification of the Adult Addiction Severity Index. The Teen Addiction Severity Index is a semistructured interview given by a trained technician in 30 to 45 minutes. Using a five-point scale, the patient rates the severity of his or her problems in the domains of chemical use, school status, employment-support status, family relationships, and psychiatric status. The interviewer also rates severity and need for treatment. This instrument is being developed to be used repeatedly as a measure of change before and after treatment for follow-up studies.

## Urine Drug Screens

Urine drug tests are commonly used to detect recent use of illegal drugs. Mental health professionals and parents need to be aware of the uses and limitations of the urine drug screen ([Wolf and Sherman, 1995](#)). The urine must be obtained under observed conditions to make sure that it is the test subject's sample, that a foreign substance such as apple juice has not been substituted, and that it has not been adulterated. Most teenagers are well versed in ways to beat the urine drug screen. A small amount of bleach or blood or the taking of a diuretic with huge amounts of fluid often makes urine drug screen results negative. Drinking large amounts of water will yield a very dilute urine sample and may make the sample negative. The urine drug test is limited to the length of time the specific drug stays in the body and will therefore be present in the urine ([Schwartz, 1993](#)). Stimulants may be detected for up to 48 hours after last use, cocaine and its metabolite benzoylecgonine may be detected for up to 3 days, and opiates (morphine, codeine) may appear for up to 2 days. Short-acting barbiturates are detected for 1 day, diazepam for up to 4 days, and methaqualone for up to 2 weeks. Marijuana, which is stored in fatty tissue, may be detected in the urine for up to 4 days in recreational users and for up to a month in chronic daily users ([Schwartz, 1993](#)).

To determine whether an adolescent has smoked marijuana after an initial positive urine test, the cannabinoid:creatinine ratio can be followed in successive urine tests. Passive inhalation of marijuana smoke in an average-size, ventilated room does not produce a positive urine test, but passive inhalation in a closed car or nonventilated small room may produce a positive urine test. Urine drug screens should not be done on a routine basis. They should be done only when there is suspicion of drug use. Adolescents' resistance to giving a urine sample, although they may claim it is based on issues of freedom, independence, and privacy, is usually related to their use and abuse of drugs. Teenagers who ask, "Don't you trust me?" should be answered with, "No, show me you are not using."

## STAGES OF DRUG USE AND CORRESPONDING TREATMENT

As a result of the evaluation, the clinician should make a determination of the stage of substance abuse involvement. The American Society of Addiction of Medicine has developed treatment levels of care ([Hoffman et al., 1993](#)), which include the dimensions of treatment resistance, relapse potential, and recovery environment. These levels are presently currently revised. The following framework describes each stage of substance abuse involvement and the corresponding treatment approaches, to provide an initial strategy for relating treatment to the degree of substance involvement. The American Society of Addiction of Medicine dimensions, as well as issues of comorbidity and availability should also be considered.

### Experimental, Recreational, or Social Use

This is the beginning stage of substance use, with curiosity and doing what peers may be doing being the important factors. Teenagers often learn that drugs are fun and seek the thrill of doing something they are not supposed to be doing. They often find that substance use helps them gain acceptance by specific peers.

STAGE	TREATMENT STRATEGIES
Experimental use	1. Education
Recreational use	2. Counseling: individual and family

### TREATMENT STRATEGIES

For adolescents at this stage (experimental, recreational, or social use), education and counseling are appropriate. Teenagers use drugs in direct proportion to availability and perceived safety. Learning about the realistic dangers of drugs and alcohol is helpful. For example, although adolescents tend to view marijuana as a benign drug, the reality is that marijuana poses a serious threat to brain functioning. It decreases attention span, impairs short-term memory, and impairs complex



behavior such as driving. Interestingly, impairment in complex visuomotor behavior is not often recognized by teenagers. Counseling is also needed for adjustment issues, and parents may need help in how to set appropriate limits with rewards and consequences.

### Substance Misuse

In this stage, teenagers are actively seeking the pleasurable experiences of using alcohol and drugs. Often they have already learned that misuse helps them to escape from feelings of frustration, anger, depression, and inadequacy. At this stage, teenagers tend to use substances primarily on weekends, and there will be some deterioration in grades and adherence to rules.

STAGE	TREATMENT STRATEGIES
Substance misuse	<ol style="list-style-type: none"><li>1. Education</li><li>2. Counseling: individual and family</li><li>3. Individual and group therapy</li><li>4. Family treatments</li><li>5. Abstinence or "honest look" contract</li><li>6. Motivational interviewing</li></ol>

At the substance misuse stage, in addition to education and counseling, individual and group therapies, family treatments, and an abstinence contract may be needed. At this stage, family therapies such as strategic, structural, systemic, and behavioral approaches are important interventions. Behavioral family therapy involves parent management training as well as contingency contracting. Here, specific clear rules are established between parents and adolescents such that there are negative consequences to any drug use. Positive reinforcement is given for going to school, doing homework, avoidance of drug-using peers, and developing other recreational activities that are incompatible with drug use.

The abstinence or "honest look" contract is often very helpful ([Bailey, 1996](#)). In this situation, the teenager expresses a willingness to stop using drugs and alcohol and to stop "druggie" types of behavior. Specific rewards and punishments related to this goal are established. Unannounced urine drug screens are included. Specific consequences are dictated if adolescents are unable to abide by this abstinence contract and include attending treatment at a more intense level of care.

Brief interventions using a combination of motivational interviewing and education and coping skills development have been developed as a harm-reduction approach for the prevention and treatment of heavy alcohol drinking at college ([Dimeff et al., 1999](#)).

### TREATMENT OF SUBSTANCE ABUSE DISORDER AND SUBSTANCE DEPENDENCY DISORDER

Substance abuse disorder (SUD) and substance dependency disorder are DSM-IV diagnostic categories indicating that alcohol and drugs are taking over the adolescent's life and are causing significant problems. Drugs and alcohol are used during the week, and the peer group is primarily a drug- and alcohol-using group. Usually the adolescent has become very secretive, deceptive, and dishonest. DSM-IV abuse disorder criteria involve direct, negative consequences. These involve the following: failure of major role obligations at work, school, or home; recurrent use in physically hazardous situations; recurrent substance-related legal problems; and continued use despite recurrent social or interpersonal problems.

DSM-IV dependency disorder criteria involve serious negative consequences in addition to evidence of physical dependency (tolerance and withdrawal symptoms) and preoccupation with substance using or compulsive use. At this stage of addiction, alcohol and drugs cannot be used in moderation. These adolescents may be able to have some periods of not using alcohol or drugs, but when they return to using these substances, the use rapidly goes out of control, with the return of severe negative consequences. Induced neurobiological changes in brain functioning, especially in the reward circuitry ([Roberts and Koob, 1997](#)), underlie this disorder. Although research has not established the validity of these two disorders or the criteria differences between them for adolescents, the DSM-IV does define clinically useful syndromes that require specific intense treatment programs.

In 1991, Catalano et al. extensively reviewed the literature on adolescent drug abuse treatment and found that, in residential programs, time in treatment was related to reduced alcohol or drug use. Family participation was associated with better outcome. No treatment modality was significantly better than any other, and these investigators could only conclude that some treatment was better than no treatment.

In the 1990s, significant progress was made. NIDA specifically increased support and direction for controlled studies on adolescent drug abuse treatment and for the development of clinical researchers. The American Academy of Child and Adolescent Psychiatry published both a 10-year research review ([Weinberg et al., 1998](#)) and the "Practice Parameters for the Assessment and Treatment of Substance Abuse in Children and Adolescents" ([American Academy of Child and Adolescent Psychiatry, 1997](#)). In addition to the specific treatments for SUD, the frequently occurring comorbid ADHD, affective anxiety, and conduct disorders should be evaluated and treated in an integrated, concurrent manner ([Riggs and Whitmore, 1999](#)).

### Family Treatments

Family therapy treatment studies have demonstrated the strongest empirical support for their efficacy. [Stanton and Stadish \(1997\)](#) conducted a metaanalysis of controlled studies for the treatment of drug abuse. They conclude that family therapy or couples therapy results in significantly greater improvement across drug use outcome measures for both adult and adolescent drug abusers than does individual therapy, peer group therapy, or family psychoeducation. [Liddle \(1995\)](#) carefully reviewed and critically evaluated studies of family therapy and family-based intervention for adolescent drug abuse from 1979 to 1995. These are random-assignment clinical trials with specific family therapy models. These studies demonstrate that family therapy can retain families in treatment and can reduce adolescent drug abuse. Methodologic limitations such as varying retention rates and outcome measures, incomplete reporting of procedures, unclear diagnostic criteria, lack of attention to therapist factors, and treatment integrity make efficacy "promising but not definitive." A representative study is that of [Joanning et al. \(1992\)](#), whose Family System Therapy yielded an abstinence rate of 54% compared with 28% for Adolescent Group Therapy and 16% for Family Drug Education.

Classic family therapy is based on the hypothesis that a connection exists between family relationships and the development or maintenance of drug abuse. Family therapy targets these specific interpersonal family processes. With structural-strategic family therapy, emphasis is on establishing a coherent family hierarchy with appropriate rules and authority. [Lewis et al. \(1990\)](#) combined various different family therapy models to develop a 12-session treatment called the Purdue Model. The goals were to decrease family resistance to treatment, to redefine substance use as a family problem, to reestablish parental influence, to interrupt dysfunctional sequences of family behavior, to assess the interpersonal function of the drug abuse, to implement strategies to change family interpersonal functioning, and to provide assertion training for the adolescent. Families receiving this treatment model significantly decreased adolescent drug abuse compared with families receiving parent skill training.

Two important extensions of family therapy have significant controlled research support. [Azrin et al. \(1994\)](#) integrates family therapy with behavior therapy techniques, whereas Henggeler's Multisystem therapy ([Pickrel, 1996](#)) integrates family therapy with direct interventions in the multiple interacting systems involving the individual, school, peer group, and community.

#### FAMILY BEHAVIOR THERAPY

In this controlled treatment study, the specific active treatment procedures involved stimulus control, urge control and family-social contracting. Stimulus control included defining safe situations versus risk situations and learning to increase the safe activities and decrease the risk activities. Urge control involved describing a drug-using activity to stimulate drug-using urges, which were then interrupted and replaced with a drug-incompatible activity. The most important treatment component was the social-family contracting in which parents reinforced drug-incompatible activities, supervised home urge-control assignments, and employed written specifications of desired behavior with contingent reinforcers. The control group received only supportive counseling. Significant results were demonstrated, with abstinence rates after 6 months of treatment of 73% for the treatment group and only 9% for the supportive counseling group. Treated youth also had improved schoolwork and family relationships ([Azrin et al., 1994](#)).

### Multisystem Therapy

Multisystem therapy views the adolescent as being affected by multiple interconnected systems involving the individual, family, and extrafamily (peers, school, and

community) environment. Adolescent problem behavior, such as delinquency and substance abuse, are proposed to be maintained by dysfunctional reciprocal interactions of any of these systems. This therapeutic intervention involves assessing the strengths and weaknesses of the various systems and intervening to change current behavior and relationships. Responsible behavior among all family members is promoted as well as developing capacities to manage their own problems. Therapists work intensely with each adolescent patient and family member and see them within their home, school, and even neighborhood peer group.

[Henggeler et al. \(1993\)](#), in randomized studies of multisystem therapy compared with individual counseling or probation for chronic juvenile offenders, demonstrated reduced criminal activity continuing through a 4-year follow-up. Preliminary data on a current study on multisystem therapy for substance abusing delinquents demonstrate excellent retention rates and favorable outcomes ([Pickrel and Henggeler, 1996](#)).

Three other specific substance abuse treatment modalities have been studied in adult patients with alcoholism who were randomly assigned and whose counseling sessions were based on treatment manuals. In this research study, called Project Match, 12-step facilitation therapy was compared to cognitive-behavioral therapy (CBT) and motivational enhancement therapy. All three were effective in decreasing alcohol consumption, with 12-step facilitation therapy the most effective in promoting abstinence ([Humphreys, 1999](#)) and motivational enhancement therapy best for those with high baseline anger ([DiClemente et al., 1999](#)). These modalities need to be modified to be appropriate for the adolescents. There is some empirical support for the efficacy of CBT in adolescents, and studies of motivational enhancement therapy and 12-step facilitation therapy are being done.

### **Cognitive-Behavioral Therapy**

This therapeutic modality uses the learning principles of classic and operant conditioning along with approaches to correct cognitive distortions and underlying negative belief systems. This treatment includes learning specific techniques to deal with drugs and alcohol. Skills to refuse alcohol and drugs are taught and are practiced by role-playing exercises. For example, adolescents are taught to say “no” immediately in a firm manner and to make direct eye contact with the person offering them alcohol or drugs. They are then to suggest an alternative activity, or if that is not successful, simply to tell the person to stop asking. Cognitive-behavioral coping skills to deal with urges, to manage substance-using thoughts, and to handle emergencies and lapses are taught and practiced. Because deficits in coping skills for negative feelings and life stresses contribute to continued substance abuse, more general coping skills such as communication skills, problem solving strategies, anger, and mood management, as well as relaxation training, are taught and are practiced. A functional analysis for adolescent substance use examines internal and external triggers, short-term positive consequences, and long term negative consequences of using alcohol and drugs, as well as nonusing behavior. This information is then used to develop positive sobriety reinforcements and cognitive-behavioral relapse prevention strategies. CBT is being studied in randomized clinical trials. [Kaminer and Burlison \(1999\)](#) compared CBT group therapy with interactional group therapy in adolescents with dual disorders. Patients undergoing CBT demonstrated a decrease in severity of substance use, but CBT was not superior to interactional group therapy at the 15-month follow-up.

### **Motivational Treatment**

[Prochaska and DiClemente \(1982\)](#) state that a person in stopping an addictive behavior goes through a series of stages of change. These stages are the following: precontemplation, in which the person is not even thinking about stopping and does not recognize any problem with alcohol or drug use; contemplation, which is the stage of ambivalence when the person goes back and forth between reasons to change and reasons not to change; preparation, in which the person increases the commitment to change; action, in which the person stops using alcohol and drugs; and maintenance, in which the person develops a lifestyle to avoid relapse. People have different levels of motivation, depending on their stage of change. Therapeutic intervention involves helping the patient in an empathetic, nonconfrontational manner to move along the stages.

Motivational treatment approaches include brief motivational interventions, motivational interviewing, and motivational enhancement therapy. Brief motivational interventions consist of one to four sessions in which, after an assessment, direct feedback and advice are given in a nonconfrontational manner respecting the person's personal responsibility for making a decision. At the November 1999 American Society of Addiction Medicine meeting, Dr. Peter Monti presented a study on a single 45-minute emergency room brief motivational interview for adolescents with alcohol use-related injuries. Six- and 12-month follow-up self-reports revealed fewer alcohol-related problems, but not fewer number of drinks per occasion for those who received the intervention.

Motivational interviewing is the individual counseling approach developed by [Miller and Rollnick \(1991\)](#) in which specific interviewing techniques are used to help the patient work through ambivalence to move out of the contemplative stage. An empathetic nonconfrontational relationship is formed in which reflection and reframing help the patient to explore the pros and cons of substance-using behavior. Self-efficacy is enhanced as the patient is helped to realize his or her capacities and options while recognizing that it is the patient's decision whether to change. Motivational interviewing is being used for one to two sessions with adolescents before a CBT program.

Motivational enhancement therapy is the four-session therapy based on motivational interviewing that was specifically developed for Project Match. After extensive assessment by standard instruments, personalized feedback is given. The patient's ambivalence is clarified, motivation for change is enhanced, a change plan is developed and commitment to change is strengthened. In Project Match, motivational enhancement therapy was best for adults with alcoholism who had a high base level of anger. There is a need for motivational enhancement therapy to be studied in substance-using adolescents.

### **Twelve-Step Approaches**

Twelve-step-based treatment is one of three common types of adolescent substance abuse treatment that is described in the [Center for Substance Treatment's protocol series \(1999b\)](#). Although this has been the most common treatment model for adolescents, there has been little research on its efficacy. Because of the many misconceptions about 12-step programs, their historical beginnings and applications for adolescents are presented in some detail.

Twelve-step programs involve the recovery group process for treatment of chemical dependency that began with the development of Alcoholics Anonymous (AA). This was the strongest group movement of the 1980s and 1990s. AA began in 1935. Bill Wilson had become involved in the Oxford Group in an attempt to stop drinking and to live a better life. The Oxford Group was a self-help program in which members surrendered their will to that of God, took a personal moral inventory, confessed transgressions, made amends to others they had harmed, and helped others without thought of financial reward. While attending Oxford Group meetings, Wilson found that trying to help other persons with alcoholism often saved him from taking another drink. In the spring of 1935, while on an unsuccessful business trip in Akron, Ohio, and being close to a drink, Bill Wilson met with Dr. Robert Smith, a physician who was also struggling with trying to stop drinking by means of Oxford Group meetings. From this famous meeting, in which they helped each other, these two men visited the local hospital, where they helped a third patient with alcoholism, and AA began. There are almost 2 million members in 63,000 groups, with memberships doubling every 10 years ([Roberston, 1988](#)).

In the development of AA, Bill Wilson set up clear guidelines that enable AA to avoid problematic issues of money, politics, and powerful leaders. AA owns no property, does no fundraising, accepts no outside contributions, and is open and free to anyone who wants to stop drinking. The 12 steps were written by Bill Wilson and other early members. The 12 steps were published in *Alcoholics Anonymous* in 1939 ([Alcoholics Anonymous, 1976](#)). The 12 steps are the guide for the changes in actions, thoughts, feelings, and belief that an individual addict slowly undergoes to establish a state of recovery and to abstain from drinking. Because an addict cannot use alcohol or drugs in moderation, abstinence is the necessary goal. Working the 12 steps is an extremely concrete process that does not require abstract thinking.

The following descriptions present the first five steps and how they can be modified to make them meaningful for adolescents. [Jaffe \(1990\)](#) uses a workbook format in which the adolescent writes answers to specific questions, which are reviewed by counselors and may be presented at a group.

#### **STEP 1**

“We admitted we were powerless over alcohol—that our lives had become unmanageable.” For adolescents, the workbook has the adolescent examine in detail the negative consequences of alcohol or drug use. Putting their own and others' lives in danger and the effects on family, school, work, mood, and self-esteem in relation to alcohol or drug use are explored. The major issue is whether drugs and alcohol are destroying their lives such that they need to stop using drugs or alcohol to make their lives better. Because adolescents desire to become more powerful, the workbook emphasizes that by abstaining from alcohol and drugs, the adolescent will become powerful to have a life. While many adult programs emphasize the concept of “surrendering” and admitting that one is an addict, these approaches are not useful for adolescents. Rather, enhancing power by doing what one needs to do (i.e., stop using alcohol and drugs) instead of doing what one wants to do (i.e., use alcohol and drugs) is emphasized.



## STEP 2

"We came to believe that a power greater than ourselves could restore us to sanity." The adolescent workbook approaches this step by recognizing that a child's first Higher Power is the person that raises them. For many drug abusing or addicted adolescents, parental figures have been neglectful or abusive. Mourning the pain and sadness from the disappointments of their childhood Higher Powers enables them to begin to develop a sense of something positive in the universe that they can turn to for help. The Higher Power concept is not a religious belief, but a spiritual feeling that one can trust something positive (e.g., the group, another person, nature) to take care of those aspects of one's life that one cannot control. One needs to have trust in the stability of the world and to realize that one controls one's own behavior but not what others say or do. For many adolescents, the concrete positive feelings of their relationships with other members becomes their first Higher Power.

## STEP 3

"We made a decision to turn our will and our lives over to the care of God as we understand Him." The adolescent workbook interprets this step to involve having the adolescents make a decision to commit themselves to working the steps and to having a positive spiritual power. The teenagers are helped to recognize that they turned over their lives to alcohol and drugs. Now they are being asked to turn their lives over to a positive program.

## STEP 4

"We made a searching and fearless moral inventory of ourselves." The workbook has the adolescents answer numerous detailed questions covering all aspects of their childhood and present life.

## STEP 5

"We admitted to God, to ourselves, and to another human being the exact nature of our wrongs." Now the adolescent verbalizes his or her inventory to a counselor or his or her sponsor.

## PROVISIONS AND RESULTS

Twelve-step programs also provide the opportunity to attend free AA or Narcotics Anonymous meetings, which are conducted several times a day in almost every city and town in the United States and most other countries. It is well recognized that adolescents will return to using alcohol and drugs if they return to contact with their alcohol-using or drug-using friends. Twelve-step programs provide a recovering peer group, which is essential for continued sobriety ( [Jaffe, 1992](#)). Twelve-step programs also provide big brother or big sister relationships in the form of sponsors. Here an older member with at least a year of sobriety provides a relationship and guidance on how to work the program toward sobriety. Twelve-step programs accept that addiction is a chronic progressive disease whereby the addict is unable to control and moderate drinking or drug using, so the only viable alternative is complete abstinence ( [Humphreys, 1999](#)). For many adolescents, it is sometimes helpful for them to view themselves as being "on the way to becoming an addict" if they do not see themselves as already being one.

Although research on 12-step adolescent programs has been sparse, the CATOR [Harrison and Hoffman, 1989](#)) residential treatment follow-up indicates that those teenagers who attended two or more meetings per week were almost six times more likely to report abstinence at 1 year than those who never attended meetings. A more recent follow-up study by Winters, as described in the [Center for Substance Abuse Treatment Protocol no. 32 \(1999b\)](#), uses improved methods with a high follow-up contact rate and meaningful comparison groups. At 12-month follow-up, those completing 12-step-based treatment had an abstinence rate or minor relapse rate of 53% compared with 27% of those who needed but did not receive treatment.

Both motivational interviewing and 12-step facilitation therapy develop motivation in the adolescent to stop using alcohol or drugs through the adolescent's personal recognition of the negative consequences of substance using. Jaffe's *Adolescent Substance Abuse Intervention Workbook* has the adolescent use this framework to answer concrete, simple questions that explore 12 areas of the adolescent's life that may have been negatively affected by alcohol or drugs ( [Jaffe, 2001](#)). These include, among others, putting another's life in danger, making depression worse, messing up the body and brain, impairing school and work, breaking the law, and inability to moderate use. [DiClemente \(1991\)](#) describes four types of adolescent drug-abuse precontemplators: the reluctant, who does not know that a problem exists; the rebellious, who oppositionally will not acknowledge a problem; the resigned, who feels too hopeless to face the problem; and the rationalizing, who has excuses not to do anything about the alcohol or drug problem. Jaffe adds the following: the restricted-thinking adolescent, who has marijuana in his or her brain for 1 to 3 weeks after use that impairs short-term memory and meaningful thinking; and the faking contemplation precontemplator, who tells therapists that he or she wants to stop using drugs and realizes the problem, but in the back of his or her mind is thinking he or she is going to tell everyone what they want to hear, to be able to resume using alcohol and drugs. The *Intervention Workbook* deals with this issue by presenting the using druggie thinking (e.g., "drugs are fun") along with healthy recovering thinking (e.g., "but my life is a mess"). This workbook is completed individually and also may be presented to a group. It is extremely useful as an initial diagnostic and treatment tool to help the adolescent to develop motivation to stop using drugs or alcohol and is appropriate for use in outpatient, inpatient, and juvenile justice programs.

### Community-Reinforcement Approach

The community-reinforcement approach is an adult alcohol treatment approach with strong empirical research support in which the person's life is rearranged so abstinence is more rewarding than drinking. Treatment strategies include increasing a person's motivation to stop drinking by clarifying negative consequences and positive reinforcers, initiating sobriety with a trial period of sobriety sampling (e.g., 1 month), doing a functional analysis of drinking patterns, establishing involvement with sober peers and alcohol-free activities, rehearsing new coping skills, and coaching significant others not to reinforce drinking patterns (e.g., stop enabling). The therapist using this approach is positive, energetic, optimistic, and sympathetic ( [Miller et al., 1999](#)).

The community-reinforcement approach closely resembles the "enthusiastic sobriety" adolescent program developed by Bob [Meehan \(2000\)](#). This 12-step based program uses young, energetic, enthusiastic, recovering, well-trained counselors. They are role models demonstrating that one can have fun without drugs or alcohol. The adolescent is asked to try 30 days without alcohol or drugs. The adolescent participates in daily groups, meetings, and social functions with recovering peers who make sobriety more fun and rewarding than using drugs and alcohol.

### Using Multiple Treatments for Adolescent Substance Abuse

No one particular treatment modality has been demonstrated to be clearly superior to the others. Multiple approaches are often needed to achieve progress. The Center for Substance Abuse Treatment has developed five different protocols to study various outpatient treatments for adolescent marijuana abuse. Motivational enhancement therapy is combined with CBT therapy in two of the protocols. A third protocol combines motivational enhancement therapy with CBT and at-home family therapy. Preliminary positive results are being obtained, with more intensive treatments most beneficial for more severe abusers.

### Treatment Setting

Adolescents rarely need medical detoxification. Reasons seem to relate to the finding that adolescents tend to use multiple drugs in an episodic time course. Moreover, adolescents are usually in better general physical health than adults. In spite of this, physical addiction to alcohol, sedatives, or minor tranquilizers may occur, with life-threatening withdrawal symptoms, that necessitates detoxification in an inpatient setting. Other indications for inpatient hospitalization include danger to self or others, psychotic symptoms, and high-risk behavior that could be of a life-threatening nature. Adolescents with SUD or substance dependency often need a short period of inpatient hospitalization to be fully evaluated and stabilized. Then they may be stepped down to a day-patient or intensive outpatient program. Within these programs, family therapy, 12-step facilitation therapy, CBT, and motivational interviewing may be used. Full biopsychosocial evaluation is needed because comorbid disorders are present in 40% to 90% of these patients. These comorbid affective, behavioral, and anxiety disorders should be defined and considered for concurrent treatment. The major issue for adolescents with abuse or dependency disorders to be treated in day or intensive outpatient programs is whether they can resist returning to contact with their substance-using peers.

## COMORBIDITY

There is a higher rate of comorbidity in adolescents with SUD. Specifically, rates of mood and disruptive disorders have been found to be higher in adolescents with SUD than in adolescents without SUD. When compared with adult samples, rates of current comorbidity of SUD in adolescents with psychiatric disorders are the same as in adults, except for disruptive or antisocial personality disorders. Adults have higher rates of the latter. This finding continues to reemphasize the importance of early intervention to prevent the development of pervasive disorders ( [Kandel et al., 1999](#)).

When relapse rates are focused on in adolescents, those adolescents with comorbid psychiatric disorders and other problems, such as high stress, low social skills, a lack of involvement in productive activities or active leisure, and no follow-up interventions, are found to have a higher risk of relapse ( [Kaminer, 1999](#)).

Costello et al. looked at the onset of several disorders to determine whether they are likely to precede the development of SUD or whether they are more likely to follow the onset of substance abuse. All disorders, including ADHD, conduct disorder, oppositional defiant disorder, and anxiety disorder, occurred well before the onset of any substance use. However, depressive symptoms occurred 1 year after the onset of alcohol abuse but 2 years before the onset of smoking. Disruptive disorder and depression had higher rates and earlier onset of substance use and abuse in both sexes ( [Costello et al., 1999](#)).

[Angold et al. \(1999\)](#) point out many reasons why clinical research on comorbidity may be flawed. First, clinical studies are not representative of the general population and may be biased. However, samples on which we base prevalence of substance abuse and dependence in adolescents are done annually in the national survey results of drug use from the NIDA-sponsored Monitoring the Future Study. However, this study is based on students in high school and middle school. Many severely substance-abusing adolescents have already dropped out of high school and are not included in this survey. Many populations may not be representative of the clinical population, as well, when surveys are done.

Second, clinical sample correlates of disorders may be correlates of the referral population. The solution is to use longitudinal studies of the general population and referred, but these studies are expensive and are often underfunded. Third, several diagnostic categories have overlapping symptoms. This would provide an explanation of how ADHD and obsessive-compulsive disorder overlap but not conduct disorder and depression. Fourth, clinical research may not distinguish between heterotypical and homotypical disorders. Heterotypical depression is an ongoing process that manifests different forms over time. Heterotypical depression would produce subtypes found under a diagnostic category. Homotypical depression, like dysthymia and depression, would be a phenomenon that changes over time.

Although these are problems that have occurred in comorbidity research, [Angold et al. \(1999\)](#) believe that it would be foolish to ignore the research and that the solution is to find research designs that overcome these problems in the future. The following comorbidity research has been divided into categories by diagnosis, to allow a more organized discussion.

### Mood Disorders

Depression in women is related to substance abuse. Among referred adolescents, conduct disorder, major depression, and ADHD may be important concomitants of substance dependence. In female patients, however, depression may be the primary variable related to substance dependence ( [Whitmore et al., 1997](#)). Another study demonstrated that posttraumatic stress disorder and depression were strongly associated with alcohol dependence in female patients ( [Clark et al., 1997](#)). Predictors of comorbid alcohol and substance abuse in depressed adolescents girls were longer depressive episodes, more conduct problems and psychosocial impairment, and school or work problems ( [King et al., 1996](#)).

In a study of female patients with conduct disorder and substance abuse, major depression was related to a more severe level of substance abuse ( [Riggs et al., 1995](#)). Another study that compared depressed adolescents girls without previous SUD to controls found the risk of SUD high in both groups (34.6% in the depressed group and 24.2% in the control group). However, depressed girls with SUD had more significant psychosocial impairment than depressed girls without SUD, and girls with depression had earlier onset of SUD than girls without depression. The girls with depression and SUD had more anxiety traits and elevated cortisol levels near sleep onset, when the hypothalamic–pituitary system is expected to be less active, than did depressed girls without SUD ( [Rao et al., 1999](#)).

In a study of 99 delinquent boys with comorbid substance abuse and conduct disorder, depression played a significant role. Depressed boys were more likely to have ADHD, posttraumatic stress disorder, and anxiety disorders as compared with nondepressed boys. Depressed boys tended to develop conduct disorder earlier than nondepressed boys ( [Riggs et al., 1995](#)). Although conduct disorder and depression seem to increase the risk of earlier onset of SUD, this may not be true for bipolar disorder. One study looked at the risk to develop SUD independent of the diagnosis of conduct disorder. In a study by Wilens, youth with adolescent onset bipolar disorder were at significant risk relative to those who developed child onset bipolar disorder. Adolescents with bipolar disorder had an 8.8 times greater risk for the development of SUD than those with child-onset bipolar disorder, and conduct disorder or other comorbidity did not account for the risk ( [Wilens, 1999](#)).

The question whether dysthymia or major depression precedes or follows the onset of PSUD has been examined in adolescents in a few small studies. Among hospitalized adolescent substance abusers with dysthymia and PSUD, 53% had dysthymia before the development of the PSUD ( [Hovens et al., 1994](#)), and in a study of adolescents with dysthymia and behavioral symptoms that were indicative of poor behavior skill, moderate to heavy alcohol consumption followed this history ( [King et al., 1993](#)).

In a study of 156 inpatient adolescent substance abusers with major depression, 60.4% had secondary depression and 16.7% had the primary form ( [Bukstein et al., 1992](#)). In a study examining comorbid conduct disorder, substance abuse, and depression in delinquent boys, the depression did not seem to be secondary to the substance abuse because the depressive symptoms were not alleviated after 4 weeks of abstinence ( [Riggs et al., 1995](#)). Research is needed to delineate better whether dysthymia or major depression with or without conduct disorder is more likely to occur before or after the development of SUD.

### Anxiety Disorders

Results of the International Consortium in Psychiatric Epidemiology demonstrate that the onset of anxiety disorders is more likely to precede that of substance disorders in all countries ( [Merikangas et al., 1998](#)). In considering anxiety disorders, different types may play different roles in the risk of development of SUD. In a treatment population with early-onset alcoholism, 50% had at least one lifetime anxiety disorder, and the most common anxiety disorder (25%) was posttraumatic stress disorder ( [Clark et al., 1995](#)).

Social phobia may play a major role in the development of SUD when anxiety disorders are considered. For instance, social phobia was highly comorbid with depressive disorder, somatoform disorder, and SUDs in a study of 1,035 adolescents, ages 13 to 17 years ( [Essau et al., 1999](#)). In one study (from epidemiologic surveys, field studies, and family studies), social phobia and agoraphobia preceded the onset of alcoholism, whereas generalized anxiety disorder and panic disorder did not ( [Kushner et al., 1990](#)). This finding may provide clinicians with unique opportunities for intervention when they evaluate children with this history who would not present in schools as likely candidates for intervention. Their compliant behavior in the classroom does not bring their needs to the attention of a teacher.

Teachers would recognize only shy children with aggression as those who need intervention. This combination is extremely important when one considers that boys with shyness and aggression have a higher risk of the development of SUD boys with than aggressiveness alone. In fact, 26 years after following-up boys with aggressiveness and aggressiveness and shyness, both traits and combinations were valid predictors of current levels of cocaine use in adults ( [Swan, 1995](#)). In another study, the most common psychiatric disorders associated with inhalant use were social phobia, major depression, and antisocial personality disorder (Compton et al., 1994).

### Attention Deficit Hyperactivity Disorder

Patients with ADHD and conduct disorder are at a higher risk of developing substance abuse than those with ADHD alone ( [Wilens et al., 1996](#)). In fact, in a prospective study following-up two groups of children for 8 years (one with ADHD and conduct disorder and one with ADHD alone), the ADHD–conduct disorder group used two to five times more alcohol and cigarettes than the ADHD group alone ( [Barkley et al., 1990](#)). Genetic research for the increased risk for substance abuse in adolescents with ADHD and conduct disorder has been confirmed in a twin study. In this study with 626 pairs of 17-year-old twins, ADHD did not increase the risk of substance abuse unless it was associated with concomitant conduct disorder ( [Disney et al., 1997](#)).

The drug of choice for adolescents with ADHD may be marijuana. In a 10-year follow-up study of children with ADHD, the most commonly used drug was marijuana



([Mannuzza et al., 1993](#)). Although a great risk for the development of substance abuse in adolescents with ADHD is not readily apparent, adults with persistent ADHD had an increased risk of developing substance abuse (52%) when compared with controls (27%). In fact, adults with ADHD without antisocial personality disorder had a twofold elevation of PSUD as compared with controls ([Biederman et al., 1995](#)).

### Conduct Disorder

Adolescents with conduct disorder that occurs before the development of SUD have a poorer prognosis than those who develop conduct disorder during the SUD ([Myers et al., 1998](#)). Differences between female and male patients with conduct disorder and SUD have been examined. Girls with comorbid conduct disorder and SUD are more likely to have experimented with nonprescription diet pills and caffeine, have a diagnosis of nicotine dependence, start drinking at a later age, have an interval between alcohol use and depression that is shorter than in boys, and have more running-away behavior ([Mezzich et al., 1994](#)).

Boys in the differences study were more likely to have had more aggressive symptoms of conduct disorder including stealing (with and without confrontation), destruction of property, weapon fights, breaking and entering, and torturing of animals. Conduct severity (aggression), ADHD, and major depression predict an earlier age of onset of conduct disorder and severe dependence in boys. As noted earlier, only major depression relates to more severe substance dependence in girls ([Riggs et al., 1995](#)).

As noted earlier, patients with early-onset ADHD and conduct disorder have a high risk of substance abuse, but late-onset conduct disorder not associated with ADHD has also been shown to be a risk for substance abuse ([Grillo et al., 1996](#)). In this study, Grillo et al. suggest that late-onset conduct disorder (which they believe is a form of personality dysfunction) is more likely to be associated with substance abuse than is early-onset ADHD and conduct disorder (which they believe is associated with a neurologic problem secondary to ADHD). More research is needed to determine whether the foregoing hypothesis is true.

[Randall et al. \(1999\)](#) studied what is known about the effects of externalizing and internalizing juvenile offenders. In this study, juveniles with externalizing disorders versus those with internalizing disorders were examined. Comorbidity for externalizing disorders was associated with higher rates of antisocial behavior and predicted a worse 16-month outcome than did substance abuse alone or substance abuse with comorbid internalizing disorders. The presence of internalizing disorders buffered the deleterious effects of externalizing disorders on substance-abusing and substance-dependent juvenile offenders ([Randall et al., 1999](#)).

### Sexual Abuse

In a study of 174 children, two groups were matched for age, race, sex, gender, socioeconomic status, and psychiatric diagnosis. The results revealed that on every measure of substance abuse, the abused group had scores indicative of more serious abuse involvement. It also revealed that the abused group used marijuana and stimulants more frequently, got drunk more often, and initiated drug use at a younger age. The abused group also was more likely to use drugs or alcohol to deal with problems or to avoid feelings of inadequacies ([Hussey and Singer, 1993](#); [Singer et al., 1989](#)).

### Suicide Risk

Substance use can increase the risk of suicide. Psychological postmortem studies of adolescents who committed suicide found that 70% were drug and alcohol users ([Shofii, 1988](#)). Among 10 to 19 year olds, those who committed suicide were found to be 4.9 more likely to have been drinking ([Brent et al., 1987](#)). In fact, white boys who were intoxicated were more likely to commit suicide using a firearm. In that study, depression, conduct disorder, and personality disorder accompanied by a precipitating event were frequently present ([Kaminer, 1992](#)).

Other risks factors found in suicide victims are active substance abuse, comorbid major depression or affective illness, suicidal ideation within the past week, family history of depression and substance abuse, legal problems, and the presence of a hand gun in the home ([Brent et al., 1993](#); [Bukstein et al., 1993](#)). With regard to suicidal thoughts, when these thoughts coincide with morbid ideation for an extended duration, the probability of suicide increases ([Deykin et al., 1994](#)). Persistently high levels of problem drinking and depressive symptoms have also been associated with higher levels of suicidal thoughts and attempts. In this study, suicide ideation could be distinguished from suicide attempts. Higher levels of depressive symptoms and greater cigarette use was found in the youth who attempted suicide (Windle and [Windle, 1997](#)).

Cultural affiliation can put youth at higher risk of suicide. A study of 3,094 high school students surveyed revealed that native Hawaiian cultural affiliation rather than ethnicity was predictive of suicide attempts. Rates of suicide attempts in native Hawaiians were greater than non-Hawaiians. Depression, substance abuse, grade level, cultural affiliation, and mean wage earner's education predicted suicide attempts in native Hawaiians, whereas depression, substance abuse, and aggression predicted suicide attempts in non-Hawaiians ([Yuen et al., 2000](#)).

### Eating Disorders

Binge eating in anorexia nervosa predicts later onset of substance abuse ([Strober et al., 1997](#)). However, patients with bulimia have been found to have a higher incidence of substance abuse than those with restrictive anorexia ([Bulik et al., 1992](#)). We have also shown that bulimic women with substance abuse have higher novelty seeking than bulimic women without substance abuse ([Bulik et al., 1994](#)). It would be interesting to study whether these high novelty-seeking bulimic women would more likely to have personality disorders, as suggested in Cloninger's type 2 high novelty-seeking individuals.

## PSYCHOPHARMACOLOGY

The number of studies on psychopharmacology in adolescents with comorbid substance abuse is minimal. The most commonly asked question is whether to treat an adolescent with a substance abuse problem and ADHD with psychostimulants. Two articles have described the effectiveness of bupropion (Wellbutrin) and the less addictive stimulant pemoline (Cylert). Both studies found these medications effective in relieving the symptoms of ADHD in a juvenile justice population ([Riggs et al., 1996](#); [Riggs et al., 1998](#)). Caution should be used in using pemoline because of the increased risk of liver failure, and monitoring of liver functions is necessary.

Clearly, if an adolescent has a history of substance abuse and ADHD, a trial of bupropion would first be warranted. If the attentional symptoms are relieved but problems with hyperactivity continue, a trial of guanfacine (Tenex) or clonidine may prove helpful. Adolescents with ADHD, a tic disorder, and a substance abuse history may be effectively treated with nortriptyline. In all cases, weekly random urine drug screens should be done to avoid any complications that may result from using psychotropic medications with illicit drugs. If other medications are not successful, stimulants such as methylphenidate may be used ([Riggs, 1998](#)). However, the medication must be allocated by a responsible parent or guardian who does not have an active SUD. Obviously, the patient should be enrolled in some type of substance abuse treatment, and periodic urine drug screens should be monitored. One article finds that if an adolescent does have a diagnosis of ADHD and is treated, the risk of developing a substance abuse problem is reduced by 85% ([Biederman et al., 1999](#)).

A few articles have addressed the psychopharmacologic treatment of mood disorders in adolescents with substance abuse. Fluoxetine proved to be effective in the treatment of depression in drug-dependent delinquents ([Riggs et al., 1997](#)). Geller et al. reported one of the most interesting findings in adolescents with comorbid bipolar disorder and substance abuse. In a double-blind and placebo-controlled study of lithium for adolescent bipolar disorders with secondary substance dependence, the lithium not only relieved the symptoms of bipolar but also decreased the use of alcohol ([Geller et al., 1998](#)).

Naltrexone (ReVia), which has been used to decrease alcohol cravings in adult patients with alcoholism, was helpful in a case report of a 17-year-old alcohol-dependent boy ([Wold, 1997](#)). Disulfiram (Antabuse) has been used as aversion therapy in adult patients with alcoholism, but it probably has limited use in adolescents because of issues of noncompliance ([Solhkhah and Wilens, 1998](#)).

## PREVENTION

As pointed out by Hansen, some of the decline of drug use in the early 1990s may be attributed to the Omnibus Anti Drug Act. This act provided funds to schools and communities to decrease drug use. However, because drug use began another upward trend in the mid-1990s, it is clear that the effects of these programs were not long term. Most prevention programs now focus on research to find effective programs with long-term results ([Hansen, 1996b](#)).

According to Hansen, evidence now exists that certain aspects of program development are essential to provide effective interventions. They are: program focus,

delivery technique, evaluation and training, and support ( [Dusenbery and Falco, 1995](#); [Hansen, 1991](#); [Tobler and Stratton, 1997](#)).

Program focus describes what the expected outcome is and what it is supposed to change. According to Hansen, the three periods of program development are institution driven, theory driven, and data driven. Earlier efforts formulated by institution-driven programs allowed researchers to test the programs and to learn from their mistakes. One such well-intentioned effort was the DARE program. Results demonstrated that drug use in control schools and those using the DARE program was almost equal. No substantial short-term or long-term effects were documented ( [Ennett et al., 1994](#)).

Some programs ( [Hansen, 1996b](#) ) grew out of research and hence were called theory-driven programs. The problem with these programs is that resources needed to evaluate these programs were minimal. Hansen reviewed the effectiveness of programming outcome components such as information, affective education, social influence, and multiple components. Overall, programs that had an informational or affective component had very little effect, whereas those that used social influence approaches, or life skills approaches with social influence approaches, were the most effective. Examples of these types of programs include Project SMART ( [Hansen et al., 1988](#) ), Life Skills Training ( [Botvin et al., 1990](#) ), and Project STAR ( [Pentz et al., 1989](#) ).

The third type of program to develop was based on data-driven prevention. Hansen pointed out that data-driven prevention programs used only variables that had strong statistical relations to drug use, whereas theory-driven programs used all variables. Hansen, in 1993, performed a metaanalysis on 242 studies and found 11 major variables in etiologic studies. The variables that had a strong statistical relationship including drug use by peers, previous drug use, bonding, commitment to school, and deviance. Variables with average statistical relationships were beliefs about psychological and social consequences of drug use and attitudes about drug use, pressure to use substances, and perceived attitudes about drug use among others. Belief about health consequences was not as strongly correlated. Weakest were home factors, psychological traits of parents, parental marital status, parental education, family composition, and socioeconomic status. Parental attentiveness, parenting style, and parental drug use had higher correlation ( [Hansen et al., 1993](#) ). Just as Newcomb's work has helped to indicate at what developmental stage risk factors for substance abuse occur and when these targets should be intervened on, Hansen's work can help to prioritize which of these risk factors should be intervened on initially because of a stronger statistical relation to the development of substance abuse.

Three targets of prevention programs have been found to have strong correlations to predicting future drug use, and therefore targeting those factors in prevention programs may have the most success: normative beliefs, lifestyle-behavior incongruence, and commitment. The only program developed to target these variables specifically has been the All Stars program ( [Hansen, 1996a](#) ). Clearly important to analyze in the effectiveness of any program is delivery technique. It has been determined that interactive programs are far more effective than noninteractive ones ( [Tobler and Stratton, 1997](#) ). All programs require training and support. A strong initial dose with booster interventions for at least 2 years is also essential ( [Botvin et al., 1995](#) ).

In the sixth triennial report to Congress, Alan Leshner, Ph.D., included some other important prevention programs, as discussed later ( [Leshner, 1999](#) ). One type of multicomponent program, the Seattle Social Development Project, has been developed to enhance protective factors. It is designed for elementary school-age children in first through sixth grades. It involves parent and teacher training. Teachers are trained to use active classroom management, interactive teaching strategies, and cooperative learning ( [Hawkins et al., 1992](#) ).

A specific prevention program aimed at high school athletes, to avoid steroid use, is called Adolescents Training and Learning to Avoid Steroids (ATLAS). Not only has this program proved effective in reducing anabolic steroid use, it also has increased belief in athletic ability and strength training and has improved nutrition and exercise behaviors ( [Goldberg et al., 1996](#) ).

A program that targets youth with poor academic achievement and potential for dropping out of school is called the Reconnecting Youth Program. The program uses skill to build resilience and also targets youth with substance use, depression, and suicidal ideation. Research also shows this program to be effective in reducing the three-targeted behaviors stated earlier ( [Eggert et al., 1995](#) ). Of course, for clinicians who are evaluating children and their families, recognizing potential risk factors and addressing them during the treatment process are important to prevent development of substance abuse.

A population that is only beginning to receive attention is that comprising adolescents in the juvenile justice system. One study looked at grouping of delinquent boys over a 2-year period. It compares pairing of boys as follows: one delinquent and one nondelinquent, two delinquents, and two nondelinquents. In the mixed pair and the pair with a nondelinquent and a delinquent member, interaction centered on normative talk and laughter. The delinquent pair interaction was typical of interaction that increased self-reported delinquent behavior. Therefore, interventions that pool together high-risk youth into homogenous groups should be avoided ( [Dishion et al., 1996](#) ).

Two family interventions have been evaluated and found to be effective on parents' child management practices and on child-parent affective quality. The programs are Preparing for the Drug-Free Years and the Iowa Strengthening Families Program. Preparing for the Drug-Free Years involves five sessions for parents and one for the children. The Iowa Strengthening Families Program involves seven sessions attended by both the parents and their children. A 1-year follow-up of the Iowa Strengthening Families Program showed increased youth resistance to peer pressure to use drugs, reduced interaction with other antisocial peers, and decreased levels of problem behaviors ( [Kosterman et al., 1997](#); [Spath et al., 1998](#) ). Project Family evaluated these programs.

One family program has been developed that has been culturally sensitive to African-American, Asian-Pacific Islander, and Hispanic families. It is targeted at substance-using families and their 6- to 10-year-old children and has three components: a parent-training program, a children's skills training program, and a family skills training program. The positive outcomes of this 14-weekly session program include the following: reductions in family conflict; improvement in family communication and organization; and reductions in youth conduct disorders, aggressiveness, and substance abuse ( [Kumpfer et al., 1995](#) ).

Another program that targets minority students is called the Jump Start Program. It targets high sensation-seeking African-American students. The most important results of this program are that high and low sensation-seeking behaviors are neutralized ( [Harrington and Donohew, 1997](#) ). A family program that is aimed at parents using methadone treatment and their children is called the Focus on Family Program. This program has shown parent drug use significantly lowered and their parenting skills improved. It consists of a 5-hour family retreat, 32 parent sessions, and 12 sessions with children, in which they practice appropriate skills to interact with their parents ( [Catalano et al., 1996](#) ).

## CONCLUSION

In summary, all mental health professionals who treat adolescents need to be knowledgeable about the role and treatment of substance abuse in this population. Since the early 1990s, escalating progress has been made as effective treatments are being developed and clinical research has been expanded.

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Betty Pfefferbaum, M.D., J.D.

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## DEFINITION

The definition of trauma varies with context. In the broadest sense, it includes both physical and psychological injury or wound ( [The American Heritage Dictionary, 1992](#)). Posttraumatic stress disorder (PTSD) and acute stress disorder (ASD), as diagnoses, refer to a characteristic set of psychological and physiologic symptoms following exposure to a stressor or traumatic incident.

## HISTORICAL NOTE

Psychological reactions to severe stress have been recorded for thousands of years. Early depictions date to the Old Testament and Roman Empire, with more scientific descriptions emerging by the 17th century ( [O'Brien, 1998](#)). Commonly applied to combat situations, a number of terms have been used to describe these reactions—battle fatigue, shell shock, combat neurosis, and war neurosis, to name a few. The descriptions include similar symptoms such as anxiety, reenactment, sleep disturbance and nightmares, amnesia, dissociation, unresponsiveness, numbness, detachment, social isolation, hypervigilance, irritability, concentration difficulty, and impulsiveness ( [Herman, 1992](#); [Kentsmith, 1986](#); [O'Brien, 1998](#); [van der Kolk et al., 1994](#)) and are consistent with the diagnostic criteria for PTSD in the Diagnostic and Statistical Manual series ( [American Psychiatric Association, 1980, 1987, 1994](#)). The Vietnam War and its aftermath brought widespread social and professional attention to the psychological sequelae of trauma, creating an intense debate about the existence of a unique disorder associated with trauma exposure. Interest in the condition has resulted in extensive research on the subject with subsequent modification in psychiatry nosology. As with many disorders, recognition of the condition in children came later.

The psychiatric nomenclature that guides diagnosis is codified in a series of manuals published by the American Psychiatric Association ( [1952, 1968, 1980, 1987, 1994](#)). The first *Diagnostic and Statistical Manual of Mental Disorders* (DSM) ( [American Psychiatric Association, 1952](#)), published in 1952, included a category of transient situational personality disorders for acute symptom responses to overwhelming situational stress in which no “apparent underlying personality disturbance” was present (p. 40). In this category were several diagnoses, including gross stress reaction in which the individual responded to “overwhelming fear” associated with severe or unusual stress (p. 40). These reactions were reversible but could progress to other diagnoses. Responses that persisted were considered an indication of more severe underlying problems and were classified elsewhere. Other diagnoses in this category included adult situational reaction and adjustment reaction of infancy and childhood.

In the DSM-II ( [American Psychiatric Association, 1968](#)), the transient situational disorders category was renamed transient situational disturbances. It was reserved for “transient disorders of any severity (including those of psychotic proportions)” in individuals with no “apparent underlying mental disorders” (p. 48). The diagnosis was to specify the cause and manifestations of the disturbance if they were known. Symptoms were thought to diminish as the stress decreased in those with good adaptive functioning. Persistence of symptoms after the stress disappeared warranted another diagnosis. The disorder was classified according to the patient's age as, for example, adjustment reaction of adult life or adjustment reaction of childhood.

Posttraumatic stress disorder first appeared by that name in the DSM-III ( [American Psychiatric Association, 1980](#)). DSM-III required a “recognizable” stressor (p. 238) “generally outside the range of usual human experience” (p. 236) that “would evoke significant symptoms of distress in almost everyone” (Criterion A) (p. 238). Symptoms in each of three clusters were required: reexperiencing, numbing, and arousal. Reexperiencing could occur through recurrent and intrusive recollections, dreams, and/or “sudden acting or feeling as if the traumatic event were reoccurring” (Cluster B) (p. 238). Numbing or decreased “involvement with the external world” not present before the trauma could be evidenced through decreased interest in “significant activities,” feeling detached or estranged from others, and/or constricted affect (Cluster C) (p. 238). Arousal symptoms not present before the trauma included increased alertness or startle response, sleep disturbance, survivor guilt, concentration or memory problems, avoidance of traumatic reminders, and/or increased symptoms when exposed to situations that symbolize or resemble the stressor (Cluster D). The Cluster C symptoms did not include avoidance; rather, avoidance was associated with arousal and classified in Cluster D. The diagnosis required at least one reexperiencing, one numbing, and two arousal symptoms.

Like DSM-III, DSM-III-R ( [American Psychiatric Association, 1987](#)) required characteristic symptoms following a psychologically distressing event “outside the range of usual human experience” that “would be markedly distressing to almost anyone” (p. 250) and that would usually be “experienced with intense fear, terror, and helplessness” (p. 247). DSM-III-R included characteristic symptoms of reexperiencing, avoidance and numbing, and increased arousal. Physiologic reactivity was categorized as a Cluster D arousal symptom and avoidance was categorized with numbing as a Cluster C symptom. At least one reexperiencing, three avoidance/numbing, and two arousal symptoms were required for diagnosis. Avoidance/numbing and arousal symptoms were required to have onset after exposure to

the stressor. New to DSM-III-R was the requirement that symptoms endure at least 1 month.

DSM-III-R also added age-specific features to describe symptoms presenting in children. These included reexperiencing in action such as repetitive play, "loss of recently acquired developmental skills" (p. 250), mutism or refusal to discuss the trauma, generalized nightmares, and physical symptoms.

The current edition of the DSM-IV ([American Psychiatric Association, 1994](#)) requires exposure to "an extreme traumatic stressor involving direct personal experience of an event," witnessing an event, or "learning about" an event (p. 424) and that the individual responds with "intense fear, helplessness, or horror" (p. 428). In children, the response may involve "disorganized or agitated behavior" (p. 428). At least one reexperiencing, three avoidance/numbing, and two arousal symptoms must be present with a temporal relationship between the event and the avoidance/numbing and arousal symptoms. The symptoms must be persistent and endure for more than 1 month. DSM-IV criteria modified DSM-III-R by moving physiologic reactivity from the arousal cluster (Cluster D) to the reexperiencing cluster (Cluster B) and added a requirement that the disturbance cause "clinically significant distress" or functional impairment (p. 429). Child-specific symptoms include repetitive play, generalized nightmares, and "trauma-specific reenactment" (p. 428).

Acute stress disorder (ASD) was added as a new diagnostic category in DSM-IV. Diagnosis requires at least three dissociative symptoms either during or after the event, at least one persistent reexperiencing symptom, marked avoidance, and marked anxiety or hyperarousal. There must be "clinically significant distress" or functional impairment (p. 432). Acute stress disorder must occur within 4 weeks of exposure and last 2 days to 4 weeks. It cannot be caused by the use of a substance, a general medical condition, a brief psychotic disorder, or an exacerbation of a preexisting mental disorder.

The changes in diagnostic nomenclature over successive editions of the DSM attest to the increase in understanding of and research about the condition over the last two decades. Of note, changes have occurred in the definition of the stressor, the clusters to which specific symptoms are assigned, the required number of symptoms from each cluster, onset and duration, and the requirement of clinical distress or functional impairment.

The application of the diagnosis to children did not occur in earnest until Terr ([1979](#), [1981b](#), [1983](#)) studied the child victims of a school bus kidnapping. Terr's detailed descriptions of the children she studied present a compelling picture of the mental suffering traumatized children may experience.

## PREVALENCE AND EPIDEMIOLOGY

Exposure to traumatic events and situations is increasingly common, especially in some environments. The estimated lifetime prevalence of PTSD in the general population ranges from 1% to 14% ([American Psychiatric Association, 1994](#); [Helzer et al., 1987](#); [Kessler et al., 1995](#)). [Giaconia and colleagues \(1995\)](#) found that by the age of 18 years, more than two-fifths of youths in a community sample of predominantly working class or lower-middle-class households met criteria for at least one DSM-III-R trauma and over 6% met criteria for a lifetime diagnosis of PTSD. Owing to the nature of disasters, most studies investigate children exposed to specific stressors and yield higher rates depending on the sample used, characteristics of the event and exposure, assessment tools, and time elapsed since the event ([Cohen et al., 1998](#)).

## CLINICAL DESCRIPTION AND DIAGNOSIS

DSM-IV ([American Psychiatric Association, 1994](#)) identifies six criteria to establish the diagnosis of PTSD. The essential feature of the disorder is the development of characteristic symptoms after exposure to a traumatic event that arouses "intense fear, helplessness, horror;" children may present with "disorganized or agitated behavior" (Criterion A) (p. 428). Exposure can occur through direct experience, with witnessing, or through learning about a traumatic event that caused "actual or threatened death," "serious injury," or "threat to the physical integrity" of oneself or others (p. 427). Symptoms are categorized into three clusters: persistent reexperiencing of the stressor (Criterion B), persistent avoidance of reminders of the event and numbing of general responsiveness (Criterion C), and persistent symptoms of increased arousal (Criterion D). DSM-IV stipulates that there be at least one reexperiencing, three avoidance/numbing, and two arousal symptoms. The symptoms must endure for more than 1 month (Criterion E) and must cause "clinically significant distress" or impaired functioning (Criterion F) ([American Psychiatric Association, 1994](#), p. 429).

### Exposure and Response (Criterion A)

Exposure and response to the stressor constitute the first criterion for PTSD, the stressor criterion. By DSM-IV criteria, the stressor need not be outside the range of normal human experience as required by DSM-III-R ([American Psychiatric Association, 1987](#)), but it must cause "intense fear, helplessness, or horror" ([American Psychiatric Association, 1994](#), p. 428). A host of stressors, both natural and man-made, have the potential to evoke symptoms. Naturally occurring stressors include, for example, tornadoes, earthquakes, and medical illnesses. Man-made events include accidents, domestic and community violence, murder, terrorism, and war. Some of these are singular events; others involve chronic or repeated exposure.

It may be difficult to identify the precise nature and aspects of the event, exposure, or initial response that create the trauma. Perceived threat, emotional states and physiologic arousal during the event, and associated experiences, including losses and disruption, sometimes are included as part of exposure ([Schwarz and Kowalski, 1991](#); [Vernberg et al., 1996](#)). This is consistent with the placement of response to the stressor as part of the stressor criterion.

### Reexperiencing Cluster (Criterion B)

DSM-IV identifies five ways in which reexperiencing may present: recurrent and intrusive distressing recollections, recurrent distressing trauma-related dreams, "acting or feeling" as if the event were reoccurring, "intense psychological distress" with exposure to trauma cues, and physiologic reactivity to traumatic cues ([American Psychiatric Association, 1994](#), p. 428). In children, reexperiencing may be evident in repetitive play with trauma themes, generalized nightmares, and trauma-specific reenactment. Criterion B requires at least one reexperiencing symptom.

### Avoidance/Numbing Cluster (Criterion C)

The avoidance/numbing cluster includes both purposeful actions and unconscious mechanisms. Specific symptoms identified in Criterion C are efforts to avoid trauma-related thoughts, feelings, or conversations; efforts to avoid activities, places, or people reminiscent of the trauma; inability to recall important aspects of the trauma; greatly decreased "interest or participation in significant activities"; feeling detached or estranged; "restricted range of affect"; and a "sense of a foreshortened future" ([American Psychiatric Association, 1994](#), p. 428). Criterion C requires at least three avoidance/numbing symptoms, and they must be temporally related to the stressor. Children may not experience numbing ([Terr, 1983](#)), be unaware of it ([American Psychiatric Association, 1994](#)), or have difficulty understanding the concept ([Earls et al., 1988](#)). Evaluations of them, therefore, should include reports by parents, teachers, and others.

### Arousal Cluster (Criterion D)

Increased arousal includes sleep disturbance, irritability or outbursts of anger, difficulty concentrating, hypervigilance, and exaggerated startle response. Criterion D requires at least two of these symptoms, and as with the avoidance/numbing cluster, arousal symptoms must have a temporal relationship to the event. The heightened arousal required by Criterion D is generalized arousal; arousal in response to reminders of the stressor is a Criterion B symptom. Over time, a repeatedly traumatized child may develop symptoms of chronic stress and arousal with other signs and symptoms such as tachycardia, anxiety, hyperactivity, and impulsive behavior ([Perry et al., 1995](#)).

### Duration (Criterion E) and Onset

Many studies fail to report duration of symptoms essential to the diagnosis of PTSD and in determining impairment. Symptoms must be present for more than 1 month and may persist for months to years. If symptom duration is less than 3 months, the disorder is acute PTSD; chronic PTSD endures 3 months or longer. Symptoms usually begin within 3 months after exposure but may be delayed for months or even years. If onset occurs 6 months or more after the stressor, PTSD is considered delayed ([American Psychiatric Association, 1994](#)). Very delayed onset appears uncommon. It may occur, however, without apparent trigger and may persist ([Yule et al., 2000](#)).



## Distress and Impaired Functioning (Criterion F)

The importance of clinical distress and functional impairment in the diagnosis of PTSD has been recognized by their inclusion in DSM-IV criteria. Few studies of children address this criterion ([de Vries et al., 1999](#); [Giaconia et al., 1995](#); [Kinzie et al., 1986, 1989](#); [Mollica et al., 1997](#); [Sack et al., 1986, 1993, 1995b, 1999](#); [Weine et al., 1995](#)), perhaps because it was not specifically required by DSM-III-R, which was in use when much of the currently available research was conducted. The use of diagnoses in clinical settings, although not necessarily in research, generally reflects the presence of distress and/or impairment.

One important measure of functioning in children is school performance, although impaired functioning also may be evident in other settings and interpersonal relationships. Although some have minimized the impact of trauma on academic functioning ([La Greca et al., 1998](#); [Terr, 1983](#); [Weine et al., 1995](#)), several studies have found diminished scholastic performance in children positive for PTSD or PTSD symptomatology ([Giaconia et al., 1995](#); [Lipschitz et al., 2000](#); [Saigh et al., 1997](#); [Shannon et al., 1994](#)). Discrepancies across studies may reflect diversity of samples, stressors, and methodology.

Perhaps the most exhaustive work on this topic is that of Sack and colleagues, who examined functioning in two series of studies of adolescent refugees—a small school-based convenience sample ([Kinzie et al., 1986, 1989](#); [Sack et al., 1986, 1993, 1999](#)) and a large community-based random sample ([Sack et al., 1994, 1995a, b, 1996, 1997](#)). Their results with respect to functioning are complicated and difficult to interpret. In the school-based sample, the diagnosis of PTSD (using DSM-III criteria) was associated with impaired functioning ([Kinzie et al., 1986](#)). Most of the youths had experienced severe disruption in their education; many were in the protective environment of special educational placement; and less than one-fourth could pass the high school graduation test ([Sack et al., 1986](#)). Their culturally derived commitment to education and respect for authority undoubtedly influenced their teachers' views of them as well as their attitudes and classroom behavior ([Sack et al., 1986](#)). At follow-up 3 years later, those who received psychiatric diagnoses were more likely to have poorer social adjustment ([Kinzie et al., 1989](#)). At 6-year follow-up, functioning was described as "impressive," with almost two-thirds in college and about one-third married ([Sack et al., 1993](#), p. 436). The 12-year follow-up revealed persistent high rates of PTSD (using DSM-III-R criteria) without major functional impairment, although the investigators acknowledged limitations in their measure of functioning ([Sack et al., 1999](#)).

In their second series of studies, Sack and colleagues ([Sack et al., 1994, 1995a, b, 1996, 1997](#)) examined a larger random sample of approximately 200 adolescents and young adults and their parents or guardians. There were few significant differences in functioning between those with and without PTSD. Many were in, or had graduated from, college and many were employed. Almost 20% of those with PTSD and/or depression had sought mental health treatment, however, suggesting that their symptoms created distress, if not functional impairment ([Sack et al., 1995a](#)).

## Symptoms and Symptom Patterns

Endorsement of some PTSD symptoms is normal following trauma exposure and alone does not imply pathology. Indeed, certain PTSD symptoms can be adaptive, especially when danger persists. Intrusive phenomena may stimulate action or planning. Arousal is associated with the fight or flight response, a process promoting survival in the face of threat. Avoidance may constitute an important psychological defense. Partial symptomatology may also be disabling ([Giaconia et al., 1995](#)), and the full symptom complex may develop over time ([American Psychiatric Association, 1994](#)). This is particularly important in children because PTSD often has a chronic course that may disrupt normal development ([Gaensbauer, 1994](#); [Perry, 1994](#)). Because of developmental influences, symptoms in young children may not correspond exactly to those in adults ([American Psychiatric Association, 1994](#); [Scheeringa et al., 1995](#)).

Reexperiencing symptoms are common in traumatized children ([La Greca et al., 1996](#); [Najarian et al., 1996](#); [Silva et al., 2000](#); [Vernberg et al., 1996](#)), although avoidance/numbing and arousal may be more frequent in some situations ([Garrison et al., 1993](#)). The avoidance/numbing cluster ([Garrison et al., 1995](#); [Lonigan et al., 1998](#); [Sack et al., 1997](#); [Silva et al., 2000](#)) and arousal ([Sack et al., 1997](#); [Silva et al., 2000](#)) may be more pathognomonic.

Identification of avoidance/numbing symptoms can be problematic with respect to children if they have difficulty understanding the concept of numbing ([Earls et al., 1988](#)). Effective avoidance and numbing may mask reexperiencing symptoms, giving the impression that the child is unaffected by the trauma ([Cohen et al., 1998](#); [Stuber et al., 1991](#)). Children, and adults observing them, may also be less aware of avoidance/numbing symptoms ([Almqvist and Brandell-Forsberg, 1997](#); [Green et al., 1991](#); [Vernberg et al., 1996](#)).

## Comorbidity

Comorbid conditions are common with PTSD. They include anxiety, affective, and substance use disorders ([Asarnow et al., 1999](#); [Bolton et al., 2000](#); [Giaconia et al., 1995](#); [Goenjian et al., 1995](#); [Helzer et al., 1987](#); [Hubbard et al., 1995](#); [Kessler et al., 1995](#); [Kinzie et al., 1986, 1989](#); [Lipschitz et al., 2000](#); [March et al., 1997](#); [Sack et al., 1986, 1993, 1994, 1996](#)). In one study of over 300 youths, a lifetime diagnosis of PTSD by the age of 18 years significantly increased the risk of other lifetime diagnoses such as depression, anxiety, and alcohol and drug dependence ([Giaconia et al., 1995](#)). It may be that factors predisposing to other conditions, such as anxiety ([Silva et al., 2000](#)) and depression ([Brent et al., 1995](#)), also predispose to PTSD. In addition, some comorbid conditions—[anxiety and depression, for example—share symptoms with PTSD \(Brent et al., 1995\). Indeed, if the diagnostic criteria for PTSD were limited to symptoms more specific to the disorder, comorbidity might decrease \(Davidson and Foa, 1991\). On the other hand, one study found severity of depression associated with a number of PTSD symptoms that did not overlap with depressive symptoms \(Goenjian et al., 1995\).](#)

Comorbid conditions may precede, follow, or develop at the same time as PTSD ([Bolton et al., 2000](#); [Deykin and Buka, 1997](#); [Giaconia et al., 1995](#); [Green et al., 1994](#)); and the course of the illnesses may vary ([Sack et al., 1993, 1994](#)). [Asarnow and colleagues \(1999\) emphasize the importance of distinguishing depressive disorders that preexist and those that occur after trauma exposure. Although PTSD stems from the primary traumatic event, depression may result secondarily from persistent severe PTSD symptoms, intervening stressors, or unresolved grief \(Goenjian et al., 1995\), and it may be less enduring than PTSD \(Sack et al., 1993, 1996\). In a long-term follow-up of children in a shipping disaster, Bolton and colleagues \(2000\) found that most of those who developed PTSD also suffered from another disorder. Most cases of comorbid major depression had onset coincident with or after PTSD, occurring after recovery from PTSD in approximately one-third of these cases. Specific phobia and separation anxiety had onset close to the disaster, and specific phobia—which may reflect persistent fear and avoidance of situations reminiscent of the initial trauma—tended to persist for many years, whereas other comorbid conditions were of shorter duration. Generalized anxiety disorder, obsessive-compulsive disorder, and substance misuse among children in the study did not differ significantly from controls.](#)

## ETIOLOGY AND PATHOGENESIS

Alarm, fear, and stress all have physiologic components that prepare the body for action. These are evident in the fight or flight response, a complex interplay among several systems of the body, which leads to increased blood pressure, pulse, and respiratory rate; increased blood supply to the muscles; focused attention; and vigilance ([Guyton and Hall, 1997](#); [Perry and Pate, 1994](#); [Perry and Pollard, 1998](#)). This response dissipates with time except in cases of severe, prolonged, or chronic stress, in which it may persist and have potentially detrimental consequences, especially for the developing child ([Perry and Pate, 1994](#); [Perry and Pollard, 1998](#)). [Perry \(1994\) submits that large increases in neurotransmitter activity associated with severe and prolonged stress in children may alter the physical development of the brain, placing traumatized children at risk for developmental disorders.](#)

[Perry and colleagues \(1995; Perry and Pollard, 1998\) maintain that the fight or flight response is not adaptive in very young children exposed to chronic or repeated trauma. The child's initial reaction to trauma, such as crying, is initiated to summon the child's caretaker who may then either fight the trauma or flee with the child. If no caretaker responds, as may be the case with abused children whose caretakers are the perpetrators of the abuse, the child will abandon the crying in favor of a surrender response leading to dissociative behaviors \(Perry and Pollard, 1998; Perry et al., 1995\). The first response is to freeze—the child becomes immobile and compliant. Freezing is adaptive because it allows more intense scrutiny of the environment and it may conceal the child's presence, decreasing the likelihood of attention from a potential perpetrator. Dissociative responses allow the child to ignore external stimuli in favor of an internal focus through mechanisms like daydreaming, fantasy, and dissociative states that result, for example, in numbing, compliance, avoidance, and restricted affect \(Perry et al., 1995\).](#)

## FACTORS AFFECTING RESPONSE

By definition, posttraumatic stress conditions emerge following exposure to a stressor or traumatic event. A number of factors contribute to outcome following trauma including aspects of the event and exposure; characteristics of the victim; and family and sociocultural factors ([La Greca et al., 1996](#); [Vernberg et al., 1996](#); [Warheit et al., 1996](#)). Because each child's exposure to a traumatic event is unique, the characteristics of that individual exposure should be evaluated.

## The Traumatic Event

Characteristics of the stressor can be important in the child's response. For example, man-made events are thought to be more traumatic than natural ones ([American Psychiatric Association, 1994](#)). Community-wide disasters may prompt an outpouring of support not found when events affect only a few residents or when victims are dispersed over wide geographic areas. On the other hand, community-wide disasters may deplete available resources and thereby deter response and recovery ([Berren et al., 1989](#)).

## Exposure

Exposure takes many forms including physical presence, direct victimization, and witnessing or learning of an event experienced by a family member or close associate ([American Psychiatric Association, 1994](#)). A number of studies demonstrate the role of physical proximity in trauma response and a dose–response relationship between exposure and symptomatology ([Breton et al., 1993](#); [Goenjian et al., 1995](#); [March et al., 1997](#); [Nader et al., 1990](#); [Pynoos et al., 1987](#)). Widespread interpersonal exposure and extensive media coverage may obscure the dose effect ([Tyano et al., 1996](#)).

Physical injury as an aspect of exposure has not been well examined in children, although it is likely to relate to PTSD symptomatology ([Green et al., 1994](#)). Investigators have explored posttraumatic stress in samples of physically injured or ill children in whom the injury, illness, and/or aspects of treatment represent the traumatic stressor ([Aaron et al., 1999](#); [Butler et al., 1996](#); [Daviss et al., 2000a,b](#); [de Vries et al., 1999](#); [Stoddard et al., 1989](#); [Stuber et al., 1991](#)). For example, in a sample of severely burned children, [Stoddard and colleagues \(1989\)](#) found a positive relationship between the extent of injury and post-burn diagnosis of PTSD.

The role of interpersonal exposure through relationship to direct victims also has been established ([American Psychiatric Association, 1994](#); [Bloch et al., 1956](#); [McCloskey and Walker, 2000](#); [Milgram et al., 1988](#); [Pfefferbaum et al., 1999a,c,d](#); [Stoppelbein and Greening, 2000](#)). The closeness of the relationship between the child and victim may influence response ([Pfefferbaum et al., 1999c,d](#)), and indirect interpersonal exposure—relationship to one who knows a direct victim—also has been documented in samples of indirect or distant victims ([Pfefferbaum et al., 2000](#)).

### INDIRECT AND DISTANT TRAUMA

DSM-IV identifies both direct and indirect exposure in the diagnosis of PTSD. Television viewing as a form of indirect exposure may be associated with symptom development ([Breton et al., 1993](#); [Nader et al., 1993](#); [Pfefferbaum et al., 1999a,c](#)). [Nader and colleagues \(1993\)](#) found that television exposure contributed to PTSD symptomatology even after controlling for the effects of other forms of exposure in Kuwait children following the Gulf War. Disaster warnings have also raised concern as potential trauma-inducing events ([Handford et al., 1986](#); [Kiser et al., 1993](#)).

[Terr and colleagues \(1999\)](#) have proposed a “Spectrum PTSD” classification for indirect trauma. They examined children's responses to the 1986 Challenger space shuttle explosion resulting from three forms of perceptual exposure—observing the launch directly from the viewing stands, watching it live on television, and hearing about it later—and from three levels of interpersonal involvement—personal relationship with the teacher on the flight, residence in the same geographic region but not students of the teacher, and residence on the West Coast with no relationship to the teacher. The spectrum classification includes distant trauma (reaction to a real event observed at the time but from a distant safe site), close call (near miss), indirect trauma (reaction to an event not directly observable), vicarious trauma (reaction to a highly threatening event that was not directly observable but was nationally threatening), mass threat (reaction to a pending or possible national or worldwide event), mass hysteria (reaction to a nonspecific threat with acquisition of symptoms through social means), and copycatting (imitation of symptoms transmitted through social means).

### MULTIPLE FORMS OF TRAUMA EXPOSURE

Traumatized children are commonly exposed to a stressor in multiple ways, yet few studies have examined the relative impact or interactions among various forms of exposure ([Macksoud and Aber, 1996](#); [March et al., 1997](#); [McCloskey and Walker, 2000](#); [Milgram et al., 1988](#); [Nader et al., 1993](#); [Saigh, 1991](#); [Silva et al., 2000](#)). [Nader and colleagues \(1993\)](#) found PTSD symptomatology correlated positively with direct exposure through witnessing and with indirect exposure through television coverage but not with indirect exposure through interpersonal relationships. Nonetheless, multiple forms of exposure, such as physical and interpersonal, may increase risk for symptom development. [March and colleagues \(1997\)](#) found greatest risk for PTSD symptomatology emanating from combined physical and interpersonal exposure.

### SECONDARY ADVERSITIES AND TRAUMATIC REMINDERS

Process trauma refers to the various secondary adversities associated with a traumatic event ([Shaw et al., 1995](#)) such as displacement and relocation, property and economic loss, family and social problems, loss of interpersonal support networks, and conflict between victims and responders. Secondary adversities constitute important aspects of exposure ([Laor et al., 1996](#); [Najarian et al., 1996](#); [Pfefferbaum, 1998](#); [Sack et al., 1996](#); [Shaw et al., 1995](#)). Several studies have examined symptomatology in evacuated, displaced, or resettled children ([Breton et al., 1993](#); [Kinzie et al., 1986, 1989](#); [Laor et al., 1996, 1997](#); [Mollica et al., 1997](#); [Najarian et al., 1996](#); [Sack et al., 1986, 1993, 1994, 1995a,b, 1996, 1997, 1999](#); [Weine et al., 1995](#)) but have not isolated the specific impact of the dislocation itself. Traumatic reminders—stimulus cues that activate symptoms—may also interfere with recovery ([Pynoos, 1994](#); [Pynoos et al., 1996](#); [Shaw et al., 1995, 1996](#)). The potential effects of secondary adversities and traumatic reminders illustrate that the traumatic event does not occur in isolation of other sources of trauma, which may intensify symptoms.

## Initial Reaction

In some studies, response during the event or initial reaction is an important predictor of posttraumatic stress symptomatology ([Aaron et al., 1999](#); [Asarnow et al., 1999](#); [Garrison et al., 1995](#); [Lonigan et al., 1994](#); [Pfefferbaum et al., 1999d](#); [Schwarz and Kowalski, 1991](#)). In fact, one's subjective appraisal of danger and life threat at the time of exposure may be a better predictor of PTSD symptomatology than more objective measures of exposure ([Asarnow et al., 1999](#); [Green et al., 1991](#); [Schwarzwald et al., 1993](#)). Emotions, such as sadness, worry, fear, and loneliness during a traumatic event may also predict later symptomatology ([Lonigan et al., 1994](#)). [Schwarz and Kowalski \(1991\)](#) recommend, therefore, that they be considered an aspect of exposure. DSM-IV criteria for acute stress disorder emphasize the importance of peritraumatic dissociation, which has received relatively little attention in children ([Carrión and Steiner, 2000](#); [Daviss et al., 2000b](#); [Perry et al., 1995](#); [Terr, 1990, 1991](#)). Current methodologic difficulties in obtaining reliable and valid measures of such initial subjective reactions must be addressed to further explore this factor.

## Personal Characteristics

### DEMOGRAPHIC FACTORS

Research findings regarding the influence of demographic factors in childhood PTSD are inconsistent because the types of trauma and populations examined are highly varied. Of the demographic factors thought to be important—sex, age, intelligence, and ethnic status—only sex has been well studied and the results are contradictory ([Foy et al., 1996](#); [Green et al., 1991](#); [Korol et al., 1999](#); [La Greca et al., 1996](#); [Lonigan et al., 1991](#); [March et al., 1997](#); [Shannon et al., 1994](#); [Shaw et al., 1996](#); [Vernberg et al., 1996](#); [Yule et al., 2000](#)). Studies with large samples commonly find girls more symptomatic than boys ([Garrison et al., 1993, 1995](#); [Giaconia et al., 1995](#); [Green et al., 1991](#); [Lonigan et al., 1991](#); [Pfefferbaum et al., 1999c](#); [Shannon et al., 1994](#)), but some studies find boys more symptomatic or find qualitative differences in symptoms and recovery related to sex ([Blom, 1986](#); [Shaw et al., 1995, 1996](#)).

The influence of age on trauma is complex ([Foy et al., 1996](#); [Garrison et al., 1995](#); [Green et al., 1991](#); [Newman, 1976](#); [Sack et al., 1994](#); [Schwarzwald et al., 1993, 1994](#); [Vernberg et al., 1996](#)). It may be difficult to ascertain or interpret symptoms in very young children who, because of less mature verbal ability, may be unable to communicate certain aspects of their distress. On the other hand, several studies have documented stress responses in very young children ([Gaensbauer, 1994](#); [Terr, 1988](#)) and even infants ([Scheeringa et al., 1995](#)). The increased cognitive capacity of older children means that they are likely to have a clearer understanding of the stressor and the danger associated with it, but children should also have better developed coping skills as they mature ([Green et al., 1991](#); [Terr et al., 1999](#)). Higher intelligence also may be protective ([Silva et al., 2000](#)), whereas a history of academic problems may place children at risk ([La Greca et al., 1998](#)).

Few studies have demonstrated ethnic or cross-cultural differences in trauma response ([Foy et al., 1996](#); [Garrison et al., 1995](#); [La Greca et al., 1996](#); [March et al.,](#)



1997; Shannon et al., 1994), but some suggest that ethnic minorities may be at risk for maladaptation (March et al., 1997; Shannon et al., 1994). It is unclear, however, to what extent poorer outcome might represent exposure to other traumatic events, family influences, or differences in socioeconomic status rather than ethnicity.

In the final analysis, demographic characteristics may prove less important than exposure and other variables in predicting trauma response (La Greca et al., 1996; March et al., 1997; Sack et al., 1994; Shannon et al., 1994; Vernberg et al., 1996). These other variables include preexisting psychopathology and prior trauma (Aron et al., 1999; Asarnow et al., 1999; Boney-McCoy and Finkelhor, 1996; Daviss, 2000a,b; Earls et al., 1988; Garrison et al., 1995; La Greca et al., 1998), parental exposure (Almqvist and Brandell-Forsberg, 1997), and other family and social variables (Laor et al., 1996, 1997; McFarlane, 1987c).

#### PREEXISTING CONDITIONS AND PRIOR TRAUMA

Preexisting emotional and behavioral problems (Asarnow et al., 1999; Daviss et al., 2000a; Earls et al., 1988; Garrison et al., 1993, 1995; La Greca et al., 1998) and prior trauma (Daviss et al., 2000a; Garrison et al., 1995) adversely impact trauma response in children. This may be especially true for preexisting internalizing symptoms such as anxiety and depression (Aron et al., 1999; Asarnow et al., 1999; Boney-McCoy and Finkelhor, 1996; Daviss et al., 2000b; Lonigan et al., 1994). Asarnow and colleagues (1999) suggest that children with anxiety disorders may appraise threat as more severe and thus may experience greater physiologic reactivity associated with trauma than those without these disorders.

Prior trauma is widely believed to increase the vulnerability of children at times of stress, but few studies have actually investigated the issue and their results are not conclusive (Boney-McCoy and Finkelhor, 1996; Daviss et al., 2000a; Garrison et al., 1993, 1995; McCloskey and Walker, 2000). It is also possible that prior trauma is desensitizing. Terr and colleagues (1999) found children with prior trauma resistant to some trauma symptoms after media exposure to the Challenger explosion. Although these children developed less fear and fewer behavioral reenactments than those without prior trauma, they experienced diminished expectations about the future, which was attributed to their earlier trauma (Terr et al., 1999).

#### Family and Social Variables

Family and social mediators of posttraumatic stress include relationships within the family and social environment, the reactions of family members, family organization, and the family's long-term adaptation (Boney-McCoy and Finkelhor, 1996; Gaensbauer, 1994; McFarlane, 1987a,b; Prinstein et al., 1996; Solomon, 1989). Social factors, such as community disruption, competition for resources, and community response also may influence adjustment (Berren et al., 1989; Pfefferbaum, 1998; Quarantelli and Dynes, 1985; Solomon, 1989).

#### FAMILY RESPONSE AND PARENTAL SYMPTOMS AND FUNCTIONING

A number of studies document an association between child and parent symptomatology following trauma (Breton et al., 1993; Daviss et al., 2000a,b; de Vries et al., 1999; Earls et al., 1988; Foy et al., 1996; Green et al., 1991; Laor et al., 1996; McFarlane, 1987b,c; Sack et al., 1995b), which in some cases may reflect similar exposure. Children's reactions, however, do not always parallel their parents. In a study of families evacuated from Chernobyl, maternal somatization and maternal perception of child somatization were not reflected in the children's self-reported symptomatology (Bromet et al., 2000). The children were only secondarily exposed through parental exposure, which along with open parent-child communication, may have accounted for their general well being.

Sack and colleagues (1995b) found that the relationship between parental and child PTSD diagnosis was not mediated by amount of trauma, family separation and loss, socioeconomic status, or treatment. Parents had higher rates of PTSD and depression than their children, and mothers had higher rates than fathers (Sack et al., 1994). Onset of PTSD was generally earlier in parents than youths (Sack et al., 1995b), raising the possibility of secondary victimization of children, which has been described in clinical reports (Rosenheck and Nathan, 1985; Terr, 1981a) but not studied systematically.

Parental symptoms and poor parental functioning constitute important risk factors for symptom development in traumatized children (Green et al., 1991; Laor et al., 1997; McFarlane, 1987b,c). Specifically, problems in children appear to be associated with irritable, depressed, and overprotective families (Green et al., 1991; McFarlane, 1987a,b), especially when these characteristics occur collectively (McFarlane, 1987a). Consistency in reaction and mood between parents may also affect the intensity of the child's reaction (Handford et al., 1986), as may the quality of the parent-child relationship (Boney-McCoy and Finkelhor, 1996).

#### FAMILY RELATIONSHIPS AND ADAPTABILITY

Boney-McCoy and Finkelhor (1996) found that the parent-child relationship is important in trauma response. Positive family relationships are generally considered protective for traumatized children (Kinzie et al., 1986; Laor et al., 1997; Pynoos and Nader, 1989; Schwarzwald et al., 1994). In fact, family contact may mitigate adverse response to severe trauma (Kinzie et al., 1986), although this protection may dissipate as other life events ensue (Kinzie et al., 1989).

Family adaptability—the capacity to change the power structure, roles, and norms within the family—is likely to affect trauma response. Family cohesion—the flexibility of affective bonds—may be even more important. Both extremes of cohesion, too distant and too close, create risk for maladaptive outcome (Laor et al., 1996), although more research is needed to help clarify these family factors.

#### VICARIOUS TRAUMA

A contagious quality of trauma has been described (Perry et al., 1995; Pfefferbaum and Pfefferbaum, 1998; Rosenheck and Nathan, 1985; Terr, 1981a, 1983, 1985). Rosenheck and Nathan (1985) reported the development of PTSD symptoms in the young son of a war veteran. Perry and colleagues (1995) suggest a possible mechanism for this process, attributing "vicarious traumatization" to persistent unchecked hyperarousal in which the parent's response becomes a source of trauma to the child (p. 285). In such situations, of course, the parent is also less able to provide needed support to the child, compounding the problem. Terr (1981a) described a similar process in siblings. She found posttraumatic play in the younger sibling of a trauma victim that was then passed on to yet another sibling and speculated that the anxiety associated with the traumatic play attracted the nontraumatized child into the play.

#### INTERPERSONAL AWARENESS

Although not a consistent finding (Breton et al., 1993), a number of studies report that parents underestimate the deleterious effects of trauma on their children (Almqvist and Brandell-Forsberg, 1997; Burke et al., 1982; Daviss et al., 2000b; Earls et al., 1988; Handford et al., 1986; Korol et al., 1999; Sack et al., 1986, 1994; Yule and Williams, 1990). When it occurs, parent-child discrepancy may represent denial on the part of parents (Almqvist and Brandell-Forsberg, 1997), but children also may be especially compliant in the aftermath of trauma and may not fully display their distress (McFarlane et al., 1987; Shaw et al., 1995), perhaps to avoid burdening their parents (Mollica et al., 1997). Adults may collude in this avoidance to escape the discomfort of the posttraumatic experience—their own, their child's, or both.

#### LABORATORY STUDIES

The most commonly described response to threat is the fight or flight response involving the sympathetic nervous system. Physiologic changes in response to trauma include elevated pulse and blood pressure that may decrease in children if they begin to dissociate (Perry et al., 1995). Glod and Teicher (1996) found differences in circadian rhythm and activity level in abused children with and without PTSD. Loss of the normal inhibitory modulation of the startle response was demonstrated in a small sample of children with PTSD (Ornitz and Pynoos, 1989). De Bellis and colleagues (1994) found severe sexual abuse associated with biochemical changes in catecholamine synthesis, and Goenjian and colleagues (1996) found intrusion symptoms related to baseline cortisol levels and cortisol suppression by dexamethasone in adolescents 5 years after an earthquake.

Although physical changes associated with heightened arousal occur as part of the posttrauma response, there are no specific laboratory tests to diagnose the condition. Measures of autonomic functioning such as pulse, electromyography, and sweat gland activity (American Psychiatric Association, 1994) may demonstrate changes, but the diagnosis is made through careful history and clinical observation.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of PTSD includes a number of conditions, most notably acute stress disorder and adjustment disorder. Acute stress disorder, like PTSD, occurs after exposure to a traumatic event in which the individual responds with “intense fear, helplessness, or horror” ( [American Psychiatric Association, 1994](#), p. 431). It is distinguished from PTSD by its onset and duration and the prominence of dissociative symptoms. In contrast to PTSD, symptoms must occur within 4 weeks of the stressor and endure for only 2 days to 4 weeks. The diagnosis of PTSD requires the endurance of symptoms beyond 4 weeks; therefore, it cannot be made within the first 4 weeks following exposure to the stressor. The diagnosis of ASD places greater emphasis on dissociative symptoms but requires reexperiencing, avoidance, and anxiety or arousal as well.

An adjustment disorder is diagnosed when emotional or behavioral symptoms emerge following exposure to a stressor or stressors if the disturbance does not meet criteria for another disorder and does not represent bereavement ( [American Psychiatric Association, 1994](#)). Symptoms must occur within 3 months of exposure and may not persist beyond 6 months after the stressor terminates. Adjustment disorders are specified according to the predominance of affective, anxiety, or conduct symptoms. An adjustment disorder may differ from PTSD because the stressor may not meet the severity and response requirements of Criterion A of the PTSD diagnosis. The diagnosis of adjustment disorder may also be appropriate if Criterion A is met but the full symptom complex necessary to diagnose either ASD or PTSD does not emerge. Diagnostic criteria for an adjustment disorder also require that symptoms be clinically significant causing “marked distress . . . in excess of what would be expected from exposure to the stressor” and significant impairment (p. 626).

Posttraumatic stress disorder shares symptoms in common with other conditions as well. For example, other anxiety and affective disorders may include sleep disturbance with nightmares, social isolation and interpersonal estrangement, decreased interest or participation in significant activities, restricted range of affect, and increased arousal evidenced in outbursts of anger and difficulty concentrating. Obsessive-compulsive disorder includes recurrent intrusive thoughts, impulses or images, and repetitive behaviors or mental acts, but these are not related to a traumatic event and are experienced as inappropriate ( [American Psychiatric Association, 1994](#)). Flashbacks associated with PTSD must be distinguished from hallucinations and other perceptual abnormalities associated with psychotic conditions.

## ASSESSMENT AND TREATMENT

### Assessment

Assessment of trauma involves the traditional methods of psychiatric evaluation emphasizing the history of exposure and prior trauma, observation, the use of projective techniques such as play and artwork, and formal psychological testing. Children may not spontaneously report their symptoms and adults may underestimate trauma in children; therefore, it is essential to ask children themselves about exposure and their responses. In some situations, such as natural disasters or reported criminal victimization, exposure to trauma is obvious, and the clinician quite naturally inquires about the signs and symptoms of PTSD. In other situations, however, exposure is obscure and the need for evaluation may be less obvious. The prevalence of violence and other stressors in modern society is sufficiently great that clinicians should routinely assess exposure and symptoms in the children they see.

### Screening

[Stallard and colleagues \(1999\)](#) recommend routine screening of children after disasters, contending that most children with PTSD go unrecognized and relatively few receive treatment. Screening is especially important when groups of people are affected by incidents such as school or community disasters.

Rating scales can be used to identify those at greatest risk and to facilitate triage. These scales typically measure physical, interpersonal, and media exposure; personal consequences; initial emotional and physiologic response; PTSD symptoms; other related symptoms such as fear, anxiety, and depression; grief; and functioning. Although rating scales are inexpensive, take little time to complete, and require minimal clinician supervision, they may trigger emotional distress, introduce participant and rater bias, and create concern that the child's symptoms are less important than data collection ( [Pfefferbaum et al., 1999b](#)).

Group interviews, conducted in the classroom or other small clusters, also may be used to assess the need for more comprehensive individual evaluation and to begin the process of healing in traumatic situations that involve multiple victims. [Allen and colleagues \(1999\)](#) used this method following the 1995 Oklahoma City bombing to elicit cognitive and emotional responses, assess current adaptation, and explore coping strategies while validating feelings and encouraging support.

### The Initial Interview and Projective Assessments

In the immediate aftermath of trauma, it is often helpful for victims to discuss their experiences, clarify and validate what has occurred, describe their emotional responses, and recognize that others have survived similar experiences. Professional contact should focus on the traumatic event and encourage normalcy ( [Vernberg and Vogel, 1993](#)).

Individual work begins with a sensitive clinical interview. Nonverbal children may express their experiences through simple projective techniques, such as play, drawing, or storytelling, often revealing their distress in repetitive reenactment and graphic depictions. [Pynoos and Eth \(1986\)](#) describe an interview appropriate for assessment and to initiate treatment. The interview consists of three stages, beginning with drawing and storytelling, which invariably contain a reference to the traumatic event. This is followed by a discussion of the event, the child's perception of the threat, the consequences, and the child's feelings of fear, self-blame, and revenge. The interview concludes with a review of the session and the anticipated course of the child's response.

### Therapeutic Considerations

Treatment involves transforming the child's self-concept from victim to survivor because the trauma is resolved in a safe setting in which painful and overwhelming experiences can be explored ( [Amaya-Jackson and March, 1995](#); [Gillis, 1993](#)). A number of therapeutic considerations guide the treatment of PTSD.

The traumatic experience must be understood in the context of previous trauma, which may be rekindled and complicate recovery. The therapy process itself can evoke reexperiencing symptoms and heightened arousal. The therapist, therefore, must consider the pace at which the child can integrate the trauma and monitor the child's responses and progress.

Avoidance is a core symptom of PTSD and may prevent the initiation of treatment and impede progress once begun. Avoidance can be protective and it can decrease stress at least temporarily, but it also may be interrupted by intrusions and increased arousal that occur spontaneously or with reminders. The therapist must not only respond to what is presented, but also must be alert for important omitted information and associated feelings ( [Gillis, 1993](#); [Pfefferbaum, 1997](#)). A discussion of avoidance should be included as part of the therapy so that children and their parents are aware that it may occur and may prevent them from obtaining help or dealing with issues.

Regardless of the approach, the child and family must be educated about common posttraumatic symptoms and the usual course of recovery. Older children and adolescents may be comfortable with a direct approach ( [Gillis, 1993](#)); for younger children, projective activities such as play, art, and storytelling provide opportunities for exploring the trauma. Treatment of prominent comorbid symptoms such as anxiety and depression must be considered.

Reenactment play may represent unresolved feelings about the traumatic experience and offers the opportunity for intervention, but the therapeutic effectiveness of reenactment play has been the subject of some controversy ( [Gaensbauer, 1994](#); [Gillis, 1993](#); [Terr, 1989b](#)). Repeated reenactment alone may reinforce pathologic patterns of response, but it also allows access to the trauma-associated affect that must be interpreted for the child ( [Gaensbauer, 1994](#)). Although she does not discourage direct interpretation within the context of the play, [Terr \(1989b\)](#) maintains that play therapy may have benefit even without interpretation and that overinterpretation may confuse the child.

Cognitive distortions associated with trauma are common ( [Gillis, 1993](#)). Inaccurate attributions, such as self-blame and omen formation, should be explored and challenged. This often involves a step-by-step analysis of the child's appraisals and attributions, which can be time consuming and emotionally difficult ( [Cohen et al.,](#)



1998).

## Treatment Modalities and Efficacy

A variety of modalities are used to treat PTSD, including individual psychodynamic and play therapy, cognitive-behavioral approaches, family therapy, group work, school-based interventions, and psychopharmacology.

Unfortunately, relatively little research documents the effectiveness of treatment ( [Cohen and Mannarino, 1996](#); [Deblinger et al., 1990, 1996](#); [Field et al., 1996](#); [Galante and Foa, 1986](#); [Goenjian et al., 1997](#); [Harmon and Riggs, 1996](#); [March et al., 1998](#); [Saigh, 1987a,b](#)) or the comparative advantages of therapeutic modalities ( [Cohen and Mannarino, 1996](#); [Deblinger et al., 1996](#); [Field et al., 1996](#)). Psychotherapy ( [Goenjian et al., 1997](#)), group work ( [Blom, 1986](#); [Galante and Foa, 1986](#); [La Greca et al., 1996](#); [Rigamer, 1986](#); [Stallard and Law, 1993](#)), and cognitive-behavioral approaches ( [Cohen and Mannarino, 1996](#); [Deblinger et al., 1990, 1996](#); [March et al., 1998](#); [Saigh, 1987a,b](#)) have been used. Novel approaches also may be beneficial. [Field and colleagues \(1996\)](#), for example, found massage therapy effective in treating children following a hurricane. In the final analysis, the specific treatment modality used may be less important than the focus on the trauma and the child's reaction to it ( [Cohen et al., 1998](#); [Friedrich, 1996](#)).

### CRISIS INTERVENTION, DEBRIEFING, AND GROUP TREATMENT

Crisis intervention, debriefing, and group therapy may be used when large numbers of children are exposed to a common event. Sessions may be held in schools, hospitals, or other settings. Groups vary with respect to structure. Some include play, artwork, storytelling, and/or role-playing ( [Galante and Foa, 1986](#); [Gillis, 1993](#)). Certain topics—such as usual responses to trauma, reactions to traumatic reminders and anniversaries, and coping mechanisms—should be addressed even if not directly introduced by participants. Parallel parent groups provide a means to address parental reactions and concerns and discuss effective management.

Group work is ideal for educating children and adults about symptoms and for providing age-appropriate explanations of the posttraumatic course. Sharing with others who have experienced the same or similar trauma can be reassuring for children who are hesitant to disclose their concerns or who believe that their experience was unique ( [Yule and Williams, 1990](#)). The group format provides opportunities to explore loss and reminisce, observe a variety of coping strategies and view others at various stages in the resolution of trauma, and gain satisfaction from helping others. Group work also provides an expedient means of identifying children in need of more intensive individual assistance ( [Gillis, 1993](#); [Terr, 1989b](#)). Group debriefing sessions may be especially helpful with respect to intrusion symptoms, perhaps by providing opportunity for ventilation, validating the child's experience, and reinterpreting attributions ( [Stallard and Law, 1993](#)).

There are also disadvantages to group work. Some children are uncomfortable sharing in a group and some need individual treatment. Group discussions may retraumatize children through reexposure to their own experiences or exposure to the experiences of others, and children may adopt the coping strategies of others before fully examining their own responses. It is important to set limits on the expression of anger and aggression, which may create anxiety in peers and signal the need for individual work ( [Gillis, 1993](#); [Pfefferbaum, 1997](#)).

### COGNITIVE-BEHAVIORAL TECHNIQUES

The literature suggests that desensitization, relaxation, and other cognitive-behavioral techniques are beneficial in treating children with PTSD ( [Cohen and Mannarino, 1996](#); [Deblinger et al., 1990, 1996](#); [March et al., 1998](#); [Saigh, 1987a,b](#); [Terr, 1985, 1989b](#)), but little research has addressed the issue. Cognitive-behavioral therapy and educational information may lend structure and support when anxiety and avoidance discourage exploration. [Saigh \(1987a,b\)](#) described the beneficial use of flooding in case studies of children with PTSD. Several studies have demonstrated benefit from structured cognitive-behavioral interventions for sexually abused children and their parents ( [Cohen and Mannarino, 1996](#); [Deblinger et al., 1990, 1996](#)), and [March and colleagues \(1998\)](#) found a group-administered cognitive-behavioral approach effective in children with PTSD related to single-incident stressors.

### FAMILY THERAPY

The family has a major role in the child's adjustment to trauma and parents should be included in treatment ( [Cohen and Mannarino, 1996](#); [Deblinger et al., 1990](#); [Gillis, 1993](#); [Pfefferbaum, 1997](#); [Rigamer, 1986](#); [Terr, 1985, 1989a](#)). Often, more than one family member is traumatized, although individual level of exposure and course of recovery may differ. The parent's own trauma may be so great that the needs of a young, perhaps less vocal child may be overlooked. [Terr \(1989b\)](#) recommends using projective techniques in family work because these engage children in sharing. Helping parents resolve their own emotional distress can increase their perceptiveness and responsiveness to their child ( [Gillis, 1993](#); [Pynoos and Nader, 1993](#); [Rizzone et al., 1994](#)). Parents also may benefit from psychoeducation about their child's symptoms and how to manage them effectively ( [Deblinger et al., 1990](#); [Rigamer, 1986](#); [Rizzone et al., 1994](#)).

The focus of family work includes validating the child's experiences and emotional reactions, helping the child regain a sense of security, anticipating situations in which additional support will be needed, and exploring ways to decrease traumatic reminders and secondary stresses. The child and parents should be educated about PTSD, its symptoms, and course ( [Pfefferbaum, 1997](#); [Pynoos and Nader, 1993](#)).

### PHARMACOTHERAPY

Few studies have examined the use of pharmacologic agents in children with PTSD, and there is little empirical support for the use of any particular medication ( [Cohen et al., 1998](#); [Pfefferbaum, 1997](#)). Pharmacotherapy, when used, is an adjunctive therapy that may be needed if symptoms are disabling ( [Marmar et al., 1994](#)). Clinicians must use their clinical judgment in determining what agents to use. Specific symptoms and the stage of the illness determine whether to use a drug, what drug to use, and the duration of use. Positive symptoms of reexperiencing and arousal may be more responsive to medication than negative symptoms of avoidance ( [Marmar et al., 1994](#); [Najarian et al., 1996](#); [Silva et al., 2000](#)). Comorbid conditions should be considered in selecting an agent ( [Marmar et al., 1994](#); [Pfefferbaum, 1997](#)).

A variety of drugs have potential efficacy in adults and appear to be appropriate if used cautiously in children. Selective serotonin reuptake inhibitors, which have a favorable risk/benefit profile, may be effective in treating childhood PTSD and comorbid anxiety and depression ( [Cohen et al., 1998](#)). Clonidine has been used with benefit in children with PTSD symptoms ( [Perry, 1994](#); [Pynoos and Nader, 1993](#)) and in severely abused and/or neglected preschool children with PTSD symptoms who did not respond to multimodal treatment in a day hospital setting ( [Harmon and Riggs, 1996](#)). [Famularo and colleagues \(1988\)](#) reported benefit from propranolol for PTSD in young children, although placebo effect was not ruled out. Carbamazepine also has been used in the successful treatment of sexually abused children with PTSD ( [Looff et al., 1995](#)).

### SCHOOL-BASED EFFORTS

School-based interventions are effective for traumatized children or children at risk for trauma. They provide access to children in developmentally appropriate settings that encourage normalcy and minimize stigma. School is also an environment in which PTSD and associated symptoms are likely to emerge. For example, intrusive thoughts and difficulty concentrating may interfere with academic performance and social adaptation; therefore, school consultation about the consequences of trauma and the recovery process may be both necessary and useful ( [Pfefferbaum, 1997](#)).

Following disasters, schools and classrooms provide formal and informal opportunities for assessing psychological response, correcting misperceptions and fears, and encouraging normalization and recovery. Such opportunities include group screening and assessment; curricular interventions addressing traumatizing events and stress responses; opportunities for disclosure and discussion; small group activities; and projective techniques such as play, artwork, and storytelling. The goals of school-based interventions must be appropriate for the setting, and it is imperative that school-based programs not supplant efforts to identify and refer children in need of more intensive individual work ( [Pfefferbaum, 1997](#); [Pynoos and Nader, 1993](#)).

### LONG-TERM TREATMENT AND PULSED INTERVENTIONS

Long-term treatment may be necessary for children with intense or enduring exposure and symptoms, preexisting or comorbid conditions, prior or subsequent trauma, or family problems. Treatment during the acute phase of trauma may be followed by planned interventions at strategic points. These may be especially important in mass casualty disasters where many have been exposed and can be reached for follow-up as groups. Patients and their parents should be advised that new issues related to trauma commonly emerge as the child matures and that these may require treatment. These pulsed interventions should anticipate and address the course

of recovery and reflect the child's developmental capabilities ( [Pynoos and Nader, 1993](#); [Terr, 1989b](#)). Intermittent treatment and pulsed interventions are especially useful during developmental transitions, anniversaries, or marker events ( [Amaya-Jackson and March, 1995](#)).

## NATURAL HISTORY, OUTCOME, AND COPING

Few studies have examined the longitudinal course of trauma ( [Bolton et al., 2000](#); [Boney-McCoy and Finkelhor, 1996](#); [Famularo et al., 1996](#); [Green et al., 1994](#); [Kinzie et al., 1989](#); [La Greca et al., 1996](#); [Laor et al., 1997](#); [McFarlane et al., 1987](#); [Nader et al., 1990](#); [Sack et al., 1993, 1999](#); [Shaw et al., 1996](#); [Terr, 1983](#); [Yule et al., 2000](#)). Specific symptoms and their intensity or severity may vary over time. For many, symptoms decrease ( [Green et al., 1994](#); [Milgram et al., 1988](#); [Sack et al., 1993, 1999](#)), but for others, they endure or even increase ( [Goenjian et al., 1995](#); [McFarlane et al., 1987](#)). In fact, trauma-related conditions may persist for years in highly traumatized youths ( [Green et al., 1994](#); [Kinzie et al., 1986, 1989](#); [Sack et al., 1993, 1994, 1999](#); [Terr, 1983](#); [Yule et al., 2000](#)). [Sack and colleagues \(1999\)](#) found new cases of PTSD emerging as late as 5 to 8 years after the end of the trauma. Very delayed onset may occur without apparent trigger and may persist but is uncommon ( [Yule et al., 2000](#)).

[Bolton and colleagues \(2000\)](#) conducted a controlled study of long-term survivors of a shipping disaster. Rates of psychopathology were higher in survivors than in the control group that was comprised of sex-, age-, and school-matched friends or acquaintances nominated by participants ( [Yule et al., 2000](#)). The development of other disorders was strongly associated with the development of PTSD, and most of those who developed PTSD also suffered from another disorder. Those with PTSD were more likely than controls to have a variety of comorbid anxiety and depressive conditions at follow-up 5 to 8 years after the disaster but not increased substance misuse, obsessive-compulsive disorder, or generalized anxiety disorder.

### Coping

The role of coping in childhood trauma victims has only recently been researched and its effects on the course of recovery remain unclear ( [Aaron et al., 1999](#); [Asarnow et al., 1999](#); [Compas et al., 1991](#); [Compas and Epping, 1993](#); [La Greca et al., 1996](#); [Prinstein et al., 1996](#); [Vernberg et al., 1996](#)). A number of conceptual models have been used to examine coping and a variety of terms have been applied to the strategies identified. No single strategy is effective in all situations for all people, and multiple strategies may be used at once ( [Compas, 1987](#)). Two conceptual models of coping are presented as examples of recent research in the area.

One conceptual model of coping identifies two basic approaches—problem-focused and emotion-focused efforts ( [Compas et al., 1991](#)). Problem-focused coping appears earlier than emotion-focused coping, which increases with age. Problem-focused coping involves attempts to change or master some aspect of the stressful situation, whereas emotion-focused coping represents efforts to regulate the emotions associated with it ( [Compas et al., 1991](#)). In a study of children confined to sealed rooms because of SCUD missile attacks during the Persian Gulf War, emotion-focused strategies involving avoidance and distraction were associated with less stress after the war ( [Weisenberg et al., 1993](#)). Indeed, avoidance, passive acceptance, and distraction may be effective in disasters where the child has no control over environmental conditions ( [Joseph and Williams, 1997](#); [Kinzie et al., 1986](#); [Weisenberg et al., 1993](#)). On the other hand, avoidant coping also may be associated with increased posttraumatic stress symptomatology ( [Aaron et al., 1999](#); [Asarnow et al., 1999](#)), and it may represent an aspect of PTSD itself.

The second model categorizes coping as avoidant, active behavioral, and active cognitive ( [Asarnow et al., 1999](#)). Avoidant coping strategies include cognitive and behavioral strategies to avoid thinking about the event or dealing with it directly. Active behavioral strategies include specific actions or behaviors to deal with the stressor. Active cognitive approaches include active thoughts about the event. Using this model, [Asarnow and colleagues \(1999\)](#) found active cognitive coping associated with PTSD symptoms and avoidant coping marginally associated with PTSD symptoms in children exposed to an earthquake. Active behavioral coping was not associated with PTSD symptoms.

Certainly in clinical work, coping is a major concern and attention to it represents a positive approach to treatment. [Allen and colleagues \(1999\)](#), for example, conducted group interviews of children in Oklahoma City schools in the year following the 1995 bombing encouraging children to identify and share the coping strategies they found helpful in dealing with the event. The children identified a host of approaches including communication, distraction, projective activities, and participation in memorial exercises; they also reported relying on parents, teachers, and classmates for support.

## PREVENTION

Prevention of PTSD involves preventing the traumatic stressor or event or recognizing symptoms soon enough to avert full manifestation of the disorder. A number of factors make this difficult: Traumatic stressors are ubiquitous and they may be unpredictable, PTSD has no single etiology, and exposure and the effects of exposure are not always obvious. Prevention at the community or institutional level entails the development of a disaster plan, which should include an organized mental health response as well as mechanisms to assure physical readiness. Prevention also involves efforts to minimize exposure and secondary adversities and educate victims and potential victims about symptoms that warrant intervention and about safeguards against emotional sequelae ( [Pfefferbaum, 1998](#); [Pfefferbaum et al., 1999b](#)).

It is important to identify high-risk situations and establish mechanisms for early detection and intervention. These measures are often educational in nature and they focus on the child, parent, school, and/or community. Facilitating disclosure of a traumatic event or stressor is important when denial, avoidance, or shame prevent the child from revealing what has occurred ( [Pfefferbaum et al., 1999b](#)).

Parents, primary care physicians, and those who work closely with children such as teachers, counselors, and other school personnel should be taught to recognize and address symptoms of trauma and should be informed about the contagious features of the condition. It is important to identify children with stress responses quickly and to refer them to an experienced mental health professional as soon as possible because the child's initial response may predict longer-term effects ( [Lonigan et al., 1994](#); [Pfefferbaum et al., 1999d](#); [Tyano et al., 1996](#); [Yule and Udwin, 1991](#)).

Health care professionals should develop relationships with local media and encourage responsible reporting to decrease transmission of posttraumatic symptoms. It is also possible to enlist the aid of the media in providing information about symptoms, factors that influence symptom development, and how and where to obtain professional help, although this must be done carefully to prevent misinformation and misinterpretation by the media ( [Pfefferbaum et al., 1999b](#); [Pfefferbaum and Pfefferbaum, 1998](#); [Terr, 1989b](#)).

The overwhelming and unpredictable nature of many traumatic events leaves some victims highly impressionable and susceptible. Those working in situations involving large numbers of individuals at once, such as in natural disasters, war, or war-like conditions, must avoid describing scenarios of response to prevent influencing symptom development in vulnerable individuals. The clinician must be sensitive to the need to explore the trauma but must avoid retraumatizing the child.

## RESEARCH ISSUES AND DIRECTIONS

As research methodology in the study of childhood trauma matures, a host of associated issues warrant investigation. These include the diagnosis of PTSD, symptom clusters, and individual symptoms; aspects of exposure, especially indirect exposure; biological parameters; the longitudinal course of the illness, comorbid conditions, and development; and treatment and coping.

### Diagnosis, Symptom Clusters, and Symptoms

Changes in diagnostic criteria over successive editions of the DSM ( [American Psychiatric Association, 1980, 1987, 1994](#)) suggest limitations in the diagnostic construct itself. For example, the DSM-IV ( [American Psychiatric Association, 1994](#)) Criterion C combines effortful avoidance and numbing into one cluster, but they may well represent distinct dimensions. Several studies using factor analysis identify three or four factors, with avoidance and numbing inconsistently placed ( [Anthony et al., 1999](#); [Dyregrov et al., 1996](#); [March et al., 1997](#); [Pynoos et al., 1987](#); [Sack et al., 1997](#); [Yule et al., 1994](#)).

The criterion requiring clinical distress and impaired functioning also awaits research attention. Given that some PTSD symptoms may represent a normal response to trauma, definitions of distress and functioning are especially important. Although both subjective and objective measures would be ideal, DSM-IV provides virtually no guidance on how to assess them. Failure to address the criterion in prior studies clouds findings that purport to establish diagnosis. A review of previously collected data and a new generation of studies may be needed to determine the degree to which PTSD symptoms and clusters result in distress or functional impairment in children and to identify qualitative as well as quantitative differences between the normal response to trauma and the PTSD diagnosis. This issue has important



implications for treatment.

Other issues related to the diagnostic construct are also of interest. For example, although DSM-IV requires that reexperiencing, avoidance/numbing, and increased arousal symptoms be “persistent,” it does not define the term and does not specify required severity or frequency of symptoms except as they, in the aggregate, cause clinically significant distress or functional impairment ( [American Psychiatric Association, 1994](#), p. 428). Clinically relevant definitions may be developed through further refinement in research-oriented operational definitions.

## Exposure

Although many studies examine the role of exposure in symptom response, a number of specific aspects of exposure have not been well explored. Of great interest are indirect forms of exposure and the PTSD spectrum along with the impact of these on functioning. Assessment of the specific aspects of initial response and their role in the development of symptoms is usually limited by retrospective assessment, yet initial response is important to our understanding of the posttrauma process and course of recovery, the relationship between the biology and psychology of the disease, the relative importance of specific symptoms and the symptom clusters, and treatment planning.

## Biological and Environmental Correlates

Neurohormonal and neurophysiologic correlates of PTSD in children are being identified. Their etiologic significance, relationship to specific symptoms or symptom complexes, and influence on long-term adjustment warrant exploration. Availability and choice of informants, inadequate measures, and complex analytic problems currently limit the study of family factors, which are vital to our understanding of the transmission of the disease, the interaction of biological and environmental influences, mediating factors, and treatment considerations. Social influences are even more difficult to assess.

## Longitudinal Course, Comorbidity, and Development

General population studies assessing response to a variety of stressors are rare but would offer the opportunity to explore effects of trauma on demographically diverse groups of children exposed to a range of events. These studies should investigate the impact of symptoms on functioning and the various factors that influence risk and resilience. Prominent among these factors are the effects of prior trauma, temperament and premorbid conditions, and family factors. Longitudinal assessment of cohorts of children would provide much needed information about the natural course of the illness and could examine comorbidity and coping. Posttraumatic stress disorder shares many symptoms in common with other disorders, requiring careful exploration of comorbid conditions and their temporal relationships to PTSD. Like many conditions, the recognition of PTSD in children lagged behind its recognition in adults, and studies in children tend to mirror those in adults. Therefore, research on the role of development in response to trauma and on the impact of trauma on development is needed.

## Treatment and Coping

Treatment approaches have developed despite a dearth of research about their effectiveness. Controlled studies assessing the impact of treatment on specific symptoms and clusters, the role of medication, the relative benefit of various modalities, and the merits of parental involvement are needed.

Coping is a complicated process ripe for investigation. Disaster coping studies suffer from methodologic problems and inconsistencies that make it difficult to interpret the results. Coping is a process that is difficult to define and operationalize. Investigators attempt to capture useful information by asking children to identify the worst aspect of their experience and the strategies they used to cope or by asking them to think of their coping in general. Studies tend to document the frequency of use of various strategies without reporting the benefit conferred ( [Asarnow et al., 1999](#); [Berman et al., 1996](#); [La Greca et al., 1996](#); [Vernberg et al., 1996](#)). Analyses also vary across studies. For example, data from the Kidcope questionnaire ( [Spirito et al., 1988](#)) used in some disaster studies can be analyzed in several ways—by categorizing strategies according to some schema such as positive/approach or negative/avoidance ( [Berman et al., 1996](#); [Spirito, 1996](#)) or by factor analysis ( [Spirito, 1996](#); [Vernberg et al., 1996](#)).

## Methodologic Issues

Methodologic concerns in PTSD and disaster research have potentially important implications for clinical work. The first involves sample selection. Most studies have examined exposed samples; instead of using control or comparison groups, samples are commonly stratified for within sample comparisons on variables such as exposure. Assessment, for both clinical and research purposes, at best uses multiple informants and measures, yet most trauma studies use survey design and query single informants. Self-report is important in child trauma research, in part, because adults underestimate the reactions of children. Children tend to be better informants of internalizing symptoms, whereas parents provide more accurate information about externalizing behavior ( [McClellan and Werry, 2000](#)).

Trauma research typically employs self-report questionnaires—ideal for screening large groups such as school children—and structured diagnostic interviews. Clinically, PTSD, like other disorders, involves a degree of symptomatology on a continuum rather than the mere presence or absence of symptoms. Therefore, measuring the frequency and severity of symptoms, as well as establishing the diagnosis, is important in understanding the development and progression of PTSD. Self-report measures are more efficient in cost and administration, but the quality and quantity of information obtained is limited. The two most widely used self-report measures in child work are the Impact of Event Scale ( [Horowitz et al., 1979](#); [McNally, 1991](#)) and the PTSD Reaction Index ( [McNally, 1991](#); [Pynoos et al., 1987, 1993](#)). Although they may be coupled with other measures, these tools alone do not measure functioning.

Structured diagnostic interviews have the advantage of establishing diagnosis, important because it represents a generally agreed on constellation of signs and symptoms warranting medical attention. The interview format allows participants to clarify questions and describe their experiences more fully. A number of structured diagnostic interviews are available, including the Child and Adolescent Psychiatric Assessment ( [Angold and Costello, 2000](#)), Diagnostic Interview for Children and Adolescents ( [McNally, 1991](#); [Reich, 2000](#); [Welner et al., 1987](#)), and National Institute of Mental Health Diagnostic Interview Schedule for Children ( [Shaffer et al., 2000](#)). The relative usefulness of structured diagnostic interviews for specific diagnoses or for specific clinical issues has not been studied, and their accuracy is limited by the unknown validity of the disorders they assess ( [McClellan and Werry, 2000](#)).

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### CASE ILLUSTRATION

Valerie was 15 years old when she entered outpatient treatment following a brief psychiatric hospitalization for depression, suicidal ideation, and conduct problems. Valerie had run away from home after an altercation with her father who disapproved of her argumentative attitude and her choice of friends. Valerie had been placed on a selective serotonin reuptake inhibitor (SSRI) during hospitalization, which was continued as an outpatient. Valerie complained of depressed mood, extreme fatigue, and excessive somnolence. She routinely went to bed immediately on arriving home after school, joined the family for dinner with reluctance, and returned to bed to sleep until awakened each morning for school. Always a poor student, Valerie's grades dropped and she disengaged from extracurricular activities including girl's basketball, which she had previously enjoyed. Valerie came from an intact family with parents who were genuinely concerned about her.

Valerie provided little information that might be useful in her treatment, including history of trauma, current relationships or activities, or feelings. She had great difficulty confiding in her therapist, and her mother often accompanied her to sessions disclosing information about Valerie's progress at home and school. Valerie remained on the SSRI with gradual benefit in mood and sleep disturbance.

Within approximately 1 year of initiating treatment, Valerie started dating a boy from her school. She was devoted to him and felt pride at his interest in her. Her mood improved and her sleep pattern returned to normal. Eventually she began having sexual relations. This introduced conflict at home, although her mother was agreeable to Valerie's use of contraceptives. Over the next several months, internal conflict also emerged, and Valerie finally confided in her mother that at the age of 11 years, she had been seduced by an older boy and then raped by him and two of his friends. Her embarrassment had prevented her from disclosing the incident earlier, but the sexual nature of her relationship with her current boyfriend created enough conflict that she felt the need to discuss what had occurred. Because the perpetrators remained in the community, the incident was reported to authorities over Valerie's objections.

Valerie became more open in therapy, no longer requiring her mother's presence in sessions. She gradually described the traumatic sexual incident, providing increased detail over time, and she reported long-standing intrusive thoughts, dreams, and guilt. She acknowledged that she had purposefully disengaged from peers and assumed no other boy would want her. Sleep, Valerie now realized, had become her escape. She continued to have an uneasy visceral feeling when she thought about the incident, but conflict with her boyfriend and at home diminished. Valerie matured and blossomed socially. Her schoolwork improved, although she was never able to achieve more than marginal success academically. She was weaned off of medication without problem approximately 18 months after entering treatment. She was seen periodically over the next 2 years at developmental transitions but, for the most part, continued to do well, eventually learning to drive, graduating from high school, and entering a junior college in her home town.

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# 75 CHILDREN EXPOSED TO DISASTER: THE ROLE OF THE MENTAL HEALTH PROFESSIONAL

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Mass disasters, whether natural, technical, or human-made, take an enormous toll in human life and exert untold physical, psychological, and economic hardships on survivors. They affect individuals, families, and whole communities. Recent years have witnessed a significant growth in mortality associated with nearly all types of disasters, apparently as a result of the increase in population density, urbanization, and climatic changes ( [Ursano et al., 1994](#)). Natural disasters killed nearly 3 million people in the 1970s and 1980s and affected the lives of at least 800 million more (WHO, 1991). Less developed countries account for a considerable proportion (about 40%) of the worst natural disasters, and an even higher proportion of disaster-related deaths. For example, three earthquakes during the last 12 years (Armenia, Turkey, and India) claimed about 30,000 lives each, mostly because of poor housing construction. In China, the costs incurred by natural disasters exceed \$12 billion annually ( [Wang et al., 2000](#)).

Modern technology and mass communication have increased the worldwide awareness of the devastating effects of earthquakes, tornadoes, floods, fires, epidemics, nuclear accidents, and wars. In December 1987, the United Nations General Assembly designated the 1990s the Decade of Natural Disaster Reduction. As part of this effort, research has been conducted on the short- and long-term physical and psychological effects of disaster on individuals at high risk, such as the very old and the very young. The present chapter focuses on the psychological impact of disasters on children. Unlike physical damage, which is usually easy to identify, internal suffering of children can remain hidden even from sensitive observers. Therefore, clinicians and researchers are attempting to elucidate the type, extent, and risks of maladaptive responses of children to mass disaster.

Earlier statements that children show only a mild response to traumatic conditions ( [Garmezy and Rutter, 1985](#)) have been shown erroneous by the application of modern empiric methodologies and direct observations. Indeed, the adverse psychological effects can be severe and long lasting, also in preschoolers ( [Pfefferbaum, 1997](#); [Pynoos et al., 1993](#); [Udwin, 1993](#); [Vogel and Vernberg, 1993](#); [Yule et al., 1999](#)), and they may persist even in the face of apparently normal social functioning ( [Laor et al., 1997](#)). Nevertheless, according to the Task Force Report of the American Psychological Association, "Few psychologists have had specific training for working with children after disasters, and discussions of children's responses to disasters have been rare in texts on psychopathology or issues in normal development" ( [Vogel and Vernberg, 1993](#)).

The aims of this chapter are fourfold:

1. To present a theoretical perspective of disaster as a systemic social phenomenon
2. To clarify the role of child mental health professionals in large-scale preparedness and community reactivation under conditions of disaster
3. To review the major findings on children's responses to disaster from the developmental aspect
4. To propose models of assessment and intervention for children, families, and communities exposed to mass disaster

## DEFINITIONS

The literature distinguishes between "trauma" and "disaster." Traumas are experiences that threaten individual health and well-being, render one helpless in the face of intolerable internal or external danger, overwhelm coping mechanisms, violate basic assumptions about survival, and stress the uncontrollability and unpredictability in the world ( [Eisen and Goodman, 1998](#)). A trauma may be caused by an isolated, unanticipated event or result from repeated exposure to several extreme external events ( [Terr, 1991](#)).

Disasters are relatively sudden, more or less time-limited, public events that extensively damage properties and lives, engendering a systemic continuously disruptive impact on the social network and basic daily routines of children and families ( [Laor and Wolmer, 1999](#); [Vogel and Vernberg, 1993](#)). The community as a whole is compromised in its capacity to negotiate the recovery of its individual members (e.g., massive displacement and relocation). Matters are often made worse when resources are overwhelmed ( [Ursano et al., 1994](#)) and the community's infrastructure is affected. This can result in unemployment, lack of housing and food, poor health and mental health services, school closures, school and job absenteeism, family dysfunction, and displacement of large populations.

Disasters differ in scope and schedule. Some result mainly in loss and disruption (loss of possessions and housing), whereas others involve also a threat to life. Some last a few seconds (e.g., earthquake), whereas others continue for years (e.g., war). Unlike traumas, disasters are characterized by the immediate, long lasting and repeated exposure of victims to reminders of the disastrous event (e.g., destroyed buildings after an earthquake). Usually, three types of experience are combined: terror owing to a danger to one's life or exposure to grotesque sights; grief following loss (e.g., human lives, basic trust, self-esteem); and the disruption of normal living ( [Austin and Godleski, 1999](#)). On the social level, there are shock, depression and mourning, confusion and social disarray, rage and blaming, norm breaking and delinquent behavior, emergence of mythic ideologies, collapse of formal leadership, ascension of informal popular leadership, and social disintegration into primary affiliations. Children feel the disruption in their family, neighborhood, and school ( [Laor and Wolmer, 1999](#)).

Theoretical, research, and intervention studies should follow both a systemic and a long-term design because the pathologic and recovery processes continue long after the disastrous event itself is over, even if it was restricted to a single point in time.

## SYSTEMIC THEORY OF DISASTER



## Disasters and the Mental Health System

Mass disaster poses a multifaceted challenge to the mental health system ([Laor, 2001](#)):

1. *Environmental*. There is an emergence of massive needs, routinely defined as pathologic.
2. *Systemic*. A multidisciplinary orientation and a multisystemic societal collaboration are needed to counter the adverse impact.
3. *Practical*. There are problems of resource allocation, extended deployment, organization, and dissemination of information and communications.
4. *Theoretical*. Similar to other relevant fields (e.g., medicine), the mental health system lacks a comprehensive and integrative “mass disaster theory,” mainly because of the lack of a general social perspective in addition to the public health perspective ([Pynoos et al., 1998](#)).
5. *Professional*. Most mental health teaching programs are not committed to disaster intervention training.

Hence, professionals have insufficient knowledge of diagnosis (of risk factors, clinical picture, and assessment) and therapeutic technology (protocol-driven, short-term, group- and community-based, and disaster-related), and little endurance owing to continuous stress (changing needs and priorities, traumatization, and burnout).

Mental health professionals operate within multiple social systems: psychiatric, medical, welfare, urban, and national. Each may be characterized by degree of adaptability and flexibility under stress. *Static* systems are rigid, indifferent to the environment, and show no adaptive change in structure or function over time. Under extreme stress, static systems may prove brittle and disintegrate. *Chaotic* systems show an anarchic response to the environment (disintegrated, disorganized, and dysfunctional). *Learning* systems respond in a flexible-reactive manner, show sensitivity to the environment and openness to some change in structure or function; however, their range of change is restricted to routine operations, based on past experience. *Meta-adaptive* systems are the most advanced, being both flexible and proactive. They are learning systems that contain units specializing in forecasting and preparing alternative scenarios for coping with change.

### Stages of Disasters

Different models have been proposed to describe the disaster response, most from the event perspective (e.g., warning, threat, impact, inventory, rescue, and recovery) ([Raphael, 1986](#)). The systemic model allows a formulation of disaster that integrates the event, the individual, and the sociocultural reaction, including the mental health response. From this perspective, disaster consists of three stages ([Laor, 2001](#)), but it may loom long before the expected event actually takes place (e.g., the months of expectation and rising anxiety preceding the outbreak of the Gulf War) ([Laor et al., 1996](#)). This *pre-disaster stage* includes warning, alert and alarm signs, and a sense of massive threat to community and personal security.

The *first stage* of a disaster consists of the damaging event itself, the primary disaster, and the attempts to alleviate its effects, that is, rescuing as many victims as possible and providing basic needs (food, water, and shelter) to the affected population. The *second stage* consists of massive changes in societal structure and function (e.g., establishment of evacuation centers and tent cities, movement of refugees), which may lead to loss of norms, structures, and functions. This loss, reflected in societal regression, may be viewed as the secondary disaster and may appear early, even as part of the first stage. Usually, the early optimism of survivors—encouraged by waves of incoming resources and VIP visits—diminishes after a few weeks and turns into disillusionment, and fears of abandonment and lack of justice and bureaucracy. For children, this stage may represent the loss of all social coordinates, causing confusion and disruption of meaning. Brittle social systems may collapse and turn chaotic, aggravating the damage. Here is where the advantage of metaadaptive systems is most conspicuous.

Life usually stabilizes in due course, generally after 18 to 36 months. At this point, there may be a *third stage* of disaster wherein the sociocultural losses, the tertiary disaster, threaten the existing collective ideology and identity (e.g., religious identity of generations of Holocaust survivors). Often, even when the first and second stages are well managed, the third stage is neglected, with effects surfacing even years later. [Note the reports on transgenerational transmission of the culture and the trauma of disaster ([Danieli, 1998](#); [Laor, 1998](#)). A recent report ([Kellerman, 2001](#)) concluded that nonclinical samples of children of Holocaust survivors and control individuals do not show different levels of psychopathology.]

Sometimes, the transition between stages and/or the increase in damage severity evolve gradually and over an extended period of time (e.g., AIDS epidemic in Africa, the recent armed conflict in the Balkans, and the Israeli–Palestinian conflict). Primary, secondary, and tertiary types of disaster coexist. The gradual pattern allows for preparation and short-term adaptation to minor increments of destruction. However, it may also engender habituation ([Solomon, 1995](#)), both within the affected and the international community, damaging the capacity for long-term forecasting and for proper coping.

## CHILDREN'S REACTIONS TO DISASTER: THE DISASTER SYNDROME

The child's protective matrix consists of various dimensions in the child's reality that could be disrupted and rehabilitated differentially: the political, cultural, social, physical, familial, maternal, and personal ([Laor, 1996](#)). Because disasters affect all these components, the disaster syndrome, unlike posttraumatic syndrome, involves all aspects of the child's developing cognitive structures and capacities and poses a more intricate pathologic threat. Children must cope with many different kinds of losses: of people, support systems, normal routines, and basic assumptions of safety and regularity. To help them, the mental health professional must recognize and help to restore the various dimensions of the destroyed reality. Children may become withdrawn and alienated from the reality they perceive as having betrayed them: nature, parents, society, and its technology. The withdrawal may take the form of psychic shock that may lead to a serious catastrophic reaction or it may partially resolve by the mobilization of coping mechanisms ([Krystal, 1968](#); [Valent, 2000](#)).

Specifically, disasters may affect children's ability to regulate the intensity of impulses and unconscious fantasies, thereby jeopardizing their sense of self-efficacy, security and autonomy, normal maturation of defensive functioning, object relations, reality testing, and attachment with caretakers. Structural developments, such as superego consolidation and its behavioral consequences (e.g., empathy, prosocial behavior); ego ideal structure, with its relevance to affiliation and ideology formation; and ego functions, with significance in areas of cognition and attention, may also be hampered during the primary, secondary, and tertiary disasters. Traumatization has a potentially damaging effect on the development of a lasting sense of identity that integrates thoughts, images, feelings, and sensations. The sense of historical continuity of the self (past, present, and future) therefore may be disrupted ([Pynoos et al., 1995](#)).

The clinical picture observed in children immediately after disaster is characteristic of acute stress and/or acute grief reaction, depending on the type of trauma and loss they have suffered. The severity of the response depends on many factors, such as level of exposure, developmental level, culture, parental support, and pre-disaster personality. Children may be in a state of shock and numbness hours or days immediately after a disaster. Dissociative symptoms, such as depersonalization and derealization—a sense of unreality of the world or oneself—may be the first line of defense against the overwhelming experience, before the prolonged process of reconstructing the internal world and external environment begins.

Preschoolers may show behavioral changes and regressive behaviors, mostly within the normal range. These may include irritability, sleep difficulties, separation problems, fears, nervousness, posttraumatic play, demanding or dependent behavior, and whining or temper tantrums ([Bingham and Harmon, 1996](#); [Laor et al., 1996](#); [Sullivan et al., 1991](#)). Older children may report disturbances in conscience functioning, although their moral functioning may seem advanced ([Goenjian et al., 1999](#)).

Studies suggest that parents and teachers tend to report fewer posttraumatic symptoms in children than the children themselves ([Korol et al., 1999](#); [Vernberg et al., 1996](#); [Yule and Williams, 1990](#)). Adults may be preoccupied with their own stress and not be attuned to their child's inner emotional states. Children also may be more reliable reporters of internalizing or dissociative symptoms. Thus, clinicians must be careful to assess children's functioning directly and not rely exclusively on external reports. They must bear in mind that although the initial response tends to predict later adjustment, initial symptomatic ratings may not correlate with later assessments ([Green et al., 1994](#)), and posttraumatic responses may show a delayed onset ([Sack et al., 1999](#)). If the disaster is limited and well controlled, most of the pathologic reactions in children will abate within the first year. In a 17-year follow-up of children exposed to a dam collapse, [Green and colleagues \(1994\)](#) found that survivors recovered from the initial symptomatic reaction and functioned similarly to nonexposed children; however, if community functioning is substantially disrupted, symptoms may persist for years ([Laor et al., 1997](#); [McFarlane, 1987a](#); [Pynoos et al., 1993](#); [Sack et al., 1999](#)). These data underscore the need for effective persistent disaster management on all levels: primary, secondary, and tertiary.

### Types of Postdisaster Symptoms

Children may show a combination of some or many of the following in reaction to disasters: specific posttraumatic stress symptoms, fears, depression and grief, and

dissociation ([Gordon and Wraith, 1993](#)).

### SPECIFIC POSTTRAUMATIC STRESS SYMPTOMS

The symptoms of posttraumatic stress disorder (PTSD) are grouped under three major domains: intrusion, avoidance/numbing, and arousal. Empiric studies have recognized certain symptoms that are specific to children, such as persistent posttraumatic play, omens, and somatic complaints ([Terr, 1983](#)). [Scheeringa and associates \(1995\)](#) proposed diagnostic criteria for the disorder in young children.

*Intrusive reexperiencing of the event* may be observed in thoughts (about the traumatic experience), feelings (as if the event were happening again), or sensations (being acutely distressed by stimuli related to the trauma). Children may tell their experiences over and over, report nightmares, and show repetitive trauma-related play. They also may describe vivid traumatic images: visual (e.g., mutilated bodies), auditory (e.g., the sound of the earthquake or screams for help), olfactory (e.g., odors of burned or decaying bodies), or kinesthetic (e.g., feeling as if they were buried under the rubble).

*Avoidance of reminders* is evidenced in the evasion of places, people, thoughts, or activities associated with the disaster. This can be both a symptom and a defensive maneuver to reduce internal stress; however, persistent avoidance coping is associated with negative mental health outcomes ([Asarnow et al., 1999](#)). The avoidance may be active (e.g., purposeful engagement in trauma-unrelated thoughts to avoid traumatic reminders) or passive (e.g., not engaging in social interactions) ([Anthony et al., 1999](#)).

*General psychic numbing* may be considered a mild dissociation response, and it is more difficult to detect in children than adults ([Yule and Williams, 1990](#)). Children exposed to disasters may lose interest in activities that were significant in the past, feel estrangement from others, show constricted affect, lose recently acquired developmental skills (e.g., toilet training or talking), and express a sense of foreshortened future.

*Increased arousal* symptoms are the most easily recognized by external observers, and include irritability, angry outbursts, exaggerated startle response, hypervigilance, difficulty in concentrating, and sleep disturbances (expectable in young children) such as difficulty getting to sleep or sleeping alone ([La Greca, 2000](#)).

In a study of Armenian children exposed to the 1988 earthquake, about 90% of those living adjacent to the epicenter met the diagnosis of PTSD 18 months later, compared to only 30% of children from the periphery of the earthquake zone ([Pynoos et al., 1993](#)). [Anthony and colleagues \(1999\)](#) found that anhedonia and attention and learning problems are the most common symptoms after disasters; however, they reflect the normal disruptive consequences of disasters rather than being markers of a pathologic reaction.

### FEARS AND DEPENDENT BEHAVIOR

Mass disasters typically induce specific fears and dependent behavior in children ([Goenjian, 1993](#); [Sullivan et al., 1991](#)). Old fears may be reactivated, current ones may intensify, and new fears may emerge with a more or less clear relationship to the event. Particularly in young children, fears may lead to dependent and clingy behavior, difficulty separating from caretakers, or refusal to attend school, thereby interrupting the separation-individuation process. [Vogel and Vernberg \(1993\)](#) explained the separation anxiety of children after disasters in terms of the child–parent attachment relationship. They claimed that disasters challenge the basic assumption that the world is a secure place, leaving the child helplessly vulnerable. The recent finding that young children's symptoms correlated with their mother's reaction 5 years after a disaster provided empirical support for this hypothesis ([Wolmer et al., 2000](#)).

### DEPRESSION AND GRIEF

Children exposed to disasters may show symptoms of depression and grief, but these are usually of lesser severity than the specific PTSD symptoms ([Vogel and Vernberg, 1993](#)). Because grief and posttraumatic stress symptoms may appear independently of one another, [Pynoos and coworkers \(1987b\)](#) stressed the need for separate diagnostic interviews for each domain. The mood symptoms, which have been suggested to be at least partially secondary to the posttraumatic reactions ([Goenjian et al., 1995](#)), are the result of different types of loss (e.g., of home, family members, personal belongings, and basic assumptions). The traumatic grief reaction, recently defined for adults, may include persistent preoccupation with thoughts of or searching for the deceased, feelings of disbelief about the death, or anger and detachment from others ([Noaghiul and Prigerson, 2000](#)). This construct still awaits validation in children.

After the 1999 earthquakes in Turkey, [Wolmer and colleagues \(2001b\)](#) found that children who had seen severely injured or dead people experienced hunger or lack of sleep after the event, or had had more traumatic experiences in the past reported more depressive and grief symptoms. [Goenjian and associates \(1995\)](#) reported high levels of depressive symptoms in the most affected Armenian children 18 months after the 1988 earthquake, apparently because of the extent of family losses and the persistence of posttraumatic symptoms.

### DISSOCIATION

Disasters may be perceived as an overwhelming interruption of human experience, thereby distorting the individual's basic assumptions, both cognitive (e.g., "What is real and what is imaginary?") as well as existential (e.g., "Is it happening to me?"). To reestablish well-being, some people define a different "spacial" arrangement of their position relative to the world: "*I am not affected by the circumstances because I am focused on my own task*" or "*I am not affected, because I am elsewhere.*" This type of distancing is adaptive. Pathologic dissociation goes one step further, with manipulation of adverse stimuli through the reconstruction of perception and the redivision of consciousness: "*What is happening to me is not real*" or "*I, the experiencing one, am not real*" ([Laor, 1996](#)). Under conditions of mass disaster, this maladaptive response may encompass extensive spheres of experience (moral, familial, social, cultural, and political) and affect a wide range of self-systems.

Dissociative reactions may be manifested by symptoms that reflect a discontinuation of personal experience (e.g., affective detachment, perceptual distortions or body- and self-distortions). Children may have out-of-body experiences, perceive life like a dream or movie, and "hear voices" of or "see" people who have died. Amnesia is apparently less frequent in children than adolescents. Under certain conditions, dissociative mechanisms provide temporary relief from the overwhelming trauma. If persistent, however, they may engender a long-term alteration in normally integrative functions of identity, memory, and/or consciousness ([Putnam, 1995](#)).

## FACTORS AFFECTING CHILDREN'S RESPONSES TO DISASTERS

Several factors influence the extent of the children's symptomatic response to disaster. Some relate to the disaster itself, others to individual, familial, and social characteristics.

### Factors Related to the Disaster

Children whose traumatic exposure is more severe tend to react in a more extreme way ([Solomon, 1995](#)). This "dose of exposure" effect is apparent, for example in proximity to the epicenter of an earthquake ([Pynoos et al., 1993](#)), the impact zone of a hurricane ([Shaw et al., 1995](#)), or the site of missile attacks ([Laor et al., 1996](#)). More severe responses have been noted in children who were exposed to the cruelest experiences, such as witnessing severely injured people and mutilated bodies, faced a direct threat to their own life, or suffered human loss, especially of family members ([Husain et al., 1998](#); [Pfefferbaum et al., 1999](#); [Yazgan et al., 2001](#)), and in children who sustained personal injuries ([Goenjian et al., 1995](#); [Green et al., 1991](#); [Laor et al., 2001](#); [Udwin et al., 2000](#)). Continuous displacement is also predictive of a worse psychological response ([Laor et al., 1997](#); [Lonigan et al., 1994](#)). Children exposed to several traumatic experiences are more likely to exhibit more posttraumatic symptoms ([Laor et al., 2001](#); [Macksoud and Aber, 1996](#); [Thabet and Vostanis, 1999](#)).

When the disaster is severe, children need to cope with a massive range of problems: lack of food, water, and shelter; property damage; inadequate housing; violence; lack of medical care; traumatic reminders; bereavement; relocation; separation from parents; and economic crisis. Their posttraumatic reactions may intensify and interfere with symptomatic recovery, at least during the first year, as well as with long-term development ([Freedly et al., 1993](#); [Husain et al., 1998](#); [McFarlane, 1987a](#); [Pynoos et al., 1993, 1995](#)).

### Factors Related to the Child



## AGE

The variations in both children's age and symptom domains examined among the various studies make generalizations difficult, although young children are considered more vulnerable ([Garbarino and Kostelny, 1996](#); [Solomon, 1995](#)). Nevertheless, behavioral problems, specific fears, regressive symptoms, and separation problems appear to be more characteristic of young children, whereas depression and anxiety are more characteristic of older children and adolescents ([Gleser et al., 1981](#)). Three months after Hurricane Hugo, preadolescent children reported more posttraumatic symptoms than early and late adolescents, who had similar responses ([Shannon et al., 1994](#)). [Green and associates \(1991\)](#) reported a similar level of some specific posttraumatic symptoms (intrusion and arousal) in preschoolers and school-age children and adolescents, although the latter reported more avoidant symptoms. This pattern may reflect different ways of expressing the same basic symptomatic domain in groups with different levels of cognitive and affective maturity, resulting in dissimilar coping styles and levels of appraisal.

## GENDER

Results are conflicting regarding gender differences. Some studies report no gender differences ([Laor et al., 1996, 1997](#); [Shaw et al., 1995](#)). Others found that girls tend to report more internalizing symptoms (anxiety, depression, fears) and boys more externalizing behavior (acting out, aggression). [Pynoos and coworkers \(1993\)](#) found that after the Armenian earthquake, girls reported more posttraumatic symptoms ([Shannon et al., 1994](#); [Udwin et al., 2000](#)); and [Gleser and colleagues \(1981\)](#) reported that after the Buffalo Creek flood, boys showed more belligerence. Girls tend to be described as more resilient than boys in childhood, but more vulnerable in adolescence ([Masten et al., 1990](#)). Girls' greater readiness to share their concerns may explain some of these gender differences.

## VULNERABILITIES AND RESILIENCY

Children with prior pathology, particularly anxiety symptoms ([Asarnow et al., 1999](#); [Lonigan et al., 1994](#); [Udwin et al., 2000](#)) and learning difficulties ([Udwin et al., 2000](#)), and children who have suffered more traumatic events in the past (e.g., divorce, surgery, car accidents, losses) ([Earls et al., 1988](#); [Laor et al., 2001](#)), are more prone to severe symptoms months after a disaster. By contrast, resilient children are those with caring adults during and after major stressors, who are also good learners, good problem-solvers, and engaging to other people. In addition, they have areas of competence and a high perceived efficacy by self or society ([Cohler et al., 1995](#); [Masten et al., 1990](#)). In a study of children exposed to the Northridge earthquake, a mild to moderate stressor, [Asarnow and associates \(1999\)](#) found that the role of heritable biology in the children's reaction was minor compared to the role of the children's subjective appraisals of stress and past psychopathology.

## COPING SKILLS

The child's coping skills also may mediate between the severity of the exposure and the response. More negative coping strategies (or a more immature defensive style) to deal with stress (e.g., blaming others, anger) are associated with greater symptomatic persistence over time ([La Greca, 2000](#); [Wolmer et al., 2001a](#)).

## Factors Related to the Family

### REACTION OF THE PARENTS

The presence of adults caring for the child during and after a major stressor is considered the most important and consistent protective factor ([Earls et al., 1988](#); [Masten et al., 1990](#)). Indeed, the reaction of the parents, especially the mother, to the disaster is generally correlated with severity of the child's responses ([Winje and Ulvik, 1998](#)). Researchers in Israel found that the reaction of preschool children to the missile attacks during the Gulf War was highly correlated with the reaction of their mothers ([Laor et al., 1996, 1997](#); [Wolmer et al., 2000](#)). This was true for 3- and 4-year old children, but not for 5-year-old children, probably owing to the latter's increasing autonomy and control of the psychological buffering systems for development ([Masten et al., 1990](#)). Five years after the war, the mothers' poor psychological functioning (increased symptoms, immature object relations and defense style) was associated with increased symptoms in their children ([Laor et al., 2001](#)).

[McFarlane \(1987a\)](#) stressed that the ability of parents to contain the anxiety generated by the extreme threats of disasters was the most important factor influencing their children's responses ([Sullivan et al., 1991](#)). Consistent love and encouragement to grieve and to discuss emotions openly within the family seem to have protected even unborn children and infants of parents evacuated after the 1986 Chernobyl disaster ([Bromet et al., 2000](#)). Parents are critical mediators of stress mainly owing to their roles in social referencing (pooling information and processing of meaning), responding emotionally, and caring for and supporting the child ([Masten et al., 1990](#)).

### FUNCTIONING OF THE FAMILY SYSTEM

The family is an important mediatory factor, particularly in young children ([Laor et al., 1996, 1997](#); [McFarlane, 1987b](#)). Families with extreme levels of cohesion—boundaries that are either too loose or too rigid—may not provide the appropriate support or allow the child to withdraw at times in order to process the traumatic experiences and reach a constructive resolution of concerns. Caring support, open communication patterns, and sensitivity to the child's needs enable parents and children to regulate dyadic processes and discuss disaster-related issues when necessary. Alternatively, parental stress may lead to a pattern of preoccupation with their own suffering and overprotection of the child, which interferes with the healthy process of resolution ([McFarlane, 1987a](#)).

## Factors Related to the Society and Culture

### FRIENDSHIPS

Friendships are valuable sources of reciprocal affection and attachment, mutual assistance, emotional security and self-esteem, and nonfamilial contexts for intimacy, thereby contributing to the child's ability to cope with stress ([Parker et al., 1995](#)). The natural sources of friendships, the family (the sibship), neighborhood, and school, may be shattered in times of disaster. Friendships may also include supportive relationships with teachers or other adults ([Udwin et al., 2000](#); [Vernberg et al., 1996](#)). Even the presence of a single concerned and caring adult may do much to offset the impact of misfortune in the lives of children ([Cohler et al., 1995](#)).

### COMMUNITY

Communities mobilize under disaster relying on their inner strength and external backup support. The inner strength of a community under disaster is a result of the operation of various factors and processes: (a) effective leadership; (b) social cohesiveness; (c) institutional empowerment; (d) available emergency services; (e) appropriate infrastructure; (f) disaster preparedness plans; (g) communal hardiness that depends on cultural factors (e.g., education, ideology).

### CULTURE

Cultural factors may also affect the clinical picture after a disaster. Cultures define the terms under which symptoms are expressed and set the parameters for the expression of personal distress. Some cultures encourage children to express their distressing feelings (anger, sadness, or longing), and others do not. For example, in some cultures, adults may admonish children who were victims of disaster to be prim and proper ([Goenjian, 1993](#)) or refrain from crying ([Laor et al., 2001](#)). Thus, cultural background, including strengths and weaknesses, needs to be taken into account by clinicians when planning treatment interventions. In addition, culture also mediates ideology and identity. As the purveyor of the meaning ascribed to disastrous events and consequences, the culture regulates the capacity of the individual to maintain an active and resilient stance. At the same time, it can lead some children to take risks that transcend their age (e.g., child soldiers, sexual exploitation).

## ASSESSMENT OF CHILDREN UNDER CONDITIONS OF DISASTER

It is extremely important that children at risk of psychopathology after exposure to disaster be identified and treated as early as possible. This is a difficult challenge for clinicians. Child mental health professionals need to be well informed about the valid assessment tools available. In the first and second stages of the disaster, they must remain sensitive to the setting in which they operate: an evacuation center, or destroyed neighborhood beset by fighting, flooding, or an infectious epidemic, or an existing natural setting, such as a school. Under both, the clinical evaluation must remain subordinate to the social one. That is, efforts must be directed not only to the treatment of the children themselves, but also to the reactivation of the society's child care systems via its existing child care workers. In a

natural setting, the assessment may be integrated into the normal institutional activity (see “School-Based Interventions” in the following). Furthermore, to identify the children's strengths and needs, professionals should also evaluate the group to which children belong (e.g., perceived support or hardiness of families, schools, and displaced communities). This mandates close collaboration among an interdisciplinary team of teachers, school counselors, social workers, psychologists, and community leaders.

The preferred clinical screening tools are those that directly assess the child rather than rely on external reporters. They should also be simple and quick to administer, accurate, repeatable, sensitive, and specific ( [Cochrane and Holland, 1969](#); [Stallard et al., 1999](#)). The criteria for identifying pathologic cases should be tempered by consideration of the psychological and economic costs of possible false-positives and false-negatives. Furthermore, the use of cutoff scores may facilitate the decision-making process, but they can obscure minor but “real” differences between children with scores slightly above or below threshold. [Green \(1982\)](#) suggested that clinicians think in terms of degree of impairment in a given sample rather than case identification. Also, the assessment of a single domain rather than the complex of posttraumatic, dissociative, and grief symptoms may decrease the sensitivity of the battery ( [Stallard et al., 1999](#)).

Brief validated assessment tools for children's posttraumatic symptoms include the Child-PTSD-Reaction Index ( [Pynoos et al., 1987a](#)) and the Diagnostic Interview for Children and Adolescents ( [Reich and Welner, 1990](#)). An abbreviated version (eight items) of the Impact of Events Scale ( [Horowitz et al., 1976](#)) has proven successful for use in children. Studies using these scales have suggested cutoff scores for case or symptom severity ( [Stallard et al., 1999](#); [Vogel and Vernberg, 1993](#)).

Symptoms of anxiety may be screened with self-report techniques such as the Revised Children's Manifest Anxiety Scale ( [Reynolds and Richmond, 1985](#)) or the State-Trait Anxiety Inventory for Children ( [Spielberger, 1973](#)). The Birelson Depression Inventory ( [Birelson, 1981](#)) and the Children's Depression Inventory ( [Kovacs, 1985](#)) are valid screens for depressive symptomatology, whereas the Grief Reaction Inventory ( [Pynoos et al., 1987b](#)) focuses on parameters of the grief process following loss.

The assessment of risk factors deserves special attention. Information should be gathered concerning the child's past functioning (traumatic experiences, mental and general health problems) as well as disaster-related events (personal injury, loss of family members or friends, witnessing severely injured or dead people, separation from parents, or experiencing hunger or lack of sleep).

## PRINCIPLES OF CHILD MENTAL HEALTH INTERVENTION

### Systemic Perspective

Mass disasters impact not only on the exposed children, but also on their families, school system, and whole sociocultural milieu. Derivative effects also appear in peripheral communities ( [Goenjian, 1993](#); [Shaw et al., 1995](#)). To cope with this complex challenge, interventions must be formulated from an integrative perspective and focus on maximizing well-being and self-efficacy and minimizing stress and disorganization; they must help victims find meaning and institute a sense of control.

In their approach to public mental health, [Pynoos and colleagues \(1998\)](#) provided guidelines for the effective utilization of government and social institutions, school communities, and interventional teams. They emphasized the need, among others, to resolve institutional conflicts over authority and resource allocation, address the teachers' own disaster experiences, and properly select and train intervention teams to work with severely traumatized victims. Population screening is useful to pinpoint areas that require specific resources and government support. Treatment interventions should be directed at the following factors: traumatic experiences and reminders, interplay of trauma and grief, and postdisaster adversities and their developmental impact.

The effect of mass disasters is so devastating because of the concomitant loss of sociocultural regulators, leading to the destruction of basic schemes, values, roles, and structures (family and individual), and leaving the community open to pain, grief, trauma, and anger. In this process, individual regression and dissociation reflect, complement and reinforce the collective ones. Working in such a milieu, professionals may find themselves embroiled in confusion and red tape among the various social/government systems (e.g., medicine, education, welfare, NGOs) and intervention teams that are trying to help. Therefore, a systemic perspective is needed to clarify the picture and help the psychiatrist: (a) formulate the newly established needs of child-oriented institutions; (b) transfer knowledge and empower professionals in related fields to resume their role; and (c) define their own role and carry out specific interventions collaboratively. This multifaceted systemic perspective was formulated and implemented after the 1999 earthquake in Turkey in the Tel Aviv-Istanbul Disaster Intervention Project ( [Laor, 2001](#)).

Mental health interventions for children and families exposed to mass disaster should follow the five AREST principles, as follows:

**Anticipate.** First, they must provide an integrated vision, foresee different scenarios, and include contingency plans. Professionals and paraprofessionals need to be trained, human and economic resources appropriately allocated, and relevant treatment protocols created. To accomplish these goals, efficient local, national, and international networks need to be developed, with collaboration among agencies (education, police, health), and sponsorship and legitimacy established ( [Vernberg and Vogel, 1993](#)).

**Redifferentiate.** The child psychiatrist must identify the extent of social loss in terms of institutional and role dysfunction and plan the process of context-related redevelopment of professional roles within and between systems (health, welfare, education) with the help of multidisciplinary teams. Attention should be addressed particularly to reconstitute the roles of parents and teachers.

**Empower.** The child psychiatrist needs to debrief (if necessary), educate, and empower social agents (e.g., teachers) who are in direct contact with children to serve as mental health mediators. They must help these agents adapt and restore their original roles, and delegate some therapeutic responsibilities to them. To this end, the child psychiatrist must take a leadership position and supply the team with a professional vision and positive expectations.

**Supervise and Assess.** The psychiatrist must define boundaries; provide knowledge, expertise, and support to therapeutic agents; assess program development; and identify needs by feedback mechanisms. As a leader, the professional needs to encourage creative initiatives in team members and provide them with individualized consideration.

**Treat and Follow-up.** Treatment focuses on the rehabilitation of individuals and families. Delayed responses should also be considered.

### Systems and Stages of Disaster

The application of the AREST principles as defined above is far from straightforward. Routine and disaster operations differ not only in intensity, speed, and expediency, but also—and primarily—in the planned systemic change. The response of the mental health professionals is determined, at each stage of the disaster, by the type of the reacting system. Preparedness and flexibility are key factors ensuring response effectiveness.

During the first stage of disaster—coping with the event itself—rigid systems tend to remain encapsulated in their normal routine, whereas learning systems may undergo structural modifications and create information centers and outreach programs. Meta-adaptive systems, owing to their forecasting capacity, may already be in state of partial readiness and will be able to initiate professional interdisciplinary teaming-up with social agencies early in the process of redifferentiation and empowerment.

In the second stage of societal regression, rigid systems treat acute referrals in existing clinics; learning systems establish field stations and initiate self-training toward the formation of larger trauma centers. Meta-adaptive systems concentrate on the rehabilitation of roles and institutions and draw on resources prepared by national and international collaborations.

In the third stage, rigid systems revert to their original constricted outlook and ignore the larger scope of the tertiary disaster—the loss of ideology and identity. They deal with suffering individuals as they are referred to their clinics, this time as chronic victims. Learning systems maintain operative trauma centers, although their perspective is narrow. They may, however, internalize some of the lessons learned from coping with the first two stages into the institutional response pattern. The main focus of metaadaptive systems is the establishment of community-based disaster intervention centers. These address the tertiary disaster by operating on both the sociocultural and communal clinical levels to enhance regeneration and growth, resilience, and hardiness. Such centers may be planned in advance and developed out of the existing community mental health system, in conjunction with the general public agencies responsible for disaster intervention.



## Professional Role Containment and Enhancement in Conditions of Disaster

First priority is given to basic survival at times of disaster. Safety, shelter, and food are the most immediate and conspicuous, and these are usually within the domain of professional relief teams. However, the members of these teams themselves may suffer from role-related problems because of the disaster-induced collapse of the sociocultural matrix of which they were a part. Therefore, to initiate an effective intervention, mental health professionals in positions of authority must respond to these needs, in their own team and teams in which they act as mediators (e.g., teachers, school counselors). They must make team members feel cared for and help them to develop a sense of belonging and purpose. Professionals may take a leadership position by formulating a vision (e.g., “developing the best intervention program,” “revitalizing our school and preventing suffering”), providing individualized consideration (being sensitive to individual needs of professionals), fostering an atmosphere of creative intellectual coping (supporting initiatives, delegating authority), and transmitting positive expectations (concerning the professionals' capacities and end results) ([Bass and Avolio, 1994](#)).

## Program Implementation

Studies of mental health interventions after disasters clearly support their effectiveness ([Galante and Foa, 1986](#); [Goenjian et al., 1997](#); [Wolmer et al., 2001](#)). However, their ongoing operation requires the continuous commitment of professionals, leaders, and local agencies. By endorsing a systemic perspective, mental health professionals may overcome the repeated adversities and challenges, inadequate professional training, limited resources, and organizational conflicts that tend to characterize the process.

Parents and teachers are known to underestimate the extent of children's suffering ([Yule and Williams, 1990](#)), perhaps because of their capacity to maintain routine functioning in the face of internal strife. Given that disaster survivors are also often reluctant to seek professional help ([Schwarz and Kowalski, 1992](#)), outreach efforts should be made to systematically screen victims at risk, optimally 1 to 3 months after the disaster ([Lindy et al., 1981](#)). Thereafter, clinical triage protocols could be utilized to match risk groups with intervention programs ([Austin and Godleski, 1999](#); [Lindy et al., 1981](#); [Pfefferbaum et al., 1999](#)).

Studies have demonstrated the applicability of Western therapeutic programs also in non-Western cultures ([Goenjian et al., 1997](#); [Wolmer et al., 2001](#)). The first step in implementing these programs is to train members of the affected community. The local staff may need continuous support and supervision. If handled correctly, this process may help traumatized survivors reduce their ambivalent resistance to what might be perceived as a foreign “intrusion” that threatens the “trauma membrane” protecting from an overload of psychic tension ([Austin and Godleski, 1999](#); [Lindy et al., 1981](#)).

## INTERVENTION MODELS

Effective treatments for traumatized children should include the psychoeducation of children and parents about the nature of the disorder, some form of exposure work, and dysfunctional cognitive restructuring ([Perrin et al., 2000](#)). Furthermore, the commonly accepted treatments specific to posttraumatic conditions need to be implemented within the broader social reality of the disaster (e.g., whole community, neighborhood, peer group) in order to alleviate the sequelae of the secondary and tertiary disasters. To this effect, the child psychiatry system must collaborate with three additional systems that provide the psychiatrist with the authority to intervene on all institutional levels: the community leadership, school, and medical clinics.

Commonly used structured treatment and rehabilitation community programs (e.g., psychotherapy, programs for the disadvantaged, adolescents, and women, as well as school empowerment and class activation), stressing participant's enhancement of initiative, activity, empowerment, hardiness, and responsibility may be adapted for disaster conditions. The adaptation rests on the formulation of disaster as destructive to both external (concrete) reality and internal functional representations—of stability (i.e., predictability and controllability) as well as of effective engagement with the environment (i.e., the physical and the communal world). Therefore, child intervention programs should aim to reconstitute the damage to both communal institutions and norms, and communal functioning (i.e., recovering communal roles: parent, teacher, worker, leader). Drawing on our understanding of the disaster syndrome, engendering massive grief, dissociation and trauma, intervention programs will be enriched by emphasizing the following transitions: from a freeze on past experience to a creative future orientation; from a fixation on death and loss to an involvement with life and revitalization; from being passively locked into grief, shame, and anger to owning up to one's potency; from withdrawal and alienation into reconciliatory involvement with nature, family, and society and its technology; and from recourse to mythologic world view into critical transformation of sociocultural identity ([Laor, 2001](#)).

In circumscribed mass disasters, effective programs may take 12 to 18 months. The first phase is dedicated to the assessment and reactivation of the community and its institutions, as well as to the introduction of clinical intervention programs. This phase may take up to a year and lead toward the first commemoration ceremony of the disaster. The community and individual taking responsibility for their future characterize the second phase: development of physical and social infrastructure and job opportunities.

## Immediate Interventions

During the acute stage, the role of the mental health professional needs to be modified because of the unique conditions, namely, the limited number of professionals and masses of individuals, often dispersed over a large area, that require help. Large populations need to be screened to identify children at risk. Other important tasks include initiating telephone crisis hotlines, supplying psychological first-aid for children and families in evacuation centers and hospitals, consulting authorities to assess immediate needs, and planning large-scale public health education programs. To prioritize needs and promote efficacy, professionals need to use medical criteria for classifying and assessing both levels of psychological damage and available individual (coping) and familial (support) resources.

At this stage, professionals become aware of the need to quickly acquire new disaster-related skills (e.g., debriefing). They may come on technically formulated protocol-based interventions, but soon discover that mastering new therapeutic techniques and implementing them under disaster conditions requires thorough training and ongoing supervision, which can be secured before proceeding with the intervention program.

The *mass media* (e.g., television, newspapers) can be helpful for confused parents. They can learn about typical reactions to stress and ways of coping with them and restoring the parental role. They should be advised to tolerate regression, encourage children to ask questions and express feelings, assure children that there are no bad thoughts or feelings, assign children appropriate activities, reestablish stability and family rituals, and convey positive expectations for the future ([Flynn and Nelson, 1998](#)). Television programs directly addressing children should cover the same issues.

Delegating age-appropriate functions to children, allowing them to take responsibility and provide active help (e.g., fill sandbags, take care of younger children), serves as an important mechanism for preventing the sense of helplessness and the passivity that leads to more severe responses.

The appropriate preparedness of a city/country can lessen significantly the consequences of a disaster. For example, as part of its general Disaster Preparedness Program, the Tel Aviv Municipality has developed an Emergency Treatment System (ETS) ([Spirman et al., 2001a](#)) that focuses on social and psychological welfare in times of emergency. The ETS is composed of eight multidisciplinary units dealing with: (a) on-site crisis (triage and evacuation); (b) family notification (of losses); (c) hospital liaison; (d) population behavior (information center); (e) brief psychological support over the phone; (f) emergency shelters (for evacuees); (g) community resources (volunteers and donations); and (h) delivery of basic needs. The ETS headquarters coordinates the units and their cooperation with relevant institutions (e.g., police, army) as well as with the clinical trauma and disaster community center, out of which the child mental health professionals operate ([Laor, 2001](#)).

## Interventions during the Second and Third Stages

### DEBRIEFING

The disaster syndrome may be viewed as a normal reaction akin to the fear response to danger or the mourning process following loss. In the long run, most affected individuals recover their functioning. It is assumed that the recovery of traumatized survivors may be facilitated by a process that allows them to review their experience (debriefing). In mass disasters debriefing is carried out in groups.

The aims of group debriefing are to allow the expression of traumatic experiences and flooding reactions; facilitate relaxation; promote cognitive organization and self-control; identify and mobilize internal and external resources; set realistic expectations; restore self-worth and hope; and prepare participants for future

experiences ([Stallard and Law, 1993](#)). The implementation of this structured technique with children may last one or more meetings and must be done by an educated professional. The technique may include play activities, such as individual or group drawing, writing, or imagery games. Participants are given time for expression and questions. The tasks of the leader are to protect limits, set rules, provide information, facilitate verbal and physical peer support, and manage containment. The leader also may identify symptomatic children and refer them for further help when the meeting ends, and distribute psychoeducational handouts on symptoms or positive coping strategies for follow-up.

Participants are encouraged to focus on thoughts, emotions, images, and sensations related to the event after reporting their trauma-related experiences. Special attention should be given to feelings of guilt and anger, because clinical experience shows that these may interfere with the process of working-through the trauma. Thereafter, participants explore personal and communal coping resources and use creative imagery to return to the "here and now," fantasize about a positive future and construct plans of action. Parallel sessions may be held with the parents.

Debriefing may be an efficient tool to help children and adults ([Chemtob et al., 1997](#); [Stallard and Law, 1993](#)); however, one should be aware that very early exposure to the memory of the traumatic event may interfere in some individuals with the normal affective-cognitive processes that lead to recovery, resulting in neutral effects or even an exacerbation of symptoms ([Mayou et al., 2000](#); [Wessely et al., 1998](#)). The specific therapeutic factors of the debriefing intervention, and who may benefit from it, remain open questions.

### *THE SCHOOL SETTING*

To assist as many children as possible, professionals may need to work with groups rather than individuals. Because teachers have trustful relations with children and parents, and most are ready to be educated and serve a therapeutic role, intact school environments are appropriate sites for societal recovery centers for early interventions ([Klingman, 1993](#); [Pynoos and Nader, 1988](#); [Vernberg and Vogel, 1993](#)).

Several researchers have found that support from classmates and teachers is a significant predictor of fewer posttraumatic symptoms after a major disaster and prevents withdrawal and isolation ([Pynoos and Nader, 1988](#); [Vernberg et al., 1996](#)). Furthermore, the class setting provides a predictable routine, clear expectations, consistent rules, and immediate feedback. It is recognized as the place to apply learning skills for exploring causes and consequences of disasters, and emphasizes that survivors may experience "normal reactions to abnormal events."

Teachers should allocate time to deal with traumatic experiences, model the children's responses, reinforce emerging coping skills, provide factual information and correct rumors, facilitate mutual support, identify suffering children, and prepare the class for future experiences. They also may encourage students to become active contributors to their family, school, and community.

For the program to be effective, mental health professionals should ensure that the teachers: (a) are not traumatized themselves; (b) are capable of mastering disaster-related educational techniques; and (c) have adapted their view of their role as teachers/educators to the new and harsh reality. To help regenerate the normal school setting after a disaster, the child psychiatrist needs to meet with the teachers, debrief them about their own experiences of the disaster, and clearly lay out the educational task at hand.

Based on these principles, [Wolmer and associates \(2001\)](#) implemented a three-stage supervision of one school's principal and teachers after the 1999 earthquake in Turkey. First, a group debriefing session was conducted to normalize responses and enable the expression of trauma-related affects (anger, guilt, helplessness, hopelessness). Thereafter, an experiential activity was introduced to help the teachers redefine their role with regard to the students as "educators" and "leaders." The authors stressed that in times of disaster, rather than merely covering the regular curricula, teachers were expected to maintain and enhance their role by providing individualized consideration, transmitting values, and conveying positive expectations. As part of this role, they were taught to implement a disaster-related classroom activation program (see the following). Finally, a continuous supervision process was begun, led by local professionals, wherein teachers not only were educated but also provided support for each other ([Goenjian et al., 1997](#)).

School-based interventions include single-session debriefings, small-group programs, and class activation programs.

### *SMALL-GROUP PROGRAMS*

Targeted small group programs within the school setting may benefit high-risk children or children who are more agitated and need closer attention than can be provided in the classroom ([Gillis, 1993](#); [Klingman, 1993](#)). [Smith and colleagues \(1999\)](#) formulated a three-session program to teach recovery techniques to small groups of children affected by disaster. The techniques used are psychoeducation, imagery and cognitive techniques, and exposure practice. Each session is dedicated to one domain of the posttraumatic syndrome: intrusion, avoidance, and arousal. The professional may also offer a fourth session for bereaved children and a session with parents, to provide them with information and suggest ways for them to help their children.

### *CLASS ACTIVATION PROGRAMS*

Programs implemented in the classroom itself may vary in focus, scope, and depth, but all are intended to minimize stigma, encourage normalcy, and reinforce the expectation that the children will soon resume their roles as students ([Klingman, 1993](#); [Pfefferbaum, 1997](#); [Vernberg and Vogel, 1993](#)). It is important to stress, however, that the teachers themselves may be struggling with severe posttraumatic symptoms and personal losses, and consequently, feel unable to help their students. Some teachers may try to avoid dealing with reminders of the event by stressing that children have no need to talk about their traumatic experiences ([Pynoos et al., 1998](#)).

[Galante and Foa \(1986\)](#) provided children who survived an earthquake in Italy seven monthly sessions for discussing and dealing with related feelings, and noted a significant reduction in symptoms. Other models also utilize expert mental health professionals in school settings with or without the presence of the teacher ([Eth, 1992](#); [Klingman, 1993](#)). Direct teacher involvement, however, is encouraged for disasters of large proportions, when expert resources prove insufficient. The class activation of [Wolmer and associates \(2001\)](#) was led by the teachers, and it consisted of an introductory meeting with the parents to provide information about the program and the children's expected reactions to the disaster, and to engage them in the process. The remaining eight 2-hour meetings of the whole class focused on various aspects of the recovery process (e.g., debriefing, establishing a safe place, learning about the earthquake, loss and death, dealing with anger and lessons of life, planning the future). The program combined psychoeducational modules, cognitive-behavioral techniques, play activities, and an ongoing documentation in personal diaries.

## **Clinical Interventions**

### *INDIVIDUAL INTERVENTIONS*

Although efficient and cost effective, group interventions may not be enough for the most affected children. Controlled and uncontrolled studies have confirmed the effectiveness of brief cognitive behavioral treatment in traumatized children ([Perrin et al., 2000](#)). Other modes of individual psychotherapy have been employed, such as play therapy, psychodynamic psychotherapy, or eye movement desensitization and reprocessing (EMDR) ([Lovett, 1999](#)). Particular attention should be directed to prior and current comorbid pathology as well as a thorough differential diagnosis (e.g., mania, ADHD).

After the 1988 earthquake in Armenia, [Goenjian and colleagues \(1997\)](#) implemented a brief treatment program combining classroom group psychotherapy and individual sessions focusing on trauma and grief. The sessions, led by therapists, allowed for open discussion of the traumatic experiences and associated feelings, assisted the children in solving intrapersonal and interpersonal problems, and offered effective cognitive-behavioral techniques to manage thought distortions, disturbing images, and stress-related sensations.

### *GROUP INTERVENTIONS*

#### *Parent-Child Groups*

Families have the potential to either protect children and mitigate their postdisaster suffering or jeopardize their adjustment to and processing of the event, thereby



exacerbating their symptoms. After disasters, children and parents tend to avoid discussing their distress, probably to avoid further disturbing each other ( [Udwin, 1993](#)). However, studies consistently show a significant association between the symptomatic response of parents (particularly of mothers) and their children ( [Laor et al., 1996, 1997](#); [Winje and Ulvik, 1998](#)), which may have a traumatic impact on the whole parent-child dyad ( [Wolmer et al., 2000](#)).

Based on the previous successful application of parent-child group psychotherapy in the treatment of child anxiety disorders ( [Toren et al., 2000](#)), and as a second stage of school-based intervention, [Laor and associates \(2001\)](#) formulated an eight-session therapeutic protocol for mothers and children (four to five dyads) with chronic posttraumatic symptoms. Led by two therapists, the group addressed dynamic, cognitive, and behavioral aspects of the disaster syndrome, and offered techniques to manage anxiety; relieve, control, and transform distressing affects; correct thought distortions; and plan for the future. Special attention was paid to identifying and correcting maladaptive family dynamics and helping mothers and children recover their attachment and roles. Preliminary clinical and empirical results showed significant symptomatic alleviation as well as a dramatic improvement in familial communication and mutual support.

### Mothers' Groups

Group interventions with mothers allow for the indirect focus on preschool children, a population that may not be reached in formal settings yet may show maladaptive behavioral reactions. These reactions are known to correlate highly with the mothers' psychological response ( [Wolmer et al., 2000](#)). Providing structured therapeutic interventions, psychoeducation and practical suggestions in regard to the children as well as strengthening the participants' confidence in their maternal role are important objectives for these groups ( [Spirman et al., 2001b](#)).

### Community-Based Interventions

Disasters affect whole communities, threatening social structures and functions. To be effective, interventions require collaborative efforts among nongovernmental organizations and formal and informal agencies. The development of a local leadership of committed individuals and its empowerment to actively meet the short- and long-term needs of the community provides a valuable source of support ( [Rappaport, 1987](#)).

In this regard, child psychiatry relief programs need to respond to disasters on three levels: (a) *The family*. Families may suffer from injured, lost, or dead members; relocation and unemployment; loss of boundaries, routines, and values (sometimes internalizing the societal anarchy); and loss of esteem and hope. (b) *The neighborhood*. During disasters, neighborhoods are subject to physical and economic destruction; loss of routines, boundaries, and safety; disintegration of informal networks, and restriction of leisure time activities; these apply also to temporary neighborhoods established by relocated families. (c) *The community*. Communities suffer from an insufficiency of proper leadership and resources, frozen initiative, dependence on external resources, destruction of social and cultural institutions (schools, community centers, religious centers) and as a result, a foreshortened sense of communal future.

Drawing on auxiliary social functions and structures introduced from the outside, as well as on professional clinical and social teams, it is possible to help temporary communities of displaced population gradually to develop coping and functioning. In a nutshell, the goal of the community-based intervention is to transform evacuated fragments of families and singles (first stage) into self-governing communities with autonomous individuals and families (third stage) ( [Laor, 2001](#)) ( [Table 75.1](#)). Professionals also may help facilitate the creation of new job resources in the community because children cannot be fully rehabilitated until their parents resume work and regain income.

Stage	Phase	Intervention	Notes
Social structures in the affected community	1	Empowerment groups	Community center
	2	Empowerment groups	Religious center
Auxiliary social structures and functions introduced from the outside	1	Self-help groups	Self-help groups
	2	Self-help groups	Self-help groups
Professional clinical health interventions	1	Individual therapy	Individual therapy
	2	Group therapy	Group therapy
Social structures	1	Self-help groups	Self-help groups
	2	Self-help groups	Self-help groups
Interventions	1	Self-help groups	Self-help groups
	2	Self-help groups	Self-help groups

**Table 75.1. The Recovery Process from a Mass Disaster in Evacuated Communities**

Using the intervention principles described in the preceding, child mental health together with child community workers and the local leadership can help set up community center programs for young mothers, children, and adolescents. Empowering- and hardiness-enhancing programs may include arts, sports, gardening and decorating, continuous education, job clubs, and volunteer recruitment and training in different areas ( [Kobasa, 1979](#)). By *empowerment* we mean a process of involvement by which individuals and communities replace their helpless stance by recovering their dignity and self-esteem, enhancing their critical self-awareness, control over resources and objectives, sense of personal and collective responsibility, and self-efficacy ( [Rappaport, 1987](#)). Individuals are able to identify specific needs and discover hidden leadership qualities, whereas communities gain a greater sense of interdependence, cohesion, and cooperation. In the case of dislocated population, communal empowerment is the process by which communities are formed and achieve greater control over their environment.

Interventions at the level of the whole community facilitate the integration of community members with the natural and social environments. For example, as part of the Community Reactivation Program after the 1999 earthquake in Turkey, [Spirman and coworkers \(2001b\)](#) developed a 2-week summer program for a whole village of displaced individuals to transform the community and its habitat as culturally internalizing death and alienation (e.g., withdrawn fathers, unemployment, uncultivated neighborhood). The method rested on five principles:

1. Team-up and train international and local youth leaders to serve as instructors on-site.
2. Identify a natural habitat adjacent to the village, representing "life" for the community, where the program's activities (e.g., arts, sports) take place.
3. Enlist the revitalization and initiative achieved, to construct recreation areas and cultivated gardens, within the original village habitat. As a result, the artificial prefab neighborhood representing "death and displacement" turns into a lively community.
4. Designate an area within the village to serve for the erection of an artistic commemoration monument, for which individuals are invited to contribute dear personal remains. The event culminates in an anniversary ceremony. This process of collective mourning allows for an honorable and lively representation of dear loss and the dynamic reconnection with historical identity.
5. Involve social and political leaders, local and international, to facilitate the social integration of the community.

These interventions allow child mental health professionals entry into the sphere of the long-term effects of the tertiary disaster, the sociocultural losses that threaten the collective ideology and identity. Professionals may take part in the modification of the school curricula to address group mourning and hardiness, collective memorials as well as celebrations of rebirth. The juxtaposition of commemoration and rebirth ceremonies help individuals to gain a new meaning of life in the face of deep mourning and lead to integration on both the personal and communal levels, which offers children an uninterrupted supportive matrix. For example, in Israel, the Independence Day celebrations follow on the heels of the Memorial Day ceremonies, and in Turkey, communities celebrated group circumcision of children about 1 month before the 1999 earthquake memorial ceremony.

### Pharmacologic Interventions

It is widely accepted that psychotherapy, rather than medication, is the first-choice modality for posttraumatic states ( [Foa et al., 1999](#); [Shiloh et al., 1999](#)). Most proposals for pharmacotherapy with children are based on studies with adult PTSD. These studies, too, invite improved controlled design ( [Marshall et al., 1998](#)). The systematic assessment of pharmacologic treatments for traumatized children has been limited so far ( [Perrin et al., 2000](#); [Vernberg and Vogel, 1993](#)). Benzodiazepines may be used in cases of acute PTSD, but only for the short-term and only intermittently ( [Foa et al., 1999](#)). They should be used with caution, particularly because, in adults, they have been implicated in worse long-term outcome ( [Gelpin et al., 1996](#)). Professionals should beware that well-intentioned donations of medications by drug companies and the urge to reach as many individuals as fast as possible may lead to anarchic management and nonprofessional

medication of children. A detailed description of pharmacologic interventions is presented in the relevant chapters.

Nevertheless, medications may be effective in alleviating anxiety or depressive symptoms ( [Terr, 1989](#)), and as part of the general principles of management in situations of disaster, they can be given even in the early stages of intervention in certain cases: to children with a known history of psychopathology prior to the disaster; individuals who do not respond to debriefing and short-term specific interventions; and members of at-risk families who are flooded by severe symptoms. These medications must be directed at specific symptoms, such as intrusion, hyperarousal, and impulsivity (e.g., carbamazepine, lithium, clonidine), anxiety (e.g., buspirone, alprazolam), depression (e.g., tricyclic medication or SSRIs), psychotic symptoms or severe aggression (e.g., antipsychotic drugs) ( [Shiloh et al., 1999](#)).

### **Taking Care of the Caretaker**

Professionals engaged in disaster intervention programs (psychologists, teachers, social workers, and nurses) are more analogous to marathon runners than to short-distance sprinters. Project coordinators need to be aware of each worker's strengths and weaknesses, personal losses, and vulnerabilities in order to regulate their exposure and prevent burnout. Program leaders need to help relief workers enhance their own coping mechanisms, tolerate the shock inherent in their work, and maintain high levels of commitment and motivation ( [Austin and Godleski, 1999](#); [Cohen, 1987](#)). Therefore, leaders should facilitate adequate training and peer supervision, encourage mutual support, lead debriefing sessions, teach stress management skills, and introduce appropriate work breaks (limiting shift durations or breaks in schedules).

## **DISASTER RESEARCH**

Disaster research is of utmost importance yet extremely hard to perform. Most disasters occur unexpectedly, and even those that are predictable have such an overwhelming impact, they exhaust all professional and economic resources. Furthermore, even when professional curiosity is maintained, assessment measures are enlisted, and questions are defined, both the victims and the clinicians resist the implementation of the research. They tend to perceive research under these conditions as hostile, foreign, exploitative, and abusive, intended to satisfy an alien agenda that is irrelevant to their priorities. There is a kernel of truth to this: The hands that give out questionnaires could have offered bread. Furthermore, from the standpoint of the clinician, many of the responses of disaster victims reflect a normal and time-limited reaction to stress and should not be structured as pathology.

The perspective offered in this chapter enables the mental health professional to deal seriously with these issues and not simply explain them away. Research initiatives need to be integrated within the systemic interventional program and rely on the direct assessment of the affected population at every stage. That way, real risks and needs can serve a basis for rational planning (e.g., resource allocation, assignment to different therapies) as well as for improvement of existing programs. It is the responsibility of the professional to educate community leaders to take practical advantage of assessment data.

The disaster scene, a large-scale natural experiment, offers access to communities that constitute different types of research groups and controls. Furthermore, in each community, one may encounter a large number of whole families whose members were simultaneously exposed and affected in different manners (direct or indirect) and degrees and for different lengths of time, as well as individuals suffering from losses of varying severity. An important issue in studies of children is the phenomenology and biological susceptibility to the disaster syndrome; that is, the interplay of traumatic grief, posttraumatic symptoms, and dissociation with psychoneuroendocrinologic and psychophysiological parameters. This particular setting provides a unique opportunity for genetic studies. Special attention also ought to be given to partial and delayed onset types of disorder as well as concurrent psychiatric and medical morbidity.

Another area of interest is the control study and comparative effectiveness of the various interventions (social interventions versus pharmacotherapies and psychotherapies), and the assessment of parameters of community hardiness and vulnerability. Other important avenues of research are the long-term sequelae of disaster in terms of developmental psychopathology of high-risk populations, the transgenerational transmission of trauma and grief, and the development of sociopolitical attitudes and preferences.

We would like to stress here that the search for answers to these intellectual questions, pressing though they may be, cannot at any time violate the privacy of the children being studied, and clinicians must be careful to comply with the accepted ethical guidelines. Institutional Review Boards can facilitate the process by offering a fast track for disaster-related research proposals.

## **CONCLUSION**

Disasters irrevocably destroy the space within which children and families thrive, thereby disrupting normal development. The overwhelming quality of the event combined with the massive extent of the loss gives rise to a complex clinical and social picture that may be termed the disaster syndrome. Children, families, neighborhoods, and whole communities are affected. Immediate damages can be only partially remedied; therefore, the physical, psychological, and social effects are long lasting.

For effective intervention, the child mental health professional needs a comprehensive systemic, social, and mental health perspective in order to develop the proper program, team-up with the proper authorities, accurately assess needs, and implement treatment. Best results are effected when the community is prepared. Some interventions are clinical and specific; educated professionals who work with children in schools and community centers can mediate others. No single community can cope on its own. This requires networking in advance on the local, national and the international levels. The challenge of disasters, thus, is offered to our vision of the brotherhood of humanity. A global community committed to the cause of children must respond to the challenge.

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# 76 PEDIATRIC PSYCHOPHARMACOLOGY I

## Pharmacokinetic and Pharmacodynamic Principles

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#### [Chapter References](#)

## OVERVIEW

In this first of two chapters on pediatric psychopharmacology, general principles of drug handling (*pharmacokinetics*) and drug mechanism of action (*pharmacodynamics*) are reviewed. The chapter highlights those aspects in which developmental factors play an important role, and serves as a complement to the following and related chapter, in which specific drug classes and individual agents are discussed in detail.

*Pharmacokinetic* principles relate to the handling and disposition of drugs within the body: to those biological processes occurring prior to engagement of drugs that lead to changes over time in drug concentration in body tissues and fluids. Drug concentration in target organs is responsible for determining how long a drug's therapeutic and adverse effects will last. Changes during development in drug absorption, distribution, metabolism, and excretion may all have an impact on the delivery of drug to target tissues. By contrast, *pharmacodynamic* principles are concerned with the biochemical and physiologic effects of drugs at their active sites, with their specific mechanisms of action. The effects of a medication may similarly change during development, as brain regions or neurotransmitter systems develop and mature at different rates. Stated succinctly, "pharmacokinetics describes what the body does to a drug; pharmacodynamics what a drug does to the body" ([Janicak et al., 1993](#)).

## PHARMACOKINETIC PRINCIPLES

In the first part of this chapter, pharmacokinetic factors that influence drug disposition during development are addressed. First, basic pharmacokinetic principles that are shared by children, adolescents, and adults are reviewed. Next, the broad pharmacokinetic differences that distinguish the pediatric population from adults are examined. This is followed by a review of specific factors affecting drug disposition, with an emphasis on developmental influences. One of the more clinically relevant applications of pharmacokinetic principles is in helping to understand (and at times predict) important drug interactions. Because of this, additional emphasis has been placed on mechanisms (e.g., the cytochrome P450 system) that are relevant to a large number of such interactions.

### Basic Principles

An understanding of pharmacokinetic principles is important for safe and effective patient care. Pharmacokinetic factors are often critical in a variety of clinical decisions, such as choosing between agents within a same drug class, switching between different medication preparations, adjusting dosages, preventing drug interactions, or correctly utilizing and interpreting therapeutic drug levels ([Preskorn, 1993b](#)).

Pharmacokinetics can be conceptualized as having four functionally distinct phases: absorption, distribution, metabolism, and excretion. Absorption and distribution are primarily responsible for determining the speed of onset of drug effect, whereas the processes of metabolism and excretion terminate the action of the pharmacologic agent by removing the active form of the drug from the body. Taken together, these four phases determine the duration of drug activity ([Paxton and Dragunow, 1993](#)).

Once a drug gains entry to the bloodstream, it is diluted in the plasma and bound at varying degrees to plasma proteins. The drug, usually protein-bound, is then either excreted by the kidneys or carried to the liver and transformed to a more water-soluble (and usually inactive) metabolite, which can then be excreted in urine, bile, or feces. This complicated and interdependent series of events is designed to reduce the effect of the foreign molecule, with the ultimate goal of eliminating the drug from the body. If the dose of a drug is sufficiently large to withstand this "pharmacokinetic assault," then a fraction of the drug molecules will endure to produce its pharmacodynamic effect ([Paxton and Dragunow, 1993](#)).

A basic working knowledge on key pharmacokinetics principles is relevant to the clinical practice of pediatric psychopharmacology so as to understand the fate of administered drugs. Practically all psychotropic medications follow *first-order* (or *linear*) kinetics, in which the amount of drug eliminated is proportional to its amount circulating in the bloodstream. First-order kinetics provide close to a 1:1 relationship between changes in dosage and plasma concentration. Such a linear association allows for clinically relevant predictions of the impact of a dose change on circulating drug levels. By contrast, *zero-order* (or *nonlinear*) kinetics prevail when metabolizing or eliminating mechanisms become saturated. This results in a fixed amount of drug being eliminated per unit of time, regardless of the plasma level. Certain drugs, such as fluoxetine, nefazodone, or alcohol, demonstrate zero-order kinetics at all clinically relevant doses, rather than just at saturation, making the relationship between dose changes and subsequent plasma levels much less predictable ([Janicak et al., 1993](#)).

For drugs that follow first-order kinetics, the concepts of biologic half-life and steady-state concentration are germane to the practicing clinician. Biologic *half-life* ( $t_{1/2}$ ) is the time required for the concentration of drug to decrease by one-half. In clinical practice, this parameter is usually assessed by measuring the decay of plasma or serum drug concentration and is referred to as the plasma (or serum) half-life. Plasma half-life values can be useful when determining dosing intervals. At consistent dosing intervals, it is the plasma half-life that determines the plasma *steady-state concentration* ( $C_{SS}$ ). This is defined as equilibrium between the amount of drug ingested and the amount of drug eliminated, resulting in no net change in plasma concentration over time.  $C_{SS}$  is reached in a time equal to approximately five half-lives. Conversely, if drug intake is abruptly stopped, then it takes a time equal to five half-lives for almost complete elimination.

### Pharmacokinetics in Children and Adolescents

There are many pharmacokinetic similarities between adults and children and adolescents. Indeed, age-independent genetic influences on protein binding, metabolism, and elimination can be more salient than those influences attributable to age and developmental change.

Nonetheless, children and adolescents do display unique pharmacokinetic properties. Unfortunately, data on children are scarce and fall considerably behind that available in adults. The result of this informational dearth is that dosage recommendations and therapeutic ranges utilized clinically in children and adolescents are, at least initially, extrapolated from adult studies, often arbitrarily ([Gilman and Gal, 1992](#)). The appropriate dose of a psychotropic medication in the pediatric age group should be empirically determined; a proportionately reduced adult dose based on body weight is frequently subtherapeutic or toxic. In addition, infants, children, and adolescents are not a homogenous group in terms of drug distribution patterns ([Jatlow, 1987](#)). These differences can be especially dramatic around the time of

puberty, when the release of gonadal hormones can strongly influence plasma drug concentrations ( [Morselli and Pippenger, 1982](#)).

Although pharmacokinetic data in children and adolescents are limited, one basic observation is suggested clinically: *Children and adolescents require larger, weight-adjusted doses of most medications than do adults to achieve comparable blood levels and therapeutic effects* ( [Jatlow, 1987](#)). This appears to be mostly a function of shorter half-life owing to an increased rate of metabolism and elimination.

## Factors Affecting Drug Disposition

### Absorption

Drugs gain entry into the body through a variety of portals. The bioavailability of a medication in the systemic circulation—its amount available to exert a biological effect on target tissues—is determined by its absorption and, for orally administered medications, by the *first-pass* effect of liver metabolism. Drugs given orally are absorbed in aqueous solutions through either the stomach or, as is the case of most psychotropic medications, the small intestine. The major factors influencing gastrointestinal absorption are pH-dependent diffusion and gastric emptying time. Absorption depends heavily on the route of entry, with intravenous administration being the equivalent of 100% absorption. Oral administration is by far the most common portal of entry but also the most unpredictable in terms of final bioavailability. An orally administered drug is absorbed from the gastrointestinal tract and must first enter the portal circulation, where it passes through the liver before reaching the systemic circulation. A considerable portion of the drug is hepatically metabolized during this first pass. Thus, only a fraction of the drug that was absorbed from the gastrointestinal tract enters the systemic circulation.

Little information is available regarding the effect of age on the absorption of psychotropic medications, although there are several theoretical considerations regarding the influence of this process in children and adolescents. For instance, stomach contents tend to be less acidic in children than in adults, causing weakly acidic drugs to be more highly ionized. Weakly acidic drugs may be absorbed more slowly in children because it is the unionized fraction that is absorbed from the stomach. Theoretically this process could affect anticonvulsants, amphetamines, and antidepressants ( [Taylor, 1994](#)). Another theoretical concern is that children generally have fewer and less diverse intestinal microflora. The fact that drugs such as phenothiazines can be absorbed or metabolized in the gut wall may help to explain why some children either are unusually resistant to phenothiazines or require a surprisingly large oral doses ( [Taylor, 1994](#)). Another factor that could reduce overall absorption is that intestinal transit time appears to be shorter in young children, which could result in incomplete uptake of slow-release preparations ( [Gilman and Gal, 1992](#)).

It is important to remember that despite the theoretical considerations outlined in the preceding, there are no data indicating a generally reduced absorption of orally administered drugs in children ( [Jatlow, 1987](#)). Anecdotal information and clinical experience suggest that absorption of certain psychotropic medications may be even more rapid in children than adults. Rapid absorption, however, may not be desirable because it results in greater peak-to-valley fluctuations in blood levels. This does not affect the final average steady-state level, but may be important because drug toxicity can be a function of peak plasma concentration, as in the case of lithium-induced tremor.

### Distribution

Drugs are distributed into intravascular and various extravascular spaces following absorption. Numerous physical factors can influence the distribution of a drug throughout the body: the size of body water compartments and adipose tissue depots, cardiac output, regional blood flow, organ perfusion pressure, permeability of cell membranes, acid–base balance, and binding to plasma and tissue proteins ( [Morselli and Pippenger, 1982](#)). Each of these factors may change during development, resulting in changes in the distribution of a drug and, subsequently, in its pharmacologic effect.

The two most important factors affecting distribution that change substantially during development are fat stores and the relative proportion of total body water to extracellular water. The relationship between the amount of drug absorbed (D), plasma concentration (Cp), and volume of distribution (Vd) can be summarized by the simple equation:  $C_p = D/V_d$ . Note that the larger the Vd, the smaller the Cp.

The proportion of body fat substantially affects the volume of distribution of highly lipophilic drugs, including most neuroleptics and antidepressants. The proportion of body fat is highest in the first year of life, followed by a steady decrease until an increase occurs prepubertally ( [Jatlow, 1987](#)). Children and adolescents at different ages have varying degrees of fat stores, and in general tend to have a proportion of body fat less than that found in adults. Hence, in children one would expect to find a larger plasma concentration with lipophilic drugs when compared with adults after being given the same weight-adjusted dose. It has been demonstrated, however, that children actually exhibit a lower plasma concentration than do adults under these conditions. Therefore, other mechanisms (e.g., increased metabolism) must explain the lower plasma concentration of lipophilic drugs in children ( [Jatlow, 1987](#)).

The relative volume of extracellular water is high in children and tends to decrease with development. For example, total body water decreases gradually from about 85% of body weight in a small premature infant to about 70% in the full-term newborn to about 60% in the 1-year-old infant, a level that is generally maintained throughout adulthood. Similarly, extracellular water decreases gradually from about 40% to 50% of body weight in the newborn to about 15% to 20% by age 10 to 15 years ( [Fetner and Geller, 1992](#)). Thus, drugs that are primarily distributed in body water (e.g., lithium) can be expected to have a lower plasma concentration in the pediatric population compared with that in adults because the volume of distribution is higher in children.

Following absorption and first pass through the liver, drugs are transported in the general circulation in two forms that are in dynamic equilibrium with each other: bound to plasma protein and unbound (free). Only the unbound drug is usually available to pass across membranes and have pharmacologic effects. Although drug protein binding may be reduced in young children, it does not appear to be an important developmental factor in older children and adolescents. A final theoretical consideration in children and adolescents is the relative permeability of the blood–brain barrier when compared with that in adults. This increased permeability could result in increased bioavailability of drugs within the central nervous system (CNS). This relative “porousness,” however, would concomitantly permit greater amounts of protein to enter the cerebrospinal fluid. Any drug that crosses the blood–brain barrier but also substantially binds to proteins, such as most psychotropics and anticonvulsants, in effect would then have its bioavailability reduced ( [Taylor, 1994](#)). Naturally, this special dispensation does not apply to unbound drugs such as lithium.

### Metabolism

Most drugs are lipid soluble, which is usually a necessary requirement for absorption, distribution, and availability at receptor sites. *Lipophilic* drugs need to be metabolized into more polar or *hydrophilic* forms (i.e., having a greater affinity for water) in order to be effectively excreted, however. Enzymes that catalyze these metabolic reactions are found in greatest abundance in the liver, the main organ of drug metabolism, but also in the wall of the small intestine, skeletal muscles, kidneys, and lungs.

Although some drugs are excreted unmetabolized (e.g., lithium, gabapentin), most undergo extensive biotransformation in the liver ( [Janicak et al., 1993](#)). *Phase I metabolic reactions*, including *hydroxylation*, *reduction*, and *hydrolysis*, convert drugs to forms more suitable for elimination. Hepatic microsomal enzymes (the cytochrome P450 system) are responsible for phase I reactions. In that role, they are of sufficient clinical relevance to warrant their being addressed separately (see following section). The products of phase I reactions, collectively referred to as *metabolites*, are usually less active and toxic than their parent compounds. A notable exception of clinical significance is desipramine, the demethylated active metabolite of imipramine.

In *phase II reactions*, *conjugation* of metabolites generated in phase I takes place with glucuronic acid, sulfate, or others. Conjugated compounds are then readily excreted in urine or other body fluids. It is clinically important to note that some drugs are never metabolized in a phase I reaction and instead simply undergo conjugation by glucuronic acid. This conjugative reaction may occur in almost any organ and therefore is not solely dependent on hepatic function. Hence, drugs such as the 3-hydroxybenzodiazepines (e.g., lorazepam, oxazepam) are rapidly cleared at equal rates regardless of age as long as renal function is normal ( [Janicak et al., 1993](#)), and are thus preferred in instances of liver insufficiency.

The relatively higher dosage requirement of most psychotropic drugs in children is most commonly explained by increased hepatic metabolic capacity. Cytochromes (CYPs), for example, come “on-line” at different developmental epochs of fetal life or infancy and reach 40% of adult capacity by the first year of life ( [Oesterheld, 1998](#)). During latency, CYPs are more efficient than in adulthood, and their activity declines to adult levels after puberty. Thus, higher doses are required on a mg/kg basis to achieve comparable steady-state plasma levels in latency children as compared to adults or adolescents, particularly for CYP-metabolized drugs.



## Excretion

The kidney is the most important organ for drug excretion. Three mechanisms are involved in the renal excretion of drugs: *glomerular filtration*, *tubular secretion* by active transport mechanisms, and *reabsorption*.

In contrast to hepatic function, renal functioning in infants approximates that of adults. Beyond early infancy, developmental changes in renal function apparently do not contribute substantially to age-related differences in psychotropic drug disposition, with the probable exception of lithium ( [Jatlow, 1987](#)).

## Drug Interactions: Focus on the Cytochrome P450 System

Given the increasing recourse to medication combinations and polypharmacy as an accepted and thoughtful ( [Wilens et al., 1995](#)) or as a controversial and potentially problematic ( [Woolston, 1999](#)) practice in child psychiatry, astute clinicians need to be aware and clinically suspicious of potential drug interactions. For children concurrently treated with nonpsychiatric drugs (including even “benign,” over-the-counter agents) the degree of oversight needs to be particularly heightened. Cytochrome-based interactions are especially common and relevant to psychotropic drugs; therefore, they are reviewed here in detail.

Cytochromes are heme-containing enzymes, located principally in the liver, which metabolize two types of substrates: endogenous (e.g., the body's own steroids, lipids, and fatty acids) and exogenous (e.g., toxins, drugs). The sequencing of CYP amino acids has led to a classification system based on similarity: Arabic numbers 1 through 4 designate family members, letters A through E label subfamily members, and Arabic numbers note specific enzyme (isoform, gene). CYP3A4, for example, is a member of family 3, subfamily A, and enzyme 4. CYP3A is the major CYP involved in human hepatic drug metabolism, constituting 30% of total CYP hepatic content that serves as a high-capacity reservoir. The other clinically relevant CYPs include 1A2, 2C9, 2C19, 2D6, and 2E1.

Most psychotropic drugs undergo both phase I and phase II reactions. A few drugs are metabolized by only one CYP (e.g., desipramine via CYP 2D6, triazolam via CYP3A). Some others undergo phase II conjugation only (e.g., lorazepam, oxazepam, and lamotrigine are glucuronidated only); however, most drugs require multiple CYPs to be completely metabolized. As examples, sertraline is *N*-demethylated via six different pathways ( [Greenblatt, 1999](#)), and clomipramine (CMI) demethylated by CYP1A2, CYP2C19, and CYP 3A to desmethylclomipramine (DCMI), an active metabolite that is then hydroxylated by CYP 2D6. In addition, CMI is directly hydroxylated by CYP 2D6.

Cytochrome-based metabolism of drugs is influenced by genetic factors. For example, 7% to 10% of whites have a genetic deficiency of CYP 2D6 and are less efficient in metabolizing 2D6 substrates, including many psychotropic agents ( [Table 76.1](#)). For these individuals, blood levels of drugs metabolized by 2D6 may increase considerably as unmetabolized drug enters the blood. Some Asian individuals have a 2D6 variant that causes them to be “somewhat slow” metabolizers; they require lower dosing of relevant drugs to achieve therapeutic blood levels. CYP 2C9 and CYP 2C19 also are genetically polymorphic. One to three percent of whites are slow metabolizers of CYP 2C9, and 18% to 23% of Japanese and 2% to 3% of whites and African Americans are slow metabolizers of CYP 2C19 ( [Bertilsson, 1995](#)). Fewer than five whites in a thousand are slow metabolizers of both CYP 2D6 and CYP 2C19, putting them at particular risk for high levels of substrates that are metabolized by *both* pathways (e.g., some tricyclics, propranolol, and citalopram) ( [Table 76.1](#)).

**Table 76.1. Cytochrome-Based Metabolism of Drugs**

## Cytochrome P450 Inhibitors and Inducers

Drugs that interact with CYPs can inhibit, induce, or have no effect on CYP activity. If a CYP is inhibited (or blocked), then more unmetabolized drug will enter the circulation, leading to increased levels of the drug (similar to a phenotypic slow metabolizer). Conversely, if a CYP is induced, additional enzyme will be available for metabolism, leading to lower drug levels. Inhibition occurs rapidly because inhibition requires blocking existing pathways. Induction takes some time to start (3 to 10 days) or stop (5 to 12 days), because protein synthesis must first occur and then cease. [Table 76.1](#) contains the five most relevant CYPs and selected common psychotropic and other pediatric drugs metabolized by them. Common potent inhibitors and inducers of CYPs are grouped alphabetically by CYP.

The table can be used to anticipate CYP-based drug interactions. If both drugs are listed on the same vertical axis, then a drug interaction is likely (especially for drugs that have only one metabolic pathway). Blood levels could be affected, depending on whether the medications are inhibitors, inducers, or substrates. Many of the most serious drug interactions occur when a potent CYP is added to a drug that is a substrate of that CYP and has a narrow therapeutic index. For example, fluoxetine or paroxetine (potent inhibitors of CYP 2D6) can increase the plasma concentration of desipramine (a substrate of CYP 2D6) about 400%. Sertraline, a less potent inhibitor of CYP2D6, increases desipramine levels only 25%. In contrast, fluvoxamine (a potent CYP3A inhibitor) can double levels of phenytoin within hours. Many of the drug interactions that result in inactivation of drugs occur when a potent CYP inducer is added to a same CYP substrate (e.g., ovulation and pregnancy can result when levels of an oral contraceptive, a CYP3A substrate, are reduced by carbamazepine, an inducer).

Although there are more limited data, glucuronidation (phase II) enzymes are also susceptible to inhibition and induction. As an example, lamotrigine is handled by glucuronidation alone, and the addition of valproate (a glucuronidation inhibitor) can significantly elevate lamotrigine levels, thereby increasing the likelihood of developing life-threatening Stevens-Johnson syndrome ( [Anderson, 1996](#)).

## PHARMACODYNAMIC PRINCIPLES

### Mechanisms of Action of Psychotropic Drugs

The second section of this chapter provides basic principles on three aspects of human brain function critical to an understanding of psychotropic drug mechanisms of action: neuronal circuitry, synaptic neurotransmission, and intracellular information processing. This information can be clinically relevant in helping to rationally guide therapeutic decision-making in pediatric psychopharmacology.

Modulation of neuronal activity via neurotransmitters is a fundamental mechanism of brain function. The premier therapeutic avenue of psychiatry, arguably, is the facilitation or inhibition of neurotransmitter systems in the brain, either through the administration of drugs or psychotherapy.

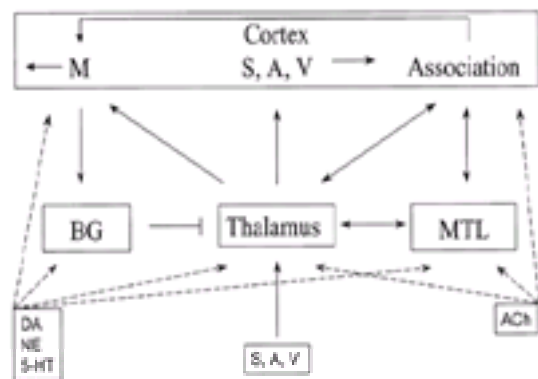
The release of neurotransmitters, their mechanisms of action, and their effect on target neurons are complex and still poorly understood; however, despite the diversity of neurotransmitters and receptors in the human brain, all forms of neural communication have the common goal of modulating neuronal activity. This is achieved by changing either the electrical or biochemical properties of the cell. The balance of intracellular and extracellular ions characterizes the electrical properties of the neuron. At rest, there are more negatively charged ions inside than outside the cell, thereby creating a negative resting membrane potential. Decreasing the resting potential leads to excitation, increasing it, to inhibition. The biochemical properties of the neuron are characterized by the expression of specific genes, the production of proteins, and the creation of a distinct metabolism. Regulation of gene expression and protein function determines the biochemical

status quo of the cell.

## Neuronal Circuitry

The adult cerebral cortex has about 1,011 neurons and each neuron establishes about 103 to 104 connections to other neurons. Neurons are arranged in distributed networks of brain regions to govern human behavior (Mesulam, 2000). This involves the collection of sensory information through perceptual modules, the creation of a representation, and the production of a response.

Here we focus on four major anatomic systems that are crucial for these three steps of information processing: the cortex, thalamus, basal ganglia, and medial temporal lobe (Fig. 76.1). The function of these four systems is modulated by several groups of neurons that are characterized by their use of a specific neurotransmitter.



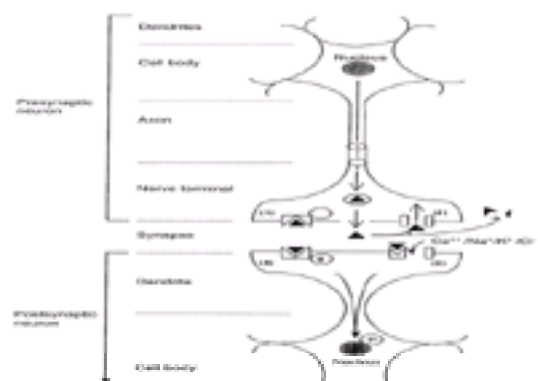
**Figure 76.1.** Neuronal circuitry: basic scheme of information processing in the human brain. Straight arrows indicate glutamatergic pathways. The BG–thalamus projection is GABAergic. The broken arrows indicate the widespread, neurotransmitter-specific projections arising from the basal forebrain (ACh) and brain stem (DA, NE, 5-HT). A1, primary auditory cortex; ACh, acetylcholine; BG, basal ganglia; DA, dopamine; 5-HT, serotonin; M, motor cortex; MTL, medial temporal lobe; NE, norepinephrine; S1, primary sensory cortex; V1, primary visual cortex. See text for further details.

1. The thalamus is the gateway to cortical processing of all incoming sensory information, here represented by the three major systems: somatosensory, auditory, and visual (S, A, V) (Fig. 76.1). Primary sensory cortices (S1, A1, V1) receive information from the appropriate input modules (sensory organ + thalamus).
2. The association cortex integrates information from primary cortices, subcortical structures, and brain areas affiliated with memory to create an internal representation of the sensory information.
3. The medial temporal lobe (i.e., hippocampus, amygdala) serves two major functions in the brain: to integrate multimodal sensory information for storage into and retrieval from memory, and to attach limbic valence to sensory information (e.g., pleasant or unpleasant, fight or flight).
4. The basal ganglia are primarily involved in the integration of input from cortical areas. The basal ganglia modulate cortical activity via a cortico-striato-pallido-thalamo-cortical (CSPTC) loop. The most prominent projections to the striatum arise from the motor cortex.

All major projections in this basic circuitry (Fig. 76.1) are glutamatergic, except for the projections from the basal ganglia toward the thalamus, which are GABAergic. The glutamatergic neurons within each of the major components of the circuit are under inhibitory control by GABAergic interneurons. In addition, four groups of densely packed neurons provide diffuse projections to all areas of the brain to modulate their functions: cholinergic neurons in the basal forebrain and brain stem, dopaminergic neurons in the substantia nigra and ventral tegmental area, noradrenergic neurons in the locus ceruleus, and serotonergic neurons in the raphe nuclei. The broken arrows in Fig. 76.1 indicate the four neurotransmitter-specific projection systems. The relay of information from one neuron to another across these neurotransmitter-specific projection systems is described next.

## Synaptic Neurotransmission

Dendrites create a network of fibers providing the neuron with input from other cells (Fig. 76.2). The cell integrates these different inputs through modulation of the membrane potential, or at the level of the nucleus (regulation of gene expression). One function of the cell body is the synthesis of all cell-specific receptors and enzymes needed for neurotransmitter production.



**Figure 76.2.** Anatomy of the neuron: the compartments of a typical neuron and the four major mechanisms of synaptic neurotransmission (1–4). See text for details.

The axon is the output station of the neuron. The axon can be short (local circuit neuron) or long (projection neuron). If a deviation from the resting membrane potential is above a certain threshold, an *action potential* is created and travels downstream rapidly. The nerve terminal is the widened terminal part of the axon. It provides a small area of close contact with dendrites of neighboring cells: a synapse. Variations of this typical scheme include synapses between two axons, two dendrites, and neurotransmitter release in medial parts of the axon (varicosities, boutons).

The presynaptic neuron, which releases the neurotransmitter into the synapse, may express two types of proteins that affect synaptic communication (Fig. 76.2): *Membrane-bound receptors* bind the intrinsic neurotransmitter (autoreceptor) or transmitters of neighboring neurons (heteroreceptor) and affect the cell via intracellular messengers. One response, for example, is the modulation of neurotransmitter release (Langer, 1997). *Membrane-bound reuptake transporters* pump the released neurotransmitter back into the cell (Amara, 1995; Lester et al., 1996).

The neuron receiving the input (postsynaptic cell) can be modulated via two different types of receptors (Fig. 76.2): *Fast-acting, class I (ionotropic) receptors*: The neurotransmitter binds to the receptor protein and within milliseconds leads to a change in the permeability of the associated ion channel, allowing the influx of ions such as  $\text{Ca}^{2+}$ ,  $\text{Na}^+$ ,  $\text{K}^+$ , or  $\text{Cl}^-$ . *Slow-acting, class II (G-protein-coupled) receptors*: The neurotransmitter binds to the receptor protein and thereby changes the protein conformation. This change is relayed to an associated G-protein, so-called because it binds guanidine triphosphate (GTP) in order to be activated. G-proteins regulate two major classes of effector molecules: ion channels and second messenger generating enzymes.

## The Major Neurotransmitter Systems

The major neurotransmitter systems can be divided into two groups based on their anatomic distribution. The first group comprises the cholinergic, serotonergic, noradrenergic, and dopaminergic neurons. These four systems originate from small groups of neurons, densely packed in circumscribed areas of the forebrain or



brain stem, and that project to their target areas typically by long-ranging projection fibers. Because these neurotransmitter-specific projection systems reach selected neural systems, their modulation leads to more circumscribed effects. These four systems are presented initially, as they are of greatest relevance to the action of commonly used psychotropic drugs.

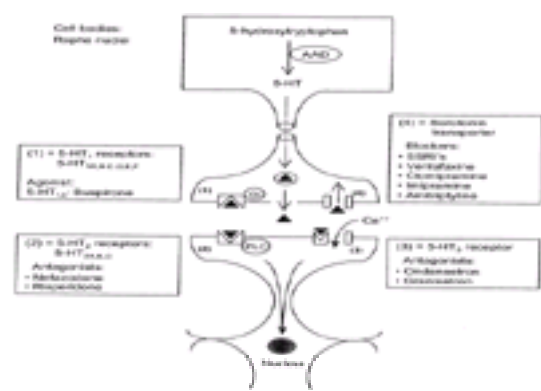
The second group includes the glutamatergic and GABAergic systems. Their neurons are by far the most prevalent and widely distributed types in the human brain. The widespread distribution of these two neurotransmitter systems has functional implications, as the modulation of glutamatergic and GABAergic neurotransmission affects many neural systems. These two systems are also relevant to psychopharmacology: for example, certain psychotropics (e.g., the benzodiazepines) and drugs of abuse (e.g., ethanol) act on GABAergic systems, and novel agents targeting the glutamate pathways are being tested for the treatment of psychotic disorders.

### Serotonergic Neurotransmission

Most serotonergic cells overlap with the distribution of the raphe nuclei in the brainstem. A rostral group (B6–8 neurons) projects to the thalamus, hypothalamus, amygdala, striatum, and cortex. The remaining two groups (B1–5 neurons) project to other brainstem neurons, the cerebellum, and the spinal cord.

Serotonin acts at two different classes of receptors: at an inotropic receptor (5-HT<sub>3</sub> receptor) or at slower acting receptors, coupled either to phospholipase C (5-HT<sub>2</sub> receptors) or to G-proteins (5-HT<sub>1,4-7</sub> receptors) (Julius, 1991).

The 5-HT<sub>1</sub> receptors (5-HT<sub>1A,B,C,D,E,F</sub>) (Fig. 76.3) are coupled to G<sub>i</sub> and lead to a decrease of cyclic AMP. The 5-HT<sub>1A</sub> receptor is also directly coupled to a K<sup>+</sup> channel leading to increased opening of the channel. The 5-HT<sub>1</sub> receptors are the predominant serotonergic autoreceptors. The atypical anxiolytic buspirone acts as an agonist at the 5-HT<sub>1A</sub> receptor.



**Figure 76.3.** Serotonergic neurotransmission.

5-HT<sub>2</sub> receptors (5-HT<sub>2A-C</sub>) are coupled to phospholipase C and lead to a variety of intracellular effects (mainly depolarization). The 5-HT<sub>2</sub> receptor may be important in mediating the therapeutic effects of agents as varied as risperidone and nefazodone. Three receptors (5-HT<sub>4,6&7</sub>) are coupled to G<sub>s</sub> and activate adenylate cyclase.

The 5-HT<sub>3</sub> receptor is the only monoamine receptor coupled to an ion channel, probably a Ca<sup>2+</sup> channel. It is found in the cortex, hippocampus, and in the area postrema, where it mediates nausea and emesis. It is typically localized presynaptically and regulates neurotransmitter release. Newer antiemetics and antimigraine drugs have effects at the 5-HT<sub>3</sub> receptor site (Fig. 76.3).

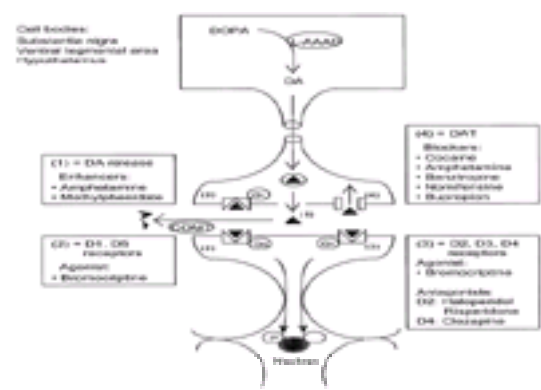
Serotonin is removed from the synapse by a high-affinity serotonin uptake site that is capable of transporting serotonin in either direction, depending on the concentration. The serotonin transporter is the primary target site for several antidepressants, including the selective serotonin reuptake inhibitors (SSRIs), venlafaxine, and tricyclic antidepressants such as clomipramine and, to a lesser extent, imipramine and amitriptyline (Fig. 76.3).

Serotonin is linked to many brain functions because of the widespread serotonergic projections and heterogeneity of the serotonergic receptors (Jacobs and Azmitia, 1992; Lucki, 1998). For example, modulation of serotonergic receptors and the reuptake site is beneficial (among others) in the treatment of anxiety, depression, obsessive-compulsive disorder, and schizophrenia (Murphy et al., 1998).

### Dopaminergic Neurotransmission

Dopaminergic neurons can be divided into three major groups based on the length of their efferent fibers: (a) ultrashort systems in the retina and olfactory bulb; (b) intermediate-length systems originating in the hypothalamus and projecting, among others, to the pituitary gland; and (c) wide-ranging systems originating from two areas, the substantia nigra (SN) and the ventral tegmental area (VTA). The SN neurons (also called A9 neurons) project to caudate and putamen, whereas the VTA neurons (also called A10 neurons) project to limbic areas such as nucleus accumbens and amygdala (i.e., mesolimbic projections) and several cortical areas such as frontal, cingulate, and entorhinal cortex (i.e., mesocortical projections).

Dopamine is released into the synapse from vesicles (Fig. 76.4); this process is facilitated by the stimulants methylphenidate and amphetamine.



**Figure 76.4.** Dopaminergic neurotransmission.

Dopamine acts at two different classes of dopamine receptors in the CNS, the D<sub>1</sub> receptor family and D<sub>2</sub> receptor family (Fig. 76.4) (Baldessarini and Tarazi, 1996). The D<sub>1</sub> receptor family includes the D<sub>1</sub> and D<sub>5</sub> receptors. Both are coupled to G<sub>s</sub> (G-stimulating) and lead to an increase of cyclic AMP. The D<sub>2</sub> receptor family includes the D<sub>2</sub>, D<sub>3</sub>, and D<sub>4</sub> receptors. All are coupled to G<sub>i</sub> (G-inhibitory) and lead to a decrease of cyclic AMP. There is a predilection of the different dopamine receptors for expression in specific brain areas (e.g., D<sub>1</sub> receptors are found in the striatum and cortex, D<sub>2</sub> receptors in the striatum and pituitary gland, and D<sub>3</sub> receptors in the nucleus accumbens). Dopamine receptor blockade is the major target of antipsychotic drugs, particularly at D<sub>2</sub> sites (Fig. 76.4). Presynaptic dopaminergic receptors are typically of the D<sub>2</sub> type and found on most portions of the dopaminergic neuron (as autoreceptors). They regulate DA synthesis and release, as well as the firing rate of DA neurons. Autoreceptors are five to 10 times more sensitive to DA agonists than postsynaptic receptors.

Dopamine is removed from the synapse by two mechanisms: first, catechol-O-methyl-transferase (COMT) degrades intrasynaptic DA. Second, the dopamine transporter (DAT) (Fig. 76.4), a Na<sup>+</sup>/Cl<sup>-</sup> dependent neurotransmitter transporter, transports DA in either direction, depending on the concentration gradient. Stimulant drugs such as amphetamine and cocaine potently block the DAT.

Dopamine affects several brain functions primarily by modulation of other neurotransmitter systems (Missale et al., 1998). Dopaminergic neurons of the SN project to the striatum and modulate the function of striatal GABAergic neurons. Dopaminergic projections of the VTA to limbic structures such as the nucleus accumbens are known to be involved in reward behavior and the development of addiction to drugs such as ethanol, cocaine, nicotine, and opiates (Diana, 1998; Koob, 1998). Dopaminergic projections from the VTA to the cortex play a role in the fine-tuning of cortical neurons (i.e., better signal-to-noise ratio) (Goldman-Rakic, 1998).

#### Noradrenergic Neurotransmission

About half of all noradrenergic neurons (i.e., 12,000 on each side of the brain stem) are located in the locus ceruleus. They provide the extensive noradrenergic innervation of cortex, hippocampus, thalamus, cerebellum, and spinal cord. The remaining neurons are distributed in the tegmental region. They innervate predominantly the hypothalamus, basal forebrain, and spinal cord.

Norepinephrine is released into the synapse from vesicles; amphetamine facilitates this release (Fig. 76.5). Norepinephrine acts in the CNS at two different types of noradrenergic receptors, a and b. Adrenergic a receptors can be subdivided into a1 receptors (coupled to phospholipase and located postsynaptically; prazosin is a typical antagonist) and a2 receptors (coupled to G<sub>i</sub> and located primarily presynaptically; clonidine and guanfacine are potent agonists, and yohimbine an antagonist) (Fig. 76.5) (Insel, 1996). Adrenergic b receptors in the CNS are predominantly of the b1 subtype. b1 Receptors are coupled to G<sub>s</sub> and lead to an increase of cyclic AMP.

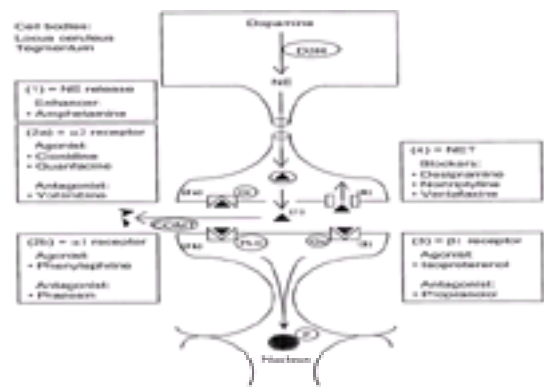


Figure 76.5. Noradrenergic neurotransmission.

Norepinephrine is removed from the synapse by COMT and the norepinephrine transporter (NET), a Na<sup>+</sup>/Cl<sup>-</sup> dependent neurotransmitter transporter. Once internalized, NE can be degraded by the intracellular enzyme monoamine oxidase (MAO).

Noradrenergic projections modulate sleep cycles, appetite, mood, and cognition by targeting the thalamus, limbic structures, and cortex. These functions are targets of antidepressant drugs, which act via blockade of the norepinephrine transporter (Fig. 76.5) (Charney, 1998). Also, the locus ceruleus (LC) receives afferents from the sensory systems that monitor the internal and external environments. The widespread LC efferents lead to an inhibition of spontaneous discharge in the target neurons.

#### Cholinergic Neurotransmission

Cholinergic neurons in the central nervous system are either wide-ranging projection neurons or short-ranging interneurons. Projection neurons in the basal forebrain (septum, diagonal band, nucleus basalis of Meynert) project to the entire cortex, hippocampus, and amygdala, and projection neurons located in the brain stem project predominantly to the thalamus. Cholinergic interneurons in the striatum modulate the activity of GABAergic striatal neurons.

Acetylcholine acts at two different types of cholinergic receptors. Muscarinic receptors bind ACh as well as other agonists (muscarine, pilocarpine, bethanechol) and antagonists (atropine, scopolamine). There are at least five different types of muscarinic receptors (M1 to M5). All have slow response times. They are coupled to G-proteins and a variety of second messenger systems. When activated, the final effect can be to open or close channels for K<sup>+</sup>, Ca<sup>2+</sup>, or Cl<sup>-</sup> (Bonner, 1989).

Nicotinic receptors are less abundant than the muscarinic type in the CNS. They bind ACh as well as agonists such as nicotine (Fig. 76.6) or antagonists such as d-tubocurarine. The fast-acting, inotropic nicotinic receptor allows influx of Na<sup>+</sup> > K<sup>+</sup> > Ca<sup>2+</sup> into the cell.

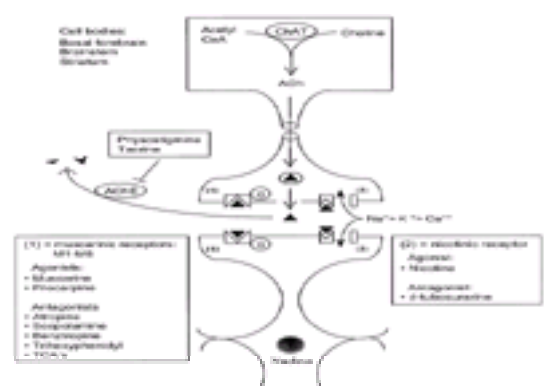


Figure 76.6. Cholinergic neurotransmission.

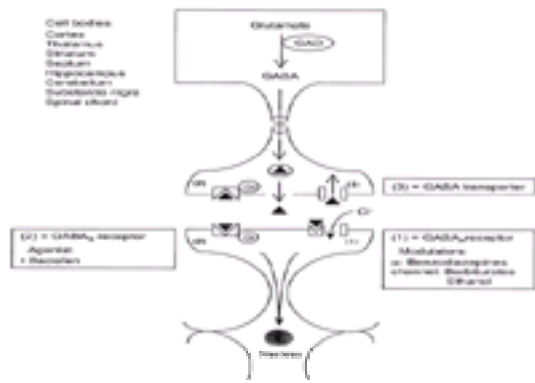
Acetylcholine is removed from the synapse through hydrolysis into acetyl CoA and choline by the enzyme acetyl cholinesterase (AChE).

Acetylcholine modulates attention, novelty seeking, and memory via the basal forebrain projections to the cortex and limbic structures. Anticholinergic delirium and Alzheimer's disease are examples of a cholinergic deficit state (Geula, 1998; Giacobini, 1998). Furthermore, cholinergic interneurons modulate striatal neurons by opposing the effects of dopamine, a function that is exploited therapeutically when using anticholinergic agents such as bethanechol or trihexyphenidyl to treat the extrapyramidal side effects associated with antipsychotics (Fig. 76.6).

#### Gabaergic Neurotransmission

GABAergic neurons can be divided into two groups (Fig. 76.7): (a) Short-ranging neurons (also called interneurons or local circuit neurons) in the cortex, thalamus, striatum, cerebellum, and spinal cord; and (b) medium- and long-ranging neurons in the basal ganglia, septum, and substantia nigra.





**Figure 76.7.** GABAergic neurotransmission.

GABA acts at two types of receptors, the GABA<sub>A</sub> and GABA<sub>B</sub> receptors.

The GABA<sub>A</sub> receptor is a receptor-channel complex comprised of five subunits ([Lüddens and Korpi, 1996](#)). Activation leads to the opening of the channel, allowing Cl<sup>-</sup> to enter the cell, resulting in decreased excitability. Five distinct classes of subunits and multiple variations in the composition of the GABA<sub>A</sub> receptor are known. The receptor can be modulated by benzodiazepines at the α subunit and barbiturates and ethanol near the Cl<sup>-</sup> channel ([Fig. 76.7](#)).

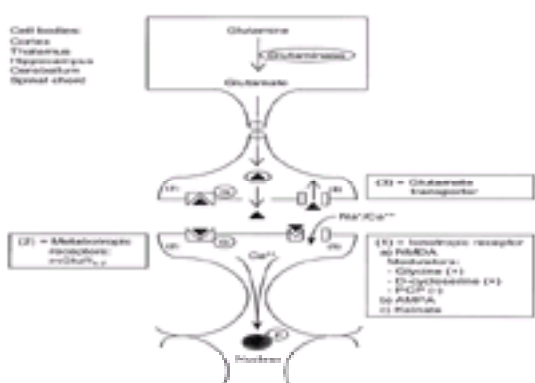
The GABA<sub>B</sub> receptor is a G-protein–coupled receptor with similarity to the metabotropic glutamate receptor ([Bettler et al., 1998](#); [Kaupmann et al., 1997](#)). The GABA<sub>B</sub> receptor is linked to G<sub>i</sub> (decreasing cyclic AMP and opening of K<sup>+</sup> channels) and G<sub>o</sub> (closing Ca<sup>2+</sup> channels). GABA is removed from the synapse by a sodium dependent GABA uptake transporter ([Fig. 76.7](#)).

Cortical and thalamic GABAergic neurons are crucial for the inhibition of excitatory neurons. GABAergic agonists such as benzodiazepines or barbiturates are efficacious in the treatment and prevention of seizures ([Bazil and Pedley, 1998](#)). Furthermore, modulation of GABA<sub>A</sub> receptors is beneficial in the treatment of anxiety disorders, insomnia, and agitation—most likely because of a general inhibition of neuronal activity.

### Glutamatergic Neurotransmission

Glutamatergic neurons are widely distributed throughout the entire brain. Prominent glutamatergic pathways are the cortico-cortical projections, connections between thalamus and cortex, and projections from cortex to striatum (extrapyramidal pathway) and to brain stem/spinal cord (pyramidal pathway) ([Ozawa et al., 1998](#)).

Glutamate acts at three different types of ionotropic receptors ([Fig. 76.8](#)) and at a family of G-protein–coupled (metabotropic) receptors ([Fig. 76.8](#)) ([Nakanishi, 1992](#); [Nakanishi et al., 1998](#); [Vandenberg, 1998](#)).



**Figure 76.8.** Glutamatergic neurotransmission.

Binding of glutamate to the ionotropic receptor opens an ion channel allowing the influx of Na<sup>+</sup> and Ca<sup>2+</sup> into the cell. NMDA receptors bind glutamate and *N*-methyl-d-aspartate. The receptor is comprised of two different subunits: NMDAR1 (seven variants) and NMDAR2 (four variants). The NMDA receptor is highly regulated at several sites. For example, the receptor is virtually ineffective unless a ligand binds to the glycine site and it is blocked by binding of ligands (e.g., ketamine and phencyclidine = PCP) to the PCP site inside the channel. AMPA receptors bind glutamate, AMPA, and quisqualic acid, whereas kainate receptors bind glutamate and kainic acid.

The metabotropic glutamate receptor family includes at least seven different types of G-protein–coupled receptors (mGluR1-7). They are linked to different second messenger systems and lead to the increase of intracellular Ca<sup>2+</sup> or the decrease of cyclic AMP. The increase of intracellular Ca<sup>2+</sup> leads to the phosphorylation of target proteins in the cell.

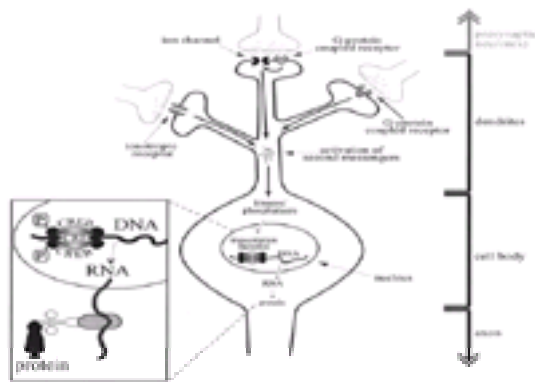
Glutamate is removed from the synapse by high-affinity reuptake; two transporter proteins are expressed in glial cells and one in neurons ([Fig. 76.8](#)).

Glutamate has an effect on many brain functions. For example, glutamatergic neurons and NMDA receptors in the hippocampus are important in the creation of long-term potentiation, a crucial component in the formation of memory ([Wilson and Tonegawa, 1997](#)). Excess stimulation of glutamatergic receptors, as seen in seizures or stroke, can lead to unregulated Ca<sup>2+</sup> influx and neuronal damage ([Coyle and Puttfarcken, 1993](#); [Dingledine et al., 1990](#); [Loscher, 1998](#)). Decreased glutamatergic function is thought to be involved in the creation of psychotic symptoms. Phencyclidine and ketamine can induce psychotic symptoms and *d*-cycloserine or glycine can decrease psychotic and/or negative symptoms in schizophrenia ([Farber et al., 1999](#); [Goff et al., 1999](#); [Heresco-Levy et al., 1999](#)).

### Intracellular Information Processing

Each neuron is the target of many projections from local and distant neurons. These influences are integrated at the level of the cell membrane and cell nucleus. The neurotransmitter-mediated activation of ion channels in the cell membrane can lead to an increase or decrease of the resting membrane potential. This may lead to the creation of an action potential. At the level of the cell nucleus, the various receptors and ion channels expressed on the cell membrane may influence gene and protein expression.

Gene expression is regulated by transcription factors that bind to specific sequences of the DNA in the nucleus ([Fig. 76.9](#)); therefore, membrane-bound receptors or ion channels in distal parts of the neuron must be able to activate intraneuronal signal transduction pathways that can span long distances and translocate to the nucleus. Because proteins assemble the neuron and determine neuronal properties, gene expression regulates neuronal function and may cause malfunction. Many psychopharmacologic agents with delayed therapeutic effects are thought to produce their therapeutic benefits through modulation of gene expression ([Duman et al., 1997](#); [Hyman and Nestler, 1996](#)).



**Figure 76.9.** Intracellular information processing. Neurotransmitters released from the presynaptic neuron activate receptors at the postsynaptic neuron. Second messengers either enter the neuron (e.g.,  $\text{Ca}^{2+}$  through ligand-gated ion channels) or are newly synthesized inside the neuron (e.g., G-protein-coupled receptors stimulate adenylate cyclase and the synthesis of cyclic AMP). Neurons usually have multiple receptor types and are able to integrate information from a variety of synaptic inputs. Second messengers stimulate protein kinases and protein phosphatases to control the state of phosphorylation of various proteins inside the neuron. Transcription factors such as CREB are regulated by kinases and phosphatases. A high level of discrimination is observed, although some transcription factors integrate information from different second messenger pathways. Thus, kinases and phosphatases regulate groups of genes under the control of specific transcription factors. Inset: Phosphorylation of CREB stimulates the transcription from DNA into RNA, which is transported out of the nucleus and translated (with ribosomes and tRNA) into protein.

Release of neurotransmitters from the presynaptic neuron into the synapse activates receptors on the postsynaptic neuron ( Fig. 76.9). Ions such as calcium enter the cell and act as second messengers on activation of ionotropic receptors. Activation of G-protein-coupled receptors facilitates the opening of neighboring ion channels or the synthesis of second messengers such as cyclic AMP. Second messengers (calcium, cyclic AMP) regulate the activity of protein kinases (proteins that transfer phosphate groups to a substrate protein) and phosphatases (proteins that remove phosphate groups from a substrate protein). In all cases investigated to date, the activation of neurotransmitter receptors changes the state of phosphorylation of neuronal proteins.

The transcription factors are one group of proteins regulated by phosphorylation. Transcription factors operate by recruiting the transcription initiation complex and RNA polymerase to particular genes. The RNA polymerase then transcribes the DNA template into an RNA molecule, which is translated into protein outside the nucleus.

Among the best-studied transcription factors in the brain is the  $\text{Ca}^{2+}$ - and cyclic AMP-responsive element binding protein (CREB) ( Montminy et al., 1990). The study of CREB has provided us with an insight into the complex consequences of transcription factor activation and gene expression on higher brain function. A variety of signal transduction pathways are integrated into CREB-mediated gene expression, such as those activated by  $G_s$ -proteins, ionotropic receptors or growth factors. Cyclic AMP-responsive element binding protein is activated by phosphorylation and regulates the expression of several target genes (e.g., genes for peptide neurotransmitters, enzymes involved in neurotransmitter synthesis, and growth factors). The discovery that CREB plays a vital role in processes such as learning and memory provided a link between gene regulation and cognitive function ( Frank and Greenberg, 1994; Stevens, 1994).

Small but persistent abnormalities in neurotransmission can have far-reaching consequences because neurotransmitters and receptors influence gene and protein expression in the brain. An understanding of signal transduction pathways and transcription factors such as CREB is instrumental in providing us with new therapeutic avenues in psychopharmacology.

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## 77 PEDIATRIC PSYCHOPHARMACOLOGY II

### General Principles, Specific Drug Treatments, and Clinical Practice

Lawrence Scahill, M.S.N., Ph.D. and Andrés Martin, M.D.

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The now-famous clinical trial conducted by [Charles Bradley \(1937\)](#) is often cited as the beginning of pediatric psychopharmacology. In that study, the racemic mixture of levoamphetamine and dextroamphetamine (Benzedrine) was administered openly to a group of 30 children with mixed behavioral and emotional symptoms. Bradley and colleagues observed that the children characterized as “noisy, aggressive and domineering” were calmer and more manageable. In the same issue of the *American Journal of Psychiatry*, [Molitch and Eccles \(1937\)](#) reported what may be the first placebo-controlled study in child psychiatry. Ninety-three boys described as juvenile delinquents were randomly assigned to gradually escalating doses of Benzedrine or placebo. The Benzedrine group showed dose-related improvements across a range of measures on learning, motor control, and short-term memory that exceeded the improvements in the placebo group. The field of pediatric psychopharmacology has made steady but slow progress since this pioneering work.

Despite clear progress in some areas, there are important gaps between research and clinical practice. For example: (a) There have been over 100 placebo-controlled studies on the efficacy of methylphenidate in the treatment of attention deficit hyperactivity disorder (ADHD), yet only a few studies have evaluated long-term effects ([MTA, 1999](#)). (b) For serious psychiatric disorders such as autism, empiric support for the use of any medication remains meager, leaving clinicians and families with inadequate guidance on how best to treat affected children. (c) Despite the demonstrated efficacy and safety of selective serotonin reuptake inhibitors (SSRIs) in the treatment of children and adolescents with obsessive-compulsive disorder (OCD), little is known about the appropriate duration of treatment. (d) Based on an evaluation of prescribing trends in three large health care systems, methylphenidate use among children 2 to 4 years of age increased from 1 to 5 per 1,000 in 1991 to 4 to 11 per 1,000 in 1995 ([Zito et al., 2000](#)). This twofold to threefold increase, depending on the health system under consideration, stands in sharp contrast with the lack of clinical research with methylphenidate for this age group. Clearly, the empiric foundations for pediatric psychopharmacology are not yet fully anchored. Over the past decade, however, there have been several initiatives that are likely to have a substantial impact on the pace of progress in the field.

The purpose of this chapter is to present general principles of clinical psychopharmacology in pediatric populations. Following the discussion of seven overarching principles, the chapter reviews the major classes of psychotropic medications and current approaches to their use in children and adolescents. Drugs within the major classes are considered in terms of their pharmacology, side effects, clinical applications and management, and available empiric support. Pharmacokinetic aspects, including drug interactions, as well as mechanisms of action and other pharmacodynamic issues are considered only briefly here, because such topics are dealt with in depth in the preceding and complementary chapter. Throughout this chapter, the reader is referred to [Table 77.1](#), in which the most salient information for each drug class and its specific agents is summarized.

**Table 77.1. Psychotropic Agents Used in Children and Adolescents**

## SEVEN GUIDING PRINCIPLES

### The Role of Development

There is general recognition that development can have a major impact on pharmacologic effects ([Vitiello and Jensen, 1995](#)). Thus, children and adults may show divergent responses to psychotropic drugs. The sources of these differences, as elaborated in the preceding chapter, are manifold. First, children often metabolize and eliminate drugs from the body more quickly than adults, resulting in shorter drug half-lives. This is apparently owing to a larger liver:total body ratio and more efficient glomerular filtration rate in children as compared to adults. One practical implication of these pharmacokinetic differences is that in order to achieve therapeutic serum levels, when compared to adults, children may require higher weight-adjusted dosages (expressed in mg/kg amounts) ([Vitiello et al., 1988](#)). In addition to pharmacokinetic considerations, there can be age effects on drug action as well because the central nervous system undergoes substantial developmental change during childhood.

These developmental changes in neurochemical systems can influence both therapeutic response and side effect profile. For example: (a) Compared to adults, adolescents have a higher risk of dystonic reactions to neuroleptics ([Lewis, 1998](#)). (b) Prepubertal children appear to be at a higher risk for the activating side effects of the selective serotonin reuptake inhibitors (SSRIs) ([King et al., 1991](#)). (c) Developmental differences in the maturation of noradrenergic pathways may explain, at least in part, why tricyclic antidepressants are less effective in children with depression as compared to adults ([Martin et al., 2000a](#)). Taken together, these findings suggest that the three major neurochemical systems that are manipulated by psychotropic drug treatments (dopaminergic, serotonergic, and noradrenergic,



respectively) are subject to age effects.

Federal policy initiatives have been instituted to promote studies specific to pediatric populations in recognition of these developmental influences ( [Riddle et al., 1998](#)). First, Congress passed in 1997 the Food and Drug Modernization Act (FDAMA), which offers pharmaceutical companies an additional 6 months of market exclusivity for products that are evaluated in children. For some drugs, this 6-month extension represents a powerful financial incentive. A second policy initiated by the Food and Drug Administration (FDA) requires pharmaceutical companies to evaluate new products in children if they are likely to be used in this population when released into the marketplace ([FDA, 1999](#)). A third and related important development has been the commitment of federal funds by the National Institute of Health for the establishment of research networks to conduct large-scale, multisite studies in pediatric populations. The combined impact of these initiatives makes it likely that in the coming years much more will be known about the safe and appropriate use of psychotropic drugs in youngsters, and about the effects of development on drug response.

### **The Limits of Categorical Diagnoses and Comorbidity as the Norm**

The second principle acknowledges the limitations of psychiatric diagnoses and of the current nosological system of classification. Most psychiatric disorders in childhood are probably heterogeneous with respect to etiology. This presumption is supported by the high frequency of comorbidity in child psychiatry observed both in clinical ([Biederman et al., 1991](#)) and community samples ([Angold et al., 1999](#)). The extent to which these mixed syndromes represent a variant of a given disorder is often unclear, but may be relevant to drug response. For example, in childhood OCD, tics are a common comorbid feature. Emerging data suggest that the tic-related form of OCD is phenomenologically different from non-tic-related forms of the disorder ( [Leckman et al., 1998](#)). In addition, the presence of tics appears to be associated with a lower probability of positive response to monotherapy with an SSRI ( [McDougle et al., 1994](#); [Scahill et al., 1997](#)). Along the same lines, children with developmental disabilities accompanied by hyperactivity, impulsiveness, and distractibility appear to have a lower rate of positive response than typically developing children with attention deficit hyperactivity disorder (ADHD) ( [Aman et al., 2000](#)). Developmentally disabled children also may have a higher rate of adverse events with stimulant treatment ([Handen et al., 1994](#)). Thus, etiologic heterogeneity may explain differences in clinical response. These issues underscore the importance of large treatment studies to identify clinically meaningful subgroups and predictors of positive and negative response.

### **Target Symptoms and the Integration of Data from Multiple Informants**

This principle concerns the importance of pretreatment assessment and the identification of target symptoms. Given how commonly psychiatric comorbidity is observed in children with mental disorders, the identification of target symptoms for pharmacologic intervention may be easier said than done. One of the practical challenges in child psychiatry is the requirement of gathering information from multiple sources, including the child and parents at a minimum, and the child's teacher or other caregiver as well in many cases. Obtaining data from multiple sources can be aided by the use of behavioral checklists, child self-reports, and clinician ratings. Behavioral checklists completed by parents and teachers permit comparison of the current patient to a normative sample. Clinician-rated instruments can assist in establishing a pretreatment baseline of symptom severity. Some checklists and clinician-rated instruments also can be used to measure change over time. Although checklists and rating instruments can be extremely useful in the evaluation phase and to measure change over time, they cannot replace the clinical interview and direct observation of parent and child. A closely related challenge is the integration of all available information, in order to identify the most pressing target symptoms and select the most appropriate medication in combination with other needed interventions.

### **Adverse Effects: Monitoring Risks and Benefits**

The dramatic increase in the availability of new psychotropic drugs, their expanded use in pediatric populations, and the uncertain impact of psychotropic medications on development underscore the importance of ongoing assessment of adverse effects. At present, there is no clear consensus on the best method of eliciting information about adverse events that occur in the context of pharmacotherapy. This lack of consensus is not limited to child psychiatry; it extends to adult psychiatry as well as other areas of medicine. Three approaches have been described: (a) use of an open-ended general inquiry; (b) the use of general inquiry augmented by a set of drug-specific queries or checklist; or (c) the use of a detailed review of body systems ( [Levine and Schooler, 1986](#)). At the center of this debate are concerns about sensitivity, specificity, and efficiency. The use of a detailed review of systems by an experienced clinician is unlikely to miss adverse effects of medication (i.e., high sensitivity). However, some experts express concern that this method may produce unacceptably high number of false positive responses. If true, this low specificity would come at a great cost in clinician time.

On the other hand, the open-ended general inquiry gives the parent and child an opportunity to express any concern that may have emerged since starting the medication or the last visit. Based on the assumption that parents and children notice important changes in behavior and/or health status, responses to the open-ended inquiry are likely to be clinically meaningful—even if they are not drug-related. The high specificity may result in missed adverse events (i.e., low sensitivity) because parents and children may not detect subtle adverse effects.

Until more is learned about the best method of eliciting data on adverse effects, appropriate clinical practice entails the combined use of an open-ended inquiry followed by a brief set of drug-specific side effects and safety concerns. In the pages that follow, we list the side effects associated with each of the medications presented. Additional safety concerns are also presented in [Table 77.1](#). Other fundamental issues to be covered in contemporary medication management of children include questions about concomitant medications, intercurrent illness, and other medical contacts since the previous visit. The need for vital sign and height and weight monitoring for neurologic examination, electrocardiogram, laboratory tests and drug levels, are medication-specific and described in appropriate sections that follow.

### **The Role of Caregivers and the Meaning of Medication**

The collaboration of the child's family is essential, and active engagement of the family is critical to the success of treatment interventions, pharmacologic or otherwise. A detailed discussion with the parents and child concerning the recommended medication, as well as an examination of the alternative treatments are necessary parts of initiating psychopharmacologic treatment. In addition, the family and child (to the extent possible) also should be partners in the final selection of medication. Once chosen, the dose schedule, side effects, anticipated results on target symptoms and the time to effect warrant explicit review. This discussion gives an opportunity to evaluate and, if needed, temper unrealistic expectations about the medication. Depending on the medication, it may be necessary to establish a contingency plan to manage specific side effects prior to their occurrence, such as being prepared to start benzotropine for a child who develops dystonia when started on a potent antipsychotic such as haloperidol. Children, and to a lesser extent adolescents, are dependent on their parents to administer medications, so that parental endorsement of the treatment plan is both ethically sound and practical. Failure to involve parents in the decision-making process may threaten treatment compliance, which in turn may undermine the success of the intervention ( [Lewis, 1995](#)). Attention also should be paid to the meaning that taking medication has for the child and family. Exploration of these issues may reveal a sense of failure on the part of the child or family. Some children may express concern that having to take medication implies that they are “crazy” or “weird.” These issues should be identified and addressed prior to initiating treatment. Even when handled prior to treatment, however, these issues often reemerge and require attention over the course of treatment.

### **Psychopharmacology in Context and the Combination of Therapeutic Modalities**

Psychotropic medications, no matter how effective, are often but one element in a multimodal treatment plan that includes other individually tailored interventions. This overarching principle, which may seem self-evident to clinicians accustomed to the concurrent use of various approaches in their practice, has become a focal point for research and practice guidelines. For example, a recent large-scale study required the failure of a brief psychotherapeutic intervention before proceeding to randomization to medication or placebo ( [RUPP, 2001](#)). The recently published practice parameters for pediatric OCD ( [King et al., 1998](#)) address questions of timing and sequence of interventions, such as when to use one modality (e.g., behavioral therapy) versus a combined medication approach. Finally, the ongoing NIMH-sponsored treatment of adolescents with depression study (TADS) is an example of formal testing of the separate and combined effects of medication and psychotherapeutic interventions.

### **Empirically Informed Evidenced-Based Clinical Decision Making**

A final principle is that treatment plans should be grounded on available empiric evidence. As the database of pediatric psychopharmacology expands, the value of this principle will become even more pressing and place a higher demand for clinicians to stay abreast of research findings. The International Algorithm Project has put forth a simple method for ranking available treatments according to their level of empiric support ( [Jobson and Potter, 1995](#)): *Class A* includes medications with good empiric support, based on consistently positive results in randomized controlled trials (RCTs). *Class B* consists of drugs with fair empiric support showing positive, but inconsistent, results in RCTs or positive results from small sample trials. *Class C* includes drugs with minimal empiric support, based primarily on accumulated clinical

experience from case reports and open-label studies. As examples relevant to pediatric psychopharmacology, *A level* of empiric support exists in the case of: stimulants for ADHD; fluvoxamine for OCD and other anxiety disorders of childhood; sertraline for OCD; fluoxetine for depression; and haloperidol and pimozide for tic disorders. *B level* of support exists for: sertraline for depression; clonidine for tics; guanfacine for ADHD; and fluoxetine for OCD. This ranking system clearly reflects the state of the science at a particular point in time. Given the rapid pace of development in pediatric psychopharmacology, clinicians have a responsibility to remain up to date with the empiric evidence to ensure the best possible match between target symptoms and treatment options.

## SPECIFIC DRUG TREATMENTS

### Stimulants

#### CLINICAL APPLICATIONS AND EMPIRIC SUPPORT

The stimulants, especially the short- and long-acting forms of methylphenidate and amphetamine, are first-line treatments for attention deficit hyperactivity disorder. In its classic form, ADHD is characterized by inattention, impulsiveness, and hyperactivity, although current convention includes primarily inattentive and impulsive/hyperactive subtypes (APA, 1994). Epidemiologic studies indicate that ADHD is relatively common in childhood, affecting 2% to 10% of school-aged children (Costello et al., 1996; Wolraich et al., 1998). Boys are affected more often than girls. It persists into adolescence in a majority of cases (Biederman et al., 1996), and into adulthood in as many as 30% to 40% of cases (Faraone et al., 2000). Studies of clinical populations indicate that the symptoms of ADHD are among the most common reasons for referral of children to mental health agencies (MTA, 1999). Nonetheless, there also are a substantial number of affected children who are not receiving treatment (Scahill and Schwab-Stone, 2000). Given the high prevalence of ADHD in school-age children, the potential for long-term functional disability and the high health-related costs associated with the disorder (Leibson et al., 2001), ADHD is a major public health concern.

The most commonly used stimulants for the treatment of ADHD include methylphenidate, dextroamphetamine and the mixed preparation of d,l-amphetamine (Pliszka, 1998; Spencer et al., 2000). Immediate-release methylphenidate has been studied more carefully than the other stimulants and remains the most commonly used agent in clinical practice (Safer et al., 1996). Although less well studied, the amphetamine products and extended-release formulations of methylphenidate have short-term efficacy and safety profiles that are comparable to methylphenidate (Elia et al., 1991; Pelham et al., 1990).

The empiric basis for the use of stimulants in children with ADHD rests on findings from hundreds of short-term, randomized, placebo-controlled studies conducted over the past 30 years (Spencer et al., 2000). Results from controlled studies over the last decade provide additional information about dose response, similarities, and differences in response across stimulant preparations, and the importance of regular clinical monitoring to achieve optimal response. Rapport and colleagues (Rapport and Denney, 1997) conducted a dose-response study in 76 subjects (66 boys and 10 girls) between the ages of 6 and 11 years. The 5-week study used four dose levels of methylphenidate (5, 10, 15, and 20 mg given twice daily (BID)) and placebo given in random order in a crossover design. All dose levels of active medication were superior to placebo. In addition, there was a clear linear trend, such that classroom behavior and the number of completed assignments improved with each increase in dose; however, the incremental improvement was smaller as the dose moved above the 10-mg dose level.

In order to compare methylphenidate and *d*-amphetamine, a placebo-controlled, crossover study was conducted in 48 boys between 6 and 12 years of age (Elia, 1991). Subjects were treated with methylphenidate, *d*-amphetamine or placebo in random order for 3 weeks in each treatment condition. During each condition the dose was increased on a weekly basis (e.g., methylphenidate doses for children under 30 kg of body weight were 12.5, 20, and 35 mg at breakfast and lunchtime for the respective weeks). Based on a global rating of response, 79% of the subjects showed a positive response to methylphenidate and 88% showed a positive response to *d*-amphetamine. Only two of the 48 subjects failed to respond to one or the other stimulant.

Pelham and colleagues (Pelham et al., 1990) evaluated the extended release products methylphenidate SR, *c*-amphetamine spansule, and pemoline in a crossover study that also included immediate release methylphenidate (10 mg morning and noon) and placebo. The sample consisted of 22 boys between the age of 8 and 13 years. The dose of active drug was fixed at 10 mg methylphenidate twice a day (approximately 0.3 mg/kg per dose) for the immediate release product, with equivalent doses of the extended release preparations. For example, 20 mg of the sustained release methylphenidate, 10 mg of *d*-amphetamine spansule or 56.25 mg of pemoline were given in the morning only. To maintain the blind across treatments, placebo was given at noon during the extended release treatment periods. Each treatment condition lasted 3 to 6 days. All forms of stimulant were superior to placebo on several outcomes, with only minor differences across preparations.

To date, only methylphenidate has been evaluated in long-term studies (MTA, 1999). With its sample size of 576 children, the Multimodal Treatment Study (MTA) of children with attention deficit hyperactivity disorder provides convincing evidence for the long-term benefits of methylphenidate. In the MTA study, children between 7 and 10 years of age were randomly assigned to one of four treatment groups: medication management ( $N = 144$ ; primarily methylphenidate administered in a systematic fashion with close monitoring); an intensive behavioral treatment program ( $N = 144$ ); combined medication management and the same behavioral treatment program ( $N = 145$ ); or community care, which served as the control group ( $N = 146$ ). The MTA research sites provided treatment to three of these groups, including the medication management group, the behavioral therapy only group and the combined medication plus behavioral therapy group. The community care group received treatment from self-selected practitioners. In most cases (84 of 146, or 58%), community care consisted of methylphenidate given on a twice-daily schedule. After 14 months of treatment, all four groups showed improvement compared to baseline. Comparisons across the four groups showed that the combined treatment group and the medication management group did significantly better than the community care group across a range of outcomes.

Several potentially important differences emerged when community care was compared to medication management provided by the MTA sites. First, community practitioners most often administered methylphenidate on a twice-daily schedule, compared to three times per day in the MTA-treated groups. Among the children randomly assigned to community care, 33% ( $N = 48$ ) stopped taking medication during the study period. Not surprisingly, this group showed the least improvement on the primary outcome measures. By contrast, only 3% ( $N = 18$ ) of the research medication management groups (both medication only and medication plus behavioral treatment) discontinued medication during the study. Finally, in the MTA medication management groups, follow-up visits were more frequent and parent and teacher ratings were used in a systematic way to inform clinical decision-making.

Taken together, the results of these studies suggest that stimulants are effective for short- and long-term treatment of children with ADHD. When considering group effects, stimulants appear to be equally beneficial, but individual patients may respond better to one preparation over another. The limited effectiveness of stimulants observed in the community care group of the MTA suggests that close clinical monitoring with dose adjustments based on systematic assessment of therapeutic and side effects contributes to compliance and optimal results.

#### MECHANISM OF ACTION

Although stimulants have become the standard treatment for ADHD, their mechanism of action is not clearly understood. In addition, the mechanism of action for amphetamines and methylphenidate may be slightly different (Solanto, 1998). Methylphenidate promotes release of stored dopamine and blocks the return of dopamine at presynaptic dopamine transporter sites. Amphetamines also block dopamine reuptake at the transporter, but appear to promote the release of newly synthesized dopamine more selectively. These combined effects enhance dopamine function in striatum and, at least indirectly, in the prefrontal cortex. It is also clear that both methylphenidate and the amphetamines affect the norepinephrine (NE) system (Biederman and Spencer, 1999). For example, both compounds decrease the firing rate in the locus ceruleus (LC), although amphetamine appears to be more potent in this action. Whether the effect on the NE system is facilitative or inhibitory is not clear at present. Nonetheless, these combined effects appear to be essential to the clinical effects of the stimulants, as drugs with more selective action (e.g., guanfacine or desipramine) tend to have smaller clinical effects.

#### PHARMACOKINETICS

Immediate release formulations of methylphenidate, dextroamphetamine, and the combined levoamphetamine and dextroamphetamine preparation are readily absorbed and show behavioral effects 30 to 60 minutes after ingestion. The peak level of immediate release methylphenidate occurs approximately 90 to 150 minutes after ingestion and the clinical effects last 3 to 5 hours. The immediate release amphetamine products achieve peak levels between 1 and 3 hours, with duration of action of 5 to 7 hours. Based on blinded studies in a research classroom setting, the d,l-amphetamine preparation appears to have a slightly longer duration of action than standard *d*-amphetamine (Swanson et al., 1998). Methylphenidate and amphetamine are broken down in the liver, and both the parent compound and its metabolites are excreted in the urine within 24 hours. Several newly developed sustained release products have been introduced, offering a range of options for clinical management of children and adolescents with ADHD. Published data on these new sustained release products are not yet available. Because some children may respond better to preparations with faster or slower onset of action, the rate of absorption may influence the choice of a particular agent. Individual



manufacturer's materials should be reviewed before prescribing these agents.

### SPECIFIC STIMULANTS AND CLINICAL MANAGEMENT

There has been considerable debate over whether stimulant dose should be weight-based or a fixed-dose ( [Greenhill et al., 1996](#); [Pliszka, 1998](#)). At least in part, this controversy can be traced to early research suggesting that lower doses, such as 0.3 mg/kg per dose, were optimal for enhancing cognitive performance, whereas higher doses (0.6 mg/kg per dose or higher) were more effective for behavioral control ( [Sprague and Sleator, 1977](#)). Subsequent studies have not supported this view. For example, a convincing linear dose-response was demonstrated across a range of outcomes in a placebo-controlled, crossover study involving 76 children and four dose levels of methylphenidate ( [Rapport and Denney, 1997](#)). Nonetheless, individual children may indeed show variability in response across a range of dose levels. Other children may show an orderly dose-response up to a threshold, above which there is little additive benefit. Thus, the mg/kg calculation can be used as a crude guide to calculate the starting dose of 0.3 mg/kg per dose and to a usual ceiling dose (e.g., 0.8 mg/kg per dose). Thereafter, the dose can be increased to establish an optimal response. For example, school-age children can be started on 5 mg thrice daily (TID) (just before breakfast, just before lunch, and 3 to 4 PM). The dosage may be increased to 10 mg BID (morning and noon) and 5 mg after school after 5 to 7 days. Subsequent increases are based on clinical response and emergence of side effects. The third dose typically remains half (or even less) of the first and second doses so as to minimize *rebound* effects and possible interference with sleep ( [Greenhill et al., 1996](#)).

To determine the optimal daily dose of methylphenidate, it is essential to get feedback from both parents and teachers. In the MTA study, children were seen weekly when starting the medication to monitor progress and side effects. The study used daily ratings to assist with the assessment of response. Although this is not feasible in clinical practice, clinicians may elect to pace dose increases with the collection of parent and teacher ratings. For example, in the first month of treatment, clinicians may increase the dose on a weekly basis. Collection of parent and teacher ratings prior to each increase would allow comparisons across dose levels. This information could be integrated with side effect data in order to select the optimal dose.

A similar approach can be used with *d*-amphetamine and *d,l*-amphetamine. These two drugs are dosed similarly to one another, but because of their greater (twofold) potency, they are used at half the methylphenidate dose. Moreover, owing to their slightly longer duration of action, the amphetamines are typically given twice a day—morning and noon. The initial dosage may be a single 2.5-mg dose in younger children or a 5-mg dose in older children. After 5 to 7 days, the medication may be raised 5 mg BID in younger children and 10 mg BID in older children. Thereafter, the dosage may be raised every 5 to 7 days to a total of 15 to 20 mg per day in younger children, and 40 mg per day in older children.

Until recently, methylphenidate sustained release, *α*-amphetamine spansules and pemoline were the only available long-acting formulations. Several new extended release products—two formulated with methylphenidate and one with *d,l*-amphetamine—have entered the market place. These longer-acting preparations are generally considered as effective as the immediate release compounds, and published results with them are forthcoming. The advantage of the long-acting preparations is that children do not need to take a dose in school, which may enhance compliance. The longer duration formulations may minimize the behavioral rebound often seen with immediate release formulations. The use of pemoline is declining since the introduction of these new extended release compounds, and given concerns about its potential hepatotoxicity.

### SIDE EFFECTS

Growth retardation, presumed to be secondary to stimulant-induced appetite suppression, has been a common concern among clinicians and families alike. Based on data from a large cohort of clinic cases treated with stimulants, Spencer and colleagues contend that slowed growth may be temporary, and that children with ADHD may be shorter than their age mates before puberty, but “catch up” in adolescence ( [Spencer et al., 1998a](#)). Appetite suppression often can be managed by giving stimulant medications with food or immediately after meals. Height and weight should be monitored regularly in children treated with stimulants, and tracked during long-term maintenance on population-normed weight- and height-for-age charts.

Other common side effects include sleep disturbance, depressed mood, stomachaches, over-focusing on details, tics and mannerisms, and picking at skin. Insomnia can be difficult to sort out, because many children with ADHD have sleep difficulties prior to receiving stimulant medications. Rebound effects associated with stimulant withdrawal may compound preexisting sleep problems. Thus, the child's sleep history should be documented prior to treatment and monitored throughout. As noted, it is common practice for the third dose of methylphenidate to be lower than the first two in order to minimize a possible rebound effect. The use of clonidine as an aid for sleep has been proposed, yet remains a controversial practice ( [Wilens et al., 1999](#)).

Results from case reports and controlled studies suggest that exposure to stimulants can be associated with the emergence of tics ( [Borcherding et al., 1990](#)) or the worsening of preexisting tics ( [Law and Schachar, 1999](#)). However, several recent studies have also shown that tics do *not* invariably worsen when children with ADHD and comorbid tic disorders are treated with stimulants ( [Castellanos et al., 1997](#); [Gadow et al., 1995](#)). Nonetheless, children with tic disorders should be monitored carefully when treated with stimulants. Dose reduction may be sufficient, but discontinuation may be warranted in some cases ( [Law and Schachar, 1999](#)).

### Antipsychotics

#### CLINICAL APPLICATIONS AND EMPIRIC SUPPORT

Antipsychotics were introduced to adult psychiatry in the early 1950s and were used in children shortly thereafter. The antipsychotics can be classified according to chemical family, such as phenothiazines or butyrophenones. Alternatively, they may be classified according to the relative potency of their dopamine blockade. Chlorpromazine and thioridazine are low-potency drugs in that relatively high doses are required to achieve usual therapeutic effects. By contrast, haloperidol and fluphenazine are high-potency dopamine blocking drugs. It is becoming commonplace to classify antipsychotics as typical or atypical with the introduction of clozapine and a short list of newer compounds. Pediatric uses of antipsychotics include the treatment of psychosis, severe behavioral problems associated with autism and other developmental disorders, aggression, tics, and as an adjunctive treatment in bipolar illness and OCD.

#### TYPICAL ANTIPSYCHOTICS

##### *Chlorpromazine*

Chlorpromazine (Thorazine), an aliphatic agent, was the first antipsychotic used in children with severe behavioral disturbances. With the introduction of newer agents, the use of chlorpromazine has declined, although it is still routinely used for the acute management of agitation or aggression, when it can be administered through either oral or intramuscular (IM) routes. When used IM, careful caution must be paid to vital signs, as significant hypotension can occur even at seemingly low doses (e.g., <25 mg).

##### *Thioridazine*

Thioridazine (Mellaril), another low-potency phenothiazine in the piperidine chemical family, was among the most commonly used antipsychotics in pediatric populations until recently. The recommended dosage for treating psychosis or severe behavioral dyscontrol in 3- to 12-year-old children is 0.5 to 3.0 mg/kg per day, given in two or three divided doses ( [McClellan and Werry, 1994](#)). In addition to the introduction of the atypical neuroleptics, recent case reports indicating that thioridazine can prolong cardiac conduction times, particularly the QTc interval, have led to decline in its use, and to an advisory “black box” label from the FDA, advising to use the agent only in special clinical situations.

##### *Haloperidol*

Haloperidol (Haldol) is a butyrophenone that is structurally unrelated to the phenothiazines. It represents the prototype of a high-potency typical antipsychotic. It has been used to treat children with psychosis, aggressive behavior, tics, and behavioral dyscontrol associated with autism since its introduction in the early 1960s ( [Anderson et al., 1989](#); [Shapiro et al., 1989](#)).

Compared to the low-potency antipsychotics, haloperidol is much more likely to cause extrapyramidal symptoms (EPS), but is less sedating. The dose of haloperidol varies according to the target symptoms. For example, in school-age children with tics or severe behavioral dyscontrol associated with autism, the dose is typically in the range of 0.75 to 2.5 mg per day ( [McDougle et al., 2000](#); [Scahill et al., 2000](#)). By contrast, doses in the range of 10 may be used to deal with an acute psychotic

episode ([Spencer et al., 1992](#)).

### *Fluphenazine*

Fluphenazine (Prolixin) is a piperazine phenothiazine. It is not approved for children under the age of 12 years, and has not been well studied in pediatric populations. At low dosages (0.04 mg/kg per day) fluphenazine decreased aggression, hyperactivity, and stereotypies in an open-label study of 12 children with pervasive developmental disorder (PDD) ([Joshi et al., 1988](#)). At a mean dose of 7 mg per day in divided doses (range, 2 to 15 mg per day) fluphenazine reduced tics and was favored over haloperidol by most patients ([Goetz et al., 1984](#)). This open-label trial of 21 patients with Tourette's syndrome (TS) included both children and adults, but results were not separately reported by age group.

### *Thiothixene*

Thiothixene (Navane) is a thioxanthene derivative. It is structurally unrelated to the phenothiazines or the butyrophenones. It is approved for the treatment of psychosis in children over the age of 12 years. The available evidence in children under age 12 indicates that thiothixene is less sedating than are low-potency neuroleptics, and that it may have a lower risk of extrapyramidal symptoms (EPS) than high-potency neuroleptics such as haloperidol ([Realmuto et al., 1984](#)).

### *Pimozide*

Pimozide (Orap) is a diphenylbutylpiperidine. It is not related to the phenothiazines or haloperidol. It is a potent blocker of dopamine at the D<sub>2</sub> postsynaptic receptors, and it is used to treat tics in TS. In placebo-controlled studies involving both children and adults, pimozide has been shown to be superior to placebo in reducing tics ([Shapiro and Shapiro, 1984](#)). It also has evaluated in head-to-head trials with haloperidol. These studies suggest that pimozide is equivalent to haloperidol with respect to tic suppression, and that it has a more favorable side-effect profile ([Sallee et al., 1994](#); [Shapiro et al., 1989](#)).

## ATYPICAL ANTIPSYCHOTICS

### *Clozapine*

Clozapine (Clozaril) is a dibenzodiazepine derivative. It is chemically unrelated to any of the typical antipsychotic drugs. Soon after it was introduced in 1960, its effectiveness in treatment-resistant patients with schizophrenia was recognized, but initial enthusiasm waned following the report of fatal agranulocytosis in a series of cases in Europe. Two open studies ([Frazier et al., 1994](#); [Siefen and Remschmidt, 1986](#)) and one controlled comparison with haloperidol ([Kumra et al., 1996](#)) have been carried out in pediatric populations. Collectively, these studies included 53 patients from ages 6 to 18 years. Clozapine was effective in 30 of 53 (56%) of these patients. This figure is impressive considering that this was a group of treatment-resistant patients. In the controlled trial, doses ranged from 125 to 525 mg/day given in divided doses. Although there were no reports of EPS, several serious side effects were observed, most notably seizures ( $N = 1$ ) and hematopoietic abnormalities ( $N = 4$ ). Weight gain is a frequently observed complication of clozapine treatment. Owing to differences in the duration of the studies, however, it is not possible to aggregate data across them. A retrospective comparison of clozapine ( $N = 20$ ), olanzapine ( $N = 13$ ), and risperidone ( $N = 38$ ) in adults indicated that clozapine and olanzapine confer a higher risk for weight gain than risperidone. Using a 10% increase in body weight as a threshold, 40% in the clozapine group, and 30% of patients treated with olanzapine had excessive weight gain. These rates were significantly greater than the 10% of patients in the risperidone group. In addition, the absolute increase in weight was significantly greater for the clozapine group compared to the risperidone group. The olanzapine and risperidone groups were not different in mean weight increase ([Wirshing et al., 1999](#)).

### *Risperidone*

Risperidone (Risperdal) was the second atypical antipsychotic to be released to the US marketplace. It is a benzisoxazol derivative that has some pharmacologic features in common with clozapine. It has not been associated with agranulocytosis. Large multisite studies in adults with schizophrenia have shown that risperidone is an effective antipsychotic with low EPS liability ([Marder and Meibach, 1994](#)). Unlike clozapine, it is associated with an increase in prolactin, suggesting more potent D<sub>2</sub> blockade. The risk of EPS increases in a dose-dependent manner at doses above 6 mg per day. This apparent threshold effect is presumed to be owing to a saturation of 5HT<sub>2</sub> receptors, followed by an increase in D<sub>2</sub> binding ([Kapur et al., 1995](#)).

To date, most studies with risperidone in pediatric populations have been limited to open-label studies in small samples. Findings from these studies suggest that risperidone may be useful for the treatment of children and adolescents with thought disorder ([Armenteros et al., 1997](#)), behavioral dyscontrol in PDD ([Fisman and Steele, 1996](#); [McDougle et al., 1997](#)), and tic disorders ([Lombroso et al., 1995](#)).

A recent placebo-controlled study of 20 subjects with conduct disorder, age 6 to 14 years of age, showed that risperidone reduced aggression by 42%, compared to 4% for placebo ( $P < 0.05$ ) ([Findling et al., 2000a](#)). In a placebo-controlled study of risperidone that included both children and adults, risperidone was superior to placebo for the treatment of tics in TS ([Scahill, 2001](#)). Thirty-four subjects (26 children and eight adults) were randomly assigned to placebo or risperidone. After 8 weeks of treatment with doses ranging from 1 to 2 mg per day in the pediatric sample, risperidone was associated with a 36% improvement on a clinician-rated measure of tic severity, which was significantly better than the 9% in the placebo group ( $P < 0.01$ ). Extrapyramidal symptoms were not observed in this study. Weight gain averaged 3.1 kg in the active treatment group compared to no change in the placebo group ( $P < 0.05$ ). Treatment emergent social phobia was observed in two cases in the risperidone group, which has been reported by others ([Hanna et al., 1999](#)).

Risperidone has also been compared to pimozide in a randomized, double-blind trial ([Bruggeman et al., 2001](#)). In that study, risperidone and pimozide were similar in their positive effects, with reductions in tic severity of 44% and 47%, respectively. Not surprisingly, the pimozide group reported more EPS. Weight gain was greater in the risperidone group (4.5 compared to 2.7 kg). Dose ranges were similar; up to 6 mg per day for each drug. The study included 50 subjects in total, but only 17 were in the pediatric age group. Results in the pediatric sample were not reported.

Several other controlled studies of risperidone are under way and published reports will be available in the near future. For example, the RUPP Autism Network recently completed a placebo-controlled study of risperidone in 101 children with autism accompanied by aggression, self-injurious behavior, and/or irritable and explosive behavior (the study's design and assessment methods are described in [Arnold et al., 2000](#); Scahill et al., in press). In most of these studies, the drug was initiated at 0.5 mg per day and increased by 0.5 mg every 5 to 7 days to a range of 1 to 3.5 mg per day in two divided doses. Findling and colleagues (2000a) started smaller children (i.e., less than 50 kg) at 0.25 mg per day and 0.5 mg for larger subjects. The dose ranged from approximately 1.0 to 2.5 mg per day given in a single morning dose.

### *Quetiapine*

Although not chemically related to clozapine, quetiapine (Seroquel) resembles clozapine in its receptor occupancy profile. It has low activity at D<sub>2</sub> receptors. Because it is a relatively low-potency drug at the 5HT<sub>2</sub> sites as well, it does not show the threshold effect observed with risperidone. Thus, even at high doses, quetiapine has low EPS liability and shows only transient rises in prolactin. As with the other available atypical neuroleptics, quetiapine has demonstrated safety and efficacy for the treatment of adults with schizophrenia. Only limited data are available on its use in children and adolescents. Preliminary results suggest that quetiapine may be useful for the treatment of psychotic symptoms in adolescents ([McConville et al., 2000](#)). Less promising results were reported in an open-label study of six adolescents with pervasive developmental disorder ([Martin et al., 1999](#)). In that study, only two of six subjects showed a positive response on global measures of behavior.

### *Olanzapine*

Olanzapine (Zyprexa) is another agent with atypical features that are preserved only in the lower dose range (<20 mg/day). The use of olanzapine has been described in open-label series of children and adolescents with treatment-resistant schizophrenia ([Kumra et al., 1998](#)), and more recently as a single ([Frazier et al., 2001](#)) or adjunctive ([Chang and Ketter, 2000](#)) agent for the treatment of juvenile bipolar disorder. Together with clozapine, olanzapine has shown the highest propensity for morbid weight gain in adults ([Allison et al., 1999](#); [Wirshing et al., 1999](#)). The high risk of weight gain associated with these agents is a pressing concern for children and adolescents and warrants more study.



## Ziprasidone

Ziprasidone (Geodon), the most recently released atypical antipsychotic medication, also has been shown to be effective in adults with schizophrenia ( [Bagnall et al., 2000](#)). Like risperidone, it is a potent blocker at the D<sub>2</sub> and 5-HT<sub>2</sub> sites. Compared to risperidone, however, much larger doses are required to achieve an antipsychotic effect. For example, the usual dose of risperidone in adults with schizophrenia is 6 mg per day. By contrast, the dose of ziprasidone is typically in the range of 80 to 160 mg per day in two divided doses ( [Tandon, 2000](#)). There is one published study of ziprasidone in a pediatric population. In that trial, 28 children (age 7 to 17 years) with TS or chronic motor tic disorder were randomly assigned to placebo ( N = 12) or ziprasidone ( N = 16) under double-blind conditions. The mean dose of ziprasidone was 30 mg per day in divided doses. Using the same outcome measure mentioned for the risperidone study in TS, ziprasidone was associated with a 35% improvement in tic severity, compared to a 7% decline in the placebo group ( *P* < 0.05) ( [Sallee et al., 2000](#)). Common side effects included moderate sedation (one subject), mild sedation (11 subjects), insomnia (four subjects), and akathisia (one subject). There were no changes in laboratory values, cardiac conduction times, or body weight in the ziprasidone group. Its apparent limited effect on weight gain makes of ziprasidone a welcome addition to the antipsychotic armamentarium, although larger studies are needed to confirm this initial impression.

### MECHANISM OF ACTION

The principal therapeutic action of the typical antipsychotics is to block postsynaptic D<sub>2</sub> receptors ( [Findling et al., 1998](#)). The observed differences across traditional antipsychotics are presumably related to the regional specificity of the D<sub>2</sub> blockade (striatum versus limbic structures), and the effects on other neurotransmitter systems. By contrast, the atypical antipsychotics block both dopamine and serotonin postsynaptic receptors to varying degrees. The addition of postsynaptic 5-HT blockade appears to lower the risk of EPS and perhaps of tardive dyskinesia (TD) as well. It may also contribute to beneficial effects on negative symptoms of schizophrenia, although this remains a matter of controversy. Some investigators argue that the beneficial effects of the atypicals on negative symptoms have yet to be demonstrated in a convincing manner, whereas others concede that these agents can improve negative symptoms, but contend that 5HT<sub>2</sub> blockade may not be the underlying mechanism involved.

Antipsychotic medications also have anticholinergic and antihistaminergic effects, and block adrenergic pathways as well. These additional pharmacologic properties probably do not contribute to the therapeutic effects of the antipsychotic drugs, but do have an impact on their side-effect profiles. For example, low-potency neuroleptics such as chlorpromazine and thioridazine cause more sedation, dry mouth, and constipation, presumably owing to antihistamine and anticholinergic effects. Their propensity to cause hypotension may be related to  $\alpha$ -adrenergic blockade.

### SIDE EFFECTS

Extrapyramidal symptoms (e.g., dystonic reactions, rigidity, and akathisia) are more common with the high-potency neuroleptics. In addition to EPS, side effects of the typical antipsychotic drugs in children and adolescents include cognitive blunting, school phobia, weight gain, and depressed mood. Drowsiness is a common side effect with the low-potency antipsychotic agents, but may occur in the high-potency antipsychotics as well. Anticholinergic side effects such as dry mouth, constipation, and blurred vision also should be monitored. As noted, thioridazine, pimozide, and other antipsychotics can prolong cardiac conduction times to varying degrees. Indeed, recent guidelines suggest that electrocardiograms be obtained at baseline and during maintenance treatment with antipsychotics ( [Gutgesell et al., 1999](#)).

Although the atypical antipsychotics are less likely to cause EPS, they are not free of adverse effects. Clozapine is associated with a low risk of agranulocytosis. For this reason, it is only used in treatment-resistant schizophrenia. Other side effects of clozapine include lowered seizure threshold and tachycardia. An adverse effect that has been recurrently reported for clozapine, olanzapine, quetiapine, and risperidone is weight gain. Although the number of subjects studied remains relatively small, all available studies in pediatric samples with these agents have reported weight gain. In the two placebo-controlled studies with risperidone, weight gain was significantly greater in the active treatment group as compared to placebo, posting 4.3 kg in one study ( [Findling et al., 2000a](#)) and 3.1 kg in the other ( [Scahill, 2001](#)). When followed for longer periods of time, the weight gain can be substantial ( [Kelly et al., 1998](#); [Martin et al., 2000b](#)). Adult studies suggest that weight gain is likely to be greatest with clozapine, followed by olanzapine, risperidone, and quetiapine ( [Allison et al., 1999](#)). Thus far, and for reasons that are not clear, ziprasidone has not been associated with weight gain. The mechanism for weight gain appears to be through an increase in appetite, which may be owing to interference with the satiety mechanism. The health consequences of obesity can be significant. For example, case reports suggest that olanzapine may contribute to increased serum triglycerides ( [Osser et al., 1999](#)) and drug-induced Type II diabetes ( [Wirshing et al., 1998](#)). Rare cases of hepatotoxicity have been reported with risperidone, prompting some investigators to recommend periodic measurement of liver enzyme levels during maintenance treatment ( [Kumra et al., 1997](#); [Landau and Martin, 1998](#)).

### Tardive Dyskinesia

The risk of tardive dyskinesia (TD) increases as a function of dose and duration, but it may occur with brief exposures as well. Tardive dyskinesia has been reported in pediatric patients, but it does not appear to be common ( [Riddle et al., 1987](#)). Thus, children and adolescents treated with antipsychotics should be monitored for abnormal movements. The largest systematic study to date ( [Campbell et al., 1997](#)) raises questions regarding the difficulty in distinguishing between TD and withdrawal dyskinesia (WD). In that study, 30% of children between the ages of 2 and 8 years showed persistent dyskinesic movements for up to 3 months of follow-up after a planned withdrawal from haloperidol. Questions of how and when to discontinue antipsychotic medication are critical ones, but data to guide clinical decisions are limited. To minimize WD, dose reductions should be done gradually while evaluating changes in symptom severity. In autism and tic disorders, discontinuation may be considered annually for cases in which good control has been achieved. If symptoms persist, the maintenance dose of the neuroleptic should be reduced to the lowest possible one sufficient to maintain symptomatic control and minimize overall exposure ( [McClellan and Werry, 1994](#)). Based on the collective experience with clozapine, the newer atypicals may also have a lower risk of TD. More study is needed, such as long-term follow-up of clinical samples treated with atypical antipsychotics and careful postmarketing surveillance of these agents in pediatric populations.

Neuroleptic malignant syndrome (NMS) is a rare adverse effect that has been associated with the typical antipsychotics. Characterized by high fever, autonomic instability, and muscle breakdown (reflected by elevated creatine phosphokinase (CPK) titers), it can pose a life-threatening risk. Indeed, a disease-fatality rate of 9% has been reported for children and adolescents ( [Silva et al., 1999](#)). Discontinuation of the agent involved is usually all that is required, highlighting the importance of early identification of the condition. In severe cases, IV fluids are needed, and some authors have advocated the use of dantrolene or bromocriptine to maximize recovery.

### Antidepressants

The antidepressants include a group of chemically diverse compounds that have been shown to be effective in the treatment of adults with major depression. More recently, several antidepressants have been used in the treatment of adults with a range of other disorders, including OCD, generalized anxiety disorder (GAD), panic disorder, social phobia, and posttraumatic stress disorder (PTSD). These broader clinical applications likewise are being implemented with increasing frequency in the pediatric population, even though the level of empiric support varies widely, and depends largely on the disorder under consideration. Antidepressants can be classified according to: (a) chemical similarity (such as tricyclics compounds, and within these, secondary or tertiary amines); (b) primary mode of action (such as the SSRIs, selective norepinephrine reuptake inhibitors, or monoamine oxidase inhibitors); and (c) miscellaneous, newer antidepressants (e.g., bupropion, venlafaxine, or nefazodone).

### Tricyclic Antidepressants

#### CLINICAL APPLICATIONS AND EMPIRIC SUPPORT

Tricyclic antidepressants (TCAs) have been used to treat several psychiatric disorders of childhood over the past three decades, including depression ( [Puig-Antich et al., 1987](#)), ADHD ( [Biederman et al., 1989](#)), OCD ( [DeVeauugh-Geiss et al., 1992](#)), separation anxiety disorder ( [Klein et al., 1992](#)), and enuresis ( [Werry et al., 1975](#)). Although TCAs have been used frequently in clinical practice, evidence for their efficacy in treating children with these psychiatric disorders is inconsistent. For example, a series of carefully conducted controlled trials have consistently failed to show the superiority of any TCA over placebo in the treatment of child- and adolescent-onset depression. This poor track record stands in marked contrast to the more compelling results in adult depression ( [Martin et al., 2000a](#)). The use of TCAs in non-OCD anxiety is equivocal. One recent study found imipramine superior to placebo in the treatment of separation anxiety ( [Bernstein et al., 2000](#)), but an earlier report found no differences between the treatment conditions ( [Klein et al., 1992](#)). In contrast to these respectively disappointing or inconclusive findings in

depression or separation anxiety, double-blind, placebo-controlled studies have demonstrated the efficacy of desipramine in children with ADHD ( [Biederman et al., 1989](#); [Singer et al., 1995](#)), and of clomipramine for the treatment of OCD in children and adolescents ( [DeVeauugh-Geiss et al., 1992](#)). Clomipramine also has been used in the treatment of children with autism accompanied by OCD-like symptoms. In a crossover study, clomipramine was superior to desipramine and placebo in reducing repetitive behaviors ( [Gordon et al., 1993](#)). Intravenous administration of clomipramine has shown promise in adults and, to a lesser extent, in adolescents with refractory OCD ( [Koran et al., 1998](#); [Sallee et al., 1998](#)).

#### MECHANISM OF ACTION

To varying degrees, the TCAs inhibit the reuptake of NE by presynaptic neurons. Over time, this pharmacologic effect is presumed to enhance noradrenergic neurotransmission. Among the TCAs, desipramine is the most selective in its capacity to block NE reuptake. This highly selective property of desipramine is presumed to play a role in its effectiveness in ADHD. Based on the favorable effects of desipramine in ADHD, there is interest in other compounds, such as tomoxetine, which have highly selective norepinephrine reuptake inhibiting properties ( [Spencer et al., 1998b](#)). Unlike desipramine, however, tomoxetine does not appear to prolong cardiac conduction times. Clomipramine is unique among the TCAs in that it is a potent inhibitor of serotonin reuptake. This property is presumed to explain the superiority of clomipramine over desipramine for the treatment of OCD ( [Leonard et al., 1989](#)).

#### PHARMACOKINETICS

Serum levels of TCAs can show wide variation across individuals taking the same oral dose because of genetic differences in CYP 450 enzyme activity. Thus, therapeutic levels for the TCAs are not well established in pediatric populations. Serum levels may be useful, however, to identify children with low or rapid metabolic activity, rule out toxicity, and assess compliance.

#### CLINICAL MANAGEMENT

An ECG, pulse, and blood pressure should be obtained prior to starting any of the TCAs. A medical and family history that focuses on syncope in the child, as well as episodes of syncope or sudden death in close relatives may be informative. Evidence of a normal physical examination within the past year also should be documented. The typical dose range for TCAs in children is up to 5 mg/kg per day for imipramine, 2.5 mg/kg per day for nortriptyline, and perhaps somewhat higher (3 mg/kg per day) for clomipramine. Imipramine may be started at a dosage of 25 mg and increased every 4 or 6 days in similar increments to 100 to 150 mg per day. In younger children, nortriptyline is typically introduced with a 10-mg dose, with increases every 4 to 6 days to a range of 50 to 75 mg per day in divided doses. Clomipramine is usually started at a dose of 25 mg, with gradual increases every 4 or 6 days to a maximum of 100 mg per day in younger children and 150 mg in older children. For all of the TCAs, repeat vital signs and electrocardiograms (ECGs) should be obtained during the dose adjustment phase and when the maintenance dose has been achieved. As part of the informed consent process, potential cardiac effects and the reason for repeat ECG monitoring should be discussed with the family and child in a developmentally appropriate manner. A heart rate corrected QT interval (QTc) above 460 ms, QRS complex longer than 120 ms, or PR interval greater than 200 ms ( [Gutgesell et al., 1999](#)) warrant dose reduction followed by a repeat ECG. Exceeding these parameters should prompt treatment reevaluation and perhaps discontinuation. For cases showing clinical benefit and persistent ECG abnormalities, consultation with a pediatric cardiologist is in order.

#### SIDE EFFECTS

The TCAs are associated with a range of side effects, including sedation, dizziness, dry mouth, excessive sweating, weight gain, urinary retention, tremor, and agitation. Lowering the dose, changing dose schedules, or switching between secondary and tertiary amines often can help manage these effects. For example, to deal with sedation, the medication could be given twice a day, with the higher dose in the evening. Switching between TCAs can be helpful at times: For example, imipramine (a tertiary amine) can be changed to nortriptyline (a secondary amine) in an effort to minimize sedation or constipation. Despite the evidence showing the efficacy of clomipramine for OCD and desipramine for ADHD, the TCAs appear to be declining in use ( [Ryan and Varma, 1998](#)). This trend is largely owing to the side effect profile and potential for serious adverse effects; the rare possibility of sudden death related to tachyarrhythmias is the most ominous. Originally reported in cases treated with desipramine, the series of case reports that accrued over ensuing years has led some (but not all) experts to recommend avoiding TCAs in the pediatric population ( [Werry et al., 1995](#)). The combination of: (a) stepwise dosing within clear weight-adjusted margins; (b) careful ECG monitoring; (c) full disclosure of the risk:benefit ratio in the treatment planning process; and (d) their selective use in nonresponders to first-line agents, provides a rational basis for keeping these important compounds in the child psychiatric armamentarium.

#### INTERACTIONS

Hepatic enzymes in the cytochrome P450 system (CYP 450) metabolize the TCAs, like most other psychotropic drugs. Several psychotropic (fluoxetine, fluvoxamine, paroxetine) and nonpsychotropic drugs (ketoconazole, cimetidine, erythromycin) inhibit the action of one or more of these hepatic enzymes. Inhibition of the enzyme specific for metabolizing the TCA can result in toxicity ( [Flockhart and Oesterheld, 2000](#)). For example, the commonly used broad-spectrum antibiotic erythromycin is a potent inhibitor of CYP3A4. Clomipramine is a substrate for this enzyme. The addition of erythromycin to ongoing treatment with clomipramine causes a dramatic rise in the level of the latter, with an attendant increase in the risk of adverse effects. This mechanism also has been exploited as a therapeutic maneuver in refractory OCD. When added to ongoing treatment with clomipramine, fluvoxamine (also an inhibitor of CYP3A4) reduces the metabolism of clomipramine and causes substantial increase in drug level. To date, only case reports showing benefit in refractory cases have been published. The proposed mechanism for the reported benefit in refractory cases is the rise in the parent compound, clomipramine, which is more selectively serotonergic in its action than the noradrenergic metabolite, desmethylclomipramine. Because this combination increases the risk of typical side effects of clomipramine, as well as the risk of prolonged cardiac conduction times and of seizures, this combined approach is reserved for refractory cases only.

#### Selective Serotonin Reuptake Inhibitors

The SSRIs are a group of chemically unrelated compounds that potently inhibit the return of serotonin into presynaptic neurons. Currently marketed SSRIs include fluoxetine, sertraline, paroxetine, fluvoxamine, and citalopram. In contrast to clomipramine, which inhibits the reuptake of both NE and serotonin, these newer compounds are more restricted in their reuptake of serotonin; therefore, their denotation as selective.

#### CLINICAL APPLICATIONS

All of the SSRIs in current use are approved for use in the treatment of adults with OCD. With the exception of fluvoxamine, the SSRIs also are approved for use in adults with major depression. More recently, paroxetine and sertraline have been approved for adults with anxiety disorders, and sertraline for PTSD. In pediatric populations, the FDA has approved fluvoxamine and sertraline for the treatment of OCD.

#### EMPIRIC SUPPORT

The introduction of the SSRIs, starting with fluoxetine in the late 1980s, has had a dramatic impact on the practice of pediatric psychopharmacology. Compared to the TCAs, monotherapy with the SSRIs is relatively simple. As a group, these medications are generally well tolerated, typically can be given once a day, and do not require blood level monitoring or ECGs. Following the early clinical trials with clomipramine and fluoxetine in children and adolescents ( [DeVeauugh-Geiss et al., 1992](#); [Leonard et al., 1989](#); [Riddle et al., 1992](#)), several large placebo-controlled clinical trials with sertraline ( [March et al., 1998](#)) and fluvoxamine ( [Riddle et al., 2001](#)) in OCD; with fluoxetine ( [Emslie et al., 1997](#)) and paroxetine (Keller et al., 1998) in depression, and with fluvoxamine in non-OCD anxiety disorders ( [RUPP, 2001](#)), have been conducted in pediatric populations. In each of these studies, the SSRI was superior to placebo in the primary outcome measure of interest.

#### OBSESSIVE-COMPULSIVE DISORDER

Sertraline and fluvoxamine have been evaluated in randomized, multisite, placebo-controlled trials of parallel groups. Using the Children's Yale-Brown Obsessive Compulsive Scales (CYBOCS) as the primary outcome measure, both drugs were superior to placebo in improving obsessive-compulsive symptoms. Sertraline was evaluated in 187 subjects ranging from 6 to 12 years of age. In that study, an average daily dose of 167 mg of sertraline was associated with at least a 25% improvement in the CYBOCS score in 53% of subjects, compared to 37% for placebo ( $P = 0.03$ ) ( [March et al., 1998](#)). In 120 children between the ages of 8 and 17 years, fluvoxamine at a mean daily dose of 165 mg was effective in 42% of children, compared to 26% among those treated with placebo ( $P = 0.06$ ) ( [Riddle et al.,](#)



[2001](#)). Based on open-label follow-up data, beneficial effects were maintained for several months in a majority of subjects (Walkup, personal communication).

An open-label study of paroxetine in pediatric OCD revealed not only promising results ([Rosenberg et al., 1999](#)), but also interestingly, reductions in thalamic volume ([Gilbert et al., 2000](#)) and caudate glutamate levels ([Rosenberg et al., 2000](#)), which paralleled clinical response. Two small placebo-controlled studies provide additional support for the efficacy of fluoxetine in OCD ([Kurlan et al., 1993](#); [Scahill et al., 1997](#)). Finally, one open-label study with citalopram has been done in children with OCD ([Thomsen, 1997](#)). In that study, 23 subjects were treated with 10 to 40 mg per day of citalopram for 10 weeks in an open-label fashion. Eleven of 23 subjects showed a clinically meaningful positive response (30% improvement or more). Five patients showed little or no response, and the remaining seven showed a partial response.

Taken together, these data suggest that the SSRIs are effective for the treatment of OCD in children and adolescents; however, the magnitude of response may not be large. In addition, some children with OCD may show only a partial response to an adequate trial of an SSRI. For example, approximately 40% of the subjects in the multisite sertraline study showed less than a 25% improvement in obsessive-compulsive symptoms ([March et al., 1998](#)). This observation indicates that clinicians should remind parents and patients not to have unreasonably high expectations for SSRI treatment. The problem of partial response raises questions about whether to switch to another SSRI or clomipramine, or embark on one of several augmentation medication strategies. Although not well studied in children, two studies have shown that the addition of low-dose haloperidol ([McDougle et al., 1994](#)) or risperidone ([McDougle et al., 2000](#)) to an SSRI can be effective in adults with refractory OCD. When evaluated in rigorously controlled studies, no other pharmacologic addition strategies have been effective in refractory OCD. These strategies have included, drugs such as lithium and buspirone among others, both of which enhance serotonergic function. Case reports suggest that the addition of clonazepam may be useful in some cases ([Leonard et al., 1994](#)) and combined treatment with an SSRI and clomipramine also may be useful ([Figueroa et al., 1998](#)). In view of the inadequate support for combined pharmacotherapy in children with refractory OCD, other interventions should be considered. For example, cognitive-behavioral therapy (CBT) has shown promise in children and adolescents with OCD ([March et al., 1994](#); [Scahill et al., 1996](#)). Further study in combined medication approaches and the combined use of CBT with medication for the treatment of children and adolescents with OCD are needed.

## DEPRESSION

Fluoxetine, sertraline, and paroxetine have each been studied for the treatment of depression in children and adolescents. The landmark fluoxetine study by Emslie and colleagues (1997) was the first to show superiority of an antidepressant over placebo for the treatment of depression in children and adolescents. A placebo-controlled, multicenter trial comparing paroxetine, imipramine, and placebo was completed recently ([Keller et al., 2001](#)). Paroxetine was found to be superior to placebo, achieving a 63% response rate, compared to 46% in the placebo group. By contrast, the response rate in the imipramine group was 50%, which was not statistically different from placebo. Imipramine was associated with common TCA side effects and with a high rate of premature discontinuations. Two open-label sertraline studies ([Ambrosini et al., 1999](#); [Tierney et al., 1995](#)) with a combined sample of 80 subjects showed promising preliminary results. An industry-sponsored, placebo-controlled study of sertraline is now under way and should provide definitive results. Collectively, these data suggest that the SSRIs are the first-line agents for the treatment of depression in children and adolescents. In support of this conclusion, the recently published Texas algorithm for depression in children and adolescents recommends fluoxetine as the first-line drug ([Hughes et al., 1999](#)).

## NON-OBSESSIVE-COMPULSIVE ANXIETY DISORDERS

To date, only fluvoxamine has been evaluated in the treatment of children and adolescents with non-OCD anxiety disorders. In a multisite study sponsored by the National Institute of Mental Health ([RUPP, 2001](#)), 128 subjects between the ages of 6 and 17 years were randomly assigned to placebo or fluvoxamine after a 3-week psychoeducational intervention. The primary outcome measure was the Pediatric Anxiety Rating Scale (PARS), a new scale developed specifically for the trial ([Walkup and Davies, 1999](#)). After 8 weeks of treatment, children in the fluvoxamine group showed a 52% improvement (mean decrease in PARS from 18.7 to 9.0) compared to 16% improvement (mean decrease from 19.0 to 15.9) for the placebo group ( $P < 0.001$ ). The dose began at 25 mg per day, with a planned increase to 25 mg twice a day after 4 days. The dose schedule continued upward in 25-mg increments every 4 to 5 days as tolerated. The findings from this study provide support for the efficacy and large effect size of fluvoxamine in the treatment of generalized anxiety disorder, social phobia, and separation anxiety. This study also paves the way for the study of the other SSRIs and combination treatments (medication and psychotherapy) in non-OCD anxiety disorders.

## MECHANISM OF ACTION

The SSRIs interfere with the return of serotonin into the presynaptic neuron by blocking the serotonin transporter located on presynaptic nerve terminals. Over time, this blockade leads to a desensitization of the serotonin autoreceptors, which typically exert an inhibitory influence on serotonin release. With continued blockade of the transporter, the desensitized autoreceptors do not exert their usual inhibitory influence and serotonergic function is enhanced. Based on a series of animal studies, Blier and colleagues suggest that the main location of the enhanced serotonergic function appears to be the hippocampus in depression, and the orbital frontal cortex in OCD ([Blier and de Montigny, 1998](#)).

## PHARMACOKINETICS

All five SSRIs have relatively long half-lives, permitting single daily dosing. Fluvoxamine, which has the shortest half-life, is sometimes given on a twice-daily schedule. A recent pharmacokinetic evaluation of paroxetine showed that children metabolize the medication faster than adults ([Findling et al., 1999](#)). Despite the shorter half-life in the pediatric population, these investigators still recommend once-daily dosing for paroxetine. The pharmacokinetic profiles of the other SSRIs in pediatric populations have not been documented. In adults, fluoxetine and citalopram have the longest half-lives of currently available SSRIs, with estimates of 48 to 72, and 33 hours, respectively. In addition, fluoxetine has an active metabolite (norfluoxetine) with an elimination half-life of 7 to 14 days. Both fluoxetine (primarily norfluoxetine) and paroxetine are potent inhibitors of CYP 2D6. Because both paroxetine and fluoxetine are 2D6 substrates, they inhibit their own metabolism, resulting in nonlinear kinetics at higher doses.

## CLINICAL MANAGEMENT

### Fluoxetine

Fluoxetine (Prozac) is available in a 10-mg scored tablet, a 20-mg capsule, and a 20-mg/5 mL liquid preparation. A typical starting dose for school-age children is 5 to 10 mg per day; smaller children may start at 2.5 mg per day. Given its long half-life, fluoxetine should be increased slowly (weekly or even at 2-week intervals) to avoid "overshooting" the optimal dose. The usual dose range for children and adolescents is 5 to 40 mg per day, although some children and adolescents may require higher doses.

### Sertraline

Sertraline (Zoloft) is available in 25-, 50-, and 100-mg tablets that can be broken in half easily. Treatment might start with a 12.5- to 25-mg dose, with similar weekly increments to a range of 50 to 150 mg in children. Higher dosages may be required in older adolescents. A recent, multiple dose study in adults with OCD showed that some patients respond to relatively low doses of sertraline ([Greist et al., 1995](#)). Thus, clinicians should review therapeutic response during the dose adjustment phase to determine whether additional increases are needed, rather than using an automatic dose schedule.

### Fluvoxamine

Fluvoxamine (Luvox) is available in 25-, 50-, and 100-mg scored tablets. Treatment usually begins at 12.5 to 25 mg per day and is increased by 25 mg on a weekly basis. The typical dose range is 50 to 200 mg per day. Although the double-blind trial in children and adolescents with OCD used a rapid dose escalation with increases every 3 days ([Riddle et al., 2001](#)), the more recent RUPP anxiety study used a slower upward adjustment ([RUPP, 2001](#)). This study started with 25 per day and increased to 25 mg twice a day within the first week. Thereafter, the dose was increased in 25-mg steps each week as tolerated. This schedule is more in keeping with current clinical practice ([Grados et al., 1999](#)).

### Paroxetine

Paroxetine (Paxil) is available in 10-, 20-, and 30-mg tablets that can be broken in half, as well as in an oral suspension (10 mg/5 mL). The typical starting dose is 5 to

10 mg per day, with weekly increases to a total daily dose of 10 to 40 mg.

### *Citalopram*

Citalopram (Celexa) is available in 10- and 20-mg scored tablets, as well as a liquid preparation. As noted, there is only one study in pediatric populations, in which 23 children with OCD were treated openly for 10 weeks at doses ranging from 10 to 40 mg per day. Based on experience with the other SSRIs, a reasonable starting dose is 5 mg per day, with increases on weekly or 2-week intervals, to a maximum of 40 mg per day.

### *SIDE EFFECTS*

The SSRIs are generally well tolerated as a group, and potentially serious side effects such as alterations in cardiac conduction times or seizures have not been reported in the usual dose range. In addition to their propensity for cytochrome P450-based drug interactions ( [Oesterheld and Flockhart, 2000](#)), common side effects of the SSRIs in children and adolescents appear to be behavioral activation and GI complaints such as nausea or diarrhea. Signs of behavioral activation include motor restlessness, insomnia, impulsive, and/or disinhibited behavior. It may occur early in treatment, with dose increases ( [King et al., 1991](#)), or following the addition of drugs that inhibit the metabolism of the SSRI (e.g., cimetidine). The potential for behavioral activation early in treatment underscores the importance of starting at low doses and moving upward slowly. As with other antidepressants, hypomania and full manic activation also have been reported. For example, in a series of 33 children and adolescents being treated for depression, two cases of sertraline-induced mania were reported ( [Tierney et al., 1995](#)). Other side effects include diarrhea, nausea, heartburn, decreased appetite, and fatigue. Sexual side effects, which are relatively common in adults, also should be considered in sexually active adolescents.

There have also been reports of suicidal ideation and self-injurious behavior with fluoxetine ( [King et al., 1991](#)). Whether fluoxetine confers greater risk for suicidal ideation than the other SSRIs or whether the SSRIs carry a higher risk for suicidal ideation than other antidepressant classes is unlikely. As with all antidepressants, especially when treating depression, clinicians should monitor suicidal thought and self-injurious potential in any child or adolescent treated with an SSRI.

### *SELECTIVE SEROTONIN REUPTAKE INHIBITOR DISCONTINUATION SYNDROME AND DURATION OF THERAPY*

A flu-like syndrome characterized by dizziness, moodiness, nausea, vomiting, myalgia, and fatigue occurring in association with the withdrawal or acute discontinuation of shorter-acting SSRIs such as paroxetine, fluvoxamine, and sertraline has been described ( [Black et al., 2000](#)). Recently, a controlled discontinuation study in 220 adults compared the withdrawal effects of fluoxetine, paroxetine, and sertraline: Paroxetine and sertraline were associated with irritability, agitation, fatigue, insomnia, confusion, dizziness, and nervousness on abrupt withdrawal, but fluoxetine was not ( [Rosenbaum et al., 1998](#)). The long half-life of fluoxetine and norfluoxetine presumably results in a gradual "autotaper," even when the oral dose is stopped abruptly. Based on these results, a slow withdrawal of the shorter-acting SSRIs is warranted. Citalopram has a 33-hour half-life, but no known active metabolites. Sudden discontinuation of citalopram also should be avoided in the absence of data on adverse effects following abrupt withdrawal.

Another clinical issue that often arises in the course of treating children and adolescents with an SSRI concerns the duration of treatment. Studies of adults with depression suggest that an episode of depression typically lasts 9 months to 1 year. Based on this evidence, the duration of treatment for depression can be set at a 1-year minimum. For OCD and anxiety disorders, however, there are no data on which to base duration of treatment. A recent review on OCD suggests discontinuation after a relatively symptom-free period of 8 to 12 months ( [Grados et al., 1999](#)). A long-term follow-up study of 54 children and adolescents with OCD found that 70% ( $N = 39$ ) remained on medication for more than 2 years ( [Leonard et al., 1993](#)). Given the potential for chronicity in OCD, children and parents should be informed that symptoms might return following a planned SSRI discontinuation. Clearly, more study is needed on the matter of SSRI treatment duration in pediatric populations.

### *DRUG INTERACTIONS*

The SSRIs are increasingly common in clinical practice because of their multiple clinical applications, ease of use, and perceived safety. In addition, the use of combined psychotropic medications seems to be on the rise. These trends underscore the importance of monitoring drug–drug interactions in clinical practice. All of the SSRIs have the potential for such interactions because they all inhibit one or more of the CYP isoenzymes. As illustrated previously, inhibition of the enzyme system responsible for metabolizing an active drug raises its serum level, thereby enhancing its beneficial or deleterious effects. For example, oculogyric crises and other dystonic reactions have been reported in youngsters when an antipsychotic was added to ongoing treatment with paroxetine ( [Horrigan and Barnhill, 1994](#); [Lombroso et al., 1995](#)). The adverse event reported by Lombroso and associates (1995) was probably the result of paroxetine's inhibition of CYP2D6, the enzyme that metabolizes risperidone. (For a more detailed discussion of drug interactions, interested readers are referred to the previous chapter.)

### **Other Antidepressant Medications**

#### *Bupropion*

Bupropion (Wellbutrin) is unrelated to all other available antidepressants. Although its mechanism of action is unclear, it appears to have both dopaminergic and noradrenergic effects. It is approved for the treatment of depression and smoking cessation in adults. Although bupropion has not been studied for depression in children or adolescents, it has been evaluated in controlled studies for the treatment of ADHD. A placebo-controlled trial of bupropion in 72 children with ADHD showed its superiority over placebo in the treatment of ADHD, although the treatment effect was smaller than that usually seen with stimulants ( [Conners et al., 1996](#)). The dose of bupropion (3 to 6 mg/kg per day) ranged from 50 to 200 mg per day in divided doses. The findings of this study are consistent with previous placebo-controlled studies ( [Casat et al., 1987](#)), and a direct comparison with methylphenidate ( [Barrickman et al., 1995](#)). In a recent open-label study conducted in 24 adolescents (11 to 16 years old) with comorbid ADHD and depression, sustained release bupropion was associated with improvements in both conditions in 58% ( $N = 14$ ), depression only in 29% ( $N = 7$ ), and ADHD alone in 4% ( $N = 1$ ), suggesting that further studies of this monotherapy appear warranted for these commonly comorbid conditions ( [Daviss et al., 2001](#)).

Side effects of bupropion include agitation, insomnia, skin rashes, nausea, vomiting, constipation, and tremor. Bupropion also may reduce the seizure threshold in a dose-dependent fashion; therefore, daily doses should not exceed 300 mg in children, and no single dose should be higher than 150 mg. Bupropion (Wellbutrin) is available in 75- and 100-mg tablets, and 100- and 150-mg sustained release (SR) tablets. Treatment is usually on a TID basis given the agent's short half-life, although BID dosing is possible with the SR preparations.

#### *Venlafaxine*

Venlafaxine (Effexor) is an agent that selectively inhibits serotonin reuptake at lower doses (<150 mg/d). It acts on both NE and serotonin reuptake at the higher dose range. To date, there is one placebo-controlled trial in children with depression. The study, which included 32 youngsters, found that the drug was no better than placebo in relieving depression ( [Mandoki et al., 1997](#)). The lack of a significant result may well relate to the small sample size, suggesting that larger studies are warranted. Indeed, a large multisite, industry-sponsored study is currently underway.

Venlafaxine is available in 25-, 37.5-, 50-, 75-, and 100-mg tablets, and in 37.5-, 75-, and 100-mg extended release (XR) capsules. Dosing is started with the smallest dose given at bedtime, and with attention to early sedation and dizziness, before moving to a twice-daily regimen. At higher doses (>150 mg/d), venlafaxine can be associated with diastolic hypertension, an effect that is clearly dose-dependent in nature.

#### *Nefazodone*

Nefazodone (Serzone) is a potent 5HT<sub>2a</sub> postsynaptic antagonist and a moderate serotonin and norepinephrine reuptake inhibitor. The clinical importance of this novel mechanism is not entirely clear, but nefazodone has been shown to be effective in the treatment of adults with major depression, especially when accompanied by prominent anxiety features. The pharmacokinetic properties of nefazodone have been studied in an 8-week trial of 28 children and adolescents. The kinetic profile was shown to be similar to that in adults, and 19 of 28 subjects were rated as clinically "much" or "very much improved" on a global impression scale ( [Findling et al., 2000b](#)). Based on these findings, an industry-sponsored, placebo-controlled in children 12 to 18 years old has been undertaken, with results still pending. Nefazodone is available in 50-, 100-, 150-, 200-, and 250-mg tablets. Dosing is divided into a twice-daily regimen, usually "weighed" toward bedtime given the agent's



sedating properties.

Several other new antidepressants have entered or will soon be introduced into the marketplace, including mirtazapine, tomoxetine, reboxetine, and various selective monoamine oxidase inhibitors (MAOIs). Data on the use of these newer agents in the pediatric population are not yet available.

## Mood Stabilizers

The prototype for the chemically unrelated drugs in this class is lithium, which has been used in the treatment of bipolar illness for nearly 50 years. Other medications that are currently on this growing list include valproate, carbamazepine, lamotrigine, gabapentin, and topiramate. Of these, only valproate and carbamazepine have been carefully studied in adults. Encouraging preliminary data are available for lamotrigine and topiramate. By contrast, empiric data for any of the mood stabilizers are scarce in the pediatric population ([Davanzo and McCracken, 2000](#)).

Data from clinical samples indicate that cases of bipolar illness resembling the adult form of the disorder can be observed in children and adolescents ([Fristad et al., 1992](#); [Strober et al., 1995](#)); however, some investigators contend that the clinical presentation of bipolar disorder in childhood may have important differences from the classic presentation in adults. In an effort to guide practice, these authors favor an expanded definition of the bipolar phenotype in children. For example, some argue that discrete episodes of mania may be harder to identify in children and adolescents ([Geller and Luby, 1997](#)). The notion that bipolar disorder resides on a spectrum encompassing the full syndrome and a cluster of related, albeit milder symptoms is supported by observations in community and clinical samples ([Lewinsohn et al., 1995](#)). What is less clear, however, is whether this wider phenotype is responsive to mood stabilizing medications. Nonetheless, the mood stabilizers are being increasingly used to treat a broad range of symptoms, including irritability, mood lability, aggression, and bipolar disorder.

### LITHIUM

Although controlled data in pediatric populations are scarce, accumulated evidence from case reports and open studies suggest that lithium is safe and effective for the treatment of children and adolescents with bipolar illness or severe aggressive behavior ([Davanzo and McCracken, 2000](#)).

#### *Clinical Applications and Empiric Support*

Lithium was compared to placebo in a crossover study of six children with various psychiatric diagnoses, including ADHD, major depression, and a "mixed" bipolar disorder ([McKnew et al., 1981](#)). Only the two subjects diagnosed with bipolar illness showed a clearly positive response to lithium. Other early lithium studies showed promising results among children ([Carlson et al., 1992](#); [Younes et al., 1986](#)). Geller and colleagues (1998) conducted the first placebo-controlled study of lithium in youngsters, including 25 adolescents with various forms of bipolar illness and comorbid substance abuse. Although lithium was associated with improvements in overall functioning and substance abuse, there was no difference between active and placebo groups on measures of manic or depressive symptoms. The failure of lithium to separate from placebo on the core bipolar symptoms may be owing to the small sample size ( $N = 12$  in active group,  $N = 13$  in placebo). Taken together with the small studies cited in the preceding, these data provide only modest support for the use of lithium for the short-term treatment of mixed bipolar illness in children and adolescents.

In a long-term naturalistic observations study, 59 children with a mean age of 10.9 years who were diagnosed with classic bipolar disorder and treated with lithium were followed for up to 10 years ([DeLong and Aldershof, 1987](#)). All patients were evaluated on and off the medication during planned discontinuation periods. Lithium was effective in 39 of 59 patients with bipolar disorder, showing both short-term and enduring benefits. In an 18-month naturalistic follow-up study, 24 of 37 adolescents with bipolar I disorder showed long-term benefit from lithium treatment ([Strober et al., 1990](#)). The 13 adolescents, who discontinued lithium treatment during the first year, were 20 times more likely to relapse than those who remained on maintenance therapy (12 of 13 versus nine of 24, respectively). These two studies provide support for the benefit of lithium prophylaxis in children and adolescents with classic bipolar disorder.

#### *Other Clinical Applications: Aggression and Self-Injury*

Lithium was superior to placebo in reducing the frequency of aggression in 66 nonpsychotic male prisoners (16 to 24 years old); however, 14 or nearly half of the 33 subjects on active medication withdrew from the study owing to adverse effects ([Sheard et al., 1976](#)). A study comparing haloperidol, lithium, and placebo in 61 treatment-resistant aggressive children (5 to 13 years old) found that after 4 weeks of inpatient treatment, both haloperidol and lithium were superior to placebo in reducing aggressive behavior; lithium was associated with fewer side effects ([Campbell et al., 1984](#)). More recently, lithium was found to be superior (16 of 20 responders) when compared to placebo (six out of 20) in the treatment of aggressive behaviors in hospitalized children with conduct disorder ( $P = 0.004$ ) ([Malone et al., 2000](#)). By contrast, the experience in the treatment of self-injurious behaviors in children with developmental disorders generally has been disappointing ([DeLong and Aldershof, 1987](#)).

#### *Mechanism of Action*

Lithium affects several neurochemical systems, including the function of serotonin, NE, and dopamine. However, its main actions seem to be mediated by effects on intracellular signaling processes, and specifically, the phosphatidylinositol and protein kinase C pathways ([Manji and Lenox, 1998](#)).

#### *Pharmacokinetics*

Lithium is readily absorbed from the gastrointestinal tract, with peak levels occurring 1 to 3 hours after oral ingestion. It is not metabolized in the liver, and the kidneys excrete approximately 95% of the ingested drug. The half-life in adults is approximately 24 hours, slightly longer than the 18 hours reported in children ([Vitiello et al., 1988](#)). This shorter half-life in children is probably owing to faster glomerular filtration rates.

#### *Clinical Management*

Dosages in the range of 10 to 30 mg/kg per day are typical for children under 12 years of age; thus, a 30-kg child would receive 900 mg per day in divided doses. Older adolescents are likely to be treated in the range of 1,200 to 1,800 mg per day during acute mania. Maintenance doses typically are lower. The optimal serum level range is in the range of 0.6 to 1.1 mEq/L. Lithium is available in tablet, capsule, and liquid (lithium citrate) forms. In outpatient settings, the dose typically is started at 300 mg twice a day for children, up to 600 mg twice a day for older adolescents. Trough lithium levels can be checked after 4 to 6 days, and adjustments in dose made as necessary ([Geller and Luby, 1997](#)). When used to treat bipolar illness, the clinical benefit of lithium may be evident within 10 to 14 days of reaching therapeutic serum level in some cases, although as many as 4 to 6 weeks of treatment may be required ([Kowatch et al., 2000](#)).

Prior to initiating a trial of lithium, a child should have a physical examination, including screening laboratory tests such as complete blood count, electrolytes, blood urea nitrogen, creatinine, and thyroid indices. Because the kidneys excrete lithium, it is generally not recommended in children with compromised renal function. The risk of glomerular damage with long-term lithium treatment appears to be minimal, but polyuria and polydipsia are relatively common because of lithium's effect on tubular reabsorption. In a few cases, nephrogenic diabetes insipidus can occur, which may warrant discontinuation. Current recommendations include repeat laboratory tests at 6-month intervals. Lithium levels should be monitored owing to its narrow therapeutic range. Levels also should be obtained when the patient's clinical status changes, if side effects occur, and routinely at 3- to 6-month intervals. Salivary lithium levels can be measured, but do not accurately predict serum levels ([Vitiello et al., 1988](#)). Serum levels should be drawn 4 to 6 days after a dose adjustment to ensure that a steady state has been achieved, and 12 hours after the previous dose to ensure a trough reading.

#### *Toxicity and Side Effects*

Lithium appears to be generally well tolerated in children and adolescents. The most common side effects may be nausea, tremor, polyuria, polydipsia, and enuresis. Other relatively frequently reported side effects include diarrhea, acne, abdominal distress, cognitive dulling, hair loss, and weight gain. The signs of lithium toxicity include tremor, blurred vision, nausea, diarrhea, ataxia, hyperreflexia, and dysarthria, which can occur at serum levels of 1.4 mEq/L or higher. Parents and children should be educated about the importance of adequate fluid intake because dehydration can increase lithium levels and may induce toxicity. At serum levels above 2.5 mEq/L, multiple organs may be affected and toxicity may prove fatal. Symptoms of acute overdose include nausea, vomiting, stupor, choreoathetosis, convulsions, and coma. Lithium, like valproate and carbamazepine, has been associated with increased occurrence of birth defects; therefore, contraception should be ensured in

sexually active teenage girls treated with any mood stabilizer.

#### *Drug Interactions*

Carbamazepine, nonsteroidal antiinflammatory drugs, tetracycline, and thiazide diuretics can all increase lithium levels when given concomitantly and therefore should be used cautiously. By contrast, theophylline and caffeine promote lithium excretion, resulting in lower serum levels.

#### **VALPROATE**

##### *Clinical Applications and Empiric Support*

Valproate is an anticonvulsant that has been shown to be an effective mood stabilizer in adults. The most commonly used formulation of valproic acid, divalproex, is an enteric-coated preparation containing valproic acid and sodium valproate in equal parts. In the pivotal three-group study demonstrating its efficacy in adult mania, divalproex ( $N = 69$ ) was compared to lithium ( $N = 36$ ) and placebo ( $N = 74$ ) (Bowden et al., 1994). Forty-eight percent of those subjects randomized to divalproex showed a positive response, which was significantly better than the 25% response rate in the placebo group, and no different from the 49% response rate in the lithium group. Largely based on these adult data, clinicians have increasingly resorted to divalproex in the treatment of a range of problems in children and adolescents, including bipolar illness and explosive behavior.

Until recently there were only case reports of divalproex use in pediatric populations (Kastner et al., 1990; Papatheodorou et al., 1995). In addition to the small number of subjects, these early observational reports studied treatment in a heterogeneous group of patients, making it impossible to merge data across studies.

Three recent studies provide useful, albeit preliminary, information about the use of divalproex in pediatric populations. In two related studies, valproate was evaluated in 30 adolescents with labile mood and explosive behavior. The first study was a 5-week, open-label trial involving 10 adolescents aged 15 to 18 years (Donovan et al., 1997). All 10 had a disruptive behavior disorder diagnosis, such as oppositional defiant disorder, conduct disorder, ADHD accompanied by school expulsion, defiance of rules, and petty crimes. Although labile mood was an entry criterion, none of the subjects met criteria for bipolar disorder. All 10 subjects showed a decline in the number and intensity of outbursts, as well as improvements in mood lability. Using similar ascertainment and entry criteria, the same investigators evaluated divalproex in a placebo-controlled, crossover trial of 20 patients (age range 10 to 18 years) (Donovan et al., 2000). During the first 6 weeks, eight of 10 subjects on divalproex showed a positive response, compared to none of 10 in the placebo group. Moreover, six of seven subjects who were randomly assigned to the placebo-first group showed a positive response to divalproex in the second phase of the study.

The use of divalproex in juvenile bipolar disorder was recently reported in a randomized, open-label trial comparing the drug ( $N = 15$ ) to lithium ( $N = 13$ ) and carbamazepine ( $N = 13$ ) (Kowatch et al., 2000). The initial sample of 42 children and adolescents, between the ages of 8 and 18 years, included 20 subjects with bipolar I and 22 with bipolar II disorder. Using a response criterion of 50% improvement or greater on the Young Mania Rating scale, divalproex was associated with a 53% response rate, compared to 38% for both carbamazepine and lithium ( $P = 0.06$ ). Although this study found a higher effect size for valproate (1.63) than for lithium or carbamazepine (1.06 and 1.00, respectively), small sample sizes urge caution when interpreting these seemingly large effects. Furthermore, divalproex has yet to be studied in a controlled fashion for this indication.

In conclusion, these emerging data provide a modest level of support for the use of divalproex in the treatment of children and adolescents with bipolar disorder and disruptive behavior accompanied by explosive outbursts and mood instability. Larger studies in equally well-characterized samples are needed to confirm these preliminary results.

##### *Mechanism of Action*

Divalproex has multiple pharmacologic effects and the details of its therapeutic actions are not fully known. It is clear that divalproex enhances GABA transmission through increased synthesis and release. Given the inhibitory role of GABA in the brain, this effect may account for the drug's anticonvulsant and antimanic effects. Based on animal studies, it has been proposed that divalproex shares with lithium a capacity to inhibit the protein kinase C signaling pathway (Manji and Lenox, 1999). This intracellular biochemical pathway plays a fundamental role in neuronal activity, transmitter release, and ultimately gene expression in the brain. Inhibition of protein kinase C is presumed to contribute to the antimanic effects of both drugs.

##### *Clinical Management*

Dosing in adolescents followed on an outpatient basis could start with 250 mg twice daily, and increase every three to five days in 250 to 500 mg increments, to a target dose of 20 mg/kg per day in two to three divided doses. Younger children might start with half the starting dose used in adolescents and move up in 125 to 250 mg increments. Levels should be checked after two weeks or sooner if a more aggressive dose schedule is used. Target serum levels are in the range of 85 to 110 (mg/L) (Kowatch et al., 2000).

Common side effects included nausea and sedation. Others include vomiting, increased appetite and weight, tremor, asthenia, dizziness, hair loss, and thrombocytopenia. There also have been reports of polycystic ovary disease in women treated for seizure disorders, manifested clinically by hyperandrogenism, accelerated weight gain, and menstrual and lipid profile irregularities (Isojarvi et al., 1993). The role of divalproex in these adverse endocrine effects is unclear and a matter of some debate, especially among teenage girls (Davis et al., 2000). Although rare, divalproex also has been associated with hepatic failure, and with fatal hepatitis in children under the age of 2. There is also a small risk of pancreatitis early in treatment. Clinical management involves monitoring weight, appetite, energy level, evidence of bruising, and of androgenism in girls. Liver enzymes and amylase levels should be obtained within the first month of treatment and periodically during chronic treatment (Davanzo and McCracken, 2000).

#### **CARBAMAZEPINE**

##### *Clinical Application and Empiric Support*

Carbamazepine is an anticonvulsant that has been used in adults for a variety of neurologic and psychiatric disorders, including seizures, trigeminal neuralgia, and bipolar disorder. It has proven effective in the treatment of lithium-resistant mania and impulsive behavior in adults. Case series and open-label studies in children have included bipolar disorder (Hsu, 1986; Kowatch et al., 2000; Woolston, 1999), aggression (Kafantaris et al., 1992), and treatment-resistant ADHD (see metaanalysis by Silva et al., 1996). To date, however, the one controlled carbamazepine trial in youngsters failed to show its superiority over placebo for the treatment of aggression among 5- to 12-year-old inpatients diagnosed with conduct disorder (Cueva et al., 1996).

##### *Clinical Management*

Carbamazepine is slowly absorbed, and peak plasma concentrations are achieved within 2 to 8 hours following oral administration. It is supplied in 100-mg (chewable) and 200-mg tablets, and in an oral suspension of 100 mg/5 mL. The initial oral dose for children ages 6 to 12 is 100 mg daily, and can be increased at weekly intervals by 100 to 200 mg. The usual maintenance dose is 10 to 20 mg/kg per day, administered in divided doses (BID or TID). Frequent dose adjustments, especially early in treatment, are common given the drug's short half-life following the induction of its own metabolism ("autoinduction"), a phenomenon related to microsomal enzyme activation.

Although the clinical utility of therapeutic plasma levels for children with mood disorders is unclear, plasma concentrations for anticonvulsant effect are in the range of 4 to 14 mg/mL, and these guidelines may be useful during dose adjustment and in order to prevent toxicity. Plasma levels should be obtained 2 to 4 days after achieving a steady-state plasma concentration. Baseline CBC, including platelet count, should be followed periodically. In addition to drug pharmacokinetic interactions, potential side effects include sedation, rash, leukopenia, and more rarely, aplastic anemia and thrombocytopenia.

Given the lack of controlled studies, the multiple drug-drug interactions, and its potentially serious side effects, carbamazepine should be considered as a second- or third-line mood stabilizer in the treatment of children and adolescents, and may be particularly useful in the context of morbid weight gain associated with lithium,



divalproex, or atypical antipsychotics.

#### OTHER (THIRD-GENERATION) ANTICONVULSANTS

Three new anticonvulsants recently approved for the treatment of epilepsy in adults have become the focus of interest as potential mood stabilizers. Although their efficacy in adults with psychiatric disorders is not yet established, and only case reports on their use in pediatrics are available to date, they are briefly reviewed here, because it is likely that they will be used increasingly in the coming years.

##### *Lamotrigine*

Lamotrigine (Lamictal) is an agent with a bimodal spectrum of efficacy; it has been used in the treatment of both depressed and manic symptoms, and may be especially useful among rapid cycling patients. Lamotrigine was reported as an effective augmenting intervention to valproate in 16 of 22 (72%) adolescents with bipolar depression ([Kusumakar and Yatham, 1997](#)). Of serious pediatric concern is the risk of skin rash reported in patients treated with lamotrigine, an effect that seems to be both dose- and age-related. The rash usually is mild and seen within the first months of treatment, but it can develop into full-blown Stevens-Johnson syndrome, a potentially lethal condition associated with widespread skin sloughing. Until additional safety and efficacy data become available, the use of this agent cannot be presently justified among children and adolescents except in the most extreme, treatment-refractory cases.

##### *Gabapentin*

Gabapentin (Neurontin) is of interest because it undergoes virtually no hepatic metabolism in addition to preliminary adult studies showing its efficacy as an antimanic agent. It is excreted largely unaltered by the kidneys and poses no serious drug interactions. However, despite a benign side-effect profile among adults, aggressive behavior and worsening hyperactivity have been reported among 12 children receiving gabapentin for the treatment of seizure disorders ([Davanzo and McCracken, 2000](#)). Consequently, its use as a mood stabilizer for children cannot be recommended until its efficacy and side-effect profile are better established through appropriate trials.

##### *Topiramate*

Topiramate (Topamax) is a glutamate release antagonist and a GABA reuptake inhibitor. It has been used in adults as an adjunctive intervention, usually in combination with traditional mood stabilizers or with atypical antipsychotics ([Chengappa et al., 1999](#)). The report of weight loss with topiramate in these initial studies is interesting. This side effect may offset the weight gain associated with atypical antipsychotic use if topiramate proves useful as a mood stabilizer. Topiramate may be associated with cognitive blunting and word retrieval difficulties, a concern that may limit its use in children.

#### **$\alpha$ 2-Adrenergic Agents**

The  $\alpha$ 2-adrenergic agents (clonidine and guanfacine) are approved for use only in adults with hypertension. Beginning with the early studies of clonidine for the treatment of TS, these drugs have become increasingly common in child psychiatry for treating tics, ADHD, and aggressive behavior in children ([Newcorn et al., 1998](#)). Indeed, between 1991 and 1995, the largest rise in the rate of psychotropic drugs used in preschoolers was for clonidine, with a 28-fold increase in one large database ([Zito et al., 2000](#)).

#### CLONIDINE

##### *Clinical Applications and Empiric Support*

There are only two controlled studies of clonidine (Catapres) in the treatment of tics, both of which included children and adults. In the first, clonidine was no better than placebo in suppressing tics ([Goetz et al., 1987](#)); however, a subsequent slightly larger trial proved it superior to placebo, producing approximately 35% improvement in tic severity ([Leckman et al., 1991](#)). Similarly, findings in ADHD have also been inconsistent: Clonidine provided modest benefit for ADHD in an early study involving 10 children in a placebo-controlled discontinuation study ([Hunt et al., 1985](#)). These findings were not replicated in a crossover study in which 34 children received desipramine, clonidine, and placebo in random order. Desipramine, but not clonidine, was superior to placebo on a parent measure of ADHD symptoms ([Singer et al., 1995](#)).

Taken together, these findings suggest that clonidine has modestly beneficial effects for ADHD symptoms and tics. These results also point out the potential limitations of small studies, even when a controlled design is used. One study shows benefit, but the next study fails to replicate the findings.

It is in this context that the multisite Treatment of ADHD in Children with Tourette's Syndrome (TACT) study was undertaken (Kurlan and McDermott, submitted). In this study, 136 children (7 to 14 years old) with ADHD and a chronic tic disorder were randomized to one of four treatments: clonidine, methylphenidate, clonidine + methylphenidate, or placebo. All three active treatments were superior to placebo on teacher and parent measures of ADHD. In evaluating the pattern of response across treatments, the investigators concluded that methylphenidate had superior effects on attention, and that clonidine had greater impact on impulsiveness and hyperactivity. Thus, the combination of clonidine + methylphenidate provided fuller coverage of ADHD symptoms in this population and produced the largest change from baseline. Tics generally declined in all active treatment groups. For 35% ( $N = 13$ ) of those children randomized to treatment with stimulant alone, however, the increase in tics limited further dose increases.

At doses averaging 0.26 mg per day for clonidine and 25.9 mg for methylphenidate, there were no serious side effects and no evidence of cardiac conduction problems with either drug alone or the combination. Sixty-four percent of the subjects receiving clonidine complained of sedation, with 42% describing moderate to severe problems with sedation. These sedative side effects limited dose increases in over half of the subjects treated with clonidine.

This study is important in several regards. First, it is the first large-scale study to evaluate a combined pharmacotherapy in children with a psychiatric disorder.

Second, it provides valuable efficacy and safety information about a treatment combination that is becoming commonplace in the field. Concern about the safety of this combination heightened following the death of three children being treated with clonidine and methylphenidate. Although a careful review of these cases concluded that neither drug nor the combination was responsible for the deaths of these children, controversy about this drug combination in children continues ([Wilens et al., 1999](#)). Given the sample size, this study cannot answer questions concerning the occurrence of rare adverse events, but the results do provide support for the efficacy and safety of this combined treatment approach. The high rate of sedation associated with clonidine in this sample confirms the narrow dose range of the agent.

Third, this study adds to the list of other recent controlled studies showing that tics do not invariably increase when children with TS are treated with stimulants. Nonetheless, this was a dose-limiting side effect in one-third of the subjects treated with methylphenidate alone.

#### GUANFACINE

Guanfacine (Tenex) is an  $\alpha$ 2-agonist that is similar to clonidine. It was developed as an antihypertensive agent. Interest in guanfacine has been prompted by three observations. First, compared to clonidine, it is less sedating. Second, it has longer half-life than clonidine. This longer duration of action could translate into a need for fewer doses per day and a lower risk of rebound effects. Third, accumulated data from a series of animal studies provides compelling evidence that guanfacine is more specific in action than clonidine ([Arnsten, 1997](#)). Until recently, there were only three open-label studies of guanfacine involving a total of 38 children ([Chappell et al., 1995](#); [Horrigan and Barnhill, 1995](#); [Hunt et al., 1995](#)). All three studies showed promising results for guanfacine in the treatment of ADHD. The study by Chappell and colleagues (1995) also reported that guanfacine had a modestly beneficial effect on tics.

The first placebo-controlled study of guanfacine in children included 34 subjects (17 in each treatment group) with ADHD symptoms and a tic disorder ([Scahill et al., 2001](#)). After 8 weeks of treatment at doses ranging from 1.5 to 3.0 mg per day given in three divided doses, the guanfacine group showed a 37% improvement in a teacher rating of classroom behavior compared to an 8% change in the placebo group ( $P < 0.001$ ). Similar results were observed for teacher ratings of inattention as well as hyperactivity and impulsiveness. Nine of 17 subjects in the guanfacine group were blindly rated as "much improved" or "very much improved," compared to 0 of

17 in the placebo group ( $P < 0.001$ ). Both groups showed modest improvement on parent-rated behavior. The 27% drop in the guanfacine group was not significantly different from the 21% drop in placebo. Children with marked tic severity were excluded from the trial because the target symptoms in this study were inattention, hyperactivity, and impulsiveness. Nonetheless, guanfacine also was associated with a 31% drop in a clinician-rated measure of tic severity, compared with 0% improvement in the placebo group ( $P = 0.05$ ).

Side effects generally were mild. One subject withdrew from the study owing to moderate sedation. Six other subjects complained of mild sedation, which resolved with continued treatment or dose decrease. Other complaints included mid-sleep awakening in three subjects, dry mouth ( $N = 4$ ), constipation ( $N = 2$ ), and loss of appetite in the morning ( $N = 2$ ). Weight was stable in both groups and there were no alterations in clinical laboratory results, including cardiac conduction parameters. The mean resting diastolic blood pressure showed an eight-point decline at midpoint in the guanfacine group that returned to baseline by the 8-week mark. There was no difference between groups on blood pressure measured from the supine to standing position. In a case by case review, six subjects in the guanfacine group showed a one-standard deviation drop (10 mm of Hg) in resting systolic or diastolic blood pressure at one visit, compared to two subjects in the placebo group ( $P = 0.11$ ). No subject showed such a reduction in blood pressure at more than a single visit.

In conclusion, these preliminary results suggest that guanfacine is a safe and effective treatment for children with ADHD and tics. The 37% improvement in teacher-rated behavior is less than the 50% to 60% improvement reported in most stimulant trials, but is similar to the level of improvement observed in other nonstimulant studies ([Biederman et al., 1989](#); [Conners et al., 1996](#); [Feigin et al., 1996](#)). Given that two-thirds of the subjects in the guanfacine trial had failed prior stimulant treatment, the results observed in this sample may not be comparable to the findings of other nonstimulant trials.

#### MECHANISM OF ACTION

The traditional view is that clonidine regulates central noradrenergic activity through its agonist effects on presynaptic  $\alpha_2$  receptors in the LC. Clonidine is indeed ten times more potent than guanfacine in reducing LC firing and inhibiting NE release. In addition to weaker presynaptic effects, accumulated data from studies in primates show that guanfacine has direct stimulating effects on postsynaptic  $\alpha_2$  receptors located in prefrontal cortex (PFC) ([Arnsten, 1997](#)). These animal studies have established that this pharmacologic mechanism improves PFC function. This action is likely to be relevant for the treatment of ADHD given the fundamental role of the PFC in attention and working memory.

#### CLINICAL MANAGEMENT

Clonidine comes in 0.1-, 0.2-, and 0.3-mg tablets. In school-age children, the starting dose is typically 0.05 (one-half of a 0.1-mg tablet) at bedtime. The dose is then increased in 0.05 mg increments every 3 to 5 days as tolerated to a maximum of 0.2 mg per day [e.g., 0.05 mg four times daily (QID)] in prepubertal children, and 0.3 mg in teenagers. To ensure even behavioral effects blood levels across the entire day, clonidine is typically given three to four times per day. Clonidine transdermal patches (Catapres TTS) delivering 0.1, 0.2, and 0.3 mg per day are available, but they have not been well studied in youngsters, and may have erratic absorption from the skin. The typical starting dose of guanfacine is 0.5 mg at bedtime, with 0.5 mg increases every 3 to 5 days to a total of 1.5 to 3.0 mg per day in three divided doses.

The side effects of clonidine and guanfacine are similar, although guanfacine appears to be better tolerated. The most common side effects include sedation, dizziness, irritability (especially when the medication wears off), and mid-sleep awakening. Hypotension is generally not a problem with either drug in children and adolescents, but warrants monitoring, particularly in the dose adjustment phase. A related concern with clonidine is the well-documented phenomenon of rebound hypertension following precipitous withdrawal ([Leckman et al., 1986](#)). In adults with high blood pressure, however, abrupt discontinuation of guanfacine did not result in rebound hypertension ([Wilson et al., 1986](#)). Nonetheless, abrupt discontinuation of either drug should be avoided. There is incomplete consensus in the field regarding the need for electrocardiograms in children and adolescents treated with the  $\alpha_2$ -agonists. A recent statement from the American Heart Association indicates that no cardiovascular monitoring other than blood pressure and pulse is required for clonidine and guanfacine ([Gutgesell et al., 1999](#)).

#### Other Agents Used in the Treatment of Children with Psychiatric Disorders

Many other agents are used in pediatric psychopharmacology. This final section briefly reviews six additional compounds. These drugs are included here because of their potential clinical usefulness, or prompted by the familiar sequence in our field: media clamor about a particular drug based on a case report, which is followed by disappointing results on careful evaluation. This sequence has recently been played out by secretin for the treatment of autism.

#### CLONAZEPAM

Clonazepam (Klonopin) is a long-acting benzodiazepine that is approved as an anticonvulsant. In adults it is also used to treat anxiety disorders and as an adjunctive treatment for tics. A study of 15 prepubertal children showed that clonazepam could be useful in anxiety disorders in some children; however, side effects—including disinhibition, irritability, and drowsiness—were common and problematic ([Graae et al., 1994](#)). Given these results, clonazepam does not appear to be a first-line agent for the treatment of anxiety disorders in pediatric populations, although it can be useful as an adjunctive intervention, especially on a time-limited basis. Treatment can begin with 0.25 mg in the morning and increasing to 0.25 mg twice daily after 3 to 4 days. Thereafter, the dose may be increased slowly to a maximum of 2 mg per day in divided doses. Clonazepam should be tapered gradually when discontinued. As with other benzodiazepines, long-term use can be associated with dependence, albeit less commonly than with shorter-acting agents. Children treated for more than 4 weeks should be gradually tapered when coming off of the drug to limit the rebound anxiety associated with abrupt withdrawal.

#### BUSPIRONE

Buspirone (BuSpar) is an atypical anxiolytic drug that is unrelated to the benzodiazepines. It acts as a 5-HT<sub>1A</sub> agonist, which results in a presynaptic release of serotonin ([Velosa and Riddle, 2000](#)). In an initial study, buspirone (10 to 20 mg per day in two divided doses) was associated with significant improvement in anxiety symptoms, as measured by a global scale ([Simeon et al., 1994](#)). This improvement was evident within 3 to 4 weeks of starting the medication. Side effects included weariness, sleep disturbance, abdominal discomfort, and headache. In a group of 25 hospitalized children with anxiety and aggressive behavior, improvement in anxiety and aggression was noted in about 75% of the cases ([Pfeffer et al., 1997](#)). Approximately 16% ( $N = 8$ ) worsened and four developed hypomania. Another open-label study was conducted in 22 subjects with pervasive developmental disorders with prominent anxiety symptoms. The subjects, who ranged in age from 6 to 16 years, were treated with 15 to 45 mg per day given on a three times per day schedule. After 8 weeks of treatment, 16 of the 22 subjects showed a clinically significant improvement in a global measure of anxiety ([Buitelaar et al., 1998](#)). Side effects included mild activation in two subjects, nausea in one, and orofacial movements in another, the last of which resolved when the drug was discontinued.

The typical starting dose is 5 mg three times per day, with gradual increases weekly to 45 mg per day (15 mg TID). In younger children, buspirone may be initiated at 2.5 to 5 mg per day and increased thereafter every 3 to 4 days to a total of 20 to 30 mg per day in three divided doses. Buspirone should be discontinued in the absence of improvement after 8 weeks.

#### ANTIENURETIC AGENTS: DESMOPRESSIN, OXYBUTYNIN, AND TOLTERODINE

Desmopressin (DDAVP) is synthetic antidiuretic hormone (ADH), a powerful inhibitor of the production of urine. It can be administered orally or via intranasal spray. One review suggested that desmopressin helps approximately 25% of children who use it, with minimal risk of adverse effects ([Thompson and Rey, 1995](#)). The beneficial effects often do not endure over time, although desmopressin is usually well tolerated. The most effective treatment for enuresis is the use of behavioral interventions, such as a “pad and buzzer” or a “moisture alarm.” However, DDAVP can be a useful short-term adjunct, as in the facilitation of sleepovers or overnight camp stays. Antimuscarinic agents occasionally can be useful for longer-term pharmacologic management of enuresis, usually in cases unresponsive or only partially responsive to behavioral interventions. Alternatives to the time-tested use of low-dose (25- to 50-mg HS) imipramine ([Werry et al., 1975](#)) include oxybutynin (Ditropan) or the less sedating tolterodine (Detrol). Beneficial effects usually disappear rapidly on drug discontinuation, as is the case of other pharmacologic interventions for enuresis.

#### SECRETIN

Secretin is a peptide that regulates pH in the small intestine. It is approved only as a diagnostic probe to assess pancreatic function. The dramatic and much publicized response of a single infusion of secretin in a child with autism produced considerable interest and hope for this treatment, but subsequent results from



placebo-controlled studies with pervasive developmental disorders cast serious doubt on its usefulness for this population ( [Sandler et al., 1999](#); [Volkmar, 1999](#)).

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## 78 PSYCHODYNAMIC PSYCHOTHERAPY

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### HISTORY

The monumental work of Sigmund Freud looms so large in the field of dynamic psychiatry that students in the 21st century easily equate Freudian psychoanalysis with dynamic psychiatry. However, it is useful to place psychodynamic psychiatry in a broader context both historically and epistemologically. [Gabbard \(1990\)](#) describes the several uses of the term *dynamic* in the late 19th century by Leibniz, Herbart, Fechner, and the renowned neurologist Hughlings Jackson. In their usage dynamic was contrasted with static. It referred to changing states of consciousness and physiologic processes as opposed to anatomic structures. Dynamic encompassed the concept mental energy. It implied functional rather than fixed organic impairment. In the 20th century dynamic psychiatry—with its emphasis on the patient's subjective experience, the workings of unconscious psychological processes, and the patient's unique inner life—was contrasted with *descriptive* psychiatry, which strove to categorize patients according to observable behavior.

Psychodynamic psychotherapy of children began with [Freud's publication \(1909/1955\)](#) of the case of “Little Hans.” Freud's interest was not the treatment of children but the centrality of childhood experience in shaping the adult psyche. Although he sought to “reconstruct” childhood experience from the analysis of adults, he encouraged his students to observe children for “a more direct and less round about proof” of the existence of infantile sexuality. It was in this context that Freud came to supervise the analysis of “Little Hans” by Hans' physician father.

Hermine Hug-Hellmuth was the first to undertake psychoanalytic therapy of children and adolescents. Her paper, “On the Technique of Child-Analysis” (1921), surveys technical issues in the psychodynamic treatment of children recognized as core issues to this day. She notes that the child does not come of his own accord. She advises against a judgmental stance or the giving of direct commissions and prohibitions. Hug-Hellmuth demonstrates her engagement of the child in “talking over things together.” She introduces the role of an educational method founded on psychoanalytic knowledge. Hug-Hellmuth examined the multiple ways in which work with children and adolescents differs from that with adults, including the complexities of the relationship between the analyst and the child's parents. Her observation that the child's spontaneous play could stand in the place of the verbal communications of the adult to reveal unconscious conflict was most significant for the development of psychodynamic therapy with children. Further, she observed that fully conscious avowal of analytic understanding was not a prerequisite for therapeutic effect in children.

[Melanie Klein \(1932\)](#) and [Anna Freud \(1946\)](#) emerged as the founders of two distinct schools of child psychoanalysis. Klein coined the term “play analysis,” emphasizing the child's play as equivalent to free association in adult analysis. Her methods encouraged early, deep interpretations and minimized the analyst's contact with parents and teachers. Anna Freud's long career from the 1920s to her death in 1982 allowed for significant evolution in her ideas regarding psychodynamic treatment of children and adolescents. Trained as a teacher, she was interested in the “educative” functions of the analyst in addition to the interpretive functions. Her work on ego mechanisms of defense ([Freud, 1936](#)) led to the development of defense analysis of children and adolescents. Her attention to observing the developmental process in children led her to conceptualize interweaving lines of development ([Freud, 1965](#)). Emigrant analysts in the wake of World War II brought both Kleinian and Freudian ideas to America. Child analytic training took root in the United States, allowing child psychoanalysts to play a major role in the training of child and adolescent psychiatrists in psychodynamic therapy.

Two distinctly American therapeutic traditions, admittedly influenced and stimulated by the frisson of psychoanalytic thought, have contributed their own unique emphases to the field of dynamic psychotherapy with children, particularly play therapy. Recognition of the contributions of the dynamic play therapy developed within the child guidance movement ([Allen, 1942](#)) and client-centered therapy ([Rogers, 1951](#)) reminds one not to equate “psychoanalytic” and “psychodynamic.” Homegrown American psychodynamic therapists such as Frederick Allen and Carl Rogers sought to counteract the impact of the American behaviorist tradition. Allen wrote:

The behaviorists advanced the belief that a child with relatively normal equipment could be made into anything parents wanted him to be if they exercised their power, adequately and intelligently, in the first few years of the child's life. The child was seen as little more than a by-product of another's desire. . . . This created a distortion of a parent's sense of responsibility . . . [and a] failure to appreciate the nature of growth and the participation of both child and parent in the child's development ([Allen, 1942](#)).

Allen regarded the child

as an individual who could be helped to grow and to become a person in his own right . . . the child was included as an active participant in the relationship designed to help him. Instead of being seen as an object to be changed, he was accepted as a human being with the capacity to change ([Allen, 1942](#), p.12).

The child's exercise of free expression, a here-and-now focus, and the rapport in the relationship between patient and therapist were emphasized as “nondirective” therapy developed through the work of [Axline \(1947\)](#) and [Dorfman \(1951\)](#).

### DEFINITION

Psychotherapy has been defined as a treatment that ameliorates psychopathologic conditions, functional impairments, and developmental disturbances by means of psychological processes and a therapeutic relationship with a trained therapist ([Gabbard, 1997](#); [Werry and Andrews, 1996](#)). In the treatment of children and adolescents these three elements—psychopathology, psychological processes, and the therapeutic relationship—should be considered in the context of the patient's social environment and ongoing development ([Brent and Kolko, 1998](#)). Psychodynamic psychotherapy aims at bringing about change in psychological processes that are largely unconscious but can be inferred from observable phenomena, such as physical actions or speech acts. The processes determining the inner world of subjective experience are of particular interest. The plethora of unconscious processes of which the human brain is capable allows for great diversity of focus for dynamic psychotherapy.

Psychodynamic therapy capitalizes on the observation that human beings are psychologically changed by relationships with other human beings. Psychodynamic technique utilizes two aspects of human relationships to achieve this mutative effect. Particular attention is paid to *transference*, the unconscious displacement onto the therapist of patterns of feelings, thoughts, and behavior originally experienced in relation to significant figures during childhood. The manifest or “real” relationship between the therapist and patient also is used to further the aims of the treatment.

The psychodynamic therapist conceptualizes psychopathologic conditions, functional impairments, and developmental disturbances in terms of the inferred unconscious psychological processes based on the symptoms and the patient's history. Similarly, social context, and developmental and maturational factors are evaluated for both their role in the etiology of the condition and potential interventions in the social environment that would affect the patient's unconscious, internal psychological functions.



## THEORETICAL BASIS

Psychodynamic therapy requires as a foundation a theory of the functioning of the unconscious mind and the internal experience of the individual as well as a theory of the psychological impact of human relationships. Psychoanalysis provides a rich tradition of observation and theory on which dynamic therapists can draw. Sigmund Freud was a research neuroscientist at the cutting edge of his era before he turned to the clinical practice of neurology and eventually psychiatry. Although he recognized that the research methods of his time did not allow for a full explication of the neuronal base of psychic events, he endeavored to keep his psychological theories consistent with biology. For instance, Freud conceptualized the mind as interplay of excitatory (drive) and inhibitory (defense) phenomena. Greatly influenced by the Darwinian theories of the German zoologist Carl Claus, with whom he trained ( [Ritvo, 1990](#)), Freud sought to construct a developmental framework for his psychology, an approach that put the psychic phenomena of childhood in the spotlight and paved the way for child psychotherapy. Psychoanalytic psychology as a theory of adaptation of the organism to its environment derived from these Darwinian roots, as did the "libidinal drive" as an aspect of the innate striving for survival and reproduction and the concept of conflicting forces within the mind.

[Freud \(1917/1963\)](#) used the Darwinian concept of a *complemental series* to position himself as a centrist in the nature–nurture controversy over the origins of psychopathology with severe constitutional deficits at one end of the spectrum of pathogenic etiology and severely traumatic experiences at the other. The psychodynamic therapist utilizes the complemental series to evaluate the interaction and impact of nature and nurture on the patient's pathology.

In dynamic theory the mind is not seen as a blank slate at birth but rather as endowed with the biological potential to develop psychic structure given an adequate environment. *Internalization* denotes the process by which experiences with the external world, usually in the realm of relationships, form stable intrapsychic structures or capacities. Once a process is internalized it no longer requires an external stimulus for its function to be executed. *Identification*, the psychological process by which one individual becomes like another, is a familiar mechanism of internalization.

Unconscious psychological processes may be considered as primary determinants of the inner world of subjective experience. The individual's total subjective experiential world, including thoughts, feelings, and fantasies as well as perceptions of the external world, regardless of whether they accurately reflect the external world as viewed by another observer, has been designated *psychic reality*. The dynamic therapist attempts to grasp the patient's psychic reality and convey to the patient an interest in understanding his experience of himself.

A primary tenet of psychoanalysis is *psychic determinism* (Moore and Fine, 1990), the principle that nothing in the mind happens by chance or in a random way. All psychic acts and events have meanings and causes and can be understood in terms of psychic earlier events. The mind retains experiences and is shaped by them. Conscious thoughts and overt behaviors provide observable clues to their underlying unconscious psychic determinants. A corollary hypothesis, *overdeterminism* or *multideterminism*, states that a psychic event (e.g., a symptom), is typically caused by more than one factor and may serve more than one purpose in the psychic framework. The multidetermined nature of symptoms provides the psychodynamic therapist with more than one way to approach a symptom. The clinical flexibility is very appealing to the practitioner but frustrating to researchers who seek standardization of method.

The concept of multideterminism is further refined in dynamic theory by the observation that the multiple determinants of a particular observable psychic phenomenon are frequently in opposition or *conflict* with one another. Psychodynamic theorists have identified several types of psychic conflict. *External conflicts* denotes the conflicts that arise between the child and the environment; the consequent frustration demands management by the child's psyche. External conflicts are most evident in early childhood when the infant or toddler has little internal restraint and struggles with the caretaker over such matters as bedtime or playing with an electrical plug. As prohibitions become internalized, the conflicts become *internal*. Not all conflicts are caused by prohibitions. Conflicts may arise between competing urges, thereby creating ambivalence. For example, conflicts arise between urges toward passive-dependence and active-mastery. An internal conflict may or may not become a fully *internalized conflict*. An internalized conflict is conceptualized as continuing in the individual's psyche when environmental forces that triggered the initial internal conflict no longer exist.

Freud realized that a conceptual tool was needed to provide an orienting and systematizing framework for clinical data and hypotheses about unconscious phenomena and subjective experience. He termed such a tool "metapsychology" and offered several different models as he strove to find a conceptual frame that accommodated all his data. The most lasting of Freud's metapsychological frameworks was a structural theory, *the tripartite model*, which divides mental functions into *id*, *ego*, and *superego*. *Id* is a concept that encompasses the mental representations of the instinctual drives ( [Ritvo and Solnit, 1995](#)). *Drive* is a term applied to a stimulus arising within the individual that arouses the mind and incites mental activity. The term *ego* encompasses all the capacities of the mind to manage and channel the arousal and activity incited by the drives. Freud, with an apt metaphor, says of ego

in its relation to the id it is like a man on horseback, who has to hold in check the superior strength of the horse. . . . Often a rider, if he is not to be parted from his horse, is obliged to guide it where it wants to go; so in the same way the ego is in the habit of transforming the id's will into action as if it were its own ( [Freud, 1923/1961](#), p.25).

Superego functions conceptualize the inner voice of conscience, which maintains ideals and values, observing and criticizing any shortfall of the self.

For many students *id* is difficult to conceptualize and to recognize in the clinical situation. "Id" did not make its way into the vernacular to the extent that "ego" did, nor is "drive" or "internal stimulus" as close to a common usage as "conscience," which makes "superego" more accessible to the untutored ear. The internal stimuli that are most familiar to the average person are feelings. Because emotions arise from genetically endowed, physiologic response patterns, they are an early, although in psychoanalytic theory not primary, step in the pathway to arousal and mental activity. In psychoanalytic parlance affects serve a signal function triggering ego efforts to manage drives. The drives themselves may be thought of as genetically endowed motivational states. Commonly we think of these as urges, needs, wishes, and desires.

Advances in neuropsychology and genetics may eventually allow the enumeration of drives on the basis of their genetic and neuronal determinants, but to date the conceptual organization of the drives is speculative and incomplete. The classical Freudian nomenclature works with a dualistic system of an aggressive and sexual drive. The sexual or *libidinal* drive includes affiliate and pleasure seeking urges. A child who enjoys being bathed by the parent and having his or her naked body admired is displaying a derivative of the libidinal drive. The pleasure the child takes from the sensuous experience of the water would also be a libidinal drive derivative. The aggressive drive in its broad connotation includes impulses toward self-assertion and the desire to prevail in one's wishes. This urge to prevail brings forth the more colloquially recognized aspect of the aggressive drive when in the face of frustration the urge to prevail becomes an urge to subjugate or destroy the perceived source of frustration. Pleasure in motoric or mental activity is an aggressive drive derivative at a basic level. Because pleasures sought in response to libidinal drive inevitably run into some requirement for delay or other frustration in the external world, aggressive and libidinal drives are intimately related; ego mechanisms must develop to regulate aggressive drives if libidinal drives are to be satisfied. A clinical formulation in this metapsychological model that failed to assess the role of aggressive drive in symptom formation would not be complete.

Many theorists and clinicians have been dissatisfied with Freud's dual drive approach. When using conceptual models other than the tripartite model they postulate other motivating forces within the individual. For example, [Miller \(1996\)](#), applying Kohut's *self-psychology* to children, proposes three primary drives: the drive toward internal integration, the will to do, and the need for others. Whichever conceptual framework is utilized, dynamic psychotherapy sees the individual's motivations, impulses, and desires as important determinants of subjective experience, mental functioning, and behavior.

In the tripartite model of the mind, ego includes all the mental capacities available to the individual for regulation of the internal milieu and adaptation to external reality. Ego's task is to optimize pleasure and gratification of wishes and needs while maintaining internal equilibrium, the health of the body, good relations with the external world, and peace with superego. Although ego includes all the mental capacities for engaging with external reality, it also focuses on internal reality. As with *id*, there is no single psychodynamic approach to describing or organizing all the phenomena that can be subsumed in the concept ego. One approach defines basic categories of ego functions: reality testing, object relations, regulation of affect, thought, defensive activity, autonomous functions, and synthetic or integrative functions. Work with children requires examining each of these functions in the context of the child's age and developmental accomplishments.

In the broad range of psychological theories, defensive ego activity and the capacity for object relations are conceptualizations of mental functioning most uniquely attributable to psychodynamic theory. *Defense* is a term describing ego efforts to protect against *internal* dangers. From experience with loved persons on whom the child is entirely dependent for survival, the child internalizes the displeasure expressed by the caretaker toward unacceptable drive derivatives, such as biting the nipple when nursing, and identifies with the pleasure expressed toward socially acceptable drive derivatives, such as biting and chewing finger foods. Ego is challenged to restrain prohibited expressions and promote approved expressions of inner urges once this process of internalizing permissions and prohibitions has

begun. Psychoanalytic observations reveal how fear of the disappearance or loss of the loved caretaker, the “object” of object relations, generates a need for defensive maneuvers. The psychological maneuver by which the inhibition is accomplished is termed a *mechanism of defense*. The fear shifts from fear of loss of the object itself to fear of loss of the object's love with increasing ego maturity and development in an environment of “good enough” parenting. Fear of loss of body integrity, often referred to as castration anxiety, enters the signaling system with further development. Finally, as superego functions and ego ideals develop, unpleasant affects of shame, guilt, and remorse stimulate restraint. Intense anxiety and mobilization of powerful and often primitive defenses occur when coping capacities are on the verge of being overwhelmed by massive overstimulation or frustration, for example, with acute or chronic trauma or the upsurge of urges associated with the onset of puberty.

Defense mechanisms are typically unconscious, automatic, psychological processes. The individual does not consciously choose to institute a defense; however, an individual can learn to recognize defensive activity as it occurs through dynamic psychotherapy, psychoanalysis, or self-analysis ( [Gray, 1994](#)). Anna Freud's classic, *The Ego and the Mechanisms of Defense* (1936) enumerated several patterns of defense already in the analytic literature: regression, repression, reaction formation, isolation, undoing, projection, introjection, turning against the self, reversal, and sublimation. To these she added: turning passive to active, denial, intellectualization, displacement, identification with the aggressor, and altruistic surrender. Following that publication, effort was devoted to developing a comprehensive catalog of defense mechanisms. Clinical observation eventually led to the conclusions that any aspect of ego functioning may be used in the service of defense and the attempt to delineate a comprehensive list of specific mechanisms was impossible and potentially misleading in its reductionism. Nonetheless, children use certain mechanisms with sufficient frequency that it is of value to psychodynamic child therapists to be able to recognize them in the clinical situation ( [Table 78.1](#)).

Denial: The disavowal of intolerable external reality factors or of thoughts, feelings, wishes, or needs that are apparent to an observer.
Displacement: The transfer of emotions, ideas, or wishes from the original object to a more acceptable substitute.
Externalization: The attribution of internal conflicts to the external environment and a search for environmental solutions. In therapy the person of the therapist is used to represent one or the other part of the patient's personality structure.
Reaction formation: The adoption of affects, ideas, or behaviors that are the opposites of impulses harbored either consciously or unconsciously.
Repression: The exclusion of unacceptable ideas, fantasies, affects, or impulses from consciousness or the sweeping out of consciousness of what has never been conscious. Repressed material emerges in disguised form in thought, speech, and actions.
Suppression: The conscious effort to control and conceal unacceptable impulses. Suppression is the exception to the rule that defenses are unconscious processes.
Substitution: The transfer of tension from drives or affects into disturbances of bodily functions or rhythms.
Turning passive to active: The management of affects and impulses directed to a passive experience with an object, more powerful "other" (by playing out in action or story the active "other" role. This includes the process of identification with the aggressor.

**Table 78.1. Defenses Commonly Exhibited By Children In Psychodynamic Therapy**

Controversy exists both within dynamic child psychiatry and between dynamic and descriptive psychiatry regarding the degree to which defensive activity and internal conflict involve such “autonomous” ego functions as perception, motility (e.g., walking, eye–hand coordination), intention, intelligence, logical thought, speech, and language. These “autonomous” ego functions were conceptualized by [Heinz Hartmann \(1950\)](#) as relatively resistant to disturbance by intrapsychic conflict. It is clear that many disturbances in this area have their origins in constitutional variations in brain functioning. However, it is also clear from clinical material that in many children and adults these presumed autonomous functions are impacted by conflict and defense; this is no surprise to psychiatrists familiar with the way cognitive function is impaired by defensive denial. The challenge to the child and adolescent psychodynamic therapist is in the differential diagnosis. Psychological testing by a professional skilled in elucidating this distinction can be very useful at times.

Superego, the moral agency in the tripartite model, encompasses conscience, morality, critical self-observation, self-punishment, and the holding up of ideals ([Compton, 1995](#)). Although available to consciousness as moral precepts and ideals, superego functions are predominantly unconscious, out of awareness. Superego is built out of the child's desire to please adults, fear of displeasing adults and thereby losing their approval and affection, experiences of consequences from the physical and social world, and identification with the models of self-control and moral values presented by important adults. There is continuing development of a set of standards, “*ego ideal*,” epitomizing the individual's beliefs of what is right, good, or desirable. Superego only can be effective in controlling behavior in conformance with this ideal to the degree that ego capacities have developed to be deployed in thwarting and channeling unacceptable impulses. When misbehavior is the presenting complaint about a particular child, the clinician must evaluate whether the behavioral expectations of the adults are “age appropriate.” The clinician also pays attention to the meaning of the behavior rooted in the child's anxiety and conflict over drive derivatives.

Superego is a concept that describes an internal, intrapsychic phenomenon derived from object relations and experiences with the external world. The clinician observes superego functions by attending to the affects signaling pleasure and displeasure. When thought and behavior conform to the internalized standards of the developing ego ideal, the child shows pleasure in performance of daily tasks, self-esteem, happiness, or contentment. The child displays signs of humiliation, shame or guilt, and low self-esteem when thought and behavior disappoint the strictures of internalized standards and superego functions. Children frequently externalize blame as a defense against the painful affect when the negative affect or sense of being wrong is unbearable. Unfortunately, when the child lies or blames others, adults may assume that the child does not know he was bad; in these instances, the child knows but cannot tolerate the knowing. Children frequently express their guilt is through enactment in which their behavior brings about a punishment. For example, a child struggling with urges to injure a new sibling may become accident-prone.

An interesting finding from psychoanalysis is that the harshness and rigidity of an individual's superego is not directly proportional to the parent's severity or the child's experiences with the parent but rather to the intensity of the aggressive wishes and relative weakness and immaturity of the individual's ego and defenses. Parental harshness is thought to weaken superego functions because when the parent's aim is to inflict a punishment that hurts the child, either physically or through deprivation, the child becomes focused on the external struggle with the parent and is distracted from the internal struggle with shame, guilt, or remorse.

Although ego psychologists have focused on defensive ego functions, an alternative metapsychology has developed under the rubric of object relations theory. Freud's emphasis on early experience as a source of both psychopathology and transference focused interest on the earliest relationships of the human infant and toddler. This emphasis and the observational studies of young children that it spawned ( [Mahler, 1962](#); [Provence and Ritvo, 1961](#); [Spitz, 1945](#)) have reaped extensive benefit for the mental health of children. Although loosely anchored in the tripartite model of the mind, psychoanalytic object relations theories as defined by [Kernberg \(1995\)](#) are

those that place the internalization, structuralization, and clinical reactivation (in the transference and countertransference) of the earliest dyadic object relations at the center of their motivational (genetic and developmental), structural, and clinical formulations ( [Kernberg, 1995](#), p. 450).

Object relations theories use a metapsychology organized around the concept of an *internal representational world*, “a world which is only gradually differentiated in the course of development as a consequence of processes of biological and psychological adaptation” ( [Sandler and Rosenblatt, 1962](#), p. 130). The child constructs representations that enable him or her to perceive sensations arising from various sources, to organize and structure them in a meaningful way. It is useful to make a distinction between representations and images. A representation is a more or less enduring organization or schema constructed from a multitude of images, each derived from a multitude of experiential impressions. Sandler gives the example of a child who experiences many images of his or her mother—mother feeding, mother talking, mother preparing food, and so on—out of which gradually is created the mother representation encompassing the entire range of mother images, all bearing the label “mother.”

The object relations metapsychological construct of the intrapsychic representational world provides a conceptual framework for the processes of internalization. Identification is the coalescence of a self-representation with an object-representation, or a change in the self-representation so that the object-representation is duplicated. En route to a stable identification are temporary identifications and imitations with transitory changes in self-image. The object-representation used as a model in identification may be based to a degree on fantasy rather than wholly on real attributes of the object from which it derived. Identification with its duplication of the object-representation within the self-representation is seen as a step in the loosening of the dependency tie to the object.

In psychodynamic therapy special attention is given to understanding and manipulating the impact of the patient–therapist relationship on the patient's intrapsychic structures and functions. A basic concept of *transference* “refers to the way in which the patient's view of and relations with his childhood objects are expressed in his current perceptions, thoughts, fantasies, feelings, attitudes, and behavior in regard to the analyst” ( [Sandler et al., 1980](#), p. 78). The therapist can observe the transference by looking for distinctive types: transference of habitual ways of relating, current relationships, and past experiences ( [Sandler et al., 1980](#)). It is



particularly useful to examine and utilize transference onto the therapist of defensive functions. Transference is frequently used as a general term for the patient's attitudes and behavior toward the therapist and as such includes *externalizations*, which [Anna Freud \(1965\)](#) specifies as

processes in which the person of the analyst is used to represent one or the other part of the patient's personality structure. . . . The child thus re-stages his internal (intersystemic) conflicts as external battles with the analyst, a process which provides useful material ( [Anna Freud, 1965](#), pp. 41–42).

Therapists' interventions differ with each therapist's assessment of transference phenomena. [Brenner \(1982\)](#) points out that the phenomenon of transference is not unique to the therapeutic relationship but rather “ every object relation is a new edition of the first, definitive attachments of childhood.” In essence, psychodynamic theory proposes that psychologically significant relationships contribute to the individual's perceptions, thoughts, fantasies, feelings, attitudes, and behavior in all future relationships.

The patient's experience of the therapist is not limited to the transference. A portion of the therapeutic action of psychodynamic therapy is thought to rest in the experience of the *real relationship* with the therapist. The child even more than the adult has receptivity for new experiences. The psychodynamic therapist provides the child or adolescent with a new object with new potentials for internalization. The therapist strives to cultivate specific qualities, some of which may be novel to the child. These include a respectful attitude toward the child, particularly toward the child's thoughts and feelings, as well as the protection of the child's confidences from intrusion by parents or teachers. The therapist strives to be reliable and predictable in the arrangements made for meeting with the child. Most significantly the psychodynamic therapist establishes a relationship with the child that is largely unilateral in its focus so that the events of the therapy evolve primarily from the child. Unlike the relationship with parents or peers in which there is a necessary and healthy reciprocity, the focus on the child in the relationship with the psychodynamic therapist allows the internal world of the child to dominate the transactions ( [Lewis, 1997](#)). The therapist as an interested and thoughtful observer becomes a model for the child's development of the capacity for self-observation, a capacity termed either *observing ego* or *reflective function*.

Although the primary focus in the patient–therapist relationship is tipped toward the patient, psychodynamic theory does address the therapist's experience of this relationship. *Countertransference* refers specifically to intrapsychic conflicts stirred in the therapist by the patient. Generically, countertransference refers to all the reactions of the therapist to the patient. Detection of countertransference requires a “constant internal vigilance of the psychiatrist, who notes the emergence of powerful positive and negative feelings toward the patient and reflects on the possible origin of those feelings in the context of past relationships” ( [Gabbard, 1990](#)). [Marshall \(1979\)](#) operationalizes the definition of countertransference into two categories: (a) reactions arising from the therapist's unresolved internal conflicts; and (b) natural reactions to a patient's provocative behavior. [Winnicott \(1949\)](#) dubbed this second type “*objective*” countertransference, objective in the sense that virtually everyone would find the patient's behavior provocative. The challenge to the therapist when the objective countertransference is recognized is to construct a response that is both honest and therapeutic. Marshall suggests that either type of countertransference may occur consciously or unconsciously. By definition, the therapist is unaware of unconscious self-derived responses. [Table 78.2](#) lists behaviors that signal the presence of unconscious therapist-derived countertransference. Many of these same factors may be purposefully instituted as adaptations of technique, *parameters*, when clinically warranted. The therapist's task is to prevent countertransferences from distorting the treatment ( [Table 78.2](#)).

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Excessive play with diminution of talk
Quick yielding to requests
Gratification of the child, particularly feeding and gift-giving
Any strong feeling, especially if accompanied by guilt or anxiety
“Lulling”: the altering of attention when a child plays out similar fantasies
repeatedly
Impulsive talk or action
Physical contact
Allowing parents to use child's time
Consultation with parents or others without child's involvement or agreement
Strong, unresolved feelings toward parents
Inability to involve parents appropriately
Preoccupation with changing behavior, especially as desired by parents or school

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Marshall R. Countertransference in the psychotherapy of children and adolescents. *Contemp Psychoanal* 15:595-628, 1979.

**Table 78.2. Clinical Clues to Unconscious Therapist-Derived Countertransference**

*Treatment alliance* and *working alliance* are terms used to refer to “all the factors that keep a patient in treatment and which enable him to remain there during phases of resistance and hostile transference” ( [Sandler et al., 1980](#), p. 53). *Resistance* is a conceptualization of the psychological mechanisms that cling to the intrapsychic status quo and seek to prevent change. In essence, resistance is a defense against affects, undesirable self-representations, or unwanted drive derivatives that are stirred and moved toward awareness by the therapeutic process. Inherent in the treatment alliance is the patient's awareness of internal difficulties and an acceptance of the need to be helped. Children with very fragile self-esteem may refuse to enter into therapy because they cannot tolerate the recognition of their difficulties.

In child and adolescent treatments the establishment and maintenance of the alliance is more complex than in adults because of the dependence on parents to bring the child to treatment. A positive alliance with the parents sustains the child's engagement in treatment even when the child's resistance is strong. Parents can subtly oppose the child's wish to come to treatment or collude with the child's resistance. The therapist must establish and maintain a working alliance with the parents sufficient to keep the therapy going.

## CLINICAL APPROACH

The clinical application of psychodynamic theory to the treatment of children and adolescents covers a broad range of settings, participants, intensities, and combinations with other modalities of treatment such as psychopharmacology, educational remedies, family therapy, and environmental change. Individual, intensive, psychodynamic therapy, based largely on psychoanalysis but drawing as well on American client-centered therapy and the child guidance approach to individual therapy, can be used to demonstrate the fundamental techniques and practices of dynamic psychotherapy.

### Indications and Goals

Individual psychodynamic therapy formulates the intrapsychic component of the child or adolescent's condition within a developmentally attuned biopsychosocial model of dysfunction. Formulation goes beyond the simple descriptive categories of the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV) or *International Classification of Diseases*, 10th revision (ICD-10) to include life events and chronology related to the emergence of psychopathology, the pathophysiology of symptoms, signs, and problematic behaviors, and the developmental status, leading conflicts, and coping and adaptive mechanisms of the child ( [Robson, 1986](#); [Shapiro, 1989](#)). Individual psychodynamic therapy is most clearly indicated in cases where the patient's difficulties are the product of intrapsychic conflict and adherence to defensive solutions or object representations that are developmentally regressive. Frequently, a child's difficult behaviors are externalizations of intrapsychic conflict. Psychodynamic therapy is indicated in cases where the etiologic factors in the social environment, such as mother's pregnancy and birth of a sibling or maternal depression over the death of grandparents, have been resolved but the child's defenses have become fixed. [Anna Freud \(1968\)](#) proposes two other categories of disturbance that psychoanalysts have attempted to treat that are not generated solely from the past but from significant present circumstances. One is the child or adolescent's difficulty coping with a current developmental imperative such as entry into school or the upheaval in self-image at puberty and the adolescent shift in relationships with the parents. The focus of therapy would still be on the intrapsychic difficulties of the youth. In Anna Freud's second category the environment or the child's own constitution, such as blindness or multiple learning disabilities, presents a current and ongoing interference with development. In these cases, Anna Freud suggests, intensive individual work may be important but is unlikely to bring the youth to health without environmental interventions.

Intensive, individual psychodynamic therapy is contraindicated in cases of severe constitutional deficits such as autism, severe pervasive developmental disorder, or moderate to severe mental retardation, particularly to the degree that these disorders affect capacity for self-observation and receptive language function. Intensive treatment is contraindicated when circumstances within the family or the treatment facility cannot sustain the continuity and integrity of the therapy, especially if disruption of the therapy repeats previous traumas of loss and unpredictability for the child. Because intensive treatment places considerable strain on family resources and the child and parents' time it should not be undertaken if a less intensive treatment is adequate.

The goals of therapy extend beyond symptom relief. A primary goal is to restore psychological development to a normal path, including management of anxiety, enhanced affect regulation, improved self-esteem, increased frustration tolerance, age-appropriate autonomy, greater capacity for pleasure and satisfaction in school work and play, and better relationships with peers. Additional goals are to enhance the child's resilience and decrease the likelihood of relapse by developing the child's capacity for understanding his or her own feelings, thoughts, and the connection between feelings and behavior. The child or adolescent comes to a more sophisticated understanding of other people's thoughts, feelings, and behaviors by learning to know and observe his own mental functions. This contributes to improved interpersonal relationships.

### Opening Phase

The challenge and gratification provided by the practice of psychodynamic therapy stems in part from the requirement that the practitioner pursue diagnostic and therapeutic goals simultaneously. Thus, the psychodynamic child and adolescent psychiatrist seek to establish a psychodynamic treatment stance from the very first contact with the adult caretaker and patient. Work with children and adolescents has the additional challenge of requiring the formation of a working alliance both with the parents and patient. Although directing and observing the evaluation process, the clinician acts in a manner that respects the parental functions of the caretaker as well as the developmentally appropriate autonomy and inner experience of the child.

### THE FIRST VISIT

With adolescents, particularly older adolescents, the clinician usually meets with the patient for one or several sessions before meeting with the parents. This allows the adolescent to explore issues of trust with the clinician and demonstrates that the clinician's primary relationship is to the adolescent. For younger children, it is typical for the parents to meet with the psychiatrist to provide background data but even more importantly to establish their own trust and confidence in the psychiatrist so that they can assure the child of the appropriateness of this undertaking. It is important to explore with parents how they will prepare the child for the first visit. If the child has expressed distress over a particular symptom, the parent may explain that the doctor is there to help with that symptom. In language appropriate to the child's age, the parents ought to tell the child that the doctor is one who helps children with their feelings and worries by talking and playing with them.

### STRUCTURING THE PSYCHODYNAMIC THERAPY

The intention in individual therapy is for the therapist and child to work together separately from the parents. The child may have difficulty leaving the parent, either because for a preschooler this is within the range of age-appropriate behavior or for an older child because of underlying conflicts. Such separation difficulties require an adaptation of technique and preliminary work with the child and parent to understand and work through the issue. The idea behind working with the child without the parent in the room is to create a zone of confidentiality and psychic safety within which the child and therapist can explore feelings, thoughts, and behaviors. If the parent is present, the child's spontaneity is restrained or stimulated in part by the possible reaction from the parent. When the child is seen alone, the therapist is in a better position to see how the child has internalized the authority of the parents.

The psychiatrist should clarify to both the parents and the child that, although he or she meets with the child's parents on a regular basis, the confidentiality in this therapy is "a one-way street," which allows the psychiatrist to tell the child what the parents have said but precludes the psychiatrist reporting to the parents what the child has said or done. Children, particularly at the beginning of therapy, frequently will not report to the therapist current events that provide a context for understanding the child's talk and play within the session because children are very oriented in the present moment and are defensive against affect. A meeting or phone call from the parents is important if the therapist is to know about these events. The "one-way street" implies that the therapist will share these communications from the parents with the child. The child's understanding of the purpose of the therapist's meetings with parents develops as the therapy continues. It is the therapist's task through word and deed to help the child understand that, although the therapist tries to help the parents understand their role as parents of a child this age and with these problems, the therapist can not control the parents and will not take sides in a specific struggle between the child and parents. On the other hand, particularly with older children and adolescents, it can be pointed out that the parent has a responsibility to the child for assuring that the therapist is doing a responsible job and thus needs some report from the therapist on the progress of the work.

There are several other elements the psychodynamic therapist uses to create a special person, place, and time for dealing with feelings and worries in addition to creating a zone of confidentiality. The therapist deals with the child in a nonjudgmental and nondirective manner such as that described by [Virginia Axline \(1947\)](#) in her classic text, *Play Therapy*. In an intensive individual psychodynamic therapy, the therapist strives to relate to the child in the style [McConville \(1998\)](#) has described as the "empathic participant" who approaches the child with the wish to know "what's it like to be you." The therapist and child explore together rather than doing something *to* or *for* the child. Eventually the therapist is *with* the child as the child examines and explores his own thoughts, feelings, and conflicts. If doctor-patient relationships are conceived of as either prescriptive, collaborative, or facilitative the psychodynamic therapist eschews the prescriptive, choosing to be collaborative and facilitative.

The office must be suitably sturdy and equipped in a manner age-appropriate to the patients who will be treated in order to work nondirectively with children. A separate playroom is advantageous when working with young children who are struggling to control intensely messy or aggressive impulses. The toys do not need to be numerous or elaborate. Children's activities in a play therapy tend to fall into the following categories: games with rules, physical activities, creative projects, solo imaginary play, and imaginary play with the therapist as a participant. It is helpful to have a deck of cards, one or two simple board games such as checkers or Trouble, a Nerf or koosh ball, paper, washable markers or crayons, tape and children's scissors, a set of blocks, puppets, action figures, and dolls or animal figures. A family grouping of animals or small dolls is useful. The child can use a ferocious animal (e.g., shark, lion, or dinosaur, either a puppet or animal figure) to express aggressive urges.

The issue of limit setting will arise no matter how appropriately the office or playroom is outfitted. Common-sense limitations are necessary to protect the room, therapist, and child from physical damage. One way to understand the limits is to say, "When we are done I have to be able to clean up and have you, me, and the office/playroom back the way it was." This creates in the child's world of expression through action the same safety from real-world consequences the adult analyst on the couch enjoys when expressing himself or herself verbally. Frequently, the therapist is able to address the impulse toward an unacceptable action while redirecting its aim. Thus one might say, "You may not cut my dress when you feel like attacking but you may cut this paper I will hold. I can see you feel strong and safe when you are doing the cutting. You let me know how scary it feels to have someone attacking." The therapist tries to prevent the dangerous action without creating a physical struggle if the child cannot be redirected. It is important that the therapist not become angry, blaming, and punitive while commenting on the child's struggle over the destructive wish and maneuvering of the therapist into being the "policeman who must keep us and the office safe." It is also appropriate for the therapist to remind the child that "We must try to find words for these feelings so you can tell me about it without things getting broken." The implementation of a set starting and ending time is the most frequently imposed limitation, as [Axline \(1947\)](#) points out. The therapist's adherence to routine in keeping the time and dates of appointments on a predictable schedule is a necessary part of creating reliability and boundaries in the therapeutic relationship. The child's reaction to waiting in the waiting room for the appointed starting time and dealing with leaving the therapist at the end of the session can serve to reveal the particular child's conflicts.

### THE INTERPRETIVE SESSION: STRUCTURING THE WORK FOR THE PARENTS

Intensive individual psychodynamic therapy is initiated after a period of evaluation, crisis management, or even pharmacotherapy. It is important that the therapist and parents meet in an interpretive session or two to review the formulation of the child's diagnosis and prognosis in dynamic and developmental terms, and to discuss the recommendation for intensive treatment and necessary arrangements. Intensive therapy implies two or more sessions per week. Psychoanalysis with sessions four or five times per week is indicated when the pathologic conflicts and maladaptive, regressive defenses are longstanding and pervasive in the child's response to a wide range of circumstances. Parents understand that frequency of practice enhances the outcome and shortens the overall time course in order to acquire a foreign language or master a musical instrument. In intensive psychodynamic therapy the child undertakes to learn a new "psychological language" and develop new skills to master feelings and impulses. The patient must be helped to counteract the resistance to change that is a natural mechanism for maintaining stability. The wise therapist is cautious in predicting the duration of treatment. Much depends on the stability of the environment and degree of current destabilizing stresses. What are the burdens and constraints on the parents in terms of effort and expense that might limit the duration of treatment? Parents should be told that intensive individual psychodynamic therapy is generally measured in years, not weeks or months. Shorter intense treatments are adaptations to specific circumstances undertaken after weighing the risks and benefits over other treatment modalities.

The frequency and purpose of parent sessions also must be discussed. As third-party coverage for intensive psychotherapy has diminished in the past 20 years, the tendency has been to decrease the parent visits in order to maximize the resources available for the child's visits, a short-sighted solution, especially with younger children. The parents and therapist need to meet to maintain an alliance and avoid undermining splits and competition for the child. Helping parents to make changes



in day care, bedtime rituals, family modesty, and discipline facilitates recovery of a healthy developmental trajectory more rapidly in the younger child.

### *BEGINNING THE PROCESS OF INTERPRETATION*

Interpretation is the process whereby the therapist, by expressing in words what he or she has come to understand about the patient's mental life, helps the patient to observe and understand his or her inner life in a new and more complete way (Moore, 1990). The therapist engages the child or adolescent in the act of observation by making *attention statements* (Lewis, 1974), comments that draw the patient's conscious attention to the content of his actions or verbalizations (Chapter 79). Beginning with ego syntonic and nonthreatening content, the therapist provides verbal commentary on the child's play, reflecting back the child's anxieties, judgments, or wishes. As the therapeutic alliance develops, the therapist begins to draw the patient's attention to coincidences, paradoxes, and remarkable absences of affects, topics, or persons who ought to be central to the child's experience.

### **Middle Phase**

Whereas the initial and termination phases may last several weeks to months, the middle phase of treatment lasts months to years. Intrapsychic structure is created or remodeled, new defenses are developed, and old patterns of response are relinquished during the middle phase.

### *THE CHILD IN THE MIDDLE PHASE*

The middle phase begins when the patient has taken in the structure of the treatment: the therapist's nonjudgmental attitude, regularity of appointment times, and maintenance of confidentiality. The patient implicitly knows that a goal of the treatment is to understand the way she or he feels and behaves, that everything she or he says or does is to be considered in the therapy. The child's play or the older child's conversation and behavior are part of the associative process revealing the patient's intrapsychic state. It does not meet the criteria of free association in adult psychoanalysis because the child's capacity for introspection is limited and the child is not instructed to say whatever comes to mind. The child or adolescent tends to maintain a consciously goal-directed stance (e.g., winning the game of checkers). This goal-directedness creates considerable tension between the child's tendency to become absorbed in the play and the capacity to self-observe. The therapist's verbalizations about the play model the observing function as well as clarify the dynamic functions of the play for the child.

The transition in late childhood and early adolescence from playing in therapy to sitting and talking reflects a developmental move from expression through action to enhanced verbal communication. Superego development contributes to this process with increasing superego autonomy and constriction in primary process expression to meet an increasingly mature ego ideal. Often in this transitional stage the patient will fiddle with objects in the office; draw or play cards; or bring in homework, magazines, books, or a pocket full of gadgets to allow an action outlet or focus that is not threatening in its pull back toward childhood play. In this period the therapist needs to allow the patient to establish a comfortable blend of action and verbal expression while being alert for the ways in which either might be used as a resistance.

Resistance is an inevitable aspect of a psychodynamic therapeutic process. Lewis and Blotcky (1997) describe three categories of resistance: active, passive, and compliant. The actively resistant patient expresses overt objections to the treatment, such as opposition to coming to appointments and complaints about interference in his or her life. The passively resistant youth may be stubbornly silent, withholding, and plead boredom. The compliant patient is described as eager to do the right things, to give "lip service" but without emotional engagement. These may be thought of as "macroresistances," appearing as an overarching response to treatment. One also finds "microresistances," moment-to-moment rejections of the therapist's offered interpretations. The child may demonstrate these by shutting down, a sudden shift of focus or behavior, or an eruption of disinhibited behavior. By observing these "microresistances," the therapist can measure the intensity of the superego in opposing awareness of the mental contents the therapist was seeking to bring to the patient's conscious awareness or the weakness of the ego when it loses control of the drive.

The patient continues to develop a relationship with the therapist during this middle phase, in addition to bringing forward the content of his inner experience. A true transference occurs, that is, the patient experiences in the current relationship with the therapist fixated conflicts, superego projections, and object representations that stem from previous experiences with the parents. The transference is affected and modified by the developmental achievements or delays of the patient. The child patient differs from the adult patient because the child is still living with the parents, who continue to exert influence over the patient. The patient may make a simple, current displacement rather than a deep transference to the therapist. The child is not the equal of the therapist because the child is still a child relating to the therapist as a child to an adult (Lewis, 1996) (Chapter 79). In addition to the emerging transference, the child or adolescent experiences the therapist as a new object for identification and satisfaction of developmental needs. The experience with an adult who respects the child or adolescent's inner life and demonstrates an approach to understanding and managing that inner life contributes significantly to the therapeutic outcome of intensive individual psychodynamic therapy.

It is important not to underestimate the contribution to the therapeutic process of the child's play and verbal self-expression, although the therapist is essential to the therapy. The play and verbalizations by expressing heretofore-unconscious mental contents in conscious communications of speech and action offer an opportunity for active mastery of components of the patient's problems apart from the specific interventions of the therapist.

### *THE THERAPIST'S MENTAL PROCESS*

The gap between the therapist's theoretical understanding of the moment-to-moment content of the therapy sessions and what it is possible to do or say at the developmental level of the child is more evident in child psychotherapy than in work with adults. Lewis (1996) describes the multistep process by which the therapist, while keeping up the play with the child, takes "mental distance," places the immediate observations into an ongoing formulation, develops an interpretation, brings the interpretation back, and presents it to the child in the context of the play and session (Chapter 79).

The therapist's interest is neither limited to nor primarily focused on the factual content of the patient's productions in the session. Even when a patient in late childhood or adolescence presents a narrative of an external event, the therapist listens for the defensive regulation of affect and drive in the narrative as well as the accuracy or potential distortions in the presentations of relationships. The therapist also assesses the patient's ability or inability to see multiple sides of the story. Referencing earlier material, the therapist considers what patterns emerge. The therapist may begin to formulate a reconstruction by thinking about how these patterns reflect a repetition of defenses and attitudes fixated by past trauma. The psychodynamic therapist continually monitors the patient's utilization of the therapist. Is the transference being manifested or is the patient turning passive to active, letting the therapist know what it feels like to be on the receiving end? Nonnarrative material is as useful in psychodynamic therapy as is narrative. In nonnarrative play the therapist observes where shifts and disruptions in the play occur in response either to frustration or the emergence of strong emotions or thoughts the child finds unacceptable. These observations inform both the content of the therapist's interpretations and their timing.

### *THE THERAPIST'S INTERPRETATIONS*

The basic process of constructing interpretations was laid out by Loewenstein (1951) and is explored comprehensively by Lewis in Chapter 79 of this volume. Interpretations may be classified as *clarifications*, *defense interpretations*, and *reconstructions*. Clarifications bring the patient's attention to bear on his or her characteristic patterns of action and interaction but do not imply a reason for the pattern. A defense interpretation picks up on the drive derivative (e.g., rivalry as an aggressive drive derivative) and points out that a defense has been instituted. The drive derivative has been changed in a way that makes it manageable and acceptable to the superego (e.g., rivalry becomes the avoidance of competition). The therapist should address the defense before addressing the drive derivative when making a defense interpretation (Freud, 1936). The defense is acceptable to the patient. It is ego-syntonic. Drawing the patient's attention to the defense reminds the patient that he or she has a way to manage the affect or drive derivative. The patient will be more likely to tolerate looking back at the intolerable drive or affect from this position of strength.

A reconstruction is an interpretation that explains current feelings, thoughts, and behaviors in terms of critical past events in the patient's life or fantasies from an earlier stage of development. Reconstructions are only offered late in the therapy process. The clinical decision to offer a reconstruction to a child or adolescent requires weighing the patient's capacity to use it to "make sense" of perplexing feelings and behaviors (Kennedy, 1971). Lewis (1996) points out that a reconstruction can be helpful to a child when it clarifies what was an essentially correct perception by the child at the time of the trauma but that has subsequently undergone distortion (Chapter 79). The therapist must consider whether a reconstruction is premature. A child too close to the trauma will reject the reconstruction in order to defend against the affects and drives the reference stirs. A reconstruction may be intellectualized or rejected because it arouses unmanageable shame or guilt.

The psychodynamic therapist must select the optimal context in addition to the level of interpretation. In play therapy the therapist decides whether to work within the

play utilizing the defensive protection displacement affords or to comment more directly to the child. If the child is playing that a tiger makes a preemptive attack on a lion, the therapist may speak about the tiger's wish to protect himself by striking first or the therapist can say to the child, "You are showing me with the tiger that you feel it is sometimes important to protect yourself by attacking first." The relationship between the child and therapist is a context for interpreting transference or externalization, clarifying the real, nontransference, relationship and offering developmental assistance. Working in the play or in the patient-therapist relationship focuses the work on events inside the treatment setting. The focus could be extended to events outside the treatment setting. The patient and therapist can discuss issues or experiences with family, peers, or school. Finally, the temporal focus may be on the past, present, or future.

#### WORK WITH PARENTS IN THE MIDDLE PHASE

Success of an intensive individual psychodynamic therapy with a child or adolescent often rests on the therapist's skill in the collateral work with the parents. The therapist must come to understand the parents' fantasies and fears about therapy, their child, and themselves as parents. Parents may come with the attitude that the therapist will redeem them from past transgressions that have affected the child. The parents may hope that the therapist will rescue the child. Alternatively, parents may see the therapist as a rival for the child's affection or as an authority figure seeking to find fault or blame the parent. Even with the most dysfunctional parent, the therapist seeks to ally with the parent's wish to be a good parent and supports the parent in those efforts. Many parents find referral for individual treatment beneficial. Where there is marital conflict the therapist tactfully explores its role in the child's difficulties. If marital therapy is indicated the parents should be referred to an appropriate clinician in order to maximize the parents' effectiveness in their parenting role, minimize the possibility that the marital conflict will disrupt the child's treatment, and protect the therapist's primary relationship with the child.

In "Problems of Termination in Child Analysis," [Anna Freud \(1957/1971\)](#) lists parental reasons for terminating treatment sooner than the Hampstead Child-Therapy Clinic recommended. Parents may be satisfied when presenting symptoms have resolved but the therapist is aware that the underlying pathologic conflicts are not stabilized. Or the parents find unacceptable transitory oppositional behaviors of the child whose therapeutic developmental gains lead to a newly achieved autonomy.

Parent work in the middle phase has the tasks of: (a) facilitating the flow of information from the parents about the child; (b) counseling and educating parents both about sensitive issues in parenting and the particular child's diagnosis, strengths, and vulnerabilities; and (c) assisting parents in advocating for the child and in case management ([Sperling, 1997](#)). The therapist must always plan interventions with parents in the context of what the therapist knows of the parents' own issues and needs. Suggestions must be given with cautious awareness that parents may resist giving up a parenting behavior or pattern of relationship with the child either because it is gratifying for the parent or represents a parental defense against negative or aggressive impulses. If such resistances are present or the parent's own conflicts can be shown to destabilize the child's progress in the therapy, the therapist has the additional task of skillfully assisting the parent to undertake therapy for herself or himself.

#### Closing Phase

The closing phase presents two distinct tasks: the decision to conclude the treatment and the *termination process* by which the treatment is ended. The psychiatrist's ability to maintain the working alliance with the patient and parents consolidates the patient's therapeutic gains and enables the patient or family to seek help in the future if needed.

#### THE DECISION TO STOP TREATMENT

Because intensive psychodynamic therapy with children and adolescents is a three-way contract ([Novick, 1990](#)) between therapist, child, and parents the conclusion may be initiated by any one, two, or all three of the parties. The optimal situation is for all three to concur that the goals of treatment have been met. A successfully completed case may be said to have "reached termination."

Premature terminations occur when there is a decision to end the therapy before the treatment goals have been met. There are two types of premature termination: disruption and interruption. Disruption refers to the decision to end the treatment because of external factors affecting any of the three parties. For example, the family may be moving or the therapist may be leaving the treatment facility. Interruption refers to a unilateral decision by one of the parties to end the treatment based on intrapsychic factors such as parents' unexamined intrapsychic conflicts aroused by the therapy. In all terminations, and particularly in the case of premature terminations, the therapist must be alert to the patient's fantasies about the reasons for ending the treatment so that they can be worked on during the termination process.

In initiating the termination process, the therapist considers the patient's ability to maintain the gains of treatment and grow in the family despite parental difficulties, the extent to which "developmental forces have been set free again and are ready to take over" ([Freud, 1957/1971](#)), and the progress the patient has made in internal consolidation of intrapsychic changes. The process of consolidation of intrapsychic changes is called *working-through* ([Chapter 79](#)).

[Sigmund Freud \(1914/1957\)](#) introduced the concept of working-through to describe the time and effort a patient requires to "become conversant with his resistance." Anna Freud describes the three major therapeutic agents of differing psychodynamic therapies in published discussions with Joseph Sandler. Abreaction is the primary therapeutic agent in client-centered play therapy. Franz Alexander's "corrective emotional experience" provides the child with a new relationship that replaces deficient past relationships. Psychoanalysis strives for a therapeutic process based on self-observation, self-awareness, and insight. Insight, according to Anna Freud, "does not occur without a working-through process," defined as the elaboration and extension into different contexts and directions of relevant interpretations. The tracing of anxieties resulting from the child's infantile perception of the world and correction of distortions of perception that were caused by the immaturity of the child's cognitive apparatus are included in her concept of working-through ([Sandler et al., 1980](#)).

#### THE TERMINATION PROCESS

All psychotherapies have a termination phase regardless of theoretical orientation ([Lewis and Blotcky, 1997](#)). Psychoanalysts give particular attention to this phase of treatment. The understanding gained of the meaning and psychological challenges to the patient inherent in "saying goodbye" are useful to the clinician in managing the conclusion of any doctor-patient relationship.

At the theoretical level, termination is conceptualized as an experience of separation and loss. The patient, parents, and therapist bring their own characteristic approaches to separation and loss to the process. Although the therapist's attention is primarily on the patient, countertransference reactions may be triggered by the loss of the patient as a real-life object. It is essential to evaluate and address the parents' needs in the face of the impending loss of the therapist as an ally.

The intrapsychic tasks for the patient are the resolution of the transference relationship and relinquishing of the real relationship with the therapist. The transference reaction to termination is colored by the patient's previous experiences with separation and loss. Ego functions may regress and symptoms recur. The therapist works to help the child verbalize feelings and ideas about the impending change and work through these reactions in light of the patient's current capacities, those appropriate to the patient's developmental level and those gained in therapy. As the patient relinquishes the real relationship with the therapist, the therapist aids the patient to internalize the therapist's functions so that the patient may carry on in developing self-understanding, particularly an ability to tolerate and examine internal conflicts and ambivalence.

The technical and practical approaches to termination are designed to promote the theoretical goals. The therapist engages the patient in *active* planning and execution of the termination seeking to avoid repetition of *passive* experiences of loss (e.g., loss of parental attention with the birth of a sibling). First, the reasons for concluding the treatment must be acknowledged and the treatment goals, child's achievements, and remaining concerns reviewed allowing the clinician, patient, and parents to gauge the patient's reaction to the impending loss. Typical reactions of children and adolescents faced with the proposition of ending therapy are: (a) fear, anger, aggression, or depression; (b) return of symptoms; (c) recapitulation of the themes of the therapy; (d) adoption of the "plan" presented; and (e) a bid to not end the therapy ([Lewis and Blotcky, 1997](#)).

Next, logistics of termination are addressed. Logistics include setting the date for the last session, deciding whether to make a clean break or a gradual diminution in the frequency of sessions, and clarifying follow-up plans. General guidelines for these decisions are tailored to the needs of the particular patient.

The clinician works to avoid precipitous endings without enough time to say goodbye and work through the patient's reactions. Optimally, the patient chooses the date by working with the therapist to understand why the patient feels one date is more suitable than another. A date at a natural time of separation, such as the end of the school year or the Christmas vacation, tends to down play the reality of the termination. This may be desirable in some circumstances but may compromise a full



working-through of the termination.

Following the model of adult psychoanalysis, many clinicians assume that therapy should be continued at the established intensity until the final session and stopped with no planned follow-up or contact. Anna Freud has seriously questioned this approach with children and adolescents ( [Sandler et al., 1980](#)). She observed that, when normal development is achieved, the child detaches himself or herself in the course of time, just as children move on from teachers and even friends. Therefore, she recommends allowing the child to reduce the frequency of visits or schedule a follow-up visit.

Classical adult psychoanalysis leaves follow-up contact up to the patient. Early follow-up is regarded as evidence of the patient's failure to resolve the transference relationship. With children and adolescents who are in psychotherapy, not analysis, one must be aware that one is working two steps removed from classical adult analysis. In conditions of significant ongoing pathology, either constitutional in the child or endemic in the family, one is well advised to establish a follow-up plan. Even in less severe cases Lewis and Blotcky point out

the clinical utility of follow-up is probably under appreciated. The follow-up process can reinforce treatment gains, provide objective assessment of any possible deterioration, sustain some continuity in the important therapist-patient relationship, and provide valuable communication between parents, clinicians, teachers and so on ([Lewis and Blotcky, 1997](#), p. 167).

All agree that it is important for the patient to feel free to return.

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## 79 INTENSIVE INDIVIDUAL PSYCHODYNAMIC PSYCHOTHERAPY

### The Therapeutic Relationship and the Technique of Interpretation: The Use of Play in Psychodynamic Therapy

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#### GOALS

This chapter describes the clinical process of interpretation in psychodynamic psychotherapy with children. Hypotheses concerning interpretation are mostly derived from descriptive accounts of clinical observations, few of which have been tested scientifically. Some empiric research on psychodynamic psychotherapy treatment outcome has been reported ([Target and Fonagy 1994, 1997](#)), providing evidence of improvement along various dimensions.

Reasons for this paucity of scientific validation include the complexity of the therapeutic relationship, issues of privacy and confidentiality, informed consent and ethical issues, costs of attempting such research, difficulties of controlling contingent variables, myriad of individual differences, difficulty of collecting a large enough number of subjects, and problems of matched controls. Some of the difficulties of even routine monitoring of the effectiveness of child psychotherapy are sometimes daunting ([Weiss, 1998](#)). Not surprisingly, the desire to undertake such research leaves many therapists ambivalent if not overwhelmed; hence, the paucity of scientific research in this area.

Having noted these difficulties, what can be said clinically about the technique of making an interpretation that might be useful to the clinician and helpful for the patient? The clinical goals of intensive individual psychodynamic psychotherapy may include reduction of anxiety, improvement in self-esteem, increased frustration tolerance, disappearance of symptoms, better coping strategies, appropriate independence, good relationships with peers and adults, satisfactory and satisfying schoolwork, feelings of pleasure and joy, and a sense of resumption of development.

#### HISTORICAL NOTE

Hermine Hug-Hellmuth (1871–1924), in 1920, was the first child analyst to describe a technique of child analysis, especially in the preparatory phase at a presentation she gave at the Sixth International Psychoanalytic Congress in the Hague. The paper was entitled "The Technique of Child Psychoanalysis," and was published subsequently in the *International Journal of Psychoanalysis* (1921). More recently, her paper was republished in a volume by [MacLean and Rapper \(1991\)](#). Also, a useful and lively exchange between Anne Polzer, a reviewer of MacLean's book, and the author, George MacLean, is available (Polzer and MacLean, 1992). Melanie Klein, in Berlin, was the first to develop the concept of play in therapy and made extensive use of play with interpretation, using a wide range of small toys and wooden human figure representations. Melanie Klein read her first paper in Budapest in 1919 ([Klein, 1920](#)). David Levy presented the first American report on play therapy in 1925 ([Harrison, 1980](#)). Later, in 1927, Anna Freud repeated Hug-Hellmuth's earliest account of the elements of child analysis ([MacLean and Rapper, 1991](#)). Meanwhile [Freud, in 1923](#), had stated the essential goals for treatment: "to make the best of him [or her] that his [or her] inherent capacities will allow and so to make him [or her] as efficient and as capable of enjoyment as is possible" ([Freud, 1923/1955](#)).

#### GENERAL CONSIDERATIONS

The general substrate for the method essentially involves a trusting, confidential, real relationship between a trained, motivated, caring, and accepting person who offers help to a person who needs that help. Usually there is agreement between therapist and parent about the type of theory used. Generally, opportunities are provided for the verbal expression of feelings, increasing self-knowledge, and improving self-mastery ([Karasu, 1977](#); [Langs, 1982](#); [Paolino, 1981](#)). The therapy is usually divided into periods of 30 to 50 minutes given from one to four times a week in a suitable office setting. Although numerous methods, techniques, and theories have been categorized ([London and Klerman, 1982](#)) and described ([Kazdin, 1988](#); [Varma, 1974](#)), unequivocal superiority of any one method has not been established ([Luborsky et al., 1975](#); [McDermott, 1977](#); [Schaefer 1977](#); [Wolman et al., 1978](#)).

#### BECOMING A CHILD THERAPIST

How does one become a child therapist? First, it has to be said that neither the amount nor kind of training are very reliable guides to the eventual effectiveness of a therapist: Highly trained psychotherapists often achieve no more with their patients than do those with much less experience ([Strupp and Hadley, 1979](#)). Victor Raimy once observed that "psychotherapy is an undefined technique applied to unspecified cases with unpredictable results. For this technique, rigorous training is required" (quoted in [London, 1964](#)). Supervision and continuous case conferences with acknowledged experts are the generally accepted methods of acquiring psychotherapy skills. The amount required varies with the endowment and needs of the individual psychotherapist. In a brief (8-month) longitudinal study of 12 beginning psychiatric residents, [Buckley and colleagues \(1982\)](#) found that appropriate use of clarification, confrontation, management of resistance, and the ability to deal with negative transference could be learned with supervision and experience, whereas the capacity for empathy and awareness of countertransference did not change during the period of the study ([Buckley et al., 1982](#)). There is some evidence that ordinary communication skills and empathic understanding can be taught very quickly ([Bird, 1980](#); [Ivey, 1980](#); [MacGuire, 1980](#); [Matarazzo, 1978](#)) and that the rest is experience. There is no evidence that a personal analysis must be part of psychodynamic psychotherapy training ([Marks, 1982](#)).

Specialist training in psychotherapy probably requires more time if more than one modality is being learned. [Bende and Crossley \(2000\)](#) believe that a curriculum and practicum for different therapeutic modalities, one of which would be the main modality, requires 3 years. However, mixing various therapeutic approaches during specialist training can be confusing and even "de-skilling," and may lead to therapist uncertainty in regard to one's own style ([Monaghan and Mooney, 1999](#)). It is worth noting that the single, focused learning over an extensive number of years that is required for practicing psychoanalysis does provide the psychoanalyst with a clear identity as an analyst, the acquisition of a set of specialized skills, and the opportunity to develop one's personal style.

Although a working knowledge of several different therapeutic modalities is clearly required in making an informed referral of a patient with a particular set of problems to a therapist who is a specialist in the form of psychotherapy that would be most appropriate, data on the outcome in terms of such referrals are not available.

Still, therapists who complete the initial evaluation are eventually in the position of having to make an informed decision regarding which kind of therapy is best, and who would be the appropriate therapist to undertake that treatment. More research is needed on this problem ([Seligman, 1995](#)).

## INDICATIONS

The question of when intensive individual psychodynamic psychotherapy in childhood is the treatment of choice nowadays often resolves into the question of to what degree multiple other forms of therapy are indicated and available for a particular child with a particular diagnosis. Diagnosis thus is an essential prerequisite and for this purpose must go beyond the categories of *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) ([American Psychiatric Association, 1994](#)) or ICD-10 to a comprehensive developmental formulation that provides a useful basis for treatment planning ([Shapiro, 1989](#)).

Rarely does a single form of psychiatric treatment suffice for the child. In most instances, various combinations of treatment modalities are necessary, including individual psychotherapy, pharmacotherapy, behavior therapy, family therapy, educational remedies, counseling, environmental changes, group therapy, and concomitant work with the parents. For example, multimodal treatment has been found to bring about significant improvement in children with attention deficit hyperactivity disorder ([Satterfield et al., 1980](#)). Nevertheless, when psychodynamic psychotherapy is used, the commonly accepted principles of psychodynamic psychotherapy are operative no matter what other treatments are required.

Pure, solitary, intensive, individual psychodynamic psychotherapy for the child alone is indicated with confidence only when the child's problem is primarily intrapsychic and there is no other known factor amenable to treatment of any other kind. Such is rarely the case. When the child's problems are a combination of external and internal or internalized processes, and other important etiologic factors are recognized, as is often the case, intensive individual psychotherapy becomes one component of a broader treatment plan. Thus, there also may be present various ongoing current conflicts, frustrations, deprivations and losses, serious physical illness, other psychiatric diagnoses, and complex neurophysiologic disturbances that may require the addition of one or more of the previously mentioned interventions.

## INTERPRETATION AS AN ESSENTIAL ELEMENT

No matter what accompanying treatments are necessary, when individual intensive or psychodynamic psychotherapy is used, the same essential elements of the process unfold, albeit modified by the age, diagnosis, and presence of these other factors, with or without the other forms of treatment. [McDermott and Chan \(1984\)](#) described the basic requirements for psychodynamic psychotherapy for children, together with a broad description of the stages and various components of the ongoing clinical work; and [Dulcan \(1984\)](#) described brief psychotherapy with children, adolescents, and their families. This chapter focuses primarily on the particular essential elements of interpretation in intensive individual psychodynamic psychotherapy.

Intensive individual psychodynamic psychotherapy is based largely on psychoanalysis, which specifically places great importance on interpretation (making the unconscious or preconscious conscious), particularly interpretation of the transference, as the principal therapeutic agent ([Lewis, 1974](#)). This holds true whether one is treating a preschool child ([Neubauer, 1972](#)), a school-age child ([Freud, 1946/1950, 1968](#)), or an adolescent and across a wide range of diagnoses ([Witmer, 1946](#)).

More recently, [Bleiberg and colleagues \(1997\)](#) conceptualized and described "mentalization" as an initial and even key element in psychoanalytic treatment and psychodynamic psychotherapy with children. They described how to help the child enhance his or her capacity to become aware of and monitor his or her mental states and the mental states of others, thus enabling the child to manage better his or her behavior. In short, the child is helped to activate his or her ability to find meaning in behavior—his or her own behavior and the behavior of others. Classical interpretation of the unconscious as described in the following, may then follow.

Cognitive perspectives also inform psychotherapy, and some mention of cognitive-developmental and information-processing considerations are made in the sections that follow. (For a more comprehensive account of cognitive development and clinical applications, see [Chapter 12](#).)

For purposes of discussion, intensive individual psychodynamic psychotherapy can be approached as a process with three major phases: initial phase, middle phase, and termination phase.

### Initial Phase

#### *THERAPEUTIC ALLIANCE*

The major goal of the initial phase is to foster a therapeutic alliance between therapist and child. This goal is usually achieved by enabling the child to experience a nonjudgmental, understanding response to his or her behavior. The actual relationship is an important therapeutic agent in its own right, as well as the soil in which the seed of an interpretation may flourish; therefore, a positive actual relationship is encouraged.

To foster a therapeutic alliance, the child must first be given some understanding by the parents of why he or she is being brought to a therapist and what therapy is like. Essentially, the child can be told that, in addition to himself or herself, others such as parents or teachers are concerned about how the child is feeling and/or behaving and that the child's parents believe the child may be troubled or upset, possibly by something of which he or she is not aware.

Next, the parents may tell the child that talking with someone who understands children may help them and the child understand what may be troubling the child. The child can be told that the parents know of such a person, and the parents can describe the person, setting, and arrangements to the child. The child should then be encouraged to ask any questions, and the parents should be prepared and can be helped to answer truthfully and accurately.

The therapist can set the scene by first seeing the child in a suitable playroom and making sure that he or she knows by what name the child likes to be called (and that the child knows the therapist's name). The child will begin to feel understood and ally more with the therapy if the therapist can offer an early interpretative statement that provides the child with some insight yet does not arouse undue anxiety. In order to do this, a series of preparatory statements, including setting statements, attention statements, reductive statements, and situational statements, is useful ([Lewis, 1974](#)).

### Setting Statements

A child may first be told in language appropriate to the child's developmental level that this is a time set aside for the child so that he or she can allow himself or herself to think freely and begin to understand why he or she sometimes feels troubled. The child also should be told that sometimes the therapist will intervene, but with the understanding that it will always be in the interest of helping the child understand himself or herself better.

### Attention Statements

Next, the child's attention can be directed to the content of his or her actions or verbalizations. Sometimes attention is drawn to a coincidence that the child has perceived but has not, or professes not to have, registered; more frequently, attention is drawn to certain paradoxes. The immediate aim is to free the child to produce new material and consolidate existing gains ([Devereux, 1951](#)). In the course of the child's play, for example, the therapist may provide a verbal counterpart to the action being portrayed, affect that might be present, or conspicuous absence of certain persons, actions, or affects.

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#### CASE ILLUSTRATION

An 8-year-old boy with a severe school phobia repeatedly enacted a war scene in which the general was attacked and almost killed. Many fantasies were contained in this play, but one prominent feature was the absence of any female, not only in this play item but also in any other play. After attention was drawn to this "fact," the child recognized his fear of attack from his mother, his wish to attack her, his resentment that his father was often attacked and offered him no protection, the displacement of his aggression toward his mother to his father, and his anxiety about even mentioning his mother.

This is different from a direct translation of possible symbolic representation in the play. The play characteristic to which attention is here drawn is in bold relief and capable of being understood by the child. This point is made to draw the distinction from more subtle paradoxes, which are not readily perceived, at least not by the younger child who is in the preoperational stage of cognitive development. In fact, attention statements can be easily understood by a preoperational child because they relate to more or less concrete perceptions within the play and require only short-term memory and limited "chunking" (defined as "aggregates of related, facts, concepts, or perceptions" [Chapter 12](#)).

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## Reductive Statements

Certain statements reduce apparently disparate behavioral patterns to a common form that has hitherto not been noticed by the child. Thus, a child may manifest certain kinds of behavior whenever he or she is, say, angry. The child may not have been aware of this anger.

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### CASE ILLUSTRATION

In the course of the treatment of a 10-year-old boy, it was noticed that there were recurring episodes of mocking, insulting, or denigrating behavior toward the therapist. Each of these episodes was related in time to one of the frequent trips away from home that the boy's mother would take. His resentment at being left behind, together with his anxiety at being left alone with his father, led to the behavior just described. This type of behavior could be reduced to a single behavioral reaction to underlying rage and anxiety precipitated by the temporary loss of his mother. When the child was told of this relationship, he reacted first of all by an intensification of the behavior pattern but subsequently was able to recognize for the first time his underlying feelings, when he stated, "My parents are always nice to me when we do fun things, but they don't help me with the serious things when I'm unhappy."

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The child here was manifesting a more or less fixed defense, which, in the example given, was brought into sharper relief. A certain level (concrete operations) of cognitive development, including some capacity to take distance from and observe affects, must be available to the child for this to occur.

## Situational Statements

Situational statements naturally follow from those previously described. For example, the child now aware of his or her anger can be shown the situations that give rise to this anger and how in certain instances he or she has repeatedly brought about such situations, either in current relationships or the transference; however, the degree of directness with which such situational statements may be made varies with the emotional and cognitive levels of the child. Children who are in the Oedipal or pre-Oedipal phases and are at the preoperational stage of cognitive development probably need to have these statements made to them in the context of the play, either through dolls and puppets, or indirectly through some other hypothetical child or children. Children who are post-Oedipal and at the stage of concrete operations usually can be approached directly.

In the very young child, feelings seem to be almost exclusively experienced in response to an external event and seem to be viewed by the child as controlled by that external event (e.g., receiving a present produces a happy feeling, mother leaving gives rise to sad or angry feelings). Subsequently, feelings are more or less contained within the body but still not under the control of the child. Next, feelings come to be felt as part of one's self; that is, the child can conceptualize a feeling, first as a somewhat diffuse internal experience and then as multiple, different emotions that are more or less controlled as the self becomes more differentiated. During the preoperational stage, children find it difficult to experience two emotions simultaneously, perhaps because they are still tied to the immediate perception (e.g., a drawing of a face has either a smile or a down turned mouth) ([Nannis, 1988](#)). Very shortly, children can experience opposite feelings, but at first only sequentially (e.g., happy first, sad later) ([Harter, 1983](#)). Once the child is at the concrete operational stage, achieves the operations of conservation, de-centers, and is more or less freed from being tied to immediate perception, the child can then experience two opposite feelings simultaneously; that is, the child can begin to experience, understand, and tolerate ambivalence (i.e., being angry at someone he or she loves).

## Middle Phase

The essence of the middle phase of psychotherapy is the interpretation of the transference and "working through." Notwithstanding the difficulties of the initial phase, a child soon understands one of the goals of psychotherapy: the attempt to gain an understanding of the way he or she feels and behaves. After the initial phase of treatment, the child realizes that everything he or she says and does is subject to use by the therapist. Consequently, the child's play that follows is part of the associative process in the context of therapy. In certain respects, however, the developmental difference between the child and adult influences the form of these associations. The child is much more susceptible to current reality, which exerts a powerful influence on his or her play. The play is often goal directed and not freely associative. Again, the child has a tendency to act rather than think, giving at least the impression of an overemphasized aggressive transference ([Freud, 1965](#)). Further, in school-age children especially, the play is often characterized by organization, reflecting a developmental shift. Last, the child may become totally absorbed in his or her play and may then be unable to exercise an observing function.

At the same time, in addition to play, a child also communicates through most of the other elements that constitute "free association." That is, the child talks, pauses, shows affects, exhibits mannerisms, portrays attitudes, and moves about in space. It is this total picture, viewed as a whole over the course of several hours, which enables the therapist to discern or infer an associative thread. And it is this overall connection that often can be conveyed to the child.

### TRANSFERENCE

In the course of discerning this total picture, a true transference—that is, those previously fixed conflicts and compromise symptoms of the child that are now being experienced currently by the child in relation to the therapist ([Harley, 1967](#))—usually can be observed. Again, the developmental differences between child and adult modify, but do not eradicate, this emerging transference in three ways. First, to the extent that the child is normally dependent, the parents with whom the original conflict was concerned are still with the child and continue to exert their influence on the child. Second, the child is not an equal of the therapist; he or she is still a child relating to an adult as well as a patient relating to a therapist. Thus, besides the mutual respect that should exist between patient and therapist, the child also has certain expectations of the adult, such as appropriate birthday and holiday greetings and gifts ([Temeles, 1967](#)). Third, the continuing development of the child continues to modify the transference, especially during shifts from the Oedipal to post-Oedipal and eventually adolescent stages. In addition, fixations that occurred earlier may be modified with the increased range, flexibility, and shifts of defenses that occur with the development of the child.

For all these reasons, the transference in child psychotherapy is usually not freestanding and is more often unstable than the transference in the therapy of an adult. Nevertheless, to the extent that there is transference, however modified, it is available for interpretation. The more common situation in work with children is one in which a relatively simple current displacement from parent to therapist is recognized and interpreted. Even within such apparently simple displacements, however, elements of transference can be found, and they throw light on the child's current behavior when interpreted.

### RECONSTRUCTION

Linking the complexities of the child's current behavior with his or her earlier fantasies may be helpful to the child. This earlier material may be derived from the personal myth of the child or may be hypothesized from reconstruction. The striking aspect of a reconstruction is that it helps the child "make sense" out of what was previously discomfiting and/or perplexing. Further, it occasionally helps a child by confirming what was probably an essentially correct perception by the child at the time, but which has since undergone distortion. The conceptual abilities of the child are such that the child often attributes affects the child thinks he or she perceives in the parents as resulting from thoughts or wishes of his or her own. Further, the child frequently projects his or her own fantasies and affects onto the parents and subsequently acts against the parents, whom the child now regards as, say, dangerous or angry. At the same time, reconstructions are sometimes barely tolerated by the child when the child is still close to the reconstructed period. The preoperational child too may have particular difficulty in distinguishing fantasies from reality ([Piaget, 1929](#)). On the other hand, the child who is at the stage of concrete operations may have particularly strong defenses—specifically, various reaction formations, such as guilt against aggressive impulses, shame about exhibitionistic urges, and disgust when regressive impulses become threatening—and may accordingly develop a resistance to any exploration of anxiety-arousing impulses.

### PROCESS OF INTERPRETATION

Some further general points on the process of making an interpretation may now be considered. [Loewenstein \(1951\)](#), whose account would have been more complete had he placed greater emphasis on the interpretation of unconscious, or at least preconscious, material, described the following steps:

1. Show the patient that certain common elements exist in a series of events.
2. Point out the similar behavior of the patient in each of these situations.
3. Demonstrate that such behavior was manifested in circumstances that all involved, for example, competitive elements, and where rivalry might have been expected.
4. Point out that, for example, rivalry does exist unconsciously but is replaced by another kind of behavior, such as avoiding competition.

5. Show that this behavior originates in certain critical events of the patient's life and encompasses reactions and tendencies that can be grouped together.

In the stages just outlined, the interpretation of mechanism, as opposed to that of content or affect, is significant. Further, it also can be seen that there is a gradual transition from preparatory intervention, through confrontation, to an interpretation containing a genetic component. Ferenczi once described his own experiences as he proceeded in the steps just outlined:

One allows oneself to be influenced by the free associations of the patient; simultaneously one permits one's own imagination to play on these associations; intermittently one compares new connections that appear with previous products . . . without, for a moment, losing sight of, regard for, and criticism of one's own biases (quoted in [Kris, 1951](#), pp. 15–30).

Essentially, one might speak of an interactive process in the therapist involving empathy, self-observation, and clinical judgment.

The description given by [Erikson \(1940\)](#) is remarkably similar. Speculations are first derived from the observer's impressions, associations, and recollections; for example, "It was as if . . ." The observer also associates past impressions in the same child, from other children, or from data derived from the parents. The therapist reflects on the latent possibilities that the associations may possibly correspond to a genetic or associative connection in the child's mind and pictures what the child is doing under the observer's eyes and what the child is said to have done in other situations. This all leads up to the interpretation. Erikson then describes three steps in making the interpretation. First, there are observations, feelings, and reflections that lead to interpretational hints. For example, a symbolic equation or metaphor may make it possible to recognize a play act as alluding to and standing for an otherwise manifestly avoided item (a person, object, or idea), or a play arrangement may prove to represent a special effort on the part of the child to rearrange in effigy his or her psychological position in an experienced or expected danger situation. Such an arrangement usually corresponds to the child's defense mechanisms. Second, these hints are then subject to further observations and reflections and emerge as a conviction in the observer's mind in the form of the reconstruction of a genetic sequence or of a dynamic configuration pertaining to the patient's inner or outer history. Finally, the therapist may proceed to convey part of these reconstructions to the child whenever the therapist feels the time has come to do so. Erikson considers the last step to be the therapeutic interpretation.

The significant point here is the step-by-step progression in working with children implied in Erikson's statement that, "the observer may proceed to convey parts of these reconstructions to the child whenever he feels the time has come to do so" ([Erikson, 1940](#), pp. 557–671). However, it is important to keep the developmental level of the child in mind here, because massive interpretations given to a young child are more likely to be heard as interfering noises than helpful statements, with a consequent heightening of resistance and play disruption or, worse, a play inhibition.

In short, the therapist, while still engaging with the child, takes mental distance from the immediate transaction and tries to place the immediate observations into the context of what has previously taken place. The therapist can do this by a kind of mental "playback." Material from previous sessions is not only "played back" mentally, but also is translated to a higher level of abstraction, which enables the therapist to formulate to himself or herself "what is going on" as a basis for formulating an interpretation. The therapist then mentally translates this back to the level of the child and the immediate situation and makes his or her interpretation at that level.

#### GENERAL GUIDELINES FOR INTERPRETATION

##### *Multiple Appeal*

As a general rule, interpretations are probably more effective when they have "multiple appeal" ([Hartmann, 1951](#)). The therapist also decides clinically when to interpret the past, current reality, the transference, or all three.

##### *Sequence*

A number of other guidelines for the order of interpretation have been suggested. Resistances or defenses should be interpreted before the instinctual derivatives ([Loewenstein, 1951](#)). One should defer interpreting a symptom representing an important unconscious conflict until a strong therapeutic alliance has been established. One should also start with interpretation of still-mobile defense traits in preference to rigid, characterological defenses. In spite of these guidelines, individual situations may be handled in several different ways, depending on the developmental level of the child and stage of therapy. Take, for example, the following situation: An 8-year-old boy wanted to take along some tracing paper belonging to his therapist on the eve of his going on a car trip with his mother and father, the anticipation of which had already aroused considerable anxiety.

What were the possibilities here for interpretation?

1. The therapist could simply have confronted the child with his wish to take something along. At the particular stage in the therapy of this child, however, this would have been redundant, although it might have been useful as a building block, or an "attention statement" for future use.
2. The therapist could have shown the child how anxious he was about the trip with his parents, but doing so might have forced the child to face his anxiety too abruptly, with insufficient time for "working through" (see the following), and without the therapist's ongoing support.
3. The therapist might conceivably have tried to link this wish with the boy's reactions in other similar situations in which he had become anxious, but this could not be done without the previous steps.
4. The therapist also might have interpreted the patient's wishes toward the therapist for protection, but in this patient such a move might have left the patient feeling exposed and defenseless.
5. The therapist might have made the connection for the child between this coming event and earlier events in his life, but this intervention might have had little use for this particular patient, because he did not yet have a clear idea of his feelings about the coming event.
6. It might then be asked whether anything should be said at all. Something should be said, but something that will temporarily buttress the child and offer support, at the same time that both the defense and fear are being interpreted. For example, the therapist could say to the child, "How nice it will be to have something to take along, especially if you are worried about the trip." There would be no need to interpret the positive transference aspect at this point, because the movement is in a forward direction. The therapist might also decide to include an aspect of the transference, but in a supportive way. The therapist might say, for example, "I think you would like to have something of mine with you on this trip." He might then allow the child to keep the paper and reserve for a later date any further exploration of the act: "Why don't you take the paper with you? When you come back we can talk again about how you feel when you have to take a trip with your mother and father."

These examples are not meant to be recommendations of specific things to say but rather illustrations of underlying principles. The choice of level and wording depends on such factors as the diagnosis, stage of therapy, and developmental level of the child. A 6-year-old child at the beginning of psychotherapy might experience his or her anxiety on the eve of such a trip as ego-syntonic and fail to understand an interpretation of the anxiety. On the other hand, a 10-year-old who is more advanced in therapy might well experience such anxiety as inappropriate and ego-alien and might find such an interpretation useful.

##### *Timing*

An interpretation is probably well timed when the therapist thinks that the statement at that moment will help to consolidate existing gains and elicit new material ([Devereux, 1951](#)). Devereux described an interpretation as being "timely" when it is capable of being utilized by the patient, and this in turn can occur only if the patient understands it.

##### *Focus of Interpretation*

When should an interpretation be made in the play, and when should it be taken out of the play? When attention statements are made, the interpretations are clearly made "in the play." Reductive statements imply that the child is receptive to statements about himself or herself; that is, "out of the play," whereas transference interpretations in their most effective form are made quite distinctly "out of the play," given the earlier steps of preparation. (The use of play in psychotherapy with children is described extensively in [Chapter 18](#) and elsewhere) ([Beiser, 1979](#); [Buxbaum, 1954](#); [Haworth, 1964](#); [McDermott and Harrison, 1977](#); [Winnicott, 1971](#)).

##### *Tact*



Tact is required when significant developmental differences are considered. A young child may have great difficulty in tolerating ambivalence and may find it especially difficult to accept a hostile or aggressive wish or fantasy. This reluctance may be overcome by placing within a fuller context the anger, say, that is to be interpreted. For example, one might more tactfully say to a child, "It is very hard to be angry at someone you love."

[Glover \(1930\)](#) tried to be more specific with regard to tact, stating that the interpretation should be delivered as a plain statement in terms devoid of active emotional stress, to prevent an immediate, overwhelming conscious conviction on the patient's part that his or her therapist was in a state of countertransference. Glover also cautioned about the use of wit, the exploitation of the comic, and the shelter provided by technical expressions. It is usually preferable to refer to the child's parents as "mother" or "father." Only with a very young child would one use such terms as "mommy" and "daddy," and then only because they are developmentally appropriate.

#### *Wording*

Wording should be specific and concrete; the interpretation also should be worded to fit the individual situation. Again, the therapist is cautioned to avoid using the same defense mechanism the patient uses; for example, laughing things off and minimizing them. Interpretations appear to gain when they contain an element of time; for example, "now," "before," "at the age of," and "after this happened."

Wording is particularly critical with young children, not only from the point of view of the level of cognitive development but also what the child can accept in his or her dependent position with respect to the parents, and the child's own struggle against regressive pulls or progressive pushes. The child attaches greater meaning to certain words than does the adult; it is necessary for the therapist to understand these special meanings.

#### *Inexact and Incomplete Interpretation*

[Glover \(1930\)](#) distinguished between inexact and incomplete interpretations in psychoanalysis. An incomplete interpretation was termed by Glover a "preliminary interpretation." For example, one would interpret a so-called genital fantasy before an anal fantasy. He contrasted this with an inexact interpretation, when one might never interpret the anal fantasy at all. That is to say, if the interpretation of the genital fantasy was regarded as the complete interpretation, then the interpretation was *ipso facto* inexact.

An interpretation is never complete until the immediate defensive reactions following on the interpretations are subjected to investigation. The complete interpretation is really the complete treatment: "Every construction is an incomplete one. . . . As a rule he (the patient) will not give his assent until he has learnt the whole truth—which often covers a very great deal of ground" ([Freud, 1937/1964](#), pp. 257–269).

#### *COUNTERTRANSFERENCE*

In work with children, the therapist is particularly prone to countertransference phenomena. For example, there is a much greater "regressive pull" ([Bornstein, 1948](#)). The therapist must learn to recognize the feelings aroused in himself or herself so that he or she can deal with the child in a helpful way. One's own feelings are a vital instrument for understanding the child. At the same time, if the therapist is not aware of, or not clear about, the nature of his or her aroused feelings and their sources (both external and internal), he or she may be hampered in working effectively with the child. Indications that countertransference feelings may be at work include the following:

1. The therapist may fail to recognize where a child is in his or her development. Expectations then are not commensurate with the child's maturational and developmental capacities. The therapist may then experience unrealistic goals, alternating with despair.
2. The regressive pull experienced by the therapist playing and working with a child may give rise to the temptation to identify, compete, and/or act out with the child, or infantilization of the child may occur.
3. A misreading of the child's relationship to the therapist may occur, in which the relationship is seen as realistic when in fact it may be a transference from the child's feelings toward his or her parents. Therapists are usually well aware of a child's aggressive feelings but may be less aware of a child's seductiveness toward an adult (parent).
4. Remnants of the therapist's own childhood relations with his or her brothers and sisters may be an important source of the therapist's ambivalence to the child. For example, excessive concern—or lack of concern—for a child's climbing on a chair or approaching a tall column of heavy building blocks may mask an underlying wish that the child will hurt himself or herself and may be associated with guilt feelings when the child does stumble or fall. Such a feeling of guilt may come about because of the partial unsuspected fulfillment of an unacceptable wish.
5. The stirring up of old conflicts within the therapist, when exposed to certain behavior in the child, may cause anxiety in the therapist. For example, uncontrolled aggression, sex play, or masturbation may be upsetting to the therapist.
6. Sometimes the therapist may transfer old feelings from his or her own childhood onto the parents of the child. The therapist may then overidentify with the child in his or her struggle with his or her parents. Rescue fantasies may then occur. Conversely, the therapist may erroneously identify with the parents (perhaps through an identification with the aggressor mechanism) and consequently exercise unnecessary, even punitive, controls against the child who is acting too aggressively or sexually for the therapist's comfort.
7. Sometimes the therapist simply cannot understand the meaning of certain behavior in a child. Of course, there are times when all of us find some item or other of behavior inexplicable. However, the persistent drawing of a blank in understanding a repeated item of behavior should lead to the suspicion of an interference by one's own conflicts—an emotional blind spot, so to speak.
8. A therapist may find himself or herself feeling depressed or uneasy during his or her work with children. Assuming the therapist is not suffering from a true depression, the possibility exists that emotions from old conflicts have been aroused and are interfering with the therapist's functioning. Occasionally, a therapist may find himself or herself aroused and experiencing great affection for a child. This too may interfere with his or her work with the child.
9. Countertransference may be at work when the therapist permits, or even encourages, acting out in the child; for example, a therapist may suggest to a child that he or she must stand up for himself or herself and hit back. On occasion, a therapist may even feel the impulse to act out with a child.
10. A therapist may need and solicit the admiration obtained by having the child like him or her. This too may represent a deeper, unconscious need of the therapist and may not be in the best interest of the child.
11. Conversely, repeated arguing with a child may suggest that the therapist has not only become involved but also enmeshed with the child.
12. Recurring countertransference problems commonly arise in relation to specific characteristics of a child. For example, a retarded child may evoke guilt and defenses against such guilt within the therapist, or omnipotent rescue fantasies, both of which may then be acted out. Passive, hostile children may arouse anger in a therapist. Aggressive children may evoke counteraggression. Sexually attractive children and adolescents of the same or opposite sex as the therapist may threaten the therapist, leading to either vicarious and "excessive" exploitation of sexual issues or denial and avoidance.

#### *WORKING THROUGH*

It is clear that working through is necessary to sustain any therapeutic effect ([Greenacre, 1956](#)). The defensive conflicts remain somewhat structured unless they are dealt with repetitively in relation to various behaviors, events, and feelings. Historically, working through was first emphasized from the point of view of being of educative value and compared with mourning and the progressive detachment of the individual libido from the organized tensions and aims that permeated the later life. This view is insufficient.

Another concept—the corrective emotional experience—is really an aspect of working through, at least in its more modern construction. Originally, the idea involved replenishment of earlier deficiencies through the current relationship. This appealing idea unfortunately proved to be too simplistic. Among other things, it failed to take into account the power of the unconscious repetition compulsion. The concept has some merit, however, if it is modified to provide the child in the here-and-now with a reaction different from his or her previous experiences, a reaction that is now more appropriate and does not perpetuate the malignant interactions to which he or she has become accustomed. This update of the concept of course is consonant with the concept of working through. Finally, with the rise of ego psychology, the recognition of the need for consistent work with the patterns of defense and the affects related to them once more becomes paramount ([Bornstein, 1948](#)).

From a developmental point of view, working through with children poses a special problem. The chief difficulty lies in the fact that the live parents are usually present and may continue to exert a reinforcing influence on the child's original conflicts. Sometimes this interference can be alleviated by concomitant work with the parents, through some form of psychotherapy or regular meetings between the parents and the child's therapist. Occasionally, it becomes clear that the child cannot work through a conflict while in the home, and an alternative plan may become necessary, such as boarding school in the case of an older child. Now and then the difficulty is insuperable at the time, and the therapy must be interrupted until the child is in a more advantageous situation for therapy. Occasionally, it is possible to hold the

child in therapy until such a situation occurs. In some instances the child can be helped to understand the repetitive and reinforcing behavior of the parents and his or her involvement in precipitating or responding to their behavior. If the influence of the parents is not too strong, the child can be helped to modify his or her own behavior in this regard, to interrupt the vicious cycle.

### Termination Phase

The criteria for termination ideally include some actual achievement (as opposed to a "flight into health") ( [Train, 1953](#)) of the goals of therapy: reduced anxiety, improved self-esteem, increased frustration tolerance, disappearance of symptoms, better coping strategies, relative independence, good relationships with peers and adults, satisfying schoolwork, feelings of pleasure and joy, and a sense of resumed developmental progress.

In almost every instance, these achievements are judged clinically rather than measured scientifically. Clinically, there is no such thing as a perfect therapy (or for that matter, a perfect patient or therapist). Every therapy eventually stops, and when it does the child should be more or less along the road toward these laudable goals. A child may even have gained a better understanding of himself or herself and may no longer need to act out so many infantile longings, frustrations, and feelings, and may have learned instead more adaptive ways to love and be loved, for example. However, in some cases, the therapy simply stops because there is a sense of diminishing returns, the resistances are too great, the treatment is inappropriate, or for various other reasons the treatment is a failure. In one survey of terminated analytic cases ( $N = 49$ ), only 14% terminated by mutual agreement among parent, therapist, and child ( [Freud, 1971](#)).

The actual phase of termination is a useful period to explore issues of separation, reactions to loss, dependency versus independence, and anxiety about progressive developmental movements. Some children undergo a temporary regression, manifested by a reappearance of their presenting symptoms, in the face of leaving the therapist and returning, as it were, to the family. Others are able to "reconstruct" and recall the beginning of their treatment rather than reenact it through regressive behavior. A reasonable period of time is required to deal with these issues depending on the frequency, duration, and intensity of the psychotherapy. Thus, when the question of whether to terminate has been decided, preferably by mutual agreement, a termination date is set that will allow a suitable amount of time for the termination phase. This period may vary from 6 weeks to 3 months or so.

Follow-up communication is often helpful in consolidating the work of the termination phase.

## THERAPEUTIC ACTION AND THEORY

Many attempts have been made to describe the therapeutic effects of interpretation in terms of the insight achieved ( [Rothstein, 1989](#)). However, children (and adults) may achieve insight yet fail to improve emotionally or behaviorally. Further, some children (and adults) may show remarkable improvement without any evidence of insight at all. This latter phenomenon suggests that other therapeutic modes may be at work, including the beneficial effects of a loving, compassionate, respectful, and dedicated relationship, the child's feeling of being understood, and the enlightening feeling that may result from new, positive life experiences.

The particular type of interpretation that will produce a so-called therapeutic insight, that is, a structural change has been given different names, as has the actual therapeutic insight. For example, "transference" ( [Fenichel, 1945](#)) or "mutative" ( [Strachey, 1934](#)) interpretations may produce "emotional," "psychological," "immediately demonstrative" ( [Richfield, 1954](#)), or "dynamic" insights as opposed to "intellectual," "descriptive," or "neutral" ( [Reid, 1952](#)) insights. The significant point presumably is that shifts from unconscious to conscious awareness are thought to occur only after derivatives of the original feeling are recognized with an experiential sense of conviction and worked through. [Bergler \(1945\)](#) considered that the whole process of working through is centered chiefly in the correct handling and mobilization of the feeling of guilt. [Devereux \(1951\)](#) felt that an interpretation (which consists of supplying an unconscious closure element) is effective when practically all conscious and preconscious material pertaining to the neurotic gestalt has been produced. [Loewenstein \(1951\)](#) believed that the therapeutic effect is owing to a psychic process in which each of the following parts has its respective place: (a) overcoming of resistances; (b) working through; (c) remembering and reliving of repressed material; and (d) effect of the reconstruction.

Anticipated changes resulting from psychotherapy are also to some extent a function of normal development. Therefore, all of the factors that facilitate normal development also will facilitate the desired changes in psychotherapy.

The experience with the psychotherapist not only is a corrective one but also represents an oasis phenomenon for the child, who now finds himself or herself, at least temporarily, in a relatively protected, facilitating environment that enables development to proceed by reducing the impact of trauma and the acting-out tendencies in the child. Another way of putting this is to say that the child in psychotherapy finds himself or herself, at least momentarily, at a distance from the acute upset of the current developmental turmoil, whether it be a too exciting, sexualized relationship with a parent, a sadomasochistic relationship with a parent, a grief reaction at the birth of a sibling, or frightening fantasies of bodily harm.

The child is also afforded an opportunity for confirmation of his or her essentially correct perception of his or her parents, leading to a strengthening of reality testing and an increase in self-esteem. The play of the child, as well as the use of words, inasmuch as they represent an intermediate stage between action and thought, provides a handle by which the child grasps affects and is enabled to delay. Most important, the play itself undergoes a development that carries with it a significant shift from primary process to secondary process.

The dyadic therapeutic relationship provides an introspective opportunity for the child and also serves to foster identification with an appropriate adult model. Indeed, the child may "borrow" strength, clarity, and organization from the psychotherapist as the child struggles to deal with an acute development crisis. Further, the loving relationship in itself and the feeling of being understood have important therapeutic effects, perhaps regardless of any insight that may have been achieved ( [Rothstein, 1989](#)). Another significant therapeutic force is the change from despair to hope, leading to a therapeutic optimism. Concomitant work with other family members also leads to shifts in the dynamic equilibrium within the family; releases the child and parents from their locked-in, fixed positions; and allows development to proceed. Last, the extended moment of time, the opportunity to examine in detail, itself contributes to clearer, more direct communications for all concerned.

## EVALUATION OF OUTCOME OF PSYCHOTHERAPY

Evaluation of the effectiveness of psychotherapy is in general a complex undertaking ( [Heinecke and Ramsey-Klee, 1986](#); [Kazdin, 1988](#); [Strupp and Hadley, 1977](#)), and such research is plagued by methodological problems ( [Hine, 1982](#)). [Parloff \(1982\)](#) reviewed nearly 500 controlled studies providing research evidence on outcome and concluded that all forms of psychological treatment are comparably effective in producing therapeutic benefits and that such benefits are reliably superior to those found in controls. Subsequent outcome studies ( [Casey and Berman, 1985](#); [Weisz et al., 1987](#)) have not contradicted this global conclusion, although [Weiss and Weisz \(1990\)](#) reported that: (a) behavioral treatments are associated with significantly larger effect sizes than nonbehavioral treatments; (b) most studies of child psychotherapy in actual clinical practice show very small effects ( [Weisz et al., 1992](#)); and (c) psychodynamic treatments have an effect size that is even smaller than client-centered treatments (0.01 versus 0.56, respectively). [Shirk and Russell \(1995\)](#), however, have expressed concern about major methodological flaws in many of the studies used in the Weiss et al. metaanalytic approach and have suggested that all such flawed studies should be excluded from metaanalytic comparisons. Improved scientific methodology in studies of child psychotherapy remains an important goal for future studies.

Other factors that may influence outcome include the motivation for help in the child and his or her family, psychiatric disorders in the parents, and current social conditions ( [Graham, 2000](#)).

In one controlled study of psychoanalytic psychotherapy it was shown to improve compliance in so-called brittle diabetes ( [Moran et al., 1999](#)).

In another albeit limited but controlled study, longer and more intensive treatment was independently associated with a greater likelihood of reliable improvement, particularly among children with severe overanxious or generalized anxiety disorders, although the majority of older (>11 years), severely disordered children remained diagnosable at termination. Moreover, if severely disordered children were treated nonintensively (once- or twice-weekly psychotherapy), more than half (56.5%) got worse or showed no improvement. Less severely disordered children were as likely to benefit from nonintensive treatment as from intensive treatment ( [Target and Fonagy, 1994](#)). (For a critical review of the psychotherapies, see [Chapter 87](#)).

From a clinical viewpoint, outcome in psychotherapy is largely a value judgment, requiring that the therapist recognize the multiple values, criteria, and factors that go into such judgments. Meanwhile, research on psychotherapy with children remains sparse ( [Barrett et al., 1978](#); [Levitt, 1971](#); [Shaffer, 1984](#)). More focused scientific



research is needed. [Marans \(1989\)](#) has suggested specific questions concerning which aspects of the therapeutic process are especially useful for a particular child.

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## 80 USE OF PLAY IN PSYCHODYNAMIC PSYCHOTHERAPY

Henry P. Coppolillo, M.D.

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The essence of psychodynamically oriented psychotherapy is the communication and exploration of the patient's subjective states and convictions even when these are dimly perceived by the patient or are unconscious. This is an arduous task when it is undertaken with an adult, who usually comes to the psychotherapeutic situation with a well-developed repertoire of signals, signs, and symbols as communicative vehicles. With the child, the task becomes more difficult because his or her complement of culturally validated signals and symbols is as yet incomplete. The play of the child can often more than compensate for this immaturity. In this chapter the use of play as a vehicle for therapeutic effectiveness and the conditions that facilitate that use are explored. A word of caution: Many of the concepts regarding psychotherapy are deceptively easy to understand. It is their application in the living situation that is difficult. For this reason, in order to become competent beginning psychotherapists must avail themselves of competent supervision as a necessary companion piece to texts and to reading and hearing case presentations.

### HISTORICAL NOTES ON THE PSYCHOLOGY OF PLAY

It is beyond the purpose of this chapter to explore the ideas of all who have been interested in play. Suffice it to say that play has been scrutinized by members of disciplines as widely disparate as military theory (war games) and philosophy of religion ( [Berger, 1969](#)). Closer to our interest, one of the first psychodynamic considerations of play is found in [Freud's \(1955\)](#) development of the concept of the repetition compulsion. A small boy whose parents were away used the repeated tossing and retrieving of a spool of thread as an example of the wish to master a trauma. [Waelder \(1933\)](#) extended the understanding of the broad and varied psychological functions that play serves.

[Hug-Hellmuth \(1913, 1921\)](#) wrote the formal articles on the use of play in the therapeutic situation when she described the psychoanalysis of children. The modality had become broadly accepted and used by therapists by the time [Klein \(1932\)](#) and [Anna Freud \(1946\)](#) published their texts on the use of play in the treatment of children.

By 1940, child therapists had begun to utilize play in therapeutic efforts that conceived of specific, more restricted goals than did psychoanalysis. [Levy \(1939\)](#) aimed at achieving a therapeutic effect with a cathartic resolution of conflict or tension through the child's play. [Allen \(1942\)](#) described the use of the play situation to develop and sustain the relationship that he felt had central therapeutic significance in the child's life. Solomon suggested a variation in the technique of using play (1955) as he guided the child's play in an effort to help the child achieve psychological integration.

A relatively more subtle conceptual variation is to be found in Virginia [Axline's work \(1947\)](#). Basing her ideas on the Rogerian concepts of "client-centered therapy," she used play to reflect back to the child his states, wishes, or convictions without attempting interpretations of unconscious motives or conflicts. The articulation that the child conveyed was considered therapeutically effective.

A moving and profoundly significant explanation of the meaning of play is offered by [Peter Berger \(1969\)](#) in his book, *A Rumor of Angels*. Berger follows the historian Huizinga, novelist C.S. Lewis, and Freud in ascribing the wish to be joyful, playful, and pleasure-seeking to the very nature of human beings. He then proposes that it is this joy and exuberance that can emerge most easily in play, because portions of reality can be suspended in play. The portions of reality that he believes are most important to suspend are pain and death. At the same time he describes the preservation and extension of parts of reality through the principle of transcendence. By transcendence he means, "phenomena that are to be found within the domain of our 'natural' reality but that appear to point beyond that reality" (pp. 65–66). Berger then proposes that human beings tend to order or organize their realities and possess an "intrinsic impulse to give cosmic scope to this order" (p. 70). Human beings can suspend certain painful aspects of their reality in play, even as they preserve other components of that reality and live out the wish to maintain order and pleasurable predictability in their universe. He summarizes his views in a beautiful passage by describing the play of children (p. 74).

Some little girls are playing hopscotch in the park. They are completely intent on their game, closed to the world outside it, happy in the concentration. Time has stood still for them or more accurately, has been collapsed into the movements of the game. The outside world has for the duration of the game, ceased to exist. And, by implication (since the little girls may not be very conscious of this), pain and death, which are the law of the world, have also ceased to exist. Even the adult observer of this scene, who is perhaps all too conscious of pain and death, is momentarily drawn into beatific immunity.

Although Berger is not a clinician and his philosophical exploration may not have direct bearing on the technical uses of play in therapy, we see in the following how closely his thoughts parallel the thinking of a clinician who used play with great therapeutic effectiveness.

[D.W. Winnicott \(1953\)](#), in his timeless paper on transitional objects, articulated concepts that are useful for understanding the developmental and therapeutic functions of play. He proposed that, in addition to the capacity of a child to experience external reality and the internal world, an intermediate area of experience is available to the child to which internal and external experiences both contribute. He called this the intermediate area of experience, and he called the objects that were experienced in this way "transitional objects." Concrete objects as well as songs, rhymes, or even the babbling of an infant can be experienced in the intermediate area. According to Winnicott, having the intermediate area of experience available to him, the child can begin to relieve the strain of relating internally generated wishes with the rigidity of external reality; once acquired, this mode of attaining self-soothing continues throughout life. Winnicott further states that "This intermediate area is in direct continuity with the play area of the small child who is 'lost in play'" (p. 96). For example, a bored or sad child may pick up a teddy bear to play with. The child can easily appreciate the external reality of its button eyes, soft fur, and the inviting demeanor its manufacturer provided. But as he brings it into the intermediate area of experience, it becomes a transitional object and, as such, is also appreciated as a soothing companion, the reciprocator of a loving hug or even the worthy opponent in a wrestling match for the championship of the child's preschool class. The teddy bear has been transformed from a toy into a character in a drama that reveals the wishes and impulses, the convictions and visions of the little author who provided him his script. Bringing a physical entity into the intermediate area of experience allows the entity to be imbued with qualities currently significant to the child. Those elements of the subject's inner world that are brought into the intermediate area and are thus attached to an object can be played with and mastered with just the right intensity. If a child becomes disturbed by the intensity of his own aggression when playing with his teddy bear he can readily turn his attention to the real qualities of the toy and thus escape the threat of his own impulses until he is ready once more to face them in play. It is this capacity's persistence that allows adults to transform a combination of sound waves into the *Eroica* or the *Appassionata* and allow great visions or tender images to sweep away the tedium of daily living without losing the ability to address the reality of daily living when necessary.

[Rapaport \(1951, 1958\)](#) envisioned how the ego would be handicapped by the extremes of being totally engrossed by events of the external world or responsive only to the impulses and images of the inner world. In the former condition the person would be oppressed by stimulus slavery, whereas in the latter state we would have to say he was handicapped by madness. Transitional objects become excellent vehicles for maintaining a balance between the tides of stimuli from the external and internal worlds. When a child takes an object or a story or song into the intermediate area of experience, he takes with it a quantity of reality that is reliably there but is unobtrusive when he imbues the object with qualities that serve to play out a wish or impulse from his inner world. If his wishes or impulses become too threatening to him, he can attend to the realistic qualities of the object more fully. If the reality of the toy is insufficient to provide him relief from tedium, he can suspend some of that reality ([Coppolillo, 1976](#)). These are the conditions of this kind of play that render it invaluable as a vehicle for the modulated, manageable expressions of the inner states of the child. The thinking of Berger, Winnicott, and Rapaport had many points of overlap and concordance even though they developed their ideas quite independently and were addressing different aspects of life and theory. The work of [Peller \(1954\)](#) on the function of play in development seems also to coincide with their thinking as she states, "Small quantities of anxiety are mastered in play" and further asserts that unconscious impulses can be acted out in play without reaching awareness (p. 181). In this chapter the work of these authors is the theory that supports the suggestions for using play as a psychodynamically therapeutic tool. (For a

more extensive review of the literature on this topic, see [Gardner, 1979.](#))

Play in this chapter is considered as both an adjunct to communication and as a vehicle of communication between child and therapist that reveal to the therapist states of the child's inner world. These states may include (but are not limited to) misconceptions and misinformation about himself or herself or misconceptions about the child's world. These states may contain unrealistic wishes, anxiety-laden impulses, conflicts, and frightening expectations of retaliations. Of course this is not to say that even as the child is struggling with these conflicts other phenomena are not occurring simultaneously. For example, it would be surprising if the child were not enjoying the growing relationship with a caring and warm therapist even as they are struggling to understand the origins of an irrational fear. Or for some children the time playing in the presence of the therapist may be one of the few moments in their lives when they feel soothed. Deconditioning or relearning may be occurring, or perhaps the narcissistic gratification of being the only object of the therapist's attention may be helpful to the child. Conflict resolution often combines with these other bonuses of human relatedness to produce therapeutic benefits.

## PRECONDITIONS NECESSARY FOR THERAPEUTIC PLAY

The following sections apply to play as used in individual psychotherapy. See [Lewis and Blotcky \(1997\)](#) for a summary of the uses of play in group therapy.

There are times in the course of a treatment process that play loses its usefulness as a communicative or therapeutic tool. This may happen even when the therapist seems to be conducting the sessions in a technically correct manner and has his or her own internal world in order in terms of fatigue, personal gratification, and countertransference ([Coppolillo, 1987](#)). Often the causes for the derailment of the process may be found in the preparation of the child and therapist to conduct therapy. For this reason some attention must be paid to conditions that will make the play an activity that is considered therapeutic by both the clinician and child, rather than one in which something will hopefully happen that will somehow benefit the child. A brief review of these conditions may be helpful.

### Conditions Regarding the Therapeutic Environment

#### PHYSICAL ENVIRONMENT

The physical conditions that are necessary to conduct safe and dignified psychotherapy are more fully discussed elsewhere ([Coppolillo, 1987](#)). Here it is sufficient to say that the physical environment needs to be safe, sufficiently well contained to convey the notion of confidentiality to the child, and allow the adults waiting for the child to be comfortable, especially if they must sit with a younger child while the patient is in the consulting room.

There is some question as to whether or not a separate "play area" is desirable. There are advantages and disadvantages on both sides of the question. A separate play area sometimes invites the child to make an artificial distinction between "talking time" and "playing time" and to therefore resent or resist inferences made by the therapist from the play. On the other hand, a single office may have rugs, furniture, and paintings that the child may damage, to the therapist's horror. If the child senses this concern, it may inhibit his or her play or spontaneity. Giving some thought to the problem allows the therapist to compensate for one or the other disadvantage.

Another specific issue that arises is how to equip the play area. Some therapists' play areas look like toy stores, with the variety of games and equipment on display. Others prefer to have only a small number of toys and drawing materials immediately available. Anna Freud is quoted as saying, "The special role of the toy as a therapeutic agent has been greatly overvalued" ([Sandler et al., 1980](#), p. 39). By this she meant that the use the child makes of the toy is by far more revealing than the choice of the toy itself. Children can use checkers to build a wall between them and some imagined threat and may use blocks to play a competitive game with the therapist. In addition, an important issue is what toys the therapist feels allow him or her to most comfortably assume an attitude of inquiry. If the therapist is vulnerable to compulsive reactions of neatness when his or her child patient is ruining a model by smearing glue all over it, it would be a good idea not to have models to build on his or her shelf. If the therapist finds himself or herself sliding into unproductive competitive feelings with a youngster, board games may not be the toys he or she would choose to have on display. One suggestion might be to have a few basic items of play in the office, such as drawing and coloring material, a doll family and simple dollhouse, toy vehicles, and toy soldiers. If the child then appears to want or need other things to play with, he can be asked as the treatment progresses if or how other toys will help both therapist and child to understand his or her worries or problems. This keeps the ultimate purpose of the play in the child's mind. What the therapist should keep in mind is that, in the optimal situation, it is the benign interest and attentive inquiry of the therapist that is helpful to the child and engages him or her, and not the allure of the toys. If the child cannot use this interest and attention (and where else in life can a child get 45 minutes to 1 hour of undivided attention from an adult), this inability needs to be addressed and worked through and not bypassed with the seductive appeal of toys.

#### PRECONDITIONS IN THE THERAPIST

We must hope that the therapist will come into the therapeutic sessions well rested, with few worries of his or her own, sufficiently confident that his or her own skills and techniques are adequate to help most children (so that the child does not *have to* get well for the sake of the therapist), and with enough play in his own personal life to ensure that the child is playing for the child's own reasons and not the therapist's. These are conditions to strive for, of course, and not ones that must be attained every minute of every day. If they were constant essentials, few of us could ever treat anyone. However, if the therapist does detect a persistent disturbance in any of the conditions mentioned, it is essential that remediation through environmental or psychological change take place in the therapist for the therapist's own and the patient's sake.

In addition to the unending self-scrutiny that the therapist must undergo, of course, is the intellectual preparation required to make play meaningful in terms of what it can reveal about the child's developmental achievements and arrests, as well as about the current state of his or her psychological organization. This requires a vivid, living picture of stages and processes in development as well as mental images of how children function in health and illness. Training in the recognition of these states as well as education in theories of human development make it possible for the clinician to utilize the rich, profound revelations that play provides.

#### PRIOR TO PLAY

Before all else, a history of the child's development and the difficulties that bring him or her to the clinical situation must be gathered ([Coppolillo, 1987](#); [Simmons, 1981](#)). Usually this history is gathered from the parents. The therapist must recall that we are all vulnerable to retrospective falsification, and especially when the historian is as emotionally involved as most parents are with their children. Remember too that the clinician is not just interested in whether a phase of development was deemed "normal." More often than not, normal is judged by whether or not the child was troublesome to the adults around him or her. It is most useful to learn of the style in which the child faced the stress of a new developmental phase; the defenses used in adapting to it, and whether or not there was pride in leaving behind old modes of adaptation and enthusiasm in adopting new talents. In short, conjuring an image of the living child as he or she developed is far more productive than pasting a label of "normal" or "abnormal" across a developmental phase.

The same considerations may be applied to the history of the difficulties that brought the child to the clinical situation. In this segment of the preparation, however, the clinician must anticipate and deal with two conditions that may become obstacles to the treatment later. The first of these is that the people who referred the child or are paying the bill may expect that the therapist will act as their agent. The situation can become critical if a school or court has referred the child and awaits guidance from the therapist. If there is any question about whom the therapist is representing, every effort must be made to clarify expectations at this stage of the treatment process. If they are not clarified, later in the treatment the child may sense divided responsibility in the therapist and feel that the therapist has betrayed him or her.

A second related issue occurs when, without meaning to do so, people bring a child to the clinical situation to be made *good* and not necessarily *well*. If the therapist inadvertently participates in trying to make the patient good, he or she joins the multitude of people who must seem to the child to wish only that he or she behave in a certain manner and to care little about the anguish or the forces that the child feels so powerless to resist. If the child reaches this conclusion, acquiescence may be possible, but revelation of his inner world is unlikely.

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#### CASE ILLUSTRATION

A 16-year-old girl was treated, with parental approval, after the juvenile court had adjudicated her "an unruly child." She had run away from home twice in 6 months and was returned both times by the police. It was only after she became convinced that her therapist was *her* agent and not insistent on her behaving in any predetermined manner that she could reveal that she was running away from an incestuous situation at home. She had turned to a number of adults in her environment for help and had encountered blank stares or deaf ears. Running away from home, the very action that was labeled "bad" and for which she had been sent to therapy, had been the most adaptive action she felt she could have taken under the circumstances. Had the therapist been intent on making her good by convincing her of the evils of running away she would have probably not revealed her secret.

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After the history is gathered, the more formal aspects of treatment begin, and it is in this phase that we come to see what an important asset the initiative of the child can be to the treatment. Yet let us review for a moment the manner in which many children are brought to our offices. One day the mother or the father says, "You are having trouble with such and so, and we are going to see a doctor about this." Often without further explanation the child is taken to the family car, where he or she sits *passively*, until he or she is escorted to a strange waiting room, where once more the child *passively* waits until initiative is further taken from the child by his being led into the consulting room. Then the child is invited to become *active*. How strange this must seem to the child! One way to avoid this dilemma is to come to an agreement with the parents about the time that the child can be seen. If it is possible, make more than 1 hour available in order to allow the child a choice in case there is some activity that is especially important to *the child* that might compete with the agreed-on hour. Then ask the parents to have the child call to make the appointment. Some parents say that the child will not call, but few children actually refuse to do so. When they do call from the safe environment of their home, the therapist can demonstrate awareness and respect for the fact that the child has activities that are important to him or her, and also demonstrate that language or concepts that will leave the child hopelessly perplexed will not be used. An added advantage of this is that it demonstrates to the child that what he or she has to say or show will be heeded ([Coppolillo, 1987](#)).

The therapeutic alliance, as essential for any child therapy as it is for adult work, has already begun with the phone call. In the succeeding hours it must be continued, developed, and fortified. It must be stated as well as demonstrated to the child that no conclusions will be reached without the child's awareness and, whenever possible, participation. The child must come to realize that the clinician will not "spring something" on him or her through a gimmick or ploy, and that the work will be accomplished with cooperative respect. The child will produce the data that the clinician and child will examine together to reach conclusions. Phrases like, "We'll put our heads together to see if we can figure out what things mean" are helpful in forging and reforging the therapeutic alliance. As part of this alliance and the therapeutic contract, the therapist must convey to the child the message that play will be primarily for the purpose of helping them both to understand what causes some of the child's troublesome feelings and behavior to occur. To fail to mention this and then, when the child is lost in play, to interpret the play tends to make the child feel what we feel when in a social situation we make a painfully revealing slip of the tongue and that is then "interpreted" by a "parlor analyst" who feels impelled to interpret the slip for all to hear. The child is far less troubled when he knows that his play will be used for revelation and the interpretation begins with a phrase something like, "I wonder if your play means that . . ."

Although the suggestions in the preceding paragraphs apply to the whole phase of the beginning of treatment, it should be remembered that the foundations for the therapeutic alliance are laid during the first contacts with the family. To ignore this may mean that, if treatment is recommended after the diagnostic workup, the therapist may have to undo some distasteful or even frightening convictions that the parents or child may have drawn about the child's treatment during the early contacts with the therapist.

Another necessary element in treatment planning that influences the therapeutic alliance is setting the number of times per week and the hours that the patient is seen. Although it may not be possible to accommodate the child's wishes as to the time that he or she can be seen, thoughtful consideration to the child's schedule is a powerful support to the alliance.

Setting the frequency of the sessions requires consideration of a number of variables. The finances of the parents and the therapist's fees are one variable; however, the nature of the pathology and what its treatment will require should be the central consideration. There is another issue frequently not considered. The child's improvement in the therapeutic sessions counts for little if there is not concomitant improvement in his or her feelings and behavior in the everyday world. This requires working through the conflicts uncovered in treatment. Often this working through cannot be accomplished in the home environment, because the parents inadvertently keep the conflict alive in the adaptation that the family group has made to the child's behaviors. In addition to consultations with the parents, the child needs a milieu in which he or she is free to try out the different behaviors that result from the resolution of conflicts. If the child is scheduled so tightly that he or she has little or no time for spontaneous play with peers or the relaxation that supports spontaneity, scheduling more sessions may be counterproductive, especially if the scheduled treatment hours are at the expense of his "free time." Therefore, setting times should be done with an eye to how the therapeutic hours will influence and be influenced by the rest of the child's daily life.

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#### CASE ILLUSTRATION

A 9-year-old girl was being seen for a delayed and inhibited grief reaction occasioned by the loss of her father when she was 4. She had been given hours before school largely because her after-school hours were filled with all manner of activities (which were also used as defenses against the recognition of her unremitting loneliness). A phase of her therapy that was shallow and unproductive had lasted for several months. During this phase she had taken to drawing a number of "wilderness scenes" in which the woods and mountains she drew contained various animals that were depicted and described as busily going about their customary activities without interacting in any way. When the therapist commented on the absence of interaction and that the animals must be lonely, she first associated to the idea that if they stayed busy they would not fight, and then to the more general notion that if they stayed busy they would not feel any disturbing affects. After the therapist likened this to her staying busy in general and in her treatment hours in specific, she could acknowledge that she was afraid that if she cried during her treatment hour she would go to school with red eyes and everyone would wonder why. It was only after a conference with her mother, a reduction of the after-school activities that she had scheduled, and the setting of her appointments after the school day that she could begin to see that the reluctance to experience and show sad affects was based on her fear that her grief would upset her mother (and in the transference, her therapist). It was only when the real concern that she would be embarrassed by her red eyes in school could be addressed that she could address *neurotic* conviction that her negative affects would disintegrate her mother. Few defenses are as effective as a bit of reality that is used as a defense.

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Having attended to the notion that events prior to formal therapy may influence the therapy itself, we can turn to those technical considerations that render the process of play useful for therapy.

### PLAY IN THE SERVICE OF PSYCHOTHERAPY

A paradox to be reckoned with in the use of play is that play is so revealing that we sometimes fail to see what it conceals. If in watching a child play we come to a conclusion prematurely and misunderstand the significance of the play or understand only one part of the play, the misunderstanding or partial understanding can be used as a defense against revealing more important significances. The therapist must be ready to wait for the cues and learn to avoid taking assumptions made from his or her own past experiences for granted.

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#### CASE ILLUSTRATION

Joey, a bright 6-year-old boy, was being treated for his tendency to isolate himself from his parents and his passive disobedience at home and school. He had been in treatment before, which his parents had deemed ineffective and had terminated. His former therapist stated that all Joey had wanted to do was to play board games. In one of the early sessions of his second treatment Joey picked up some checkers and went to the table where children usually played. The therapist, sure that the child again wanted to play a board game picked up the checker board on his way to the table, and as he picked it up he remembered the comments of Joey's former therapist. He replaced the board and sat down to see what Joey would do. Joey said nothing but began to use the checkers as blocks. He built a wall between himself and the therapist, commenting on how difficult it was to build a good wall with checkers. The therapist answered that if Joey really wanted to show him how important a wall was, he would get some blocks with which he could more easily build a wall. The blocks became unnecessary. The discussions about walls between people were necessary and revealing.

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Even the therapist's very natural presupposition that Joey wanted to play a board game with him would have led to a compliant and counterproductive defense on Joey's part in this instance. Thus the adage: "Wait until the cues are clear to make an inference or an interpretation."

Generally there are four ways that children play during their treatment hours. They can become lost in play and play alone in the presence of their therapist. They can play alone but with a demand, either explicitly stated or conveyed implicitly through body language, that the therapist watch their play attentively. They may play with the therapist in unstructured play with no rules other than that the game evolves. They may express a wish to play a structured game such as a board game, guessing game, or a hiding game. The meaning of each of these modes will eventually become clear if the therapist keeps a number of principles in mind.

The first of these principles is that while minding the content of the play the therapist must also notice the process by which the play is chosen and how it progresses.

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#### CASE ILLUSTRATION

Tommy was a 10-year-old boy with little self-esteem despite a remarkable talent for drawing and better than average intelligence. In the course of his treatment he would suddenly break off discourse or some form of play and would ask to draw. He would then engross himself so deeply in his drawing that he would not attend to questions about the content of his picture or the techniques he had used to produce it. When finished with what was usually a remarkably good drawing he would present it to the therapist and give bland, nonrevealing answers to anything that he was asked about the picture. One day he came to the office ahead of his mother and announced that he wanted to draw a picture. Before the therapist and Tommy could move to the inner office the mother arrived and announced that she had to tell the therapist that Tommy had gotten grades that were far below his competence in a number of his classes. The therapist waited until they were in the inner office then recalled that Tommy felt his compelling desire to

draw on a number of occasions when they had discussed or gotten close to discussing issues that Tommy felt would cast him in a bad light. When the therapist mentioned that he had just begun to notice when Tommy wanted to draw, Tommy himself said, "Yeah. When I get in trouble." By noticing the process rather than being stuck on the content of the drawings, the therapist and Tommy found a great amount of material to explore together.

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Another issue to contemplate is that of body language that serves communication during play. Recall the pinched, tight control of the perfectionistic youngster who is attempting to draw the perfect picture to win approbation. Think of the increasingly less purposive movements of the impulsive child who gets "carried away" by aggressive impulses while playing with toy soldiers. And who can ignore the apathy and anhedonia conveyed by the lack of tone and movement of the depressed child?

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CASE ILLUSTRATION

Annie, a 4-year-old girl who had lost her mother, displayed both the longing for close contact as well as the rejection of substitute relationships in her play. On a number of occasions she demanded that the therapist sit on the floor near a dollhouse as she played with the dolls. Several times she stumbled "accidentally" or had another accident that would land her in the therapist's lap. On other occasions she would clearly avoid any physical contact with the therapist. In one instance she asked the therapist to fix the clothes on the doll with which she was playing. She shrank away as the therapist reached for the doll she was handing him. The therapist, by asking her to notice her movement, made it possible to discuss her ambivalence about feeling close to anyone else while mourning her mother.

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The third issue that requires attention as play progresses is the previously mentioned countertransference that can occur at any point. These are subtle and can be insidiously destructive to the therapeutic process. Countertransference may take any number of forms. They are facilitated by the regressive pull of the child's immaturity and his or her play and generated by a variety of states and conflicts in the therapist. Lewis offers a list of examples of countertransference in the preceding chapter on intensive, individual psychotherapy of children ( [Chapter 79](#)). The only comment to add here is that when countertransferences occur in a positive form, as in the wish to rescue or defend the child, or be protective of the child or when he or she is already being protected, they are much more difficult to detect. Negative feelings toward the child become a foreign body in the awareness of the therapist more readily than those that are compatible with our self-image of loving, caring adults. However, positive countertransferences can be as destructive to the process as negative ones ( [Coppolillo, 1969](#)). Rather than think of these as sins that must be abolished, it is useful to view them as additional cues to consider regarding the nature of the transaction. The therapist who understands that he or she is vulnerable to rejection, for example, may come to understand that a feeling of irritation or boredom is being occasioned by a withdrawn child's need or wish to become "lost" in solitary play in order to avoid the burden of relating. Again, the therapist who depends on the affection of the child may find that the child seeks play that he or she thinks will please the therapist in order to charm or seduce the therapist away from recognizing conflicted, negative feelings in more spontaneous play.

Finally, an eye must be kept on the content of the play. Here the help of the child is needed in order to ascertain in what way the play is significant *to the child*. Questions about what is happening in the play can be asked both when the child is playing alone and when the play is interactive. When the child has assigned the therapist a role in the play and demands that he or she act out the role, asides or stage whispers can be used as parentheses in which the questions may be posed.

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CASE ILLUSTRATION

A 4-year-old child, whose mother had suffered a serious accident after the child had been sent to bed for her afternoon nap, was playing out her fear and chagrin about being coerced to go to sleep. She made the dolls with which she was playing be sent to bed by the therapist and then had them hop out of bed to ask for drinks, food, and other favors.

Child, holding the doll in the therapist's face: "I want some milk."

Therapist, in a stage whisper behind his hand: "What does he say?"

Child, in a stage whisper: "He says 'go to bed.' "

Therapist, gruffly: "Go to bed!"

Child puts the doll in the doll bed while she pretends to be crying.

Therapist, again in a stage whisper: "What do you wish he had said?"

Child: "Why can't you sleep, little girl?"

His inquiry allowed the therapist to then meet her plea for understanding instead of coercion.

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In addition to inquiry about the content of the play, the therapist must continually look for the associations that may explain the shift from one play activity to another.

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CASE ILLUSTRATION

An 8-year-old boy, the son of a physician, had played "going to doctor school" in the therapist's office by looking through an anatomy text. When it became clear to the therapist that the child was demonstrating more than a passing interest in the genitalia and breasts in the female illustrations, he asked him if the medical school play was to hide his embarrassment at his curiosity about women's bodies. The boy was able to acknowledge this. Later in treatment the therapist noticed that on several occasions the boy returned to the medical school play after having talked about his sister. With some gentle questioning the therapist discovered that the boy was chronically anxious and stimulated because of the fondling and exploration to which he had been subjected by an older sister.

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On occasion, a seemingly disconnected or spontaneous comment during play offers a revealing association.

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CASE ILLUSTRATION

A 4-year-old boy, brought to treatment for voluntary retention of feces and fear of using the potty, became obsessed with flat tires in play. The toy cars he played with always developed flat tires. He drew cars with flat tires and repeatedly requested that the therapist draw cars with flat tires for him. On one occasion when the therapist was drawing one of these cars for him, the boy commented that the therapist had a big belly. When the therapist asked him what he meant and how it was connected to the cars, the boy answered, "Your belly is round and not flat." Having made the connection of flatness between car tires and bellies, the boy could tell of when he was riding with his father in the car when they had a blowout that sent the car out of control and frightened him. He could then say that he was afraid of getting flat (out of control) if he went to the bathroom.

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Finally, the therapist must allow some disciplined play to his own creativity and imagination in making abstractions about the content and process of the play he is observing. For example, if a child is playing with cars and labels one a fire truck, another a police car, and still another an ambulance, the abstraction that would occur to the therapist that would tie them together would be responses to an emergency. Then, depending on the age of the child, and with his history in mind, the therapist might be able to determine if the child was in the process of mastering a trauma that he had experienced, like a home fire, or was symbolically conveying anxiety about something unpredictable or uncontrollable occurring such as an impulse getting out of hand.

And so, by having the patience and interest to wait for the cues, the serenity to keep an eye on his or her own countertransference, the imagination and creativity to envision patterns of significance in the content and process of the child's play, the therapist can, with tact and kindness, return to the child, in a manageable form that which the child has anxiously revealed symbolically in his or her play.



## CONCLUSIONS

Theoretical considerations often provide frames of reference that permit the user of a technique of disciplined creativity in the use of that system. Instituting rules for the use of a technique often does just the opposite, by simply constricting rather than enabling. For this reason, a brief review of how the mentioned concepts of Winnicott and Rapaport are indicated, because they specifically apply to the therapeutic use of play.

[Winnicott \(1953\)](#) demonstrated how bringing an object into the “intermediate area of experience” alleviated the strain of relating external reality to the inner world of wishes and subjective convictions.

[Rapaport \(1951, 1958\)](#) described how attending to the inner world of drives and impulses, as well as to one's own values and self system, presents a stimulus barrier to the world of external reality and shelters one from slavery to external circumstances. He continued his exploration by noting that, conversely, human beings were not simply expressions of sublimated drive activities. Both the autonomous functions of the ego and attentions to external reality provide the human being with a measure of autonomy from the inner world.

Integrating the work of these two authors ([Coppolillo, 1976](#)) permits us to appreciate the powerful effects of play in the adaptation of the human being to his or her environment. In the therapeutic use of play, we may join Winnicott in considering the immersion into play as providing a “holding environment” ([Winnicott, 1965](#), pp. 50–51). The privacy, and nonthreatening and nonintrusive qualities of the ambiance then act as a shield against any coercive qualities the child may ascribe to the external world, and thus increase the child's sensitivity to his or her inner world. At the same time, “the reality” of the therapist's presence, his or her absence of fear or anxiety about the child's impulses, the regularity of the hours, and the therapist's commitment to understanding rather than judging, reassure the child that his or her own impulses will not be overwhelming. Into this setting a toy, game, or idea can be taken into the intermediate area by the child and there further regulate and titrate the amount of anxiety the child can tolerate as either fear of his or her impulses or dangers ascribed to the external world. For example, if the child is playing out destructive fantasies with the use of soldiers and an impulse to destroy becomes too transparent or intense, the child may turn his or her attention to the realistic quality of the soldiers and decide that they are too small, too few, or just toys. If aspects of reality become too coercive or boring, the child may allow himself or herself to be a bit more lost in the unrealistic quality of the play and imbue the toy with more aggression or creative ways to bring down the opposing general. With each new awareness achieved through these adventurous explorations, the child becomes more secure, expansive, and above all, more confident that the revelations his or her play brings will be useful and not threatening or destructive. In this way, more profound and subtle issues can be addressed in the process of play until one day, with that bit of sadness that accompanies all developmental steps, child and therapist recognize that the healing brought by play no longer needs the catalytic action of the therapist. Even without being aware of it, the child has developed another function for play, another way to find healing and soothing.

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# 81 CHILD AND ADOLESCENT BEHAVIOR THERAPY

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## HISTORY

The field of child and adolescent behavior therapy has become much more accepted into the mainstream practice of psychotherapy, but it nevertheless remains often misunderstood and thus misapplied. The general definition of behavior therapy is quite broad and frequently refers to a variety of approaches related only by their reliance on standardized methodologies and measurable outcome results. Although the more widespread use of behavior therapy is encouraging, the increase in untrained clinicians who bill themselves as behavior therapists is unfortunate. Too often, unsuccessful treatment attempts are attributed to inadequate behavior therapy techniques rather than inept clinical applications of appropriate interventions. This chapter attempts to define and clarify the basic assumptions of behavior therapy, introduce its applications, and discuss the current issues in the field.

Behavior therapy refers to a set of related assumptions, principles, and techniques that are rooted in learning theory and used to change human behavior. Although the intellectual foundations of behavior therapy date back to the 17th century empiricism of Locke and the 19th century utilitarianism of Bentham, behavior therapy's more recent past can be divided into two relatively distinct periods: an early history dating from approximately the turn of the 20th century to the early 1950s, and a more recent period dating from the 1950s to the present.

The early history of behavior therapy is characterized by seminal contributions from key figures that frequently were not recognized for their relevance to human problems until the mid-20th century. Examples of this are the work of [Ivan Pavlov \(1927\)](#) on the conditioned reflex, Edward [Thorndike \(1911\)](#) on connectionism and the law of effect, and John B. [Watson \(1913\)](#) on behaviorism. Watson and his colleagues, Rosalie Raynor ([Watson and Raynor, 1920](#)) and Mary Cover Jones ([Jones, 1924a, 1924b](#)), were particularly influential in drawing popular attention to the importance of behavioral principles of conditioning in shaping human behavior. In addition, they were the first to show that these principles could be used to ameliorate infant and childhood problems. Despite these and other efforts, behavioral approaches were overshadowed during this period by the prevailing *zeitgeist*, then dominated by psychoanalysis and the emerging child guidance movement. Even a 1938 appeal by Arnold Gesell, a leader in that movement, for professionals to pay greater attention to behavioral principles had little impact on the use of behavioral approaches ([Ross, 1981](#)). The subsequent influence in the 1940s of Carl Rogers and his client-centered therapy further delayed the acceptance of behavioral approaches ([Meyers and Craighead, 1983](#)).

It was not until the 1950s that mental health professionals began to adopt behavioral approaches more widely for the treatment of mental disorders. This period was marked by a critical evaluation of the limitations of traditional therapeutic approaches to human problems and an eagerness to consider more scientifically based approaches. Works by [Skinner \(1953\)](#), [Wolpe \(1958\)](#), [Eysenck \(1960\)](#), and [Bandura and Walters \(1963\)](#) were particularly influential in demonstrating how learning theory principles could be used to ameliorate human problems. Patients with psychoses, severe anxiety neuroses, and autism became candidates for the application of the behavioral strategies and techniques identified in these works because few of the traditional approaches offered much hope for symptom improvement ([Ferster and DeMyer, 1962](#); [Ross, 1981](#)). The success of these approaches in the 1950s and early 1960s with the most seriously disturbed adults and children enhanced the credibility of behavior therapy and increased its acceptance among professionals ([Kazdin, 1978](#)). This heralded the appearance of a host of behavioral journals during the 1960s, which began to provide a scientific basis for the field. Success with more disturbed patients also prompted professionals to begin to apply behavioral approaches to a wider range of problems, particularly to those presenting in outpatient settings. This prompted behavior therapists to reconceptualize the role of cognitions in changing behavior, such that they came to be viewed not only as a potential *target* of treatment but also as a possible *mechanism* for behavior change. The integration of cognitions into behavior therapy, coupled with developments in the emerging field of information processing, sparked the development of social learning theory and cognitive-behavioral approaches in the late 1960s and early 1970s ([Meyers and Craighead, 1983](#)). The latter approaches dramatically increased behavior therapy's scope, the settings in which it could be practiced, and its potential for human betterment. Today, behavior therapy includes such a diverse array of therapeutic principles, practices, targets, and settings that it may be more accurate to use the term behavioral *interventions* to describe the additional preventive, promotive, and consultative efforts in the field.

## BASIC ASSUMPTIONS

Behavior therapy rests on a common set of assumptions that distinguish it from other forms of therapy. The primary assumption is that most behavior develops and is maintained according to the principles of learning ([Herbert, 1988](#); [Johnson et al., 1986](#); [O'Leary and Wilson, 1987](#); [Rimm and Masters, 1979](#)). Learning may be defined as a process in which behavior is initiated or is changed in reaction to internal or external stimuli that are not the result of "native response tendencies, maturation, or temporary states of the organism" ([Hilgard and Bower, 1966](#), p. 2). Specific behavior therapy techniques are derived from each of the four types of learning: respondent conditioning, operant conditioning, cognitive behavior modification, and social learning.

The principles of respondent conditioning are perhaps the most widely known among these. Respondent conditioning is based on the observation that certain behaviors, known as *respondents* (e.g., salivation, increased heart rate), are elicited involuntarily in response to certain stimuli (e.g., having food in one's mouth or being startled by a loud noise). Such behaviors are known as *unconditioned reflexes* or *unconditioned responses* because they occur "automatically" in response to a particular stimulus. Stimuli that are able to elicit such responses are known as *unconditioned stimuli*. When unconditioned stimuli are paired repeatedly with a previously neutral stimulus, eventually the neutral stimulus is able to elicit a conditioned response that usually resembles the unconditioned reflex. This was first demonstrated by [Pavlov \(1927\)](#) in a now-classic experiment with dogs, in which he paired a previously neutral stimulus—a bell—with the presentation of food, such that eventually his dogs began to salivate merely at the sound of the bell. Respondent conditioning in humans was first demonstrated by [Watson and Raynor \(1920\)](#) in their experiment with Albert, an 11-month-old infant. After allowing Albert to play comfortably with a furry white rat, the experimenters began to startle Albert with a loud noise (an unconditioned stimulus) whenever he began to play with the rat. After only seven pairings, Albert's pronounced startle to the noise was capable of being elicited exclusively by the sight of the rat, thus revealing a conditioned fear response. Importantly, Albert's fear also was elicited to a lesser degree when he was



in the presence of other furry animals and objects, illustrating the related principle of *stimulus generalization*. [Seligman \(1971\)](#) has argued that humans are more “prepared” to be conditioned to certain objects, thus accounting for the observation that persons are more likely to be conditioned to some objects than to others.

Another form of learning is *operant conditioning*. Based on Thorndike's observation that behavior is strengthened or weakened by its consequences, operant conditioning was first identified by [Skinner \(1938, 1953\)](#) as involving behaviors that could be modified or maintained by their consequences. Skinner termed such behaviors *operants* because they operate on the surrounding environment to generate consequences. Thus, behavior that is followed by pleasant consequences is likely to increase in frequency, whereas behavior followed by unpleasant consequences is likely to decrease in frequency. Operants differ from respondents in two primary ways: (a) They involve voluntary behavior that is “emitted” (i.e., as thoughts or actions) rather than “elicited” (i.e., as involuntary reflex responses); and (b) in the stimulus–response science of behavior, they are linked to properties of the response (e.g., its strength, frequency, duration) rather than to those of the stimulus ([Hilgard and Bower, 1966](#)). Because [Skinner \(1953\)](#) argued that most human behavior is operant in character, he advocated that the principles of operant conditioning be used to solve many types of behavior problems, an approach known today as *applied behavioral analysis*.

A third type of learning is *cognitive behavior modification* ([Beck, 1976](#); [Ellis, 1962](#); [Mahoney, 1974](#); [Meichenbaum, 1977](#)). This has been described as an attempt to integrate internalism with behaviorism because of the primacy of cognitive and symbolic processes in mediating behavior ([Meichenbaum, 1977](#)). A basic assumption of cognitive–behavioral approaches is that cognitive processes (e.g., attributions, cognitions, expectations, beliefs) influence one's behavior and affect. Irrational and faulty cognitive processes foster the development of maladaptive behavior patterns that are best reversed directly through the modification of maladaptive cognitions. Techniques such as cognitive restructuring and problem solving are common tools used by cognitive–behavioral therapists to correct maladaptive cognitive processes that are believed to cause behavioral problems. Cognitive–behavioral approaches do not concern themselves with intrapsychic, biological, neurologic, or genetic factors in the development of child behavior problems ([Kendall, 1985](#)). Rather, a major focus of cognitive–behavioral approaches is determining how cognition and behavior influence the development of child behavior problems. Although cognitive–behavioral approaches place less emphasis on the influence of affective, familial, and social processes on human behavior, they seek to integrate understanding of these processes with cognitive and behavioral factors to produce change (Kendall, 1993).

*Social learning theory*, also known as *social cognitive theory*, describes the final category of learning ([Bandura, 1969, 1977, 1986](#); [Bandura and Walters, 1963](#)). Social learning theory is an attempt to integrate the two types of conditioning theories with our understanding of cognitive processes. According to [Bandura \(1977\)](#), learned behavior is governed by three types of regulatory processes: paired stimulus–response events (as in respondent conditioning), environmental consequences (as in operant conditioning), and symbolic cognitive processes (described partially in cognitive behavior modification). The most important among these are cognitive processes, and particularly those that involve observational learning, a process in which behavior change occurs by observing a model. For example, a child who views another child being rewarded for a particular behavior is more likely to perform similar behaviors. The centrality of observational learning in social learning theory is one of its distinguishing features. Another important feature is the view that behavior is characterized by “triadic reciprocity”; that is, behavior is determined by the reciprocal interplay among three forces: behavior, cognitive and other personal factors, and environmental influences ([Bandura, 1986](#)). A final distinguishing characteristic of social learning theory is that it emphasizes the person's capacity for self-directed change. The latter two characteristics are clear departures from the more deterministic view of human nature and causality offered by respondent and operant conditioning theories, and to a lesser extent by cognitive behavior modification ([Bandura, 1986](#)).

A second major assumption of behavior therapy is that observable behavior should be the primary focus of therapy ([Kazdin, 1985a](#); [Rimm and Masters, 1979](#)). Subjective experiences such as feelings and cognitions, as well as psychophysiological processes such as electrodermal activity, are viewed as integral and essential elements of the therapeutic enterprise as long as observable behavioral referents for their occurrence are identified. In general, behavior therapists eschew treatment formulations that hypothesize a distal underlying cause for an observed behavior, preferring instead to focus on environmental contingencies proximal to the behavior in question. In this connection, although behavior therapists acknowledge the importance of biological and genetic factors that may contribute to the emergence of a problem behavior, they seek to identify and modify environmental contingencies that are presumed to maintain it in the here and now.

A third major assumption of behavior therapy is that treatment techniques should be empirically based ([Johnson et al., 1986](#); [O'Leary and Wilson, 1987](#); [Rimm and Masters, 1979](#)). Many of the techniques behavior therapists use were derived from laboratory experiments and have their roots in experimental psychology. As a result, behavior therapists view their work as an “applied science” ([O'Leary and Wilson, 1987](#)) because it involves the application of these techniques to clinical problems. A related byproduct of this development is that behavior therapists emphasize the setting of clear and precise goals in treatment, which enables them to validate treatment goals and outcomes empirically.

A fourth major assumption of behavior therapy, related to the previous one, is that treatment should be action oriented and directive ([London, 1986](#); [Rimm and Masters, 1979](#)). This assumption is based on the belief that action, rather than insight, is the primary mechanism for change, and that symptom change is the appropriate focus of treatment rather than some ill-defined and unspecifiable “underlying cause.” The primacy of action means that behavior therapists take an active, directive role in treatment (“homework” is assigned for patients to complete between sessions, specific treatment goals and strategies are discussed directly with patients, behavioral contracts between therapist and patient or between parents and child are negotiated in the session, and so forth). Practice is encouraged, interpretation rarely is used, and empathic listening is used as long as it promotes the accomplishment of behaviorally based treatment objectives.

A fifth and final major assumption of behavior therapy is that behavior change is best maintained through enlisting the help of key persons in the client's environment ([Herbert, 1988](#); [Kazdin, 1985a](#)). In the case of children, this usually means teaching behavioral principles to parents, teachers, and other family members and establishing a treatment plan that provides for the generalization or “transfer” of treatment from the clinic to the child's natural environment, such as the home or school. Underlying this assumption is the belief that much behavior is situation specific and that proximal environmental contingencies can best be addressed by training key persons in the child's environment who can help maintain therapeutic gains.

## BEHAVIOR THERAPY TECHNIQUES

The techniques of child and adolescent behavior therapy can be categorized as attempting to produce either of two kinds of behavior change: (a) to strengthen, develop, or maintain behavior; or (b) to reduce or eliminate behavior. Although these techniques can be used for a wide range of problem behaviors, only the most common applications are summarized in the following sections.

### Techniques to Strengthen, Develop, or Maintain Behavior

In operant conditioning, *reinforcement* is the process by which behavior is strengthened by its consequences. In the case of *positive reinforcement*, a reward (or reinforcer) is presented after the occurrence of the desired behavior; in *negative reinforcement*, the reward involves removal of an aversive stimulus after the desired behavior occurs. Both positive and negative reinforcement differ from punishment because their purpose is to *increase* the likelihood that a particular behavior will occur. Punishment, on the other hand, is aimed at *reducing* the incidence of a particular behavior. Reinforcers can be either tangible (i.e., involving concrete, material items, such as food, money, privileges, opportunities to engage in specific activities or behaviors, and removal of sanctions) or intangible (i.e., involving social or related items, such as encouragement, praise, and smiles) ([Herbert, 1988](#)). In addition, reinforcers can be external (i.e., obtained from others) or internal (i.e., provided to oneself). An important property of reinforcement is that behavior is strengthened differentially by the schedule of reinforcement used. Reinforcement that is administered immediately after each time a desired response occurs is known as *continuous reinforcement*. Other types of reinforcement schedules involve some form of *intermittent reinforcement*. In a *fixed interval* schedule, the child is reinforced after a specific time, regardless of what response is emitted, whereas in a *variable interval* schedule, the rate at which reinforcement is administered varies randomly around a specific average. A *fixed ratio* schedule administers reinforcement after a specified number of the child's desirable responses, whereas a *variable ratio* schedule administers a reinforcer randomly around a specific average of desirable responses on the part of the child. In general, learning that results from intermittent schedules of reinforcement—and, in particular, variable interval or variable ratio schedules—is more stable and difficult to change than that acquired through continuous reinforcement. The treatment of compulsive gambling is an example of how an intermittent reinforcement schedule can lead to behavioral persistence and high rates of responding.

Positive and negative reinforcement are techniques frequently used to teach children skills to compensate for behavioral, learning, and other skill deficits. In addition, reinforcement often is used in combination with other techniques designed to strengthen or eliminate behaviors.

A second important behavioral change technique is *stimulus discrimination training*. This involves reinforcing a particular response in the presence of one stimulus while not reinforcing that response in the presence of another stimulus. Stimulus discrimination training enables the child to learn how to discriminate when and under what conditions it is most appropriate to emit a particular response, and when it is not. Three techniques are used in stimulus discrimination training: shaping, fading, and chaining ([Martin and Pear, 1983](#)). *Shaping* involves reinforcing closer and closer approximations of behavior to produce the final desired behavior. Thus, a

teacher of a severely mentally retarded child might initially reward the child for standing near a desk, then sitting in the desk, then engaging in playful behavior while sitting in the desk, then for on-task classroom behavior while sitting in the desk. This is accomplished by varying when and under what conditions reinforcement is provided. *Fading* involves gradually changing a stimulus that controls a response so that eventually a new, alternative stimulus produces the same response. Thus, an autistic child who only mimics the word *shoe* might be provided with a reinforcer (e.g., praise, hugs, tokens) every time he or she repeats “shoe” in the presence of an additional prompt, such as “What’s on your foot? Shoe.” Gradually the prompt “shoe” is faded until the child is able to respond directly to the question “What’s on your foot?” *Chaining* involves reinforcing more and more of the stimulus–response links that comprise a complex chain of behavior, such as using a fork to eat. This procedure usually involves the use of shaping or fading techniques to achieve stimulus control of the desired behavior. Thus, in chaining the therapist might demonstrate an entire behavior to be learned and then reinforce a child for his or her attempts to complete the entire behavior, as well as for specific successive approximations of the behavior, as is done in shaping. Shaping, fading, and chaining commonly are used in conjunction with other behavioral techniques, such as reinforcement, to teach children skills to compensate for behavioral, social, or other skill deficits.

*Conditioned reinforcement* also is used to strengthen, develop, or maintain behavior. In conditioned reinforcement, a particular stimulus—the conditioned reinforcer—signals the likelihood that reinforcement is forthcoming. Over time, the stimulus itself becomes reinforcing (i.e., it becomes conditioned) because of its association with the receipt of reinforcement. Thus, parental praise is a conditioned reinforcer because it signals the likelihood that other reinforcers, such as food, privileges, affection, and so on, are forthcoming. Tangible conditioned reinforcers that can be earned and exchanged for other reinforcers typically are referred to as *tokens*. A behavioral program that uses tokens systematically to produce behavior change is known as a *token economy*. Token economies have been used with considerable success in a wide variety of settings, such as schools, inpatient units, and group homes, as well as in the home by parents ( [Kazdin, 1977](#)).

*Contingency contracting* is another behavioral technique that is used primarily to increase specific behaviors, but also may be used to reduce inappropriate or unwanted behaviors (Herbert, 1987; [Kazdin, 1989](#); [Spiegler and Guevremont, 1993](#)). Contracts may be verbal or written and may be used in conjunction with conditioned reinforcers, such as tokens, to reinforce target behaviors. Written contingency contracts should specify the following in unambiguous terms: (a) the responsible parties involved (e.g., the parent(s) and the child); (b) the target behavior(s) to be increased (e.g., studying, school attendance) or eliminated (e.g., smoking, physical aggression toward a sibling); (c) the consequences for completing or failing to complete the target behavior (i.e., the behavioral contingencies); and (d) the time interval during which the contract is in effect (e.g., weekly, monthly). For some contracts, it also may be necessary to specify who will monitor some target behaviors (e.g., a teacher) or how those behaviors will be monitored (e.g., daily school report). An example of a written behavioral contract that parents might use to increase several specific target behaviors of an adolescent is found in [Fig. 81.1](#).

Child agrees to do	Parent agrees to do
1. Get ready for school (wake up on the alarm, get dressed, eat breakfast, brush teeth, and get out to the school bus on time every day).	1. Make breakfast, get ready, and send child off to school on time every day. If child is late, parent will bring to school every day and a reward of \$10 or the value of the week.
2. Complete homework. Complete daily homework, complete all projects on time, and complete all projects on time.	2. Give child \$10 if the child completes all homework on time. If the child does not complete all homework on time, parent will give the child a reward of \$10 or the value of the week.
3. Complete chores. Take out trash on Tuesday nights, clean shower on Wednesday and Thursday, and clean the kitchen on Friday.	3. Pay weekly allowance of \$10 on Friday if child completes all chores on time.
4. Engage in "family" time (reading with parent, watching TV, playing board games, etc.)	4. Engage in "family" time (reading with parent, watching TV, playing board games, etc.)

Notes: 1. If more than one child is involved, the contract will be modified for each child.  
2. If the child fails to complete a task, the parent will be grounded for 1 week (not to be used as a punishment).  
3. I agree to abide by the above contract to the best of my ability.

Child: \_\_\_\_\_  
Parent: \_\_\_\_\_

**Figure 81.1.** Sample contract between (child \_\_\_\_\_) and (parents \_\_\_\_\_) from the week beginning Sunday, \_\_\_\_\_.

Modeling represents a behavioral change technique that involves having a child observe a model engage in a particular sequence of behavior for the purpose of producing behavior change. Three types of modeling have been identified: live, symbolic, and participant ( [Bandura, 1969](#)). In *live modeling*, a child observes a model engaging in the target behavior *in vivo*; in *symbolic modeling*, a child views the model on film or videotape or imagines himself or herself engaging in the behavior; and in *participant modeling*, a child observes a model engaging in the target behavior *in vivo* and then attempts that same behavior. Modeling can have a number of different effects: acquisition of a new behavior, social facilitation of observed behavior, enhancement of environmental influences, arousal of specific emotions, disinhibition of behaviors that previously were avoided, and extinction of behaviors associated with persons or objects that were observed ( [Bandura, 1986](#)). (The latter two involve effects that reduce or eliminate problem behaviors.)

A final set of behavioral change techniques that can be used to strengthen, develop, or maintain behavior, as well as reduce or eliminate behavior, is skills training approaches. Two types of skills training approaches are described most commonly: *social skills training* and *cognitive skills training*. The aim of social skills training approaches is to teach children the skills necessary to interact in socially appropriate ways with peers and adults. Typically, skills are taught in an analog or laboratory setting through a combination of behavioral techniques, such as positive reinforcement, modeling, coaching, instruction, role playing, and behavioral rehearsal ( [Hops et al., 1985](#)). For example, an extremely shy boy may be taught how to interact more assertively with peers by first viewing a model perform the target behavior and then being praised for each successive approximation of that behavior during role play. Therapist and child then rehearse this procedure until the child is ready to interact assertively with peers. Such techniques have been used extensively to teach appropriate social skills to socially isolated and withdrawn children, to mentally retarded and autistic children, and to children who are overly aggressive and antisocial ( [Dumas, 1989](#); [Hansen et al., 1989](#); [O’Leary and Wilson, 1987](#)).

Cognitive skills training approaches are a related type of technique that emphasizes teaching children cognitive skills that are presumed to mediate successful task performance. Also known as *self-instructional training*, *problem-solving training*, or *self-control training* (depending on the goals of the treatment), cognitive approaches are based on the cognitive–behavioral assumption discussed earlier, in which faulty cognitions mediate maladaptive behavior. As opposed to social skills training approaches, which emphasize teaching behavioral skills, these approaches focus on teaching children covert cognitive mediational techniques, such as corrective self-statements and covert verbalizations, to help regulate their behavior in various situations. In most training programs, the children use self-statements to identify the problem, generate alternative solutions, decide on one solution, evaluate the outcome of their choice, and provide covert positive self-reinforcement ( [Dumas, 1989](#)). This approach has been used successfully to promote adjustment to a wide variety of behavior problems in very young children, as well as in older children and adolescents. Although summarized here as a technique to strengthen, develop, or maintain behavior, cognitive skills training approaches often are used to reduce or eliminate childhood impulsivity, aggression, or fears ( [Christoff and Myatt, 1987](#); [Dumas, 1989](#); [Kendall and Braswell, 1985](#); [Meyers and Craighead, 1983](#)).

### Techniques to Reduce or Eliminate Behavior

In operant conditioning, *extinction* refers to the process in which reinforcement is withheld after an operant response so as to reduce the frequency of its occurrence. Ironically, undesirable behaviors usually *increase* in frequency for a brief period immediately after the introduction of an extinction procedure, although gradually they disappear in the absence of reinforcement. A common example of extinction is when parents attempt to help their young infant sleep for longer periods through the night. If a parent responds immediately to the infant’s cries after it is put to bed by holding it, the infant learns to associate crying at bedtime and being held. To unlearn or extinguish this operant response, a parent allows the infant to cry a few minutes before responding. If this practice is followed on successive nights by gradually increasing the parent’s response delay intervals by 5 minutes on each occasion, the infant’s crying will slowly be extinguished, so that he or she learns to fall asleep alone. Although useful as a procedure to reduce or eliminate some undesirable behaviors, extinction usually is not appropriate for the elimination of destructive or disruptive behaviors, which may require more direct intervention ( [Johnson et al., 1986](#)).

A technique for the reduction of problem behaviors that combines some of the properties of extinction with those of reinforcement is *differential reinforcement*. In this technique, reinforcement is given for the nonoccurrence or low rate of occurrence of the problem behavior. Three types of differential reinforcement have been identified: *differential reinforcement of other behavior* (DRO), *differential reinforcement of incompatible behavior* (DRI), and *differential reinforcement of low rates of behavior* (DRL). In a DRO procedure, a child is reinforced for not emitting the problem behavior during a specified time or response interval. In effect, what is being reinforced is every behavior *other* than the problem behavior. In a DRI procedure, a child is reinforced for emitting an appropriate behavior that is topographically incompatible with the problem behavior during a specified time or response interval. For example, a child who has trouble remaining on-task during class is reinforced for completing a specified number of assignments or for working on an assignment for a specified length of time. In a DRL procedure, a child is reinforced for emitting low rates of the problem behavior during a specified time or response interval. Thus, a child who frequently leaves his or her seat during class is reinforced for leaving the seat only twice during a specified time interval. DRO and DRL procedures provide the least amount of stimulus control in that alternative problem behaviors may be reinforced during a specified interval, along with appropriate behaviors. This is not the case with a DRI procedure because the emission of appropriate or prosocial



behaviors is required for reinforcement to occur. Differential reinforcement procedures are readily incorporated into school or institutional settings and are particularly effective in the reduction of disruptive, aggressive, and self-injurious behaviors ( [O'Leary and Wilson, 1987](#); [Martin and Pear, 1983](#); [Repp and Deitz, 1979](#)).

*Punishment* is a technique used to reduce or eliminate undesirable behavior through the introduction of an aversive stimulus or removal of a positive stimulus after an operant response. Scolding, spanking, or removal of privileges (e.g., watching television, going outside to play) are examples of punishment, if these are presented contingent on a child's behavior and reduce the frequency of that behavior. Punishment procedures differ from extinction procedures because they usually bring about a rapid decrease in the frequency of problem behaviors, thus making them particularly effective techniques for the reduction of self-injurious and aggressive behaviors and certain kinds of disruptive behaviors. Nevertheless, punishment also has many disadvantages that recommend against its regular use. It tends to suppress behavior temporarily rather than actually change it and may produce serious and unwanted side effects ( [Johnson et al., 1986](#); [Johnston, 1972](#); [Kazdin, 1982](#)), such as (a) the elicitation of fear responses that may become conditioned to the punisher or punishing situation; (b) the promotion of escape responses, such as physical or emotional withdrawal, after punishment is terminated; (c) the strengthening of behaviors that motivate the child to avoid the aversive situation and thus reduce the chance that more desirable behaviors can be learned; (d) reinforcement (and thus strengthening) of the undesirable behavior through negative attention the child receives during punishment; and (e) observational learning of other undesirable behaviors that may be modeled by the punisher for the child, such as aggressive physical or verbal behavior and disrespect for the rights of others.

Three common punishment procedures are time out, response cost, and overcorrection. In a *time out* procedure, the child is removed from the setting in which the misbehavior occurs and is placed in a restrictive environment for a brief period. The lack of opportunity to obtain reinforcers by having to remain in such an environment usually results in a rapid suppression of misbehavior. In the typical application of this procedure, the child is warned that a time out will follow if the misbehavior continues. If the child does not heed the warning, he or she is sent to the time out room (or area) and asked to remain there for 10 minutes (3 to 5 minutes for very young children). At the end of the designated time, the child is allowed to return to the previous setting. Time out has been widely used with success in classroom and institutional settings, as well as in the home. It has been found to be a particularly effective punishment technique for the reduction of disruptive or aggressive behavior, which is easily reinforced by peers ( [Foxy and Shapiro, 1978](#); [Hobbs and Forehand, 1977](#)).

*Response cost* is another common punishment procedure in which a reinforcer is removed contingent on misbehavior so as to reduce the future probability of its occurrence. Typically, response cost procedures are used in conjunction with a token economy program, but they also may be used in the home by parents who have established contingency contracts with their child, usually an adolescent. In such contracts, the parents agree to provide the child with rewards or privileges (e.g., use of the family car, extended use of the telephone) in return for completion of specific behaviors on the part of the child (e.g., coming home before curfew, cleaning one's bedroom). If the child does not keep the contract, payment is made by removal of agreed-on rewards or privileges. In the appropriate use of this procedure, the child is informed in advance that he or she will be penalized a specified amount of rewards, privileges, or tokens for a particular misbehavior. When a misbehavior occurs, rewards, privileges, or tokens are removed. This procedure has been used to reduce the incidence of a wide range of problem behaviors, such as disruptions in the classroom, crying, perseverative speech, aggressiveness, and hyperactivity ( [Rimm and Masters, 1979](#)). Response cost differs from time out because the child is not removed from the opportunity to gain reinforcers, making it particularly effective in promoting self-control and self-management.

A final, frequently used punishment procedure is *overcorrection*. Overcorrection involves having the misbehaving child correct the consequences of the misbehavior. In *restitutional overcorrection*, the child is required not only to restore the environment to its original state before the misbehavior but to improve it (e.g., a child who defaces a roommate's poster may be required to save enough money to replace the poster, as well as to do the roommate a particular favor). In *positive practice overcorrection*, the child is required to practice positive behaviors that are incompatible with the misbehavior (e.g., a messy eater may be required to practice eating correctly). Although these two procedures usually are not used in combination, for some problem behaviors, such as bed wetting, their combined use has met with success (e.g., a bed wetter is required to remove wet linen, remake the bed, and then repeatedly practice leaving the bed, walking to the toilet, and returning to the bed) ( [Azrin et al., 1974](#)). Major characteristics of overcorrection procedures are (a) they relate directly to the misbehavior by requiring the child to complete a chain of behavioral responses that are topographically similar to a correct response; (b) they are intended to have the child experience the effort required by others to correct the consequences of the misbehavior; (c) they are instituted immediately after the misbehavior so as to reduce the likelihood that positive reinforcement from the behavior can occur; and (d) "graduated guidance" is used to enforce completion of the overcorrection requirement, whereby the enforcing adult manually assists the child through completion of the overcorrection response ( [Foxy and Bechtel, 1982](#)). Although overcorrection has been shown to be effective in reducing the incidence of a wide range of aggressive, self-injurious, and self-stimulatory behaviors, as well as inappropriate toileting and oral behaviors (e.g., vomiting, drooling), it is an aversive procedure that holds considerable potential for abuse if not used with caution and discretion.

*Desensitization* refers to a broad class of related "exposure training" behavioral techniques that are based on classical conditioning principles and used to reduce children's fears. In desensitization, a child is gradually exposed to a conditioned stimulus (e.g., a dog, taking a test, being separated from one's mother) until the fear is extinguished. *Participant modeling* and *symbolic modeling* have each been used to desensitize a child from a conditioned stimulus. In participant modeling, the child observes another person (usually a child) in the presence of the feared stimulus and then is reinforced for gradually approaching the stimulus until the fear disappears. In symbolic modeling, the conditioned fear is extinguished by having a fearful child view a film in which an initially fearful child gradually overcomes the fear.

The most common form of exposure training to reduce fears is *systematic desensitization*. In this technique, the child first works with the therapist to establish a hierarchy of fears about the anxiety-provoking stimulus. For desensitization to have the potential for success, this hierarchy must be as detailed as possible. A sample hierarchy is presented in [Table 81.1](#) for a child with test anxiety.

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10	Attending class
20	Hearing the teacher announce that there will be a test at the end of the week
30	Attending class the day after the test has been announced
40	Doing homework in that same subject the day after the test has been announced
50	Attending class 3 days before the test
60	Studying for the test at home 3 nights before the test
70	Attending class 2 days before the test
80	Studying for the test at home 2 nights before the test
85	Attending class the day before the test
90	Talking to classmates about the test 2 days before the test
100	Sitting in class just before the exam is passed out

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<sup>a</sup>Items are ordered according to the amount of subjective anxiety they elicit from 10 (lowest) to 100 (highest).

**Table 81.1. Sample Items of a Desensitization Hierarchy for Test Anxiety for a 14-Year-Old Student<sup>a</sup>**

Once the fear hierarchy is established, the child is helped to feel relaxed by talking to the therapist or a parent, playing quietly with peers, or eating a favorite food. An alternative approach used regularly in systematic desensitization with adults is to have the child engage in deep muscle relaxation before exposure training ( [Cautela and Groden, 1978](#)). Relaxation is intended to function as an incompatible response to anxiety. Once the child is relaxed, the therapist asks the child to imagine the least distressing scene in the fear hierarchy and to indicate if the scene remains distressing (e.g., if the child is seated in an armchair, he or she may be instructed to lift an index finger to indicate distress). If the scene continues to elicit anxiety, the child is instructed to relax for a few seconds, and then the procedure is repeated with the same scene until it no longer elicits anxiety on three successive presentations. The therapist repeats this procedure with each successive distressing scene in the hierarchy until the child no longer reports being distressed by the feared stimulus. Typically, no more than three to five scenes can be presented per session.

A variation of this technique called *in vivo desensitization* has been developed, in which the child undergoes exposure training to the hierarchy *in vivo* rather than through imaginal responses ( [Morris and Kratochwill, 1983](#)). The effectiveness of both systematic and *in vivo* desensitization is enhanced when the therapist has the child practice between sessions those items that no longer elicit anxiety and when practice is accompanied by positive reinforcement ( [Johnson et al., 1986](#)).

A related exposure training technique that may be used as an alternative to desensitization is *flooding* (also known as *implosion therapy* when applied *in vivo*). Widely used with adults, flooding involves having the child come in contact with the most feared item in the hierarchy, either *in vivo* or in imagination, until the fear is extinguished. Although flooding has been effective with children when graded desensitization procedures have not met with success ( [Yule et al., 1974](#)), its use with

children raises ethical concerns about children's informed consent and voluntariness of participation in such a potentially upsetting procedure.

In addition to other behavioral reduction techniques discussed previously, a number of the techniques described earlier have met with considerable success in reducing or eliminating problem behaviors in children. Techniques such as modeling, self-instructional training, problem solving, and contingency contracting have been used extensively to treat problems such as aggressiveness, impulsivity, antisocial behavior, fears, and substance abuse.

## BEHAVIORAL ASSESSMENT

Behavioral assessment is the process of identifying target behaviors and their controlling conditions by several different methods to assist the clinician in developing an appropriate treatment plan. Traditional assessment approaches hypothesize the operation of traits or personality constructs from *within* the individual. Behavioral assessment approaches hypothesize the operation of specific behavior patterns or states *outside* of the individual. Although the distinction between these two approaches may be extremely subtle in practice (e.g., in the assessment of a child's cognitions and affects), conceptually it is an important one because it focuses the assessment on current circumstances in the client's environment rather than on enduring underlying traits or states.

Traditional assessment approaches attempt to uncover the individual's personality structure, arrive at a diagnosis, and, finally, offer a prognosis. In contrast, behavioral assessment methods gather data that are directly, rather than inferentially, related to a treatment program and its evaluation. Behavioral assessment can be viewed as a dynamic process that involves (a) problem identification and target behavior selection, (b) choice and design of a treatment program, (c) periodic measurement of results during treatment, and (d) evaluation of the final treatment outcome ([Bornstein et al., 1984](#)). Because all treatments are not equally effective and vary in their efficacy according to several client–therapist factors, such as the client's age and potential for compliance, family support, and the therapist's expertise in behavioral assessment, the choice and design of a treatment program is based, as much as possible, on the relevant therapy outcome literature. Once a treatment program has been selected, periodic reassessment of its efficacy and suitability throughout the treatment phase is essential.

### Models of Assessment

There are several widely used models of behavioral assessment. Kanfer and Saslow's S-O-R-K-C model ([Kanfer et al., 1969](#)) is a comprehensive method that assesses the stimulus (S), the organism's biological conditions (O) and behavioral response (R), the contingencies and schedules of reinforcement (K), and relevant consequences (C). The BASIC ID model of [Lazarus \(1973\)](#) is a multimodal assessment of behaviors (B), affects (A), sensations (S), imagery (I), cognitions (C), interpersonal relationships (I), and drugs and general physical state (D). [Peterson \(1968\)](#) has proposed a functional analysis of behavior model that involves systematic observations of the problem behavior and the stimulus conditions (antecedents and consequences) that give rise to it, followed by experimental manipulation of a functionally related condition that causes the behavior.

One particularly useful and comprehensive model for behavioral assessment is the Behavioral Assessment Grid (BAG) developed by [Cone \(1978\)](#). This model divides assessment into a tripartite conceptual scheme of behavioral contents, methods of assessment, and types of generalizability. The behavioral *contents* area of the BAG refers to the three domains that behavior therapists typically assess for behavioral responses: cognitive (subjective), motor, and physiologic. For example, a child's fear may be assessed as a worry in the cognitive domain, as behavioral avoidance in the motor domain, or as a biochemical response to a stimulus in the physiologic domain. These are believed to be distinct yet potentially overlapping areas of behavioral functioning. The *methods* component of the BAG refers to the many types of indirect and direct measurement techniques that can be used to make a behavioral assessment. Indirect methods include behavioral interviews, self-reports, and ratings by others. Direct methods include self-observations or observations by others in analog or naturalistic situations under free play conditions or role play instructions. Last, the *universes of generalizations* component of the BAG refers to the extent to which measurements obtained are generalizable across multiple contexts. Specifically, six “universes” of generalizability have been identified as particularly relevant to consider when making a behavioral assessment: dimension (of behavior that is assessed), method (used to make the assessment), setting (in which the assessment is made), time (when the assessment is made), item (which aspect of a dimension is assessed), and scorer (who makes the assessment). Thus, the BAG model allows for three domains of behavioral contents by eight methods of data collection by six universes of generalization, resulting in a theoretically possible matrix of 144 different combinations of behavioral assessments. Although existing assessment measures do not fit into each of these 144 categories, the BAG model provides a useful framework in which the clinician can conceptualize and classify behavioral assessment instruments and measures.

### Assessment Strategies

There are a variety of behavioral assessment strategies that range from the least to the most direct in terms of the method of data collection ([Cone, 1978](#)).

#### INTERVIEWS

Behavioral interviews are an integral element of the assessment process ([Morganstern, 1988](#)). The objective during the initial phase of the behavioral interview is to establish rapport with the child, regarded as a necessary but not sufficient ingredient for successful treatment. During this phase, the child is assured of the confidential nature of the relationship and informed of under what conditions of danger to self or others that confidentiality will be broken. After the child presents the problem, the therapist probes for additional information regarding the initiation, frequency, and controlling conditions surrounding the problem behaviors. In the case of very young children, it is common to conduct a verbal interview with the parents and a play interview with the child to obtain a sample of the child's behavior.

Structured interviews are another type of indirect assessment strategy and a relatively recent addition to the classic behavioral assessment protocol for children ([Morrison, 1988](#)). There are three major structured interview measures in use with children: the Diagnostic Interview for Children and Adolescents (DICA) ([Herjanic et al., 1975](#)), the Diagnostic Interview Schedule for Children (DISC) ([Costello et al., 1984](#)), and the Schedule for Affective Disorder and Schizophrenia for School-Age Children, Present Episode Version (K-SADS-P) ([Chambers et al., 1985](#)). The DICA is a 60- to 90-minute interview of both the child and parent in three parts, conducted by trained interviewers. The DISC involves two 45- to 60-minute separate interviews with both the child and parent by trained interviewers and provides a *Diagnostic and Statistical Manual of Mental Disorders* (DSM) diagnosis. Last, the K-SADS-P is a semistructured interview of both the parent and child that assesses four areas (affect, anxiety, conduct, and psychosis) in two time periods (the past week and when the current episode was at its worst). It takes approximately 60 minutes to complete each interview.

#### SELF-REPORTS AND RATINGS BY OTHERS

Self-reports and ratings by others can be obtained through the use of various rating scales, which vary in terms of their validity and reliability ([Morrison, 1988](#)). Such self-report scales increasingly are being used by behavior therapists to assess symptom severity and also to monitor treatment effects. Some of the most widely used self-report instruments are discussed here.

The Children's Depression Inventory (CDI) is a 27-item scale adapted from the Beck Depression Inventory that provides information on a child's cognitive, affective, and behavioral signs of depression ([Kovacs and Beck, 1977](#)). It can be self-administered or read to children, as well as completed by parents. The Child Behavior Checklist (CBCL) is an empirically derived questionnaire that asks respondents to rate the occurrence of 118 problem behaviors of childhood or adolescence on a 3-point scale ([Achenbach, 1978](#); [Achenbach and Edelbrock, 1979](#)). Although the version that is the most widely used is the parent form, teacher and youth self-report forms also have been developed. The CBCL is one of the most carefully designed, empirically sound, and comprehensive instruments available for the assessment of children's problems. It includes additional items that provide a gross assessment of a child's social competencies. The Conners Teacher Rating Scale ([Conners, 1973](#)) is a 39-item self-report measure whose items are rated on a 4-point scale. It is used most commonly in the assessment and treatment of hyperactivity because it is sensitive to changes due to both behavioral interventions and psychopharmacologic treatment. The Conners Parent Rating Scale ([Conners, 1970](#)) is also available for parent reports, and both scales come in short-form versions. The Fear Survey Schedule for Children—Revised ([Ollendick, 1983](#)) is an 80-item self-report measure that is rated on a 3-point scale. It differentiates phobic from nonphobic children and also identifies etiologic factors such as separation anxiety, school phobia, and recent physical illness.

#### SELF-OBSERVATION

Self-observation, one of the oldest methods of direct behavioral assessment, includes the simple tabulation of behaviors by check marks or counters as well as behavioral monitoring by more sophisticated electronic devices that measure physiologic responses such as heart rate and blood pressure. These measures are used in several self-control procedures, such as weight reduction, tic control, and nail-biting cessation. Self-observation is less expensive and intrusive than the use of outside observers, allows monitoring to occur for longer periods and across a greater number of settings, and is particularly appropriate when monitoring infrequently



occurring behaviors. Self-monitoring can be a highly reactive process for clients, one that can precipitate dramatic behavior change in a favorable direction.

### DIRECT OBSERVATION

Direct observation can be conducted in the natural environment as well as in analog situations. An example of the use of direct observation in an analog situation is the use of the Behavioral Avoidance Test (BAT) to measure phobic behavior in children. The BAT is the most commonly used direct observational procedure to assess specific fears ([Wells and Vitulano, 1984](#)). During the BAT, the clinician records the distance from and time a client spends with a feared object. Although this procedure is very useful in providing concrete evidence of the nature and extent of a specific fear under controlled conditions, it can be quite reactive to demand characteristics, such as telling a child to try harder.

Direct observation of role plays also is used in behavioral assessment, in either naturalistic or analog situations. During a role play, clients are asked to imagine themselves in various situations and then to respond in their typical manner. Role plays in naturalistic and analog situations are used frequently in the assessment of social skills deficits in children.

## TREATMENT APPLICATIONS

### Anxiety Disorders

Anxiety disorders include that group of disorders whose symptoms are characterized primarily by anxiety and avoidance behavior. This category of disturbances includes phobic disorders, panic disorders, generalized anxiety disorders, and obsessive-compulsive disorders. Although fears and anxieties are common and transitory throughout childhood ([Lapouse and Monk, 1959](#)), more severe and persistent phobic reactions in childhood that are not considered to be age or stage specific require the attention and treatment of professionals. Thorough assessment of children's fears should include an evaluation of the three-channel response system of subjective feelings and thoughts, behavioral avoidance, and physiologic activity ([Graziano et al., 1979](#)).

Assessment in all three channels facilitates treatment planning and enhances treatment efficacy. For example, for a child who exhibits avoidance behavior (i.e., the motor system or channel), reinforced practice would be an essential component of the treatment; for a child who reports considerable subjective anxiety (i.e., the cognitive channel), self-control strategies would be recommended; and, finally, for a child who experiences somatic symptoms when confronted with the feared stimulus (i.e., the physiologic channel), relaxation training would be indicated. Typically, however, treatment plans for anxiety disturbances usually are more complex because they involve two or three different channels of behavioral disruption. For example, a treatment plan for a child who experiences fear in all three channels might begin with relaxation training (physiologic), move to self-instructional training (cognitive), and then advance to the use of participant modeling (motor) in combination with self-instructional training. Thorough reviews of the use of various behavioral techniques, such as relaxation training, systematic desensitization, flooding, implosion, modeling, self-control training, and operant procedures to treat childhood anxiety disorders are available ([Siegel and Ridley-Johnson, 1985](#); [Straus, 1987](#); [Wells and Vitulano, 1984](#)). [Kendall and colleagues \(1992\)](#) describe a cognitive-behavioral treatment program in detail for use with anxious children, and [Kendall \(1994\)](#) demonstrates its effectiveness with school-age children in a randomized clinical study. [Flannery-Schrieder and Kendall \(2000\)](#) compare group and individual cognitive-behavioral approaches for anxious youth.

Despite the controversy over the etiology and definition of school phobia, behavior therapy usually has been successful in its remediation. Techniques such as systematic desensitization, participant modeling, and contingency contracting have been particularly efficacious ([Ollendick and Mayer, 1984](#)). Obsessive-compulsive disorders, on the other hand, have been shown to be responsive to *in vivo* exposure procedures when combined with response prevention techniques ([Baer and Minichiello, 1990](#); [Grayson et al., 1982](#); [Scahill et al., 1996](#)). For example, compulsive hand washers are encouraged to touch contaminated objects and then refrain from washing to break the negative reinforcement chain. Although there has been less success in the treatment of obsessional behaviors, the most promising strategy appears to be imaginal exposure to obsessional thinking for a prolonged period of 30 minutes or more (i.e., avoidance response prevention) to extinguish the anxiety response ([Rachman and Hodgson, 1980](#)). The treatment of obsessive-compulsive disorder symptoms in children and adolescents with Tourette's syndrome is best approached as part of a comprehensive, multimodal therapeutic plan ([King et al., 1993](#)). Finally, much of childhood anxiety may in fact be secondary to social skill deficits in anxious children. Such children initially may lack the necessary skills to interact in a feared situation and fail to acquire those skills because of successful avoidance behaviors, while their peers continue to learn interaction skills ([Wells, 1981](#)). The obvious treatment program for these children is to augment fear reduction techniques with social skills training ([Christoff and Myatt, 1987](#)).

### Depression

Behavioral researchers and clinicians maintain that childhood depression represents a complex amalgam of symptoms that vary in degree and kind according to a child's developmental level. Thus, children who are depressed in early childhood may show evidence of excessive crying, loss of appetite, and temper tantrums, whereas those of middle childhood (6 to 8 years of age) may exhibit dysphoria, somatization, and impulsive behaviors, and those of late childhood (8 to 12 years of age) may display poor self-esteem, social isolation, self-deprecating behaviors, and hopelessness ([Kaslow and Rehm, 1985](#); [Kovacs and Beck, 1977](#)). The complexity of this symptom picture, coupled with the considerable overlap between these and other symptoms of disorder in childhood and adolescence ([Kendall et al., 1989](#)), has led behavioral investigators to argue that the most effective intervention for childhood depression is to treat specific symptoms of the disorder, such as self-deprecating behaviors, social withdrawal, and low activity level ([Kaslow and Rehm, 1983](#); [Puig-Antich, 1982](#)).

There is growing evidence that this strategy is effective ([Reynolds and Coats, 1986](#); [Stark et al., 1987, 1990, 1991](#)). Among the various behavioral techniques, self-control training has demonstrated the greatest efficacy in the treatment of depression for children (9 to 12 years of age) and adolescents. Self-control training in the treatment of depression involves teaching the child to monitor negative self-attributions, evaluate the validity of those attributions, and reinforce positive alternative attributions. Children also are taught to set realistic goals and standards for performance, refrain from punitive self-statements, and use positive self-statements as reinforcers. Finally, to compensate for the tendency of depressed children to attribute success to luck or chance, children are taught to view success as resulting from their own stable and internal competencies and to view failure as emanating from external factors outside of their control ([Hughes, 1988](#); [Rehm, 1977](#)). Controlled outcome studies using these techniques in either 10- or 12-session interventions delivered over a 5-week period have resulted in significant reductions of depressive symptomatology immediately after treatment as well as at 5 to 8 weeks' follow-up, compared with waiting list control groups ([Reynolds and Coats, 1986](#); [Stark et al., 1987](#)).

Two other behavioral approaches also have been successful in the treatment of depression: problem-solving training and relaxation training. Problem-solving training has emphasized teaching children problem-solving skills for use in social and interpersonal situations, such as identifying and expressing feelings appropriately, generating alternative courses of action and considering their consequences, and implementing the solution that best fits the situation ([Stark et al., 1987](#)). Relaxation training, on the other hand, has been found to be successful in reducing depressive symptoms in adolescents ([Reynolds and Coats, 1986](#)). Adolescents are taught to identify the link among stressors, muscle tension, and depressive symptomatology and then are trained in relaxation techniques.

The success of self-control and problem-solving training with children and of relaxation and self-control training with adolescents attests to the considerable promise of behavioral treatments for childhood and adolescent depression. At present, however, the key ingredients of behavior change remain unknown. Among the cognitive-behavioral treatments (i.e., self-control training and problem-solving training), there is a common emphasis on having the child engage in more pleasant activities and use self-monitoring to track depressive symptoms ([Hughes, 1988](#)). Among all three treatments, there is a common emphasis on providing the child with an enhanced sense of control over symptoms.

### Conduct Disorder and Antisocial Behavior

Conduct disorder and antisocial behavior in children are characterized by externalizing behaviors such as aggression, truancy, firesetting, stealing, and other social rule violations. The level of dysfunction it entails is extensive, involving significant disruption in the home, at school, in peer relations, and in cognitive processes (Kazdin and Wassell, 2000). Its prevalence in community samples is relatively high, and its demand on clinical services is great, accounting for up to one-third to one-half of clinic referrals ([Kazdin et al., 1989](#)). In addition to its significant social and personal costs in the short run, the effects of conduct disorder and antisocial behavior frequently extend well into adulthood, manifested in such problems as criminal behavior, alcoholism, problems at work or school, significant disruptions in interpersonal relations, and other psychosocial difficulties ([Kazdin, 1987](#); [Robins, 1981](#); [Rutter and Giller, 1983](#)).

Two types of behavioral interventions have demonstrated success in the treatment of conduct disorder and antisocial behavior: problem-solving skills training (PSST) and parent training. PSST uses behavioral rehearsal, role playing, modeling, corrective feedback, and social reinforcement to teach children interpersonal cognitive



problem-solving skills, such as generating solutions, means–ends and consequential thinking, and perspective taking ( [Kazdin et al., 1987](#)). The intervention is delivered in 20 to 25 sessions once or twice per week. Controlled studies have demonstrated that PSST, as opposed to relationship therapy or an attention placebo control intervention, is effective in reducing antisocial behavior in severely disturbed, clinic-referred children 7 to 13 years of age at posttreatment as well as after a 1-year follow-up ( [Kazdin et al., 1987, 1989](#)). Despite these reductions in antisocial behavior, the incidence of problem behaviors of children who had undergone PSST remained significantly higher than that found in community samples ( [Kazdin et al., 1989](#)).

A second approach to the treatment of conduct disorder and antisocial behavior in children and adolescents is behavioral parent training. A variety of such treatment programs have been developed and have demonstrated considerable success in reducing antisocial behavior compared with no treatment or alternative interventions ( [Ducharme et al., 2000](#); [Wells and Forehand, 1985](#)). The most widely evaluated parent training approach is the program developed by Patterson and colleagues ( [Patterson, 1975, 1982](#); [Patterson and Guillion, 1968](#); [Patterson and Reid, 1973](#); [Patterson et al., 1982](#); [Vuchinick et al., 1992](#); [Wiltz and Patterson, 1974](#)). In this program, parents are taught basic behavioral principles and techniques (e.g., reinforcement, time out, response cost, conditioned reinforcement, contingency contracting) and how to apply these to specific interactions with their child. Next, parents are trained extensively through program manuals and weekly training sessions to observe, identify, track, and record deviant and prosocial behaviors on the part of their child. As part of this training, parents are asked to identify two prosocial and two problem behaviors to track and monitor over a 3-day period. To facilitate monitoring, it is not uncommon for the therapist to call parents daily to assist in the completion of this task. Finally, the therapist works directly with parents to help them design and implement a token reinforcement system for use in the home. Children are praised for appropriate behavior as well as provided with points that can be exchanged daily for reinforcers. Noncompliance or misbehavior is managed through the use of a time out (from reinforcement) or response cost (loss of points) procedure. The therapist uses a variety of behavioral procedures to teach parents necessary skills, such as modeling, behavioral rehearsal, and role playing.

Related behavioral parent training programs also have been developed for use with younger clinic-referred children, 2 to 8 years of age ( [Forehand and McMahon, 1981](#); [Webster-Stratton et al., 1988](#)). Such programs usually teach parents how to (a) observe, identify, and monitor their child's behavior; (b) reinforce prosocial and appropriate behavior; (c) extinguish minor aggressive or destructive behavior through ignoring; (d) give clear and concise one-time commands on the appearance of misbehavior; (e) provide a single warning of impending consequences for failure to comply with a command; and (f) use a 3- to 5-minute time out to punish noncompliance ( [Wells and Forehand, 1985](#)). In general, because positive reinforcement for prosocial behavior often is quite successful in reducing aggressive and destructive behavior in younger children, it is recommended that these techniques be implemented first before more punitive techniques are applied.

Overall, behavioral parent training programs for children and adolescents with conduct disorder or antisocial behavior have been very effective in reducing the problem behaviors of referred children to levels observed in nonreferred peers. These effects are maintained up to 1 year posttreatment ( [Fleischman and Szykula, 1981](#)), with some benefits persisting for over 10 years ( [Baum and Forehand, 1981](#); [Forehand and Long, 1988](#)). Such programs also appear to have some impact on reducing maternal depression and enhancing the prosocial behavior of other children in the family ( [Kazdin, 1985b](#)). Importantly, these programs have been found to be least effective when implemented in brief, time-limited treatment (10 hours or less rather than 50 to 60 hours) or by an inexperienced therapist, or with families who have significant marital, psychological, or social adjustment difficulties ( [Kazdin, 1988](#); [Wells and Forehand, 1985](#)). When such difficulties are observed during the course of treatment, the therapist is advised to deal with marital conflicts and other potential barriers to effective treatment implementation directly, as part of a more comprehensive intervention strategy ( [Griest and Wells, 1983](#)). Studies have shown that a combined approach of PSST and parent training is most effective in the treatment of antisocial behavior in children ( [Kazdin et al., 1992](#)).

### Attention Deficit/Hyperactivity Disorder

Evidence is compelling that behavior therapy in combination with psychostimulant medication is the treatment of choice for attention deficit/hyperactivity disorder (ADHD) ( [Hinshaw, 2000](#); [Rapport, 1987](#)). In studies comparing these two treatments, neither alone has been found to demonstrate efficacy consistently across relevant outcomes, such as reduced impulsivity and motor activity and improved attention, concentration, academic performance, and social relations ( [Barkley, 1985, 1990](#); [O'Leary and Wilson, 1987](#); [Rapport, 1987](#)). Although this may be due in part to the complexity inherent in diagnosing this disorder accurately ( [Barkley, 1981, 1991](#); [Rapport and Ismond, 1984](#)), a picture is beginning to emerge that suggests that these two treatments may each have a differential impact and thus be most effective when used in combination.

The psychostimulant medications most frequently prescribed for the treatment of children with ADHD are methylphenidate, dextroamphetamine, and pemoline, with methylphenidate by far the most commonly used and investigated of these in the United States ( [Safer and Krager, 1983](#)). Controlled outcome studies have shown methylphenidate to be effective in reducing impulsivity ( [Ayllon et al., 1975](#); [Brown and Sleator, 1979](#); [Rapport et al., 1985](#)) and motor activity level ( [Porrino et al., 1983](#)), improving attention, concentration, and on-task behavior ( [Rapport et al., 1986, 1987](#)), and enhancing social relations with adults and peers ( [Barkley, 1977](#); [Barkley et al., 1984](#); [Whalen et al., 1981, 1989](#)). Despite widespread positive effects in these domains, psychostimulant medication in general has fared poorly in demonstrating short- or long-term effectiveness on learning and performance on academic tasks ( [Barkley and Cunningham, 1978](#); [Hechtman, 1985](#); [O'Leary, 1980](#)), although there is evidence that some kinds of learning may be responsive to methylphenidate at higher dosage levels than are commonly used ( [Rapport, 1987](#); [Rapport et al., 1982](#); [Vyse and Rapport, 1989](#)).

Behavioral treatments for children with ADHD, on the other hand, consistently have demonstrated efficacy in enhancing learning and improving academic performance ( [Ayllon et al., 1975](#); [Barkley, 1985](#); [O'Leary, 1980](#); [Pelham et al., 1980](#)), in some cases by as much as 1.6 years of growth over the course of 1 year of treatment ( [Satterfield et al., 1979](#)). Behavioral treatments also have been shown to be effective in reducing impulsivity and motor activity and enhancing attention and concentration, although findings from these studies are less clear and at times contradictory ( [Barkley, 1985](#); [Gittelman-Klein et al., 1976](#); [O'Leary and Wilson, 1987](#)).

Behavioral interventions are most effective when implemented in three arenas: the home, the classroom, and with the individual child. Home interventions usually involve teaching parents observational and behavior management skills for use in rewarding appropriate behavior and extinguishing inappropriate behavior. Typically, parents are taught to use positive as well as conditioned (token) reinforcement to strengthen their child's ability to play independently and to engage in prosocial interactions with others. In addition, parents are trained in the use of time out and response cost procedures so that they can provide mild punishment for misbehavior and noncompliance with house rules. Successful school interventions have emphasized similar techniques, although a token economy system used in combination with response cost usually is an essential component of such programs. Finally, self-control techniques, such as self-monitoring, self-evaluation, and goal setting, have also been used with children with ADHD ( [Binder et al., 2000](#); [Davies and Witte, 2000](#)). Although these techniques have demonstrated only limited effectiveness, when used alone or in combination with psychostimulant medication they hold promise when combined with the parent or teacher components of a comprehensive behavioral treatment program ( [O'Leary and Wilson, 1987](#); [Whalen et al., 1985](#)).

### Pervasive Developmental Disorders and Autism

Pervasive developmental disorders (PDDs) consist of infantile autism and pervasive developmental disorders (not otherwise specified). The essential features of effective treatment of PDD include a thorough assessment of functional behaviors and their controlling variables. Because PDD involves impairment in social interaction and communication skills, the selection of target behaviors for modification should focus on key deficits that have a generalizing effect on several areas of functioning. Attentional and social skills training are two such areas that appear to have far-reaching educational and interpersonal ramifications.

Behavioral treatment techniques for these disorders generally fall into two categories: suppressing unwanted behaviors and teaching new skills. Early attempts at perceptual and sensory processing and integration training ( [Ornitz and Ritvo, 1968](#)) based on a neurologic theory of deficits have shown inconsistent results. Several different techniques have been used since with success to reduce unwanted behaviors, such as time out ( [Foxy and Shapiro, 1978](#)), overcorrection ( [Foxy and Azrin, 1973](#)), and punishment ( [Rincover et al., 1979](#)). New skills are taught by using reinforcement, stimulus discrimination training, and social skills training, and by providing clear instructions ( [Harris and Handleman, 1987](#)). For example, a child who has difficulty responding to social cues might respond to shaping and prompting techniques that can be used to teach such basic skills as making eye contact and sitting still. Once these skills are acquired, they can be used to teach the more complex and related skills of attention and observation ( [Koegel and Schreibman, 1977](#)). Parent training and sibling involvement in behavioral interventions also have been used to enhance the effectiveness of such training programs ( [Harris, 1983, 1984](#); [Lovaas et al., 1973](#); [Schreibman et al., 1983](#)).

Historically, the long-term prognosis for a return to normal functioning in children with PDD or autism has been very poor. However, a controlled outcome study by [Lovaas and colleagues \(1987, 1989; McEachin et al., 1993\)](#) on the intensive treatment of autistic children younger than 4 years of age is very encouraging. The treatment involved a minimum of 40 hours per week of one-to-one adult contact with the child for a period of over 2 years. Student therapists as well as the child's parents were trained to provide the intensive home-based treatment, which consisted of operant procedures aimed at promoting cognitive, verbal, and social skills. At the conclusion of the controlled trial, 47% (9 of 19) of the children who participated in the treatment were enrolled in regular first grade classrooms and were reported as "indistinguishable" from their classroom peers by school personnel. None of the children in a nonintervention group were enrolled in regular first grade classes. In addition, the nine children attending first grade classes exhibited dramatic gains in IQ from pretreatment to posttreatment. This pattern of differences between



experimental and control groups was maintained at a subsequent follow-up of these children when they reached a mean age of 11.5 years ( [McEachin et al., 1993](#)).

## **Mental Retardation**

The dramatic expansion of behavior therapy techniques has revolutionized the field of mental retardation therapy since the early 1970s ( [Reber, 1992](#); [Whitman and Johnston, 1987](#)). The most effective behavioral interventions have been developed for the mildly and moderately retarded, a group that comprises approximately 98% of the total population of retarded persons ( [Grossman, 1983](#)). In general, the goals of behavior therapy with the mentally retarded do not involve treatment for psychiatric disorders but rather focus on promoting adaptation in three major areas of skill deficits: self-control, social skills, and academic performance.

Self-control problems have been treated with a wide variety of techniques, such as overcorrection for self-stimulatory behavior ( [Foxy and Azrin, 1973](#)), differential reinforcement of other behaviors for the reduction of stereotypies ( [Eason et al., 1982](#)), and a variety of other operant techniques ( [Berkson, 1983](#)). As a last resort, punishment has been used effectively to reduce unwanted behaviors ( [Barrett et al., 1981](#)). Remarkable success also has been demonstrated in the acquisition of social skills among the mentally retarded, especially in the areas of communication skills and daily living skills. In general, reinforcement techniques have been applied in programs designed to improve expressive language skills ( [Handleman et al., 1984](#)), to foster understanding and ability to follow directions ( [Lancioni et al., 1984](#)), and to acquire basic personal and home living skills ( [Schadlock et al., 1984](#)). Finally, academic performance has been enhanced through the implementation of behavioral interventions. Basic classroom learning skills have been taught with the help of interventions that promote eye contact and increase on-task behavior ( [Bornstein and Quevillon, 1976](#); [Zigler and Hodapp, 1985](#)).

## **Enuresis and Encopresis**

Behavior therapy has been found to be particularly effective in the treatment of enuresis and encopresis. Of the two kinds of enuresis, diurnal (daytime) and nocturnal (nighttime), the latter usually is more persistent and troublesome to parents and child. Two types of behavioral treatment have been used with success in the treatment of nocturnal enuresis: the urine alarm and dry bed training (DBT). The urine alarm was first popularized as the bell and pad treatment for enuresis in 1938 by Mowrer and Mowrer. Although a number of modifications of the original bell and pad procedure have been developed over the years, the essential procedure remains the same. The child sleeps with a pad under a sheet that has been wired with an electrical current and serves as a urine sensor. When urine wets the pad, an alarm goes off and wakes parents and child. After using the toilet, the child makes the bed and resets the alarm before returning to sleep. Conditioning is believed to occur through either one or both of the following mechanisms: (a) the repeated pairing of the alarm and voiding (i.e., classically conditioned reinforcement), such that the child comes to inhibit voiding and sleep through the night; or (b) stimulus discrimination training, whereby the child is awakened when bladder distension cues are near their peak (i.e., when there is a full bladder) and, as a result, learns to wake and void when these cues are present.

Reviews have found this technique to be effective in 75% to 80% of cases, with relapse rates of 30% to 41% ( [Doleys, 1977, 1985](#); [O'Leary and Wilson, 1987](#)). Of those children who relapse, just over two-thirds remain continent after reinitiation of the procedure. Typically, training is completed in 5 to 12 weeks.

Two modifications of the standard urine alarm treatment have been found to reduce relapse considerably ( [Doleys, 1985](#)). The first involves use of an intermittent reinforcement schedule in which the child is awakened on only 50% to 70% of wetting episodes. The second involves use of an overlearning procedure in which the child is asked to consume liquid 1 hour before bedtime, usually beginning with 10 ounces and building up to 32 ounces over a few weeks. Overlearning is believed to promote generalization of learning to an increased range of bladder capacity and distention ( [Young and Morgan, 1972](#)).

A second behavioral treatment found to be effective in eliminating nocturnal enuresis is DBT ( [Azrin et al., 1974](#); [Said et al., 1991](#)). This treatment incorporates a number of behavioral techniques: positive practice, positive reinforcement, punishment, and the urine alarm. In DBT, the child is awakened during the night, asked to go to the toilet if any urge to void is present (positive practice), and then allowed to return to bed with praise for completing this onerous task (positive reinforcement). The urine alarm also is used as part of the treatment, and in the usual manner, the child is required to clean and change wet sheets in the event of an accident (punishment). Usually the child also is verbally reprimanded for a wet bed (punishment) and praised for staying dry (positive reinforcement). This procedure is followed on successive nights (and, in some applications, repeatedly throughout a single night) until the child remains dry for 2 weeks. Parent training manuals have been written to make this treatment accessible to the public ( [Azrin and Besalel, 1979](#); [Azrin and Foxx, 1974](#)).

There is some evidence that DBT is slightly more effective than the urine alarm, particularly in reducing relapse and achieving faster results, although evidence from various studies is inconclusive ( [O'Leary and Wilson, 1987](#); [Ronen et al., 1992](#); [Young and Morgan, 1972](#)). Because this procedure requires considerable effort on the part of parents and child, clinicians must determine whether the additional benefits of DBT over and above those of the urine alarm outweigh the very real possibility of treatment noncompliance.

Behavior therapy also has been shown to be effective in the treatment of functional encopresis, which is defined in DSM-IV as the repeated passage of feces in inappropriate places more than once per month after the age of 4 years and occurring for a period of 6 consecutive months ( [American Psychiatric Association, 1994](#)). Recommended effective behavioral treatments for encopresis involve the use of (a) positive and conditioned reinforcement, sometimes in combination with mild punishment; or (b) regular pants checks in combination with positive and conditioned reinforcement plus full cleanliness training (FCT). Laxatives or suppositories also often are used as an adjunct to treatment to promote the passage of fecal material. Treatments based on reinforcement (with or without mild punishment) involve training the parent to praise the child or deliver a conditioned reinforcer contingent on the child's passage of feces on the commode ( [Ashkenazi, 1975](#); [Wright and Walker, 1976](#); [Young and Goldsmith, 1972](#)). In the initial stages of this treatment, sometimes it is necessary to provide reinforcement for successive approximations of the appropriate behavior, for example, rewarding the child for passing feces in the pants while in the bathroom. This treatment usually eliminates encopresis within 15 to 20 weeks in most cases.

A second, slightly more efficient treatment for encopresis, which is also carried out by parents and supplemented by laxatives or suppositories as necessary, consists of three components: (a) periodic pants checks (usually 1 to 2 hours apart), (b) FCT after soiling, and (c) positive reinforcement for clean pants checks ( [Young and Morgan, 1972](#)). FCT is implemented when soiling is discovered during a pants check. After a show of displeasure by the parent, the child is required to wash the soiled underpants for 20 minutes and then must take a 20-minute bath in cool water. Successful pants checks result in praise as well as the awarding of conditioned reinforcers, such as tokens or points, which can be accumulated and exchanged for privileges, treats, or special activity time with one or both parents. This technique usually results in the elimination of functional encopresis in 10 to 15 weeks and is effective with most cases ( [Young and Morgan, 1972](#)).

## **Applications of Behavior Therapy in Diverse Settings**

One of the major advantages of behavior therapy is its applicability to a wide range of settings. Behavior therapy has been used with success in family interventions, school and community interventions, residential settings, and in the delivery of pediatric services. These diverse applications of behavioral approaches have promoted the delivery of services and treatments to previously underserved populations.

### **FAMILY INTERVENTIONS**

Two types of behavioral interventions have been widely used with families: behavioral parent training and behavioral family therapy. Parent behavioral training programs differ according to the behavioral skills emphasized (e.g., contingency management, conflict resolution, problem solving, positive parent-child interactions, contingency contracting) and the problem behavior being addressed (e.g., fostering independence for a mentally retarded child, reducing a child's fears, decreasing a parent's likelihood to engage in physically abusive behavior, promoting effective parent-child communication, and reducing parent-adolescent power struggles) ( [Graziano, 1977](#); [Twardosz and Nordquist, 1987](#)). Despite this wide-ranging focus, behavioral parent training programs have shown a promising record of efficacy relative to other forms of treatment.

Behavioral family therapy, on the other hand, is an emerging area of family intervention that combines family systems theory with learning theory. Family systems theory is used to formulate the presenting problem in the context of key parameters of family functioning, such as family communication, boundaries, power, affectivity, and family life cycle status. Learning theory is used to formulate the presenting problem in terms of behavioral contingencies that maintain or exacerbate the problem or that function to block effective problem solving. These perspectives are then combined in a treatment plan that may include teaching family members to identify feelings and thoughts during family interactions; reinforcing effective communication in the session; pointing out behavioral contingencies that inadvertently strengthen problem behaviors in the family; using modeling, role plays, and behavioral rehearsal in the session to teach contingency contracting or behavioral management skills (e.g., time out); and assigning homework for family members to practice skills taught in the session.

## SCHOOL AND COMMUNITY INTERVENTIONS

School and community interventions are another rapidly growing arena for behavioral interventions. Behavioral treatments are well suited for implementation in school settings because they often can be delivered effectively in a group context. The school, in particular, provides an excellent opportunity for implementing behavioral interventions at multiple levels. Children referred for treatment often can be seen individually or in groups in the school setting by a school psychologist, social worker, or counselor (Hughes, 1988). In addition, high-risk or normal children can participate in behaviorally based classroom interventions that seek to promote competencies or prevent problem behaviors, such as substance abuse (Snow et al., 1987). These programs frequently are compatible with other types of behavioral interventions, such as parent training programs, delivered in the same setting (Tebes et al., 1989). Community interventions provide further opportunities for behavioral interventions implemented at multiple levels. Such programs can augment school-based or parent training programs, can involve a variety of community organizations in their implementation, and can use the popular media to enhance the impact of the intervention (Pentz et al., 1989).

## RESIDENTIAL SETTINGS

Behavioral interventions also are common in residential treatment programs for children and youth. The most common form of behavioral intervention in these settings is the use of a token economy system as part of the treatment milieu. Such interventions typically provide steps in which children can gain more and greater privileges as they progress through the program. Token economies have been implemented with success in a variety of settings, such as those used to treat delinquent youths and mentally retarded children and adolescents (Kazdin, 1977).

## PEDIATRIC BEHAVIORAL MEDICINE

Finally, since the early 1980s, behavior therapy has been expanding its applications to pediatric settings in hospitals. Pediatric behavioral medicine is the new, rapidly expanding, interdisciplinary field involved with the prevention and treatment of emotional and behavioral problems related to child and adolescent health and illness. Although there is no specific theoretical orientation to the field of pediatric behavioral medicine, in practice it is the application of behavior modification principles in the medical arena. Pediatric behavioral medicine and its parent field of behavioral medicine usually are characterized by three types of interventions: (a) direct treatment of medical problems such as pain (Varney et al., 1982) and hypertension (Lutzker and Martin, 1981); (b) the development of programs to enhance compliance to medical procedures and regimens, such as fear reduction programs before surgery (Lutzker and Martin, 1981) and compliance programs for juvenile patients with diabetes (Lowe and Lutzker, 1979); and (c) health education, in which knowledge and skills training are provided in such areas of health behavior as accident prevention, nutritional compliance, and medical advice (Finney and Christopherson, 1984). The emergence of the relatively new field of behavioral medicine has expanded traditional medicine beliefs to include a more important role for the emotional factors that influence the development and course of many physical disorders and their most efficacious treatment.

## RELATED APPROACHES

### Hypnosis

#### HISTORY

A field related to child and adolescent behavior therapy is *hypnotherapy*. Although hypnosis has been practiced for thousands of years, much skepticism remains regarding the legitimacy of its therapeutic use (Kroger, 1977; Olness and Gardner, 1988). The history of modern hypnosis begins with Franz Anton Mesmer (1734–1815), who in the late 18th century claimed that magnetic fluid imbalances in the human body were the cause of many somatic and psychological diseases. His dramatic “cures” involved staring into his patients’ eyes while they held onto iron rods attached to a large tub of iron filings and allegedly were demagnetized of their illnesses (Tinterow, 1970). Mesmer was discredited by the distinguished Franklin Commission, which had been established by King Louis XVI to investigate mesmerism. Several of Mesmer’s followers tried to defend mesmerism, but not until the late 19th century did Jean-Martin Charcot (1835-1893), the French neurologist, give hypnosis a new measure of scientific credibility by describing it in neurologic terms. James Braid (1795-1869) coined the term *hypnosis* from the Greek word for “sleep,” *hypnos*, and he is now remembered as “the father of hypnosis.” Finally, Freud initially was interested in Charcot’s work with hysteria through hypnosis but abandoned this interest in favor of free association in psychoanalysis.

#### DEFINITION

The definition of hypnotherapy is complicated because it encompasses a wide variety of theories and techniques. Basically, hypnotherapy is a treatment modality that uses an altered state of consciousness (i.e., hypnosis) in its overall treatment plan. Much of the work in hypnotherapy is viewed as instilling “an attitude of hope in the context of mastery” (Olness and Gardner, 1988, p. 89). Most popular theories define hypnosis as an altered state of consciousness involving some combination of heightened awareness, increased suggestibility, intense interpersonal relationship, or direct unconscious access. These theories are primarily physiologic, psychological, or some combination of the two. The physiologic theories suggest selective inhibition of brain centers, whereas the psychological theories emphasize the subject’s hypersuggestibility. It is clear, however, that the process of undergoing hypnotic trance can affect physiology through altering autonomic nervous system responses, such as lowering blood pressure or reducing muscle tone. However, the studies of physiologic responsivity are unreliable because a person’s exact physiologic response also is related to the level of arousal developed by the imagery of the specific hypnotic trance. Not all hypnosis induces the same response.

#### TECHNIQUE

Formal hypnotic trance induction can be achieved in a variety of ways. In general, the child is first offered an explanation of the process that includes a methodology for relaxing and imagining, while therapeutic rapport is established. During the induction phase, the child often is asked to concentrate and focus on a visual fixation point on the ceiling until his or her eyelids became heavy and he or she closes them. Then, a deepening technique would enhance relaxation by means of any simple counting or breathing procedure, for example. Obviously, there is a limitless number of hypnotic induction techniques using visual imagery, auditory imagery, movement imagery, storytelling, ideomotor techniques, progressive relaxation, eye fixation, distraction, and other individual and group variations (Olness and Gardner, 1988). Although children younger than 7 years of age have a limited responsiveness to standard techniques, children between 7 and 14 years of age reach their peak in hypnotic susceptibility (Place, 1984). Often there is a temporary decline during adolescence, when trust in adults typically is diminished, until the pattern is stabilized again during adulthood. Olness and Gardner (1988) report the use of tactile and kinesthetic stimulation techniques with very young, preverbal children. There appear to be no significant sex differences during childhood regarding susceptibility (Cooper and London, 1966). However, the data are confounded when we consider informal hypnosis in the style of Milton Erickson and his followers (Erickson and Rossi, 1981). These methods involve indirect unconscious suggestion without the use of a bona fide trance induction. The child may not even know that a hypnotic technique was used because the doctor appears simply to be talking in some strange metaphor or asking the child to imagine something silly and absurd. These techniques can become quite sophisticated and appear subtle to the untrained eye. Some of the best professional training programs in hypnotherapy are organized by the American Society of Clinical Hypnosis in Des Plaines, Illinois, and the Society for Clinical and Experimental Hypnosis in Liverpool, New York.

#### APPLICATIONS

The clinical uses of hypnotherapy are limited only by the creativity of the therapist. In general, hypnotherapy is underused in the treatment of children, but it often is considered valuable in the areas of pain and habit control, pediatric medical problems, and stress reduction. As long as hypnosis is not used as an exclusive technique outside of a professional therapeutic context, there is little danger involved in its use. The major contraindications are situations where its use aggravates existing problems (ignoring acute pain, which is a warning signal) or avoids more effective treatment of the problem (medication or surgery). Hypnotherapy can be helpful in the treatment of headache (Olness and MacDonald, 1981, 1987; Olness et al., 1987), chronic and acute pain (Crasilneck and Hall, 1973; Hilgard and Hilgard, 1975; Russo and Varni, 1982), severe burns (Wakeman and Kaplan, 1978), obesity (Haber et al., 1979), compliance and pain control during medical procedures (Olness and Gardner, 1988), asthma (Kohen, 1986), dermatologic problems (Mirvish, 1978; Olness, 1977), enuresis (Mirvish, 1978; Stanton, 1979), nausea associated with chemotherapy (Cotanch et al., 1985; Hockenberry and Cotanch, 1985; Kohen et al., 1984; Zeltzer et al., 1984), stress reduction, and other problems of childhood and adolescence. There have been several dramatic claims of clinical cures of terminal disease through the use of hypnosis and positive imagery, but there are no properly controlled studies to support these claims (Lucas, 1985). In the broader context of good treatment, hypnosis can be a useful adjunct to behavioral treatment. The reader is referred to *Hypnosis and Hypnotherapy with Children* (Olness and Gardner, 1988) for a further review and excellent discussion of this topic.



## Biofeedback

Biofeedback is a treatment method that uses physiologic feedback instrumentation to train individuals in the regulation of their body responses, such as muscle tone, heart rate, blood pressure, and cortical brain activity ([Blumenthal, 1985](#)). Originally, biofeedback technology developed out of experimental attempts to operantly condition autonomic nervous system responses ([Miller, 1969](#)). Several models since the late 1970s have attempted to explain the process of biofeedback: learning theory, body awareness, cybernetics, hypnosis, and others. Some of the major applications of biofeedback are (a) electromyography feedback for muscle relaxation in the treatment of tension headaches ([Budzynski, 1978](#)), hypertension ([Patel, 1977](#)), cardiovascular disease ([Cheatle and Weiss, 1982](#)), muscular rehabilitation ([Basmajian, 1981](#)), and temporomandibular joint pain ([Scott and Gregg, 1980](#)); (b) thermal feedback of skin temperature in the treatment of migraine headaches ([Blanchard and Andrasik, 1982](#)) and Raynaud's disease ([Surwit et al., 1978](#)); (c) feedback of electrodermal activity for treatment of hyperhidrosis; and (d) electroencephalographic feedback of cortical brain arousal in the treatment of neurologic disorders. Because of the cost and inconvenience of the equipment, biofeedback has lost some relative popularity among the vast array of relaxation therapies, yet it still remains helpful for some clients.

## ISSUES IN CHILD AND ADOLESCENT BEHAVIOR THERAPY

### Common Criticisms

Two of the most common criticisms of behavior therapy is that it results in symptom substitution as opposed to lasting therapeutic change and that it fails adequately to take into account clinical process issues that arise during the course of treatment.

#### *SYMPTOM SUBSTITUTION*

Perhaps the best-known criticism of behavior therapy is that it results in symptom substitution. This criticism is based on the belief, grounded in psychoanalytic theory, that the goal of behavior therapy is to provide symptomatic relief as opposed to treatment for a symptom's underlying cause. According to this view, the alleviation of a particular symptom will result in the appearance of a new symptom until the underlying cause is addressed.

A number of behavior therapists have questioned the logical basis for this view ([Bandura, 1969](#); [Kazdin, 1982](#); [Sloane et al., 1975](#); [Wolpe, 1978](#)). For example, because critics who argue for the inevitability of symptom substitution after behavioral treatment do not specify which symptom will appear, when, and under what conditions, how can a valid test ever be made of the charge? Furthermore, how would one know whether a symptom arising after behavior therapy was a consequence of the treatment and not merely a new symptom? And finally, because all types of intervention—behavioral or otherwise—inevitably result in both intended and unintended positive and negative effects, why should behavior therapy be singled out for criticism when such effects are endemic to therapy *per se*? Logical considerations aside, evidence is lacking for symptom substitution after behavioral interventions ([Kazdin, 1982](#)), although there is considerable evidence that behavioral interventions have wide-ranging beneficial effects beyond specific target symptoms ([Casey and Berman, 1985](#); [Durlak et al., 1991](#); [Herbert, 1988](#); [Hersen and Bellack, 1985](#); [Weisz et al., 1987](#)).

#### *CLINICAL PROCESS*

Another frequent criticism of behavioral interventions is that clinical process factors that emerge over the course of treatment are not taken into account. Factors such as the therapist–client relationship and the nature of the therapeutic interaction are focal examples of such processes. Critics have asserted that this is because behavior therapists do not adequately attend to these issues in treatment, or even eschew the clinical value of doing so.

To some extent, these criticisms are accurate. For many years, behavior therapists spent much of their time emphasizing the need to attend more to the *tasks* of treatment than to process issues. An example of this can be found in the early behavior therapy literature, in which the therapist was described essentially as a “social reinforcement machine” ([Krasner, 1962](#)). Someone with only a cursory knowledge of actual behavior therapy practice could easily be led to believe that behavior therapists regard themselves merely as technicians who find little merit in fostering the therapeutic alliance and responding genuinely and with empathy to their clients. Although the reasons for this early emphasis are not entirely clear, a prevailing experience among behavior therapists in the 1950s and 1960s was excitement over the emerging technologies being used in treatment and a belief that this represented significant strides away from previous approaches. The cost of embracing this “techno-therapist” vision, however, was that much of the writing about behavior therapy excluded some of the *essentials* of successful treatment, behavioral or otherwise: the need to build a strong working alliance with one's client and to use that alliance to weather disruptions (i.e., resistances) in the treatment so as to facilitate therapeutic gains.

More recently, behavior therapists have begun to focus on the merits of the clinical process in promoting the therapist–client relationship and in facilitating effective clinical interactions ([Fishman and Lubetkin, 1983](#); [Herbert, 1988](#); [Hersen and Bellack, 1985](#); [Kazdin, 1988](#); [Sloane et al., 1975](#)). Ironically, some behavior therapists have argued that such factors are even more important to the success of behavioral interventions because the therapist usually is more actively engaged with the client over the course of treatment in setting goals collaboratively and in implementing them through the use of a variety of interactive techniques ([Fishman and Lubetkin, 1983](#)).

### Ethical Issues

The ethical concerns of child and adolescent behavior therapy are no different, in principle, from those found in any other area of psychotherapeutic practice ([Keith-Spiegel and Koocher, 1985](#)). The 1970s saw a growing legal and public demand for increased accountability on the part of all therapists for their clinical practices. This led to a published report on the ethical practice of behavior therapy by the Association for the Advancement of Behavior Therapy ([Azrin et al., 1977](#)). The report sought to protect clients' rights in such areas as therapeutic goal setting, quality and appropriateness of treatment, voluntary participation, confidentiality, and the competency of the therapist.

Ideally, behavior therapists encourage young clients together with their parents to set their own goals for treatment in consultation with their therapist. This minimizes the chance for the treatment to be unduly influenced by the therapist's personal biases. Behavior therapy practice insists that therapists make clear their biases to their clients, establish an explicit treatment contract, and monitor progress periodically to determine whether continued treatment is warranted. Alternative methods of treatment always should be considered, and clients must remain aware of their treatment options as well as their voluntary and confidential participation in any treatment program.

Because so much of behavior therapy occurs in the context of research to evaluate treatment efficacy, these standards apply to clinical as well as research protocols. Special guidelines also have been developed for use whenever more aversive techniques of punishment are implemented as part of the treatment ([American Psychological Association, 1981](#)). Although interventions based on such techniques can affect incidental behavior not targeted for change, they are justifiable as a final treatment of choice for self-injurious and otherwise unmanageable children exhibiting dangerous behaviors. Finally, behavior therapists believe that the ethical practice of therapy involves selecting an intervention strategy well suited to the presenting problem. Such a choice should be based on empirical evidence of the treatment's long-term effectiveness, relative efficiency, likely side effects and response covariation, overall safety, the potential for compliance, and the potential consequences of not offering treatment.

### New Directions

Since the early 1980s, behavior therapy has clearly established itself as a major force in mental health treatment, prompting a reconsideration of previous approaches to practice. Two examples of this changing view are the current value placed on the use of integrative approaches to practice and the considerable interest in more and better treatment outcome studies. Currently, the trend in mental health care is toward a greater integration of proven therapeutic approaches. Therapists of all persuasions are moving toward a pragmatic eclecticism rooted in empirical evidence ([Bornstein et al., 1985](#)), rather than defending their own turf. This open-mindedness is prompted by the reality that most clinical data are equivocal and that therapeutic behavior change is difficult to achieve. Such a perspective will permit clinical researchers to begin to specify which treatments work with which persons and under what conditions ([Kazdin, 1988](#)).

The emphasis on treatment specificity is a major issue in the field of child and adolescent behavior therapy and is one that underlies the current interest in more and better treatment outcome studies. Such studies need to use more adequate controls, use larger sample sizes to be able to detect true differences among sample groups (not only between treatment versus placebo or waiting list control groups, but among groups that receive differing treatments), and examine more comparable

treatments to determine relative differences in their effectiveness on similar disorders. At present, clinical researchers in general have failed to use samples that are large enough to detect the small differences that presumably exist between two or more treatments (Kazdin and Bass, 1989) and usually have compared a heterogeneous mix of treatments for widely varying clinical populations (Kazdin et al., 1992). In addition, treatment outcome studies need to include three-channel assessments of children's functioning as well as assessment of other relevant domains in the child's life, such as family functioning, school performance and adjustment, and peer relations.

Another promising direction for behavior therapy is the increased design and implementation of self-control and self-management interventions. Not only are these approaches efficacious (Durlak et al., 1991; Dush et al., 1989), but they foster the development of such basic competencies as independence and self-control, are relatively easy to use, and are readily accessible through self-help books and tapes that can serve as an adjunct to treatment and, in some cases, produce equivalent results (Webster-Stratton et al., 1989).

Pediatric behavioral medicine is another exciting and growing field in child and adolescent behavior therapy that holds future promise (Lewis and Vitulano, 1988). As one of the fastest-growing interdisciplinary fields, its potential to improve the care and treatment of children with health-related problems is great, particularly in such areas as stress management, pain reduction, treatment compliance, and the care of chronically ill children. Many of these innovative applications will need to be community based to reach the populations for whom they are intended.

Finally, the development of preventive and promotive interventions also is likely to be a major arena for behavioral approaches. Such interventions often include common behavioral techniques (e.g., skills training, role playing, behavioral rehearsal, self-monitoring) that can easily be incorporated into natural settings for children, such as schools (Weissberg et al., 1991). The recent implementation of life skills training programs in schools in which children are taught social skills, self-control, and other behaviorally based skills to reduce high-risk behavior that may lead to human immunodeficiency virus infection and other problems faced by children and youth is one example of a preventive behavioral intervention (Barth, 1993). Behavioral approaches also figure prominently in interventions aimed at promoting resilience among children at risk, such as those surviving a natural disaster, being regularly exposed to community or family violence, growing up in extreme poverty, or being reared by a severely mentally ill parent (Rolf and Johnson, 1994; Tebes, 1994).

Behavior therapy's emphasis on an empirically based science of behavior, coupled with its applicability to a wide range of settings, has enabled it to grow into a field that addresses many of the problems of childhood and adolescence and offers efficacious innovative interventions for their prevention and treatment.

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## 82 COGNITIVE THERAPIES

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Cognitive therapy (CT), rational-emotive therapy (RET), and cognitive-behavioral therapy (CBT) are among the leading empirically tested psychosocial treatments of child and adolescent psychiatric disorders. Clinicians can significantly increase their assessment and treatment skills by learning the basic theory and strategic techniques of CT, RET, and CBT. RET and CT both focus on the role of cognitive distortions in the development and maintenance of psychopathologies. Differences include CT's emphasis on modifying underlying dysfunctional beliefs, whereas RET aims to change specific and dysfunctional self-statements ( [Meyers and Craighead, 1984b](#)). CBT is a hybrid of cognitive, behavioral, affective, and social strategies in which behavior and feelings are altered through changes in thinking patterns. The focus is on behavior and the cognitions needed for controlling and guiding feelings and behavior ( [Reinecke et al., 1996](#)).

### COGNITIVE THERAPY

#### Background and Conceptual Framework

The contributions of such diverse theorists as A. Adler, F. Alexander, K. Horney, S. Freud, G. Kelly, M. Arnold, R. Lazarus, A. Ellis, C. Rogers, and J. Piaget have been acknowledged in the development of CT ( [Weishaar and Beck, 1987](#)). The empirical nature of the approach is ascribed to developments in behavior modification, especially those related to collaboratively developing an agenda for each session, operationalizing problems, testing hypotheses, eliciting feedback, assigning homework, and using problem-solving techniques. In the CT model, feelings and behavior are determined largely by how people structure situations or events in their own minds. Thoughts or images of an event are *cognitions*, which are shaped by classifying, interpreting, evaluating, and assigning meaning to the event based on underlying attitudes or assumptions (schemata) derived from earlier experiences. When, for example, danger is perceived, egocentric (i.e., Piagetian preoperational stage) mechanisms are activated, and primitive thinking is used. Objective reality cannot be clearly distinguished from subjective thoughts and feelings. Cognitions also have been described as stream-of-consciousness awareness for verbal or pictorial events ( [Beck, 1967](#); [Beck et al., 1979](#)). Personal schemata usually develop early in life and can be either adaptive or maladaptive, universal or idiosyncratic, positive or negative ( [Harter, 1977](#); [Wright, 1988](#)).

The schemata are influenced by the child's developmental history before the onset of psychological distress. In stressful situations, latent negative schemata can be activated by circumstances similar to the original event or by an overwhelming of the child's coping abilities. *Automatic thoughts*, cognitions that tend to be repetitious and to occur without deliberation, are then invoked. The normal corrective process—refinement of the perception through testing against reality and prior experience, and then modification to reflect reality—fails to function in these instances. The result is the person's responding with exaggerated affect and deviant behavior. The automatic thoughts, only partially available to consciousness, are accepted as plausible without question. Closer scrutiny of these thoughts can reveal distortions and errors in logic. Psychiatric disorders result when the patient is sensitized to situations that evoke such *cognitive distortions* (systematic errors in reasoning). The consequent dysfunctional beliefs, attitudes, and assumptions become “silent” regulators of behavior. They override common sense and realistic responses, resulting instead in pathologic and disturbing emotional reactions ( [Beck, 1967](#); [Beck et al., 1979](#)). The negative cognitive triad of a negative view of the self, world, and future illustrates this principle ( [Beck, 1967](#); [Beck et al., 1979](#)).

The related depressogenic schemata (e.g., “I am really dumb. Nothing is ever going to go right for me.”) are latent and may be activated by a situation.

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#### CASE ILLUSTRATION

Don, an 8-year-old boy with dysthymic and developmental reading disorders, felt overloaded with homework because of procrastination in completing assignments and not understanding a key concept needed to complete an English paper. As his depression deepened, Don lost the ability objectively to assess his negative thoughts and adaptively process incoming information. Cognitive distortions then followed. Don believed that, because he had problems with one concept for one paper, he was stupid and would never learn English. His automatic thoughts precluded his seeking clarification in his textbook or from class notes. As a result his performance suffered.

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Several types of cognitive distortions or systematic errors in reasoning are associated with depression and require attention in therapy ( [Beck, 1967](#); [Beck et al., 1979](#); [Weishaar and Beck, 1987](#)).

1. *Arbitrary inference*: Specific conclusions are made contrary to or without confirming evidence (e.g., even though Don had always submitted papers in a timely fashion, he began to tell himself, “This material is so complicated that I will never get it and may as well give up now.”).
2. *Selective abstraction*: Details are taken out of context, and relevant data are ignored in conceptualizing an entire experience (e.g., Don thought, “The teacher doesn't like me at all. So I'll probably get a bad grade anyway.” This was based on the teacher's not calling on Don when he had the answer to a question.)
3. *Personalization*: Responsibility for external events is attributed to the self, with no basis for a connection (e.g., “I'm to blame for my parents' fighting.” This caused Don to daydream considerably, to not concentrate, and to miss class material or assignments.)
4. *Absolutistic, dichotomous thinking*: All experiences are placed in extreme categories, and the patient selects the most negative one (e.g., “I'll never learn to do that.”).

Anxiety disorders, agoraphobia, and other phobias likewise are conceptualized as responses to incorrect processing of situations ( [Kendall et al., 1991](#)). CT has been used in treating a number of childhood disorders and usually is combined with other cognitive and behavioral methods ( [Robins and Hayes, 1993](#)).

#### Strategies and Techniques

The goal of CT is to relieve symptoms and prevent future disorder. The therapist is active and directive. Questioning opens the belief system to scrutiny, with behavioral “experiments” serving as means to determine the validity of the underlying dysfunctional assumptions or beliefs. The behavioral experiments usually are developed and agreed on in the session. This process has been labeled a *collaborative empiricism*, with the patient's active involvement being a critical feature. The patient must understand the rationale and objectives of each assignment or experiment and agree to its implementation. The collaborative relationship is enhanced through active elicitation of feedback from the patient regarding his or her perceptions of therapy-related issues, and it aids in handling transference and resistance issues.

Initial treatment strategies are directed at clarifying the model and its theory, defining the problem, and relieving symptoms. Training patients to identify their automatic thoughts and to treat them as testable hypotheses is crucial. Also central is a listing of problems and priorities for treatment, which is incorporated into an overall treatment plan and used to set agendas for each session. *Guided discovery* is a term denoting the process by which Socratic dialogue is used in helping the patient learn to identify maladaptive conditions and underlying assumptions and to test them through consideration of alternatives. Behavioral change is expected to occur



through the testing of these hypotheses, the subsequent development of more realistic, adaptive assumptions, and the increased use of congruent behaviors ([Weishaar and Beck, 1987](#)).

“Cognitive” techniques allow for identifying, testing, and correcting specific distortions in thinking through monitoring negative automatic thoughts; recognizing the interrelatedness of cognitions, affect, and behavior; examining evidence supporting the distorted cognitions; substituting reality-based interpretations for the distorted ones; and changing the dysfunctional, distorting beliefs ([Beck, 1967](#)). They explore the basis of faulty assumptions by operationally defining the assumptions, refining terms, developing measures of probability, and reassigning attribution. Other useful techniques include charting the occurrence of automatic thoughts and disturbed affects, exploring worst-case scenarios, and using imaging to convey self-concepts and fantasies. [A listing of additional cognitive or verbal techniques is available elsewhere ([Beck, 1967](#)).] Once an assumption is identified, the patient and therapist engage in a process of validity testing: determining its universal application, listing supporting evidence, and considering the pros and cons of modifying the assumption or implementing an experiment contrary to the expected result.

“Behavioral” techniques help patients bring forth and test their dysfunctional cognitions and change their behavior. Homework assignments are used in practicing social skills, increasing activity, time structuring, and carrying out exercises targeted to specific situations. Monitoring activity through mastery and pleasure ratings, behavioral rehearsals and role playing, cognitive rehearsals, self-reliance training, graded task assignments, use of “diversion techniques,” exposure to dreaded situations, and relaxation training are additional behavioral techniques of value in conducting CT ([Beck, 1967](#); [Petti et al., 1982](#); [Robins and Hayes, 1993](#)).

### Application to Children and Adolescents

Several strategies to facilitate the successful use of CT in adolescents have been described ([Bedrosian, 1981](#); [Bowers et al., 1996](#); [Leahy, 1988](#); [Petti et al., 1982](#); [Wilkes and Rush, 1988](#)). The need to interview the whole family to learn the context within which the symptoms occur and to get the family involved in treatment distinctly differs in all three models from standard work with adults. [Bedrosian \(1981\)](#) outlines a series of questions useful in addressing this issue. Often the parents, who have brought the youth for treatment, have their own distorted perceptions related to developmental issues of adolescence, which require modification. Similar distortions exist in teenagers (e.g., regarding physical appearance, sexuality, autonomy and control, competency, and peer status).

### SPECIAL ISSUES

As in all psychotherapies, the nature of the therapeutic alliance with children and adolescents differs from that with adults. It is difficult to evaluate and test automatic thoughts in children, even after they have been acknowledged. The alliance can be jeopardized by premature confrontation of the dysfunctional cognitions. The youngsters may not perceive a need for treatment, believing that it is the parents' problem. Modifications to address these issues include examining everyone's view of the problem and acknowledging and accepting the teen's negative view of treatment. A more symptom-focused plan can be developed once the relationship jells. [Wilkes and Rush \(1988\)](#) suggest that offering to teach teens alternative ways of perceiving events is preferable to identifying and correcting logical errors. Use of a triple-column technique, with the headings “fear,” “automatic thought,” and “rational response” placed at the top of the columns, to deal with specific areas (e.g., noncompliance in keeping appointments), is recommended as well.

The techniques and principles of establishing rapport differ little from those applied in adult practice; conflict and threats to the relationship are minimized by modeling a style of information processing that contrasts significantly with that experienced in relation to the parents. For adolescents, the therapist must clarify that he or she cannot control his or her behavior, and that the therapist's evaluation of the teen is quite separate from their reaction to the adolescent's behaviors. The adolescent must feel in control of the flow of information considered and be involved at least in helping to set the agenda for the session ([Bedrosian, 1981](#)). Juveniles frequently have difficulty with lengthy, hour-long sessions; durations of 30 minutes supplemented by meetings with family members usually are better tolerated. Accepting teens' wish to end therapy on cessation of symptoms often is necessary unless a risk of danger is evident ([Bedrosian, 1981](#)). Likewise, expectations for homework assignments are less than those for adults. Flexibly set standards and concrete assignments are necessary. Recordings of activities and behavioral experiments should be expected to be completed more often than cognitive tasks, which can provoke performance anxiety. Some children have difficulty learning to use the techniques. Determining their cognitive capacity to complete the assignments is critical ([Forehand and Wierson, 1993](#)).

### DEVELOPMENTAL PERSPECTIVE

[Leahy \(1988\)](#) highlights the importance of taking a developmental perspective in the use of cognitive approaches with children and describes levels of depression related to development. For example, the therapist could use a blackboard to illustrate concepts such as the “Bad Thought Monster” and the “Smart Thought Man (or Woman)” and to depict examples of types of thoughts for each, have them fight, and so forth, to illustrate conflict, maladaptive and adaptive cognitions, and the relationship between thoughts, feelings, and actions. He also suggests creating an exciting persona such as a “Zen warrior” with the “force” for correct thinking and gaining competence. [Stark and Kendall \(1996\)](#) use similar methods to bring childhood interest to the challenge of automatic thoughts. They suggest that the child be like a detective when challenging maladaptive thoughts that cause depression. Teaming up with “Detective Dan,” the child tests assumptions and beliefs to see if they are realistic. Thus, the same treatment principles used with adults are creatively implemented in socializing youngsters to treatment (e.g., accepting behavioral assignments), identifying and changing underlying assumptions, and setting up experiments to challenge dysfunctional cognitions, commensurate with the cognitive–developmental level of the child. Facilitative modeling is a modification of treatment in which a parent is invited to some sessions to demonstrate interrelationships between thoughts and feelings and to serve as a role model for such behaviors as the sharing of negative self-statements and their correction.

Challenging assumptions is difficult with children, and the therapist may need to draw inferences from limited verbalizations. The Bad Thought Monster, Smart Thought Man (Woman), and Detective Dan are particularly useful in providing examples. Puppets that easily look the part of such representations also can be useful. For children younger than 12 years or those lacking formal operational thinking and the ability to abstract, [Leahy \(1988\)](#) recommends having the child commit adaptive thoughts to rote memory, with the therapist modeling such during the sessions. Less emphasis is placed on Socratic dialogue and more on role playing and reverse role playing in rational responding. Rational thoughts can be carried on flashcards and practiced as self-verbalizations during the day, with the parents being involved with this type of homework. In the initial socialization of the youngster to principles of the cognitive approach, the therapist may be usefully portrayed as “like a teacher” ([Leahy, 1988](#)). Drawing the analogy of therapist as coach also may assist in this process.

The following case represents an attempt to demonstrate the direct application of Beck's CT principles to the treatment of a disturbed child ([Petti et al., 1982](#)).

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#### CASE ILLUSTRATION

John, age 12 years, was referred for suicidal ideation, recurrent depression, and refusal to eat or to go to school. We agreed that these problems, particularly his fear of eating outside his home, had contributed markedly to his dysphoria and would be the focus of our work. He developed an understanding of the interrelatedness of thoughts, feelings, and behaviors through the use of diagrams depicting his own feelings in positive and negative situations. We exposed several major cognitive distortions through role playing, assessing probabilities, monitoring predicted outcomes, homework (“experiments”), and other techniques. Our handling of an invitation he received for holiday dinner at a favorite relative's home illustrates some of these techniques. John's first response was to refuse adamantly to go or even to discuss the possibility. Exploration of this during the session revealed the underlying dysfunctional belief: “I'll get sick and puke all over. I'll be very embarrassed, and everyone will be upset with me.” Thus the resulting automatic thought, “No! I am not going,” and the block to further discussion became more understandable. John then estimated the probability or likelihood that any of the feared events expressed in his dysfunctional thoughts might actually occur (i.e., someone getting sick, getting scared of becoming ill, getting a nauseated feeling, being extremely embarrassed) on a scale from 0% to 100%. The estimated probabilities ranged from 80% to 100%. These were substantially reduced as we discussed his actual past related experiences. Probabilities for pleasant experiences on the visit likewise were analyzed and modified. We then role played the visit and ultimately devised a set of thoughts that could be used when the automatic thought occurred. He practiced them during the session and agreed to do further practice at home and to record the actual outcome as part of our “experiment.” John carried out his homework and reported that no one, including himself, had gotten sick, and he compared this outcome with his early and modified predictions. This became another part of our developing a more complete picture of his distorted cognitions and the means to address situations causing his fears and dysphoria. The parents also were involved in supporting his positive cognitions, adaptive responses, and overall program. Systematically collected data revealed progress in all areas.

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## RATIONAL–EMOTIVE THERAPY

### Background and Conceptual Framework

Rational–emotive therapy or rational–emotive behavior therapy (REBT) ([Ellis and Dryden, 1997](#)) represents a bridge between CT and CBT. RET has been used with children since the 1950s and was among the first cognitive approaches in the psychotherapy of disturbed youth ([Bernard and Joyce, 1984](#); [Ellis and Bernard, 1983](#); [Waters, 1982](#)). As with CT, RET focuses on cognitions and on the identification, then elimination, of sources causing symptoms. Unlike CT, it seeks profound and lasting personality change, emphasizes active disputation with the patient concerning fundamental dysfunctional thoughts, and teaches evaluation of actions. It focuses on the core of the underlying dysfunctional belief system within the structure of an interactional theory of personality and personality disorder, and may be used with developmentally advanced teens but requires levels of cognitive functioning generally beyond those of younger youth. For these younger children, general

RET, "virtually synonymous with CBT," is used ([Ellis and Bernard, 1983](#)).

Rational–emotive therapy applies a panoply of action-oriented activities. In RET, *beliefs* express values and consist of one's appraisal and evaluation of the interpretation of reality and not the interpretation itself. *Assumptions* comprise both interpretations made about reality that can be assessed as true or false and the actual appraisal and evaluation of the interpretations, which also can be evaluated regarding extent of validity. *Irrational beliefs* in the RET framework are antiempirical assumptions held about the self or surrounding world that are formed early in life and often are fixed. In the RET model, emotional disorders are considered to occur following cognitive errors, either from (a) the interpretation of one's perceptions of a situation through empirical distortions of reality, or (b) the faulty *appraisal of the interpretation* through a negative evaluation of the inference with respect to one's life ([DiGiuseppe and Bernard, 1983](#); [Ellis and Bernard, 1983](#); [Waters, 1982](#)). The faulty appraisal of interpretations is considered the key to most emotional disturbance and to treatment. Distortions of reality can be corrected by adequate appraisal. A distorted perception will not result in significant levels of extreme arousal and pathology if it is appropriately appraised ([Bernard, 1988](#)).

Rational–emotive therapy, like CT, aims to modify or replace dysfunctional cognitive–emotive links and thus ameliorate specific emotional and behavioral problems, and to provide broad-based skills and strategies for solving diverse problems during and after treatment ([Ellis and Bernard, 1983](#)). Labeled "ABC(DE)," the therapeutic process is as follows: An activating event (A) is interpreted and analyzed in view of the person's belief (B) about what happened, which creates the emotional or behavioral consequence (C). In and after treatment, disputation (D) uses systematic challenge and question of the untenable hypotheses or irrational beliefs of absolutistic and imperative assumptions held about self, the world, and others in the environment. The new trust in the resulting cognitive–emotive and behavioral changes (E) occurs when the unrealistic assumptions and beliefs are reformatted into more sound and empirically valid statements.

Targeting distortions of inferences drawn during interpretation is a limited, less elegant, and short-lived solution (nonpreferential RET), whereas challenging the appraisal or evaluation of the interpretation (preferential RET) is the approach of choice if the child is capable of and willing to engage in such an effort. RET assumes that children and adolescents are especially influenced by affective and behavioral interventions and susceptible to cognitive appeals through didactic teaching, persuasion, and information giving ([Ellis and Bernard, 1983](#); [Waters, 1982](#)). The basic goal for both preferential and nonpreferential RET is the internalizing of a "philosophy of life/cognitive strategy that is more rational and realistic than the one [patients] commonly abide by when they get into difficulties" ([Ellis and Bernard, 1983](#)).

Adolescents and older children may in addition have the following as goals of treatment ([Young, 1983](#)): learning self-acceptance; learning to dispute personal imperatives (e.g., "musts," "shoulds," and "oughts" and "awfulizing"); and correcting misperceptions of reality. Young also suggests that adolescents can most easily grasp the irrationality of "I can't stand it" thinking and learn to challenge their conviction of being unable to handle inconvenience or discomfort.

[Waters \(1982\)](#) has listed seven goals of RET for younger children: identifying emotions correctly, establishing a vocabulary of terms to describe emotions accurately, distinguishing harmful from constructive feelings, distinguishing thoughts from feelings, being alert to self-talk, connecting self-talk and feelings, and developing rational coping statements.

### Strategies and Techniques

The stages of RET consist of relationship building, assessment, skill building, and application of skills. A high level of rapport may not be required for successful treatment with children and adolescents ([Bernard, 1988](#)). The factors for developing a working relationship are similar to those for CT.

### ASSESSMENT

Problem identification is the first phase of assessment. In this stage, determination is made of the actual existence of a problem and whether it belongs to the child or to individuals in the environment (e.g., parents, teachers) ([DiGiuseppe and Bernard, 1983](#)). The problem analysis phase follows and continues as an ongoing part of therapy. The dysfunctional cognitions, emotions, and behavior contributing to the disorder are determined and the subsequent insights integrated into the evolving treatment plan. Central concerns are expected to be revealed after higher levels of rapport are achieved in the therapeutic relationship. The assessment also addresses the child's cognitive strengths and weaknesses, the extent to which language may control the child's behavior, and the child's capacity to gain distance from the problem. The assessment should identify the antecedent and consequent conditions of the target behavior and the type and severity of maladaptive emotions before the analysis of such dysfunctional cognitions as (a) irrational self-statements and beliefs—self-defeating and irrational appraisal ("awfulizing," self-downing); and (b) causal attributions—appropriateness of beliefs regarding negative and positive events (e.g., negative outcomes are caused by factors related to the self; positive events are attributed to external factors). Additional components of the analysis are areas of strengths and skills, including coping self-statements and practical problem-solving skills—for example, generating several solutions to an interpersonal problem (alternative solution thinking) and predicting consequences of specific behavior (consequential thinking) ([DiGiuseppe and Bernard, 1983](#)).

Direct assessment measures, elaborated by [DiGiuseppe and Bernard \(1983\)](#) to access the self-talk of children and teens, are of great practical use in helping youngsters access their own thoughts and feelings, as well as in providing a mechanism for reporting self-talk. For the RET practitioner, they provide a window into the child's perceptions and verbal-language range and a basis for cognitive restructuring efforts. The following techniques may be of value for conducting most types of psychotherapy with children and some teens ([DiGiuseppe and Bernard, 1983](#)):

1. *Subjective Units of Discomfort Scale* ([Wolpe, 1982](#)): The child is asked to think of the worst feeling he or she has had or can imagine. This feeling is given a 100 rating and then compared with absolute calm, rating 0. The child is then asked, "From this scale, for (situation), how do you rate yourself at this moment, or when you are exposed to—?"
2. *Emotional (feeling) scales*: Children are asked to rate the impact of a particular emotion experienced in a given situation on a 10-point scale. This enhances the child's ability to quantify the strengths of feeling states and to perceive the experience of several emotions from weak to strong.
3. *Emotional flashcards*: The therapist and child take turns play acting the emotion named on the card, while the other guesses at it or tells stories about the emotion. This increases emotional awareness.
4. *Emotional detective*: The child is asked to work on a "case" by investigating how the child or others handle feelings, with the report due at the next session.
5. *Peeling the onion*: The therapist keeps offering verbal prompts until the level of thought activating the dysfunctional thinking is exposed.
6. *Feeling charts*: Descriptors (e.g., pleasant–unpleasant, weak–strong, short–long) are used by the child to depict feelings.
7. *Emotional vocabulary building*: Children list the names of all the feelings they know, then are asked to consider a situation in which a particular feeling was experienced and, if possible, the preceding thoughts. This allows labeling each feeling with a definition and sensitizes children to a wider range of emotions. It also demonstrates the level of emotional understanding and provides an introduction to RET ([Waters, 1982](#)).
8. *Thought bubbles*: The aim here is to convey the notion of thoughts creating feelings and to assist children in making the connections. This can be done with a series of cartoons depicting temporally related scenes of a problem. The cartoons contain characters with dramatic expressions, and the child is asked to fill the accompanying bubble with thoughts of the character.
9. *Sentence completion*: Incomplete sentences concerning problem areas are presented, which the child is requested to complete. These elicit the presence or absence of coping self-statements, beliefs, and practical and emotional problem-solving skills (e.g., "When the girl gets teased before class, she thinks ...").
10. *Think aloud*: Children are asked to think aloud as they attempt to complete a task. This provides a picture of the affective quality of their self-talk (e.g., fear of visiting the dentist for an examination).
11. *Thematic Apperception Test–like approach*: Pictures of ambiguous situations are shown, and the child is asked to create a story about the thoughts and feelings of the characters and explore alternative options in that situation.
12. *Expansion*: Verbal prompts expand abbreviated self-talk; for example, as the child expresses thoughts experienced in a problematic situation, the therapist asks questions and gives verbal instruction to expand the content ("What thoughts came after . . .?" "Tell me more about ...").
13. *Words "and," "but," and "because"*: These are used to facilitate tuning in to and reporting automatic self-talk when the child ends an incomplete sentence about a thought.
14. *Instant replay*: Developed for parents and children to keep track of unpleasant emotions arising from specific situations and events occurring during the prior week, the "rerun" asks them to play back their related thoughts and feelings.
15. *Guided imagery*: The child is asked to relax, to imagine vividly a problem situation, and to describe feelings and related self-talk.

### SKILL BUILDING

The third stage, skill acquisition and skill building, consists of training to use problem-solving skills and help the child or adult take responsibility for their emotions by controlling their feelings through rational thinking and use of such skills. Teaching youngsters to change inappropriate to appropriate feelings frequently involves changing irrational, extreme responses to moderate and more realistically appropriate feelings and actions (e.g., changing rageful anger to annoyance or changing



panic to apprehension) to allow more goal-oriented actions ( [Waters, 1982](#)).

[DiGiuseppe \(1981\)](#) suggests that initial efforts with children should be directed to helping them build a schema with a vocabulary of feelings and responses and a framework for their application. To illustrate how changes in feelings and behavior are possible, the contrasting differences in results between a child's usual negative behavior and what can be expected from more appropriate, less disturbing behavior, achieved by use of alternate strategies, should be discussed. The need to get out of an "emotional fog" and to calm down before taking action is another principle to discuss early on in the process of problem solving.

Teaching the specifics of "ABC," the next step, focuses on awareness of self-talk and the connections between beliefs, feelings, and antecedent events. Older children are taught the difference between rational and irrational beliefs and other critical thinking skills (e.g., absolute vs. conditional thinking; consequential thinking; discriminating fact from opinion, inference, and assumption; learning to take a perspective on a situation; and disputing irrational thinking). Younger children practice coping self-statements in learning how cognitive change can occur and influence B and C in the ABC paradigm ( [DiGiuseppe and Bernard, 1983](#); [Waters, 1982](#)).

Because younger children may have difficulty with the terms *rational* and *irrational*, [Waters \(1982\)](#) suggests substituting *helpful* and *harmful*, respectively. Helpful beliefs are based in reality and result in more acceptable feelings and actions and in getting what you want. Harmful beliefs result in self-defeating thoughts, destructive and nonacceptable feelings, and failure to attain one's desires. A set of questions based on the question, "Is there enough evidence to conclude that this thought is true?" allows the child reasonably to challenge beliefs.

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#### CASE ILLUSTRATION

Fred, a verbal young teen with borderline and schizotypal features, is overly attached and dependent on his grandmother. He asked her to help monitor his homework. Learning disabled but very bright, he frequently makes errors. When she suggests that a word may be misspelled or a sentence incomplete, an argument ensues because he inevitably challenges her point. Fred was finally able to share his underlying belief that being wrong was a sign of weakness and defeat. To accept her correction meant "submitting" and defeat. He learned to challenge the rationality or helpfulness of the belief by questioning the correctness of the appraisal and seeking evidence to try to support it. He decided that it was not so awful to have her point out errors, that he was really strong enough to accept such assistance, and that it could be much worse if she corrected in a belittling fashion or not at all. He learned that the harmful belief ran counter to his own needs and desires. Most critically, he came to understand that making errors did not make him any less worthwhile and that accepting corrective feedback could improve rather than diminish his stature as a person of worth.

Fred's case also illustrates a more sophisticated intervention. He had just received a poor grade from a recent examination. Beyond learning to dispute the concepts of "ain't it awful" or "I'm just stupid" with regard to that particular episode, he was able to look at the more general issue of what needs to be done when you have a learning handicap, and that it is different from being stupid or not having any control over the situation. He discussed what might be done differently in the future. The next step was to apply empirical analysis, a strategy to modify dysfunctional interpretations. As in CT, therapist and child agreed to design an experiment to evaluate his belief of being stupid. Fred agreed to pay closer attention to the teacher and write assignments and tests down in a notebook that would be kept up and brought home daily. We then compared the results of the next examination with his earlier experience. He tested whether he (a) would do poorly again, to see if he did have some control over the situation, and (b) could do well, contrary to his belief that he was always doomed to fail. His belief was that he had done everything possible and that he had no control over his grades. Fortunately, the behavioral structure he imposed on his schoolwork demonstrated that organizational skills and not stupidity accounted for his poor performance.

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Rational-emotive imagery ([Ellis and Bernard, 1983](#); [Waters, 1982](#)) is another strategy that asks the child to imagine a picture depicting a situation that generates an extreme emotion. The child is asked to change the feeling to a milder form (e.g., from hating and greatly fearing a bully to a strong dislike and worry about the bully). Once this is done, the link between thought and emotional change is highlighted for present and future use.

#### APPLICATION OF SKILLS

Implementation of the newly learned rational thinking skills outside the therapy session is the final stage of RET. Homework assignments are the major vehicle in effecting this task. This involves, for example, practicing the skills; monitoring their effect and associated feelings; listing personal demands; listing thoughts, feelings, and behaviors related to specific events; completing a self-concept inventory; and using positive self-talk as a reinforcer ( [Bernard, 1988](#)). This phase requires considerable effort by both therapist and child because major changes in thinking, feeling, and acting are being attempted. The relationship must be sufficiently established to weather the expectations placed on the child. Developing a system of reinforcers in cooperation and collaboration with the parents can be useful in overcoming resistance to completing the homework or in overcoming a well entrenched set of bad habits. The therapist also can focus on irrational beliefs in the session to deal with this difficult issue ( [Bernard, 1988](#)). In children with cognitive deficits rather than distortions, training in such CBT models as interpersonal cognitive problem solving (ICPS) ( [Spivack and Shure, 1974](#)) is most appropriate.

#### ROLE OF PARENTS

Involvement of the parents in all stages of assessment and treatment facilitates a successful outcome. Assessment provides a guide to the extent to which parental behaviors and beliefs influence the child's problems. Goals and principles to use in work with the parents have been developed ( [Bernard, 1988](#)), and the same four stages are applicable, from the development of rapport to practice of skills outside the sessions. Likewise, problem identification and analysis of difficulties that exist in the family are analogous to those for the children. The focus is to assist the parents in overcoming their own and the child's difficulties by developing an understanding of their basic dysfunctional beliefs and an awareness of more constructive alternatives. Participation in their own homework also is expected, including better control of emotions, distinguishing rational from irrational ideas, implementing behavioral changes, and use of child-related literature ( [Bernard, 1988](#)). The RET approach of [Block \(1978\)](#) has been found to be one of the probably efficacious treatments for adolescents with conduct problem behaviors ( [Brestan and Eyberg, 1998](#)). The use of RET in the family context is articulated by [Ellis and Dryden \(1997, pp. 152-161\)](#) in their case example of family therapy for a family of five, including a disturbed and disturbing 15-year-old daughter.

## COGNITIVE-BEHAVIORAL THERAPY

### Background and Conceptual Framework

Cognitive-behavioral therapy, a major force in the treatment of children, is based on developments from the behavior therapy movement of the late 1960s and the thrust of psychology in a "cognitive" direction. A number of workers have been credited for their major contributions to this growth ( [Kendall, 1993](#); [Mahoney, 1993](#); [Petti, 1989](#)). The evolution occurred along three paths: (a) advances in cognitive and developmental psychology ( [Bandura, 1977](#); [Meichenbaum, 1977](#); [Shirk, 1988](#)); (b) evolution of self-control procedures from the operant model, including [Bandura's \(1977\)](#) concept of reciprocal interaction between individuals and their environment; and (c) the contributions of CT and RET, as noted previously. CBT reflects an integrative approach that incorporates CT and RET. As such, it has become the primary cognitive therapy approach used with children and adolescents. In addition to being widely used, CBT has been shown to be efficacious in studies of several disorders ( [Kazdin and Weisz, 1998](#)). Empirical research has shown CBT to be superior to no treatment and to some other types of psychotherapy (including placebo support) for depression (Clarke et al, 1999; [Kaslow and Thompson, 1998](#)), anxiety ( [Ollendick and King, 1998](#)), obsessive-compulsive disorder ( [March and Mulle, 1996](#)), and conduct/oppositional disorders ( [Brestan and Eyberg, 1998](#); [Kazdin and Wassell, 2000](#)). Many CBT interventions are considered probable empirically supported treatments (ESTs). ESTs are defined as "clearly specified psychological treatments" shown to have their efficacy "established" or "as probably efficacious" in controlled research with a delineated population ( [Chambless and Hollon, 1998](#)). To be designated an EST, a treatment must meet rigorous research efficacy criteria ( [Chambless and Hollon, 1998](#); [Lonigan et al., 1998](#)) that are roughly analogous to those required of medications for clinical indication. ESTs are becoming one of the major standards of care for psychotherapeutic intervention. Hence, consistent findings of empirical support for the use of CBT with diverse disorders suggest that CBT interventions are some of the most effective modalities of psychotherapy.

Primary CBT goals are concordant development of efficient and adaptive modes of perceiving, interpreting, solving, and responding to problematic situations, leading to an increase in adaptive thinking and behavior and the elimination or reduction of maladaptive and inappropriate behaviors. This is accomplished by developing and internalizing self-control skills and reflective problem-solving strategies. CBT integrates the concept from behavior therapy that antecedent and consequent environmental factors contribute to overt behaviors, with concepts from cognitive and rational-emotive therapies that emphasize the role cognitions play in dealing with environmental stimulation and changing overt behavior ( [Craighead et al., 1985](#)). Cognitive distortions and deficiencies associated with deviant or dysfunctional behavior are the focus of concern ( [Kendall, 1993](#)). A multimodal approach to treatment is considered necessary to address the multidimensional factors that contribute to and maintain the psychopathology. Underlying assumptions have been summarized by [Cohen and Schleser \(1984\)](#): (a) active problem solving is expected in childhood; (b) discrimination, extraction, and analysis of data and subsequent planning are characteristic of problem solving; and (c) styles of problem solving change with developmental progression. The child is assumed to be an active participant in and interpreter and recorder of experiences. The developmental implications and skill levels of the child are considered essential to planning treatment strategies. Cognitive therapies with adults can successfully focus on their subjects' distorted processing of information and world views. In most children and many adolescents, however, the issue of distorted cognitions is considered of less importance than the lack of effective strategies for controlling behavior ( [Braswell and Kendall, 1988](#)) and the prominence of impulsive cognitive styles over more reflective styles ( [Cohen and Schleser, 1984](#)). The need to differentiate between the presence of cognitive deficiencies, distortions, or both is critical in helping the

child build a new “cognitive template” to identify and solve problems ( [Kendall, 1993](#)).

With age, children become more reflective ( [Leahy, 1988](#); [Roberts and Nelson, 1984](#)). Strategies for implementing CBT interventions also must consider the developmental level of the child’s cognitive skills ( [Durlak et al., 1991](#)) and attentional, memory, and related skills. Achieving a level of formal operations allows the teen to attend selectively to tasks, to use deductive reasoning and problem solving, and to learn strategies that can be applied in a flexible manner. Those who have attained the concrete level of operations can appreciate the usefulness of a strategic approach to problem solving, but may not be able to focus their attention or may get excessively involved in the problematic situation and thus may lose details in the process. They need to be made aware of the value of using a mnemonic strategy. Preoperational and preschool children function on a here-and-now basis. They are incapable of planning for a problem-solving situation ( [Cohen and Schleser, 1984](#)).

Generalization of change to other situations, settings, or environments is a major consideration in conceptualizing strategy. Problem identification, goals of treatment, and sequence of planned intervention strategies depend on the child’s level of development ( [Roberts and Nelson, 1984](#)). Increasing a child’s ability to report experiences accurately, evaluate his or her own performance situation, and self-monitor mood is considered a major objective of CBT interventions.

## Strategies and Techniques

Many types of CBT models exist; the names of these approaches include *cognitive–developmental* ( [Harter, 1977](#)), *cognitive training*, *cognitive behavior modification* ( [Meichenbaum, 1977](#)), *cognitive–behavioral–psychosituational* ( [Griegeer and Boyd, 1983](#)), and others ( [Dobson, 1988](#); [Dryden and Golden, 1987](#); [Meyers and Craighead, 1984a](#)). The basic strategies in general are similar. Most of the variants emphasize one facet of CT, RET, or CBT over another, depending on the developmental level of the child, the targeted behavior, and the techniques to be used.

## ASSESSMENT AND TREATMENT

[Roberts and Nelson \(1984\)](#) review assessment issues for general use with CBT. These include the role of developmental processes, identification of target behaviors, evaluating outcome, and relationships between measures for verbal and motor behavior. They also outline cognitive and behavioral assessment strategies related to academic and social problem solving and consider rating scales, checklists, measurement of academic performance, and behavioral observations.

Several standardized scales and approaches have been devised to assess the family structure in CBT with conduct-disordered children and teens ( [DiGiuseppe, 1988](#)). Understanding the extent to which children have the cognitive repertoire needed to understand therapeutic interpretations and their causal/attributional/locus of control style is considered a critical aspect of assessment ( [Shirk, 1988](#)). [Braswell and Kendall \(1988\)](#) list a number of other behavior rating scales, performance measures, and self-report instruments used to assess children’s self-talk, expectancies, and attributional style.

Differentiation between internalizing and externalizing disorders is one way to select treatment strategies. Internalizing disorders (e.g., depression and anxiety disorders in older children and teens) might best be treated through recognizing and testing “mistaken misperceptions, expectations, and/or attributional preferences,” whereas externalizing conduct-disordered children with deficiencies in effectively mediating their behavior might be approached through training in verbal mediation techniques (e.g., self-control and more reflective problem-solving approaches) ( [Braswell and Kendall, 1988](#); [Kazdin and Weisz, 1998](#)). Even within the same diagnostic category, several different CBT methods and strategies might be considered. Multiple approaches to particular types of disorders are available. [Kendall \(1993\)](#) lists general treatment strategies of CBT as modeling, building a coping template, rewards, enactive procedures, affective education, and training tasks, and provides specific methods for each. The following describes representative CBT models.

## MODELS

### Verbal Self-Instruction Training

Among the most widely used CBT procedures, verbal self-instruction training (SIT) has been demonstrated to be efficacious in assisting children with self-control and self-monitoring problems ranging from anger outbursts to social skills deficits and impulsive behavior. SIT involves learning and practicing a set of procedures for interpreting situations, guiding behavior, and solving problems. Initially, the procedures are practiced by saying them aloud as problems are solved. Later, the child learns to use SIT procedures with internalized talk and well rehearsed responses to problem situations.

[Kendall \(1992\)](#), for example, uses SIT as part of a manual of CBT treatment for impulsive behavior called *Stop and Think*. The Stop and Think treatment program begins with the child learning a five-step problem-solving plan for slowing impulsive behavior: (a) identify the problem (“What am I supposed to do?”); (b) generate alternatives (“Look at all the possibilities”); (c) select the best solution (“Pick an answer”); (d) evaluate the solution (“Check out your answer”); and (e) self-praise or redirection back to task (“I did a good job” or “I’ll pick another answer and do better next time”). Children initially say these problem-solving steps aloud as they solve a variety of word and math problems for practice. Later in treatment, they practice the problem-solving steps with increasingly difficult and realistic situations (such as social interaction). Homework assignments encourage children to practice their problem-solving steps at home and to learn to monitor their feelings and behavior. Other components of Stop and Think teach children to anticipate feelings, control emotional responses, and understand what others might be thinking or feeling. The ultimate goal of Stop and Think is to teach the child to use the SIT problem-solving steps in complex, provocative (e.g., emotional, social) situations to produce more planned, careful behavior.

Self-instruction training also is a part of programs that address depression ( [Stark and Kendall, 1996](#)) and anxiety ( [Kendall, 1990](#)). These interventions use SIT (as well as several other CBT techniques) as a part of problem solving and learning new patterns of interpreting situations. They help because negative cognitive appraisal of (e.g., making sense out of) situations often leads to depressed or anxious symptoms. Although SIT programs for depression, anxiety, and impulsivity differ somewhat in content, they share in common the use of internal cognitive and behavioral directions to carry out the goals of treatment.

There is strong evidence that SIT improves children’s problem-solving ability and coping skills ( [Kendall and Braswell, 1993](#)). Research has shown Stark’s cognitive–behavioral treatment for depression and Kendall’s cognitive–behavioral treatment for anxiety to be efficacious in reducing symptoms of these disorders in children ( [Kaslow and Thompson, 1998](#); [Ollendick and King, 1998](#)), although SIT is only one component of these interventions. Treatments for attention deficit/hyperactivity disorder (ADHD) that use SIT alone have not been found to be very effective for the core inattention–disorganization and hyperactive–impulsive symptoms of the disorder ( [Hinshaw, 1996](#)). However, SIT may be more effective for impulsivity and aggressive behavior related to social and anger control problems ( [Hinshaw, 1996](#); [Kendall and Braswell, 1993](#)).

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## CASE ILLUSTRATION

Nine-year-old Joe was referred for severe hyperactivity and oppositional behavior present from the time of his adoption at 6 years of age. His dysthymia was successfully treated with imipramine, but his impulsive and negative behavior persisted. His adoptive parents were most concerned about his ravenous “stealing, wolfing, and secreting” of sweets. He and his parents agreed to target this behavior for treatment. After collecting baselines for missing goodies, a self-control program was initiated. First Joe’s thoughts, feelings, and actions were explored using the thought, “I want/need a cookie.” A written sequence was developed, which was practiced out loud in the sessions for Joe to learn to use at home:

I want cookies!  
What AM I supposed to do? (Think of available choices.)  
I need to look at all my alternatives!  
Grab it and take a chance on being punished, feeling guilty, etc.  
Ask my mom or dad and expect ...  
Wait till later to ask and expect ...  
Which is my best choice?  
How should I do it? and, later to discuss in therapy ...  
How well did I do? [Petti, 1989](#).

Joe refused to keep a log monitoring his thoughts, feelings, and success in maintaining control when faced with the urge. He did report verbally what occurred, and this compared well with parents’ reports. His probability estimate for how successful he would be in controlling his behavior around sweets was less than 50%. Both he and his parents were therefore delighted at his close to 100% success rate. A similar approach was taken with other impulsive behaviors.

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Interpersonal cognitive problem solving is an approach that mixes problem-solving skills with interpersonal challenges. ICPS makes intuitive sense because good social behavior is based on processes such as means–end thinking, accurate appraisal, evaluating alternative responses, and choosing an appropriate response for a problem. Conversely, fixation on a goal and failure to think through the means to reach the goal can lead to problems in social behavior. When problems in social behavior occur, the child may resort to less skilled means of achieving or avoiding social goals, such as aggression and withdrawal. By carefully appraising, planning, and choosing problem-solving behaviors, children can therefore improve their social behavior.

Children are taught ICPS by first learning that many alternative behaviors may occur in a given social situation and that their behavior is their choice. Next, means–end thinking, connecting behavior and social consequence, is encouraged ( [Spivack and Shure, 1974](#)). Later, children are taught problem-solving steps, using SIT techniques. A typical progression of problem-solving steps involves identification (accurate appraisal) of the problem, generating possible solutions, evaluating the solutions, choosing a solution, and evaluating the adequacy of the solution (if the solution fails, the problem-solving steps are used again). The child practices the problem-solving steps on increasingly difficult social problems. Role playing is used to make the practice more realistic and to anticipate difficulties. Most ICPS programs include a reinforcement component in which children can earn rewards for adequate performance of ICPS steps and goals ( [Kendall, 1992](#); [Kazdin, 1996](#)).

Interpersonal cognitive problem solving has been shown to be an efficacious preventive intervention in poor urban preschools ( [Shure, 1999](#)), although the relationship between ICPS skills and later social behavior has been questioned ( [Braswell and Kendall, 1988](#)). ICPS, especially when combined with parent management training, has been shown to lead to decreased aggressive and externalizing behavior, as well as to improved prosocial behavior ( [Kazdin, 1996](#)). ICPS also involves developing the skills to name unconnected, alternative solutions (alternative solution thinking), to weigh pros and cons of those solutions to a situation, and to think of the different outcomes that might occur (consequential thinking). Implementation of ICPS by teachers and parents of preschool and early school-age children has been shown to prevent violence and high-risk behaviors. Manuals and books are available for this purpose ( [Shure, 1999](#)).

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#### CASE ILLUSTRATION

In the treatment of a depressed, impulsive 12-year-old ([Petti and Wells, 1980](#)), situations provoking anxiety or anger were hierarchically arranged from least to most arousing. Scenes evoking the lowest level of arousal were addressed first through role playing of appropriate cognitive, behavioral, and verbal responses. The responses were first modeled by the therapist. The youngster was then coached in their use and provided positive feedback as he role played each situation. The emotion-arousing situations were practiced until the patient's responses were satisfactory to both patient and therapist. Follow-up indicated better than adequate functioning.

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#### CASE ILLUSTRATION

Jean is a 14-year-old girl with low-average intelligence and a long history of being sexually abused in a chaotic "family" setting. Her episodic dyscontrol became prominent after placement in a supportive preadoptive home. During an inpatient stay, her lack of interpersonal skills was evident. She had developed a knowledge of the basic language concepts required for this approach and had some appreciation for the feelings of herself and others. However, she was wedded to "satisficing"—taking the first available option that came to mind, which was usually based on long-standing distrust of others and was predominantly hostile, aggressive, and negative in nature. Several episodes on the unit were used to develop the concept of seeking alternative explanations and responses, then to tie the options to possible consequences, and finally to anticipate the consequences of her actions when confronted with conflict. She then practiced this evolving skill around her desire to do what she wanted on an outing, in conflict with the desires of her caretaker. She was able to negotiate a compromise acceptable to all. This allowed her then to trust and learn to discuss conflicting views and desires without assuming the worst before she even began the process. She ultimately felt more confident in relating to peers, staff, and family members. Initial preparation of significant others in the environment to be receptive to these changes can be the critical factor predicting the degree of successful outcome.

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#### Self-Evaluation and Self-Management Skills

Self-evaluation skills consist of learning accurately to recognize and label internal states and external behaviors. Typically, a self-evaluation protocol begins with a discussion of the behavior or internal state to be identified. Concrete, specific examples of the behavior are given to teach the child to recognize it. [Kendall \(1990\)](#), for example, uses facial expressions and bodily reactions to teach children to understand anxiety states. The child's improved self-evaluation allows him or her both to identify anxiety states and understand the impact of anxiety states on expression and physical functioning. After discussing the characteristics of the behavior or the internal state, the child then practices accurately identifying it in the normal course of daily life. [Hinshaw \(1996\)](#), for example, uses a gamelike format to teach children with ADHD about their behavior. Both therapist and child rate the child's target behavior on a scale of 1 (worst) to 5 (best). If therapist and child ratings match, the child receives a reward. Self-evaluation is a part of many cognitive–behavioral programs that require the child consistently to monitor target behaviors. Used in isolation, it may promote better control over behavior by making the child more aware of the target behavior itself or the precursors of the behavior.

Self-management skills apply the knowledge gained in self-evaluation in the service of modifying behavior. One type of self-management skill, self-regulation, involves (a) self-evaluation, (b) setting acceptable objectives, (c) evaluating the response, and (d) reinforcing oneself if the standard is met. [Williamson and associates \(1981\)](#) describe a modification of this approach with an extremely hyperactive, distractible young boy and the subsequent dramatic decrease in his need for stimulant medication. Correspondence training, on the other hand, is a self-management intervention that operates by rewarding children for doing what they say they will, for accurately describing what they did, or for showing an appropriate response to a targeted situation described by the therapist. [Hinshaw \(1996\)](#) describes an anger self-management program that consists of self-evaluation/monitoring followed by use of strategies to reduce anger/aggression and practice of the skills during mock provocation by peers in a group. [Hinshaw \(1996\)](#) has shown in a series of studies that self-evaluation/self-management training for anger control results in improvements in angry-aggressive behavior.

## CONCLUSIONS

The cognitive therapies are varied and comprise amalgams of diverse contributions. It has been argued that they are simply extensions of the behavior therapies ([Wolpe, 1982](#)). However, collectively they do address in a systematic manner virtually all nine of the skills axes and methods to promote these skills considered by [Strayhorn \(1983\)](#) to be relevant to psychotherapy and preventive mental health. Combinations of treatment components usually are used in most studies ( [Durlak et al., 1991](#)).

Overall, the cognitive therapies are recognized as research-validated, efficacious psychotherapeutic approaches for depressive, anxious, and some disruptive behavior disorders ([Kazdin and Weisz, 1998](#); [Kendall, 1991, 1993](#); [Lochman, 1992](#)). They provide opportunities for briefer, empirically supported, cost-effective interventions and allow developmentally appropriate, multimodal assessment and intervention. Combined treatments developed for specific disorders ( [Bernstein et al., 2000](#)) can be expected. Manuals (e.g., [March and Mulle, 1998](#)) to train therapists in implementing CBT represent a major advance in the field. They provide researchers with the means to ensure uniformity of techniques across investigators and individual clinicians. CBT often is used in a group format, especially in early intervention in schools ( [Clarke et al., 2001](#)). Limitations ascribed to CBT research related to its evaluation with nonreferred populations fail to consider the major value of this efficacious approach to early intervention and the secondary prevention of more severe forms of psychiatric illness when delivered in schools and related settings ([Petti, 1999](#)).

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# 83 GROUP PSYCHOTHERAPY FOR CHILDREN AND ADOLESCENTS

Fern J. Cramer-Azima, Ph.D.

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Between 1995 and 2000, somewhat more than 500 articles, chapters, books, and dissertations were written about group psychotherapy with children and adolescents. From the clinical perspective, there have been advances in the treatment of previously contraindicated populations (low intelligence, organic cases, the very young, and the very difficult, acting-out child.) This chapter updates the previous review ( [Azima, 1996](#)) and includes new group applications to high-risk psychiatric disorders, focusing specifically on research advances in these areas. It is clear that there has been a marked shift in theoretical orientations from psychodynamic/psychoanalytic to cognitive-behavioral/interactive ones, especially for short-term modalities. This shift has encouraged structured and manualized approaches that can be operationalized and subjected to research investigations.

Increasingly, child group psychotherapy is becoming part of a multimodal integrative approach for outpatient and inpatient, day hospital, and residential facilities. Psychoeducational approaches with children and parents have become more prevalent, as has their use in schools and communities. As is illustrated, the emerging problems of our society that have affected youth have dictated the increased use of groups for anxiety, depression, suicide, violence, oppositional defiance, drug addiction, posttraumatic stress, social phobia, medical disorders, family separation, and sexual abuse.

An early report by [Toseland and Siporin \(1986\)](#) that group psychotherapy is an efficient and cost-effective treatment has been further confirmed by the meta-analysis of [Hoag and Burlingame \(1997\)](#). These authors analyzed 56 outcome studies published between 1974 and 1997 on the effect of group treatment with children and adolescents (4 to 18 years of age). A variety of types of group treatment were assessed, including preventive programs, psychotherapy, and guidance, counseling, and training groups. The results indicated that group treatment was significantly more effective for children than wait-list and placebo-controlled groups (effect size = .61), with the average child or adolescent treated in groups better off than 73% of those in control groups. Some findings were challenging and surprising, in that variables that were significantly related to improvement included socioeconomic status of the patient, the allegiance of the therapist, setting of the therapy, and publication year of the study. Variables not related to improvement included diagnosis and content and source of the outcome measures. Future meta-analyses are necessary to confirm these findings. The current clinical and research overview corroborates positive outcomes in most cases for both clinical and research reports.

Clinicians have continued to be enthusiastic that group psychotherapy for children and adolescents is a treatment of choice. Young, active children can communicate with one another, often without words or symbolic reasoning, as if they have a private language. Therapists have the opportunity to observe the actual behavior of the children, to clarify the diagnosis, and gradually to interpret the meaning of their play and interactions. Psychotherapeutic and educational goals are provided in a safe, supportive, empathic setting where boundaries and rules are established by the therapists. As for adolescents, group settings are particularly advantageous because of their specific need for relationships with their peers. Reciprocal exchange of thoughts and feelings permits self-disclosure in the group that often is not possible in individual therapy, where rebellious silences pervade the transference to parental authority figures. In the group, the adolescents themselves are clear about what is acceptable and proper for others in their age group. Learning that problems are not unique and that they are shared by peers can promote faster sharing of information.

## APPROACHES

Current group psychotherapeutic approaches for children stem from modifications of traditional activity therapy ( [Schiffer, 1977](#); [Slavson, 1952](#)) and group analytic models ( [Anthony, 1965](#)). For young children, these approaches are blended with psychoanalytically oriented play therapy ( [Ginnott, 1961](#); [Phillips, 1985](#); [Winnicott, 1971](#)).

### Activity Group Therapy

This approach, introduced by [Slavson \(1952\)](#), focuses on observation of the children's behavioral and motoric communications and is carried out in a permissive, empathic, supportive setting. The focus is on seeing how the child relates to and copes with other children and physical objects in the group context. Transference interpretation is minimized. The therapist provides little structured activity and intervenes only when a child is in danger, is hurt, or threatens another child. Toward the end of the session, refreshments are provided.

### Activity-Interview Therapy



This modification was formulated by [Schiffer \(1977\)](#) to provide additional structure to the original technique. The first part of the session allows group play with peers, followed by a structured discussion period. The fantasy produced in the play period is explored, with the therapist imparting meaning, insight, and transference interpretation. In addition, the group dynamics and bonding with peers and the therapist solidify over time and provide the vehicle for the therapeutic change.

### Group-Analytic Psychotherapy

Current activity models are more integrative with psychodynamic theory. [Johnson et al. \(1998\)](#) outlined a guide of activities useful in many settings. This model was adapted by [Anthony \(1965\)](#) for nursery, latency, and adolescent groups. The therapist intervenes rarely with the nursery children, keeps to the background, and fits interpretation into the play themes. A specified "small table" technique was evolved, which concretized the space or territory of the table and room for each child and therapist and permitted observation of changes over time. For the latency group, a "small room" technique was used, in which a discussion group was followed by an activity phase. Repeated themes identify the children's conflicts, whether they are expressed in symbolic play, free associations, or fantasies and dreams. The older latency children begin to demonstrate more of the specific group analytic factors introduced by Foulkes, such as socialization, mirroring, the condenser, and chain phenomena. The condenser is a sudden reaction to the group that is discharged by a particular event. The chain phenomenon is likened to the group's free-floating discussions, in which the topic is carried forward by each individual's response, followed by a condenser outburst. The specific roles of the scapegoat, stranger, and historian in the latency-age group are still highly relevant today. Although the technical aspects of the approach may be modified, the therapist's psychodynamic understanding remains central.

Some 60 years later, current group approaches include pure or blended psychoanalytic, psychodynamic, behavioral, and cognitive models, combined with family and network interventions. The precise applications appear to be the result of the group therapist's theoretical training, the particular setting's philosophy, and the type of population that is in need of treatment.

Most therapists working in the field today use a developmental framework involving parents in parallel treatment for the younger children. The two major theoretical trends are psychoanalytically oriented and behavioral–cognitive approaches, both emphasizing here-and-now interactions. Unfortunately, in this age range there has been insufficient attention paid to the actual interactions in the matrix, the reciprocity patterns, and the changes over time in leader dominance and submissive followers within the group ([Azima, 1982](#)).

The group therapy area in the past suffered from an undue borrowing from individual therapy theory. For group theorists, the historical premise is that the infant is embedded in a symbiotic group fusion and, developmentally, only gradually becomes individuated. There has been a revival of interest in interpersonal ([Sullivan, 1953](#)) and peer theories. [Grunebaum and Solomon \(1987\)](#) concluded that the ability to master peer relationships and define friendship patterns involves a separate line of development from that of the mother–child dyad. These authors do not negate the importance of the parent–child relationship, but emphasize the independence of peer competency. These authors have postulated that self-esteem and peer relationships are such interconnected phenomena that self-evaluation may be viewed in large measure as the inner experience of the esteem in which one is held by one's peers. Nowhere is the study of peer relationships more important than in child and adolescent group therapy.

As in all therapies, the essential ingredient is the analysis of content and process, which over time allows the unfolding and recapitulation of the past in the present context. The group affords an increased expression of thoughts and feelings, including projective biases that allow each member to be understood from many more points of view than a single therapist can hypothesize. Further, the projections open a window into the speaker's own pathologic process. The allies and dissidents actively demonstrate the variation in projective identifications, as if it were a system of revolving mirrors, reflecting intrapsychic and interpersonal coordinates for each member. The differing responses to the individual speakers promote the working-through process and constitute a corrective monitoring system for the group therapist. Among other important features of multiperson therapy is the provision of a social context and the rebuilding of more accurate interpersonal relationships; the development of bonding, an esprit de corps or group climate that motivates and energizes the loyalty, intimacy, openness, and cohesion of the membership; the development of a group composition that permits stronger, better-functioning members to interact therapeutically with ego-weak, more pathologic children; and the working through of dominant/submissive, independent/dependent roles ([Garland, 1992](#); [Pfeifer, 1992](#); [Spinner, 1992](#)).

### APPLICATIONS AND TECHNIQUES FOR CHILDREN'S GROUPS

Increasingly, community demands have led to the introduction of short-term models ([Epstein, 1976](#); [Scheidlinger, 1984](#)) and the introduction of structure and innovative techniques to focus the attention of the emotionally disturbed child to allow self-disclosure. [Frank \(1983\)](#) introduced the concept of dramatic play for greater structure and therapeutic support for ego-weak children. Central to the controversy was the dilemma of whether the children's verbalizations were necessary for an interpretive approach ([Charach, 1983](#); [Sugar, 1974](#)). Not only were many of the children nonverbal, but they lacked the symbolic ability to communicate their thoughts and feelings.

Significant sociocultural changes have altered the concepts of latency and adolescence and have led to a modification in group composition, the roles of the members, and the functions and countertransferences facing the therapist ([Azima, 1977](#)). The bulk of referrals for clinic and day hospitals are for defiant, acting-out children from single-parent families in which the father is absent or plays a minor role. These children often are both witnesses and victims of psychological and physical abuse. Simultaneously, they are encouraged by television, music, and dance to act out more of their sexual and aggressive fantasies. These youngsters in the group are boisterous and outspoken, and use sexualized language and behavior.

[Aronson and Scheidlinger \(1997\)](#) demonstrate the current trend to integrate psychoanalytic and psychodynamic and developmental theories in therapy groups. [Canham and Emanuel \(2000\)](#) and [Privat \(1999\)](#) both use concepts from Bion as a basis for their approach. The former authors describe a 1-year psychoanalytic psychotherapy group for young children (4 to 8 years of age). Privat outlines a strict, "closed," classic monotherapy for seven children, aged 9 to 11 years.

The literature review demonstrates a decrease in the use of the psychoanalytic model and a significant increase in cognitive-behavioral, psychoeducational and multimodal approaches. In contrast, cognitive–behavioral and psychoeducational models have proliferated, with accompanying clinical research. These trends have encouraged the goals of short-term, managed care, and the integration of parents, community, and psychopharmacologic treatments. Psychoanalytically oriented theory remains central for long-term groups, and more traditionally trained clinicians whose goals may be more significant ego changes for their patients.

### COMPOSITION AND STRUCTURE

A major task facing the therapist is the selection and balancing of the composition of the group, orchestrating the number of acting-out children with the quieter, less demanding, more compliant ones, and deciding on the play, activities, or toys used to engage and interest the children. The reality is that there is no such thing as an ideal composition, and a group is formed with the available referred candidates. The stronger the group, the more very disturbed children can be included.

As a rule, the children are seen and evaluated with their parents. Often, observing the children over time helps clarify the diagnosis ([Anthony, 1965](#); [Liebowitz and Kernberg, 1986](#)).

#### Age Groupings, Heterogeneity, Homogeneity, and Frequency

Preschool children, boys and girls 3 to 5 years of age, are seen in small groups of three, four, or five and usually by two therapists in a play or activity group. The more active or pathologic the children, the greater the need for auxiliary personnel.

The latency therapy group usually is separated into early (5 to 7 years of age), middle (8 to 10 years of age), and late (10 to 12 years of age) groups. Frequently these age groups are intertwined, and more attention is placed on the composition of the range of intelligence, physical size, and diagnoses of the children. In treatment groups, latency boys outnumber girls, at a ratio of 4:1 to 8:1, depending on the population being serviced. There is some current indication of a rise in girl referrals, however. The preponderance of boys in the groups necessitates that one of the therapists or special care counselors be male to provide a role model and to diminish acting-out behavior. Some of the children from single-parent families are threatened by the presence of both male and female therapists.

At times, the late latency group is combined with the preadolescent group. The older boys and girls usually do better in homogeneous groups with the same-sex

therapist ([Kennedy, 1989](#)).

Frequency of sessions varies from once or twice a week to every day in a day hospital. A beginning group may tolerate only 15 minutes and gradually work up to 45 minutes or an hour.

### Play, Toys, and Activities

Play and activities are the natural vehicles for child therapy ([Bratton and Ferebee, 1999](#); [Sweeney and Homeyer, 1999](#)) and, in general, the less complicated and fewer the toys, the better fantasy play is encouraged. The fundamentals are a portable table and chairs, paper, pencil, crayons, playhouse, dolls, and play telephones. The toys should not be unduly stimulating but rather should focus on the projective nature of the action and production. Among the techniques useful in getting the children focused and "grouped" are the following: creating large murals or collages and playing the game of "Guess What I Have Made." Art therapy and psychodramatic techniques ([Skaife and Huet, 1999](#)) are useful procedures, both when the children invent the characters and when the therapists provide a fishbowl from which a variety of themes outlined on paper slips may be drawn. This technique is especially helpful when a theme is being avoided, for example, dealing with the group bully, the sexually provocative child, a child who feels responsible for a divorce, or a child in the group who faces residential placement.

Through the play, the therapist and children begin to understand the meaning of the disclosures. Preparing the children to express themselves is antecedent to further working through of their problems.

### Specialized Group Approaches

Humor adds a special, and necessary, dimension to work with children (Dana, 1995). A *clown club* ([Smith et al., 1985](#)) has been introduced to provide a structured fantasy approach. The therapists dress up and play clowns, to the delight of the children. The psychodrama can be expanded to include the playing of good and bad witches, angry teachers or parents, and the like.

A variety of *video techniques* ([Gardano, 1994](#); [Mallery and Novas, 1982](#)) have been used with school-age children. Children can produce and watch their own videos. Replaying and redoing scenes allow the children actually to see their behavior and attempt to correct it by activities that strengthen organizational skills and memory. The video camera is an invaluable tool for diagnosis, research, training, and follow-up ([Smead, 1996](#); [Tellerman, 1998](#)).

*Kinetic group psychotherapy* ([Schachter, 1984](#)) is a technique involving an activity period of exercises or games, followed by a verbal discussion period. The technique has been used with a wide range of children's problems, including childhood autism and depression.

The use of *genograms* ([Davis et al., 1988](#)) is a technique borrowed from family therapy with latency-age children. The children, aided by the therapist, map out the family constellation, which helps them to focus on and question the events of the parents' marriage or separation, new alliances, and catastrophic events. The genograms are shared in the group and encourage each member to divulge hidden fears and learn to distinguish between reality and fantasy.

Other innovative techniques include storytelling ([Gersie, 1997](#)), group sandplay, ([De Domenico, 1999](#)), music therapy ([Plach, 1996](#)), and use of masks, puppet plays, and group emblems ([Prokoviev, 1999](#)). All such projective activities allow the children to reveal their problems in a nonthreatening manner. A sign of a maturing group process is when the children need less prepared structure from the therapists and suggest and create their own object world. Older, more verbal, intelligent, and stable children are capable of using a talking group as opposed to a holding group for the ego-weak children.

For impulse-ridden children ([Crawford-Brobyn and White, 1986](#)), changes in the traditional models may be necessary. Some children can progress from working with another child in a dyad for a time to joining the group. The acting-out child may be able to tolerate only one of four group sessions, until tolerance is slowly built up. [Evans \(1998\)](#) uses an individual therapy session before the group for children (and adolescents) who act out, are defiant, or fearful of their peers. This author terms this approach *active analytic*. [Strieder et al. \(1996\)](#) reviewed a wide variety of differential diagnoses and corresponding group structures operating in outpatient treatment of latency-age children.

## INTERACTIONAL PSYCHODYNAMIC GROUP PSYCHOTHERAPY

The model described in this chapter is applicable to all age groups. The specificities of the approach for children and adolescents include the provision of activity, play, and fantasy according to developmental level, temperament, diagnosis, and goals of the treatment. The interactive context in the here and now, among the peers and the therapists, amplifies the precise nature of the communication difficulties and conveys over time the dysfunctional intrapsychic conflicts within the group paradigm. The degree of permissiveness, structure, and limit setting depends on the activity level and explosiveness of the group and the degree to which regressive acting out is desired or can be tolerated. Greater vigilance is necessary with children who are overly aggressive than with shy children. The thrust of the model is to define the emerging object relations, symbolized by the choice of play objects, and the actual interactions with other group members. The psychodynamics of the group activities are partially translated into meaningful dialogues and understanding by the children. As the therapy progresses, modifications of the explanations and interpretations are appropriately made.

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### CASE ILLUSTRATION

David was a husky, attractive 7-year-old bully when he first came to the day hospital. He kicked the therapist and refused to have anything to do with the other children. His bravado covered his horrifying nightmares, his daily nausea in the car, and his inability to function in the classroom. When he started in the group, the members were working on a large world mural. One youngster was drawing the clinic, another the roadway, and another the school car. At this point David became very agitated and wanted to scribble over the drawing. The group members became somewhat intimidated, and the therapists attempted to calm him down, but to no avail. He was then told that, unfortunately, he could not manage the group that day and was asked to leave, with the comment "When you feel you are able to return and join the activity, please tell Sally [the Special Care Counselor who escorted the child from the room], knock at the door, and let us know when you are ready." It took several weeks before David was able to return to the group; when he did, he announced, "I'll try it out." The other children greeted him with understanding. The group members were drawing different emotions on faces. David first drew an angry face with teeth; when he noticed that others were drawing happy or sad faces, he remarked that he often had such feelings himself. In a subsequent session two or three of the members played with hand puppets and through the play told David that they did not like to be hit. Two years later David was present when a new child joined the group. By chance he and this child again chose the puppets. When the younger child kept smashing the head of the puppet on the table, David said in a soothing voice, "I know what it is like when you are so mad that your head feels like thunder." David worked through a considerable amount of rage. His somatic symptoms, including car sickness, largely disappeared, as did his repetitive drawing of cars. Such a child needed a gradual progression from dyadic to group therapy. His mother profited from parenting management. She was not a psychologically minded individual, but was motivated to help her child and cooperated well with the program.

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## APPLICATIONS FOR SPECIAL POPULATIONS (CHILDREN)

### Anxiety Disorders

Increasing attention has been paid to the group treatment and outcome assessment of anxiety in children. [Dadds et al. \(1997\)](#) evaluated the effectiveness of a cognitive-behavioral and family-based group intervention for preventing the onset and development of anxiety problems in children. In this study, 1,756 children, 7 to 14 years of age, were screened for anxiety problems using teacher nominations and children's self-reports. After recruitment and diagnostic interviews, 128 children were assigned to a 10-week, school-based, child-and-parent-focused psychosocial intervention or to a monitoring group. Both groups showed improvements immediately postintervention. However, at 6 months' follow-up, improvement was maintained in the intervention group only, with reduction in the rate of existing anxiety disorders and prevention of the onset of new anxiety disorders. These results indicated that anxiety problems and disorders identified by child and teacher reports can be successfully targeted through an early intervention school-based program.

[Barrett \(1998\)](#) evaluated a cognitive-behavioral, family-based group intervention with 60 children, ranging in age from 7 to 14 years, with a diagnosis of anxiety. The study divided the sample into three treatment groups: group cognitive-behavioral therapy (CBT), group CBT plus family management, and wait list. Posttreatment, 64.8% of children no longer fulfilled diagnostic criteria for an anxiety disorder, compared with 25.2% of children on the wait list, and the treatment groups maintained the gain at 12-month follow-up. Comparisons of self-report measures and clinician ratings of children receiving group CBT with those receiving group CBT plus family management indicated marginal benefits from the addition of family management to the group CBT.

A randomized clinical trial by [Silverman et al. \(1999\)](#) evaluated the efficacy of group CBT with concurrent parent sessions versus a wait-list control. The treatment group of children showed substantial improvement on the main outcome measures posttreatment and at 3-, 6-, and 12-month follow-ups, compared with no gains on the wait list. A group CBT treatment of childhood anxiety focusing on the role of parental involvement was carried out by [Mendlowitz et al. \(1999\)](#) in a study of 62



parents and children. One group was subdivided and randomly assigned to one of three 12-week treatment conditions: parent and child intervention, child-only intervention, and parent-only intervention. A battery of tests was used to assess child anxiety, depression, and coping strategies before and after treatment. The results showed that all treatment groups reported fewer symptoms of anxiety and depression posttreatment, but children in the parent and child intervention used more active coping strategies post treatment compared with the other two treatment conditions. Parents in the parent and child intervention group reported significantly greater improvement in their children's emotional well-being compared with the other treatment conditions. The short-term effectiveness of this group CBT intervention was demonstrated. Comparison of these three studies assessing the effectiveness of parental involvement is difficult, but suggests this issue needs further classification.

### **Social Incompetence and Phobia**

Groups for children emphasizing cognitive-behavioral and educational models demonstrate effectiveness in reducing social anxiety, shyness, and incompetence. [Blonk et al. \(1996\)](#) studied the short-term effect of group CBT for 72 socially incompetent children (8 to 12 years of age) who were experiencing poor peer relationships. The sample was divided into treatment groups (six children per group) and a wait-list control. Treatment outcome was assessed by teacher and parent reports on social behavior, sociometrics, and self-reported anxiety and self-evaluation. Posttreatment groups showed more appropriate social behavior and an increase in peer acceptance and number of friendships. These effects were sustained at 4- and 5-month follow-up assessments.

[Shechtman \(1993\)](#) reported increased self-esteem and close friendship in 52 elementary school children placed in 6 small counseling groups compared with matched control subjects. As might have been predicted, there was an intercorrelation between intimate friendship and self-esteem.

### **Depressive Disorders**

There has been an increase in the use of groups to alleviate mourning in children. [Schoeman and Kreitzman \(1997\)](#) used 12 parallel sessions for caretakers and children, and a joint session to work through the death of a parent. [MacLennan \(1998\)](#) reported on the use of children's groups for both expected and sudden death of family or friends. [Glazer and Clark \(1999\)](#) describe a family-centered intervention for grieving preschool children, and a multifamily and psychoeducation group was described as helpful by [Fristad et al. \(1998\)](#). A group play and activities therapy was described by [LeVieux \(1999\)](#), and an overview of loss and grief groups was provided by [Keitel et al. \(1998\)](#).

An outcome study by [Tonkins and Lambert \(1996\)](#) demonstrated the effectiveness of a short-term, 8-week bereavement psychotherapy group of 16 children, aged 7 to 11 years, divided into a treatment group and a wait-list control group. In the treatment group that shared feelings about the death of a parent or sibling, there was a significant decrease in symptomatology on multiple measures from multiple sources, and participants were able to develop new coping strategies.

[Clark et al. \(1993\)](#) introduced a group for mothers exhibiting postpartum depression and their newborns. Mother and infants participated in 12 weekly group sessions, 2 of which included spouses or partners. The authors' report is based on 5 years of time-limited groups, which showed improvement in the mothers' depression, problem solving, mutual support, and empathy.

[Trad \(1994\)](#) elaborated a sequential model of mother-infant psychotherapy, integrating the mother's individual therapy with the mother's observation of her infant's behavior with the therapist, participation in a mother's group, and family therapy. These last two studies suggest a preventive intervention for high-risk infants.

### **Groups for Abused and Traumatized Children**

Trauma groups have multiplied since the early reports of [Green \(1978\)](#), [Cunningham and Mathews \(1982\)](#), and [Mara and Winston \(1990\)](#).

In 1994, Reichert reported on the use of play and animal-assisted therapy for sexually abused Appalachian children. The focus was on the use of play and fantasy for the children to reverse their role from victims to survivors. [DeLuca et al. \(1995\)](#) evaluated the effectiveness of brief (9 to 12 weeks) structured therapy groups with 35 girls, 7 to 12 years of age, with a history of sexual abuse, showing an increase in self-esteem and a decrease in anxiety and behavior problems. Parents also felt that the treatment was helpful at 9- to 12-month follow-up. [Zamanian and Adams \(1997\)](#), using a time-limited (16-week) psychotherapy group with four sexually abused boys, describe the loss of power, helplessness, and the defenses of identification with the aggressor, splitting, dissociation, and so forth. The therapist's conflictual countertransference is discussed.

[Strieder et al. \(1996\)](#) outline a comprehensive ego-enhancing program of 10 session psychotherapy groups for cumulatively and repetitively traumatized children. As part of an elementary school-based violence prevention/intervention program [Murphy et al. \(1997\)](#) introduced trauma/grief-focused psychotherapy groups for children exposed to intrafamilial and extrafamilial violence. The multimodal, interdisciplinary team's goals were both psychological repair and social adjustment.

A comparison between a psychodrama group with young girls and a control group showed significant decreases in self-reported difficulties, withdrawn behavior, and anxiety/depression ([Carbonell and Partelano-Barehmi, 1999](#)).

[Peled and Edelson \(1992\)](#) reported on a 10-session group format for children of battered women. Children who are witnesses to violence and abuse of their mothers sustain significant trauma. The ability to speak about these events with their peers and therapists provides significant support in short-term, manually guided psychoeducational groups. Activity groups ([Nisivoccia and Lynn, 1999](#)) and play therapy ([Gallo-Lopez, 2000](#)), as well as a multimodal programs, have been used with children who have witnessed abuse. [Crockford et al. \(1993\)](#) introduced an integrated program, "Play Friendly and Safe," in which there were separate and combined groups for children and abused parents, as well as the inclusion of a nonoffending parent support group. A psychoeducational group for grandmothers raising inner city, abused, helpless, and depressed children focused on practical issues of school, home maintenance, and daily problem solving ([Vardi and Bucholz, 1994](#)).

Child victims of extrafamilial sexual abuse have been treated in separate, combined, group, and family approaches ([Grosz et al., 1999](#)). Group play therapy combined with psychoeducational techniques, drawing, and story telling have been used for family traumatized latency-age children ([Leavitt et al., 1997](#); [de Ridder, 1999](#)).

Children who were abused by a school employee were treated individually, in play groups, and with the family ([Pelcovitz, 1999](#)). Children and adolescents who have abused others have been treated in trauma-alleviating groups ([Erooga and Masson, 1999](#)).

Working with these abused children in all settings is difficult because they are fearful of divulging secrets about their abusive parents ([Schacht et al., 1990](#)). In residential settings, they are treated in homogeneous groups, whereas in outpatient clinics and day centers they more frequently are seen in heterogeneous groups. Negative countertransference feelings toward the parents must be faced. Often these abused children become the perpetrators, and tend to victimize weaker children. In play groups, several cotherapists often are necessary to control and moderate the acting out.

### **Learning Disorders and Underachievement: Clinic- and School-Based Groups**

[Gaines \(1986\)](#) outlines a variety of strategies helpful in the treatment of the retarded children and those with attention deficit disorders, including computer games, videotaping, and expressive arts. The use of structured, time-limited activities is critical with this group ([Azima, 1986](#)). Various group models for these underachievers have been used in various settings. [Mishna \(1996\)](#) used a psychodynamic interpersonal model stressing mutual recognition and trust in an outpatient setting. [Slavin \(1997\)](#), using a psychoanalysis-based approach in schools, addressed both academic and behavior problems.

[Gupta et al. \(1995\)](#) used a method of classification and diagnosis of school-age children seen in clinic groups. Working with ego-impaired groups in a residential program, [Winek and Faulkner \(1994\)](#) used a psychoanalytic, insight-oriented group, conceptualized as a collective superego, to encourage maturation. In school settings, groups using art therapy ([Prokoviev, 1999](#)) and psychotherapy ([Merydith, 1999](#)) have been used with underachieving students.

In an assessment of brief group therapy with low-achieving elementary school children, [Shechtman \(1996\)](#) examined 142 low achievers, in grades 2 to 6, who were randomly divided into an experimental and a control group. In addition to receiving assistance with school work 4 to 6 hours per week, the experimental children participated in a weekly psychotherapy group. The results indicated significant gains for the psychotherapy group in both academic progress and social well-being, which increased over time.

[Montello and Coons \(1998\)](#) compared the behavioral effects of an active, rhythm-based group music therapy with a passive, listening-based group music therapy with 11- to 14-year-olds in special classes for emotional, learning, and behavioral disorders. The Achenbach's Teacher Report Form showed that both music therapy interventions (not only the hypothesized active music groups) produced a lowering of scores on the aggression/hostility scale. It was suggested that music was a helpful modality for increasing creativity and self-mastery.

### Medical and Neurologic Conditions

Increasingly, medical and neurologic conditions are being treated in groups for children, adolescents, and parents. Some of the following subgroups overlap with former ones in the review (e.g., learning disorders, school, trauma). *Hyperactive* children were treated in a semistructured activity group to enhance self-esteem and social competence, diminish sense of shame, and work through unmet exhibitionistic needs ([Gnoulati, 1999](#)).

Group therapy with *siblings of autistic children* increased knowledge of the disorder and allowed the expression of thoughts and feelings related to despair, guilt, and alienation from society ([Carmi, 1997](#)). Children with *Aspergers' syndrome* have been treated in a social skills group for boys ([Marriage et al., 1995](#)), and in a 2-year interpersonal group stressing peer interaction ([Mishna and Muskat, 1998](#)).

A parents' psychoeducational and experiential group for *developmentally disabled adolescents* was integrated into a total treatment program ([Lynn, 1994](#)).

A structured group intervention for siblings of children with *cancer* was conducted for a younger (7 to 11 years of age) and an older (12 to 17 years of age) group. The 6-week program revealed statistical and clinical improvement on posttreatment measures in interpersonal and intrapsychic problems, improved mood and communication, and greater cancer-related knowledge ([Dolgin et al., 1997](#)).

A 3-year play group for three *hearing-impaired* latency-age boys showed improvement in school, home, and community posttreatment and 2 years after termination ([Troester, 1996](#)). A social skills group for boys with *Gilles de la Tourette's syndrome* showed small improvements in self-esteem and ability to voice social and academic problems with each other and families. Subsequently, a monthly support group was formed for parents and children ([Lambert and Christie, 1998](#)).

Habit reversal training for *trichotillomania* in a group format showed decreases in measures of global severity of obsessions and hair-pulling behavior at 1- and 5-month follow-ups ([Mouton and Stanley, 1996](#)).

*Epileptic* adolescents were treated in psychoanalytically oriented group therapy for 2 years. The goals were a better understanding of the illness and provision of information on questions such as the effects on sexuality, pregnancy, and work. The goals included psychological support, comprehension, acceptance, and coping with the disorder ([Rossi et al., 1997](#)). A cognitive-behavioral group for adolescents and adults with *spinal cord injuries* led to improved feelings of self-control compared with a control group at a 2-year follow up ([Craig et al., 1998](#)).

The results of these studies of various group models suggest important group applications to these long-standing, chronic medical and neurologic illnesses.

### Family Issues

Groups for *children of divorce* are used in various formats, including psychoeducational, cognitive-behavioral, drawing, and story-telling activities. [Epstein and Bordium's game \(1985\)](#), "Could This Happen," helps focus disclosures of anxiety about angry, "bad" parents.

[Roseby and Johnston \(1997\)](#) introduced a group treatment manual for school-age children dealing with violent separating families. The manual includes drawings, cartoons, and specified themes and activities.

A group intervention for *children and separated families* revealed differences posttreatment and 6 weeks later. [Durkin and Mesie \(1999\)](#) suggest that children should not be regulated as to with whom they communicate, or how, but rather that caution be used to allow children to make their own choices regarding visitation rights and the like.

*Parent, family, and multifamily groups* have used psychoeducational, psychotherapeutic, parallel combined, or integrated programs ([Epstein, 1976](#); [Hoffman et al., 1981](#); [Paramenter, 1976](#)).

A comparison of multifamily group therapy (42 families) with traditional family therapy (39 families) in the treatment of abusive and neglectful caregivers showed that the children in the multifamily therapy group become more assertive, had fewer behavior problems, and showed greater self-confidence ([Meezan and O'Keefe, 1998](#)).

## ADOLESCENT GROUP PSYCHOTHERAPY

### Young Adolescent Group

The techniques used with the pubertal group (12 to 14 years of age) approximate those used with latency-age children, namely, a combination of activities, play, drawing, psychodrama, and discussion periods. Most therapists tend to treat pubertal children in homogeneous groups with a same-sex therapist. These adolescents often are gauche and active and have difficulty in verbalization, especially the more pathologic, who are hospitalized or live in residential care. This age group works best on structured themes related to dependency, attachment, separation, and competition, among others. Sessions in general are a maximum of 45 to 50 minutes in length. Both short-term and long-term models are used. In the latter category, [Gordon \(1989\)](#) has reported a 2-year group with aggressive boys that used the model of working through symbiotic attachment and gradually working toward individuation. Interpretations were made to the group as a whole, dealing with ongoing interpersonal themes, rather than on the intrapsychic material of any one member. Videotaping, music, projective art techniques, and board games ([Kraft, 1986](#)) often are stimulating for children of this age, who often are too timid to talk openly about their sexual abuse, drug use, inferiority fears, and marked ambivalence to parents.

### Middle and Late Adolescent Groups

The age group of 15 to 19 years is most amenable to verbal psychotherapy. Outpatient models usually group the 15- to 18-year-olds. Increasingly, the adolescents referred for group psychotherapy are characterized by depressive reactions, suicidal attempts, and borderline symptomatology, in addition to the usual range of behavior disorders linked to delinquency, rebellion against parents, school dropout, and drug and alcohol use. The more severely disturbed adolescents are hospitalized and placed in residential and treatment centers. School groups focus on learning disorders, low motivation, disruptive behavior, and the like. Outpatient clinic groups treat the largest number of adolescents, whereas private practice groups tend to cluster in the more affluent areas.

The average psychotherapy group size ranges from six to eight and includes both sexes (with the exception of the pubertal group); where possible, heterogeneous composition is preferable. Acutely psychotic, autistic, or very handicapped borderline youngsters are suitable for outpatient groups but may be placed in modified group forms in hospital and residential settings ([Speers and Lansing, 1965](#); [Stengel, 1987](#)). The inclusion of borderline and very fragile adolescents depends on the strength of the total membership, which acts as a type of absorption filter and control mechanism. Group sessions vary from 1 to 1½ hours, at the rate of once or twice a week, and may be either short or long term.

## THEORETICAL CONSTRUCTS

Theories and techniques include psychoanalytic, psychodynamic, psychodramatic, gestalt, transactional analysis, behavioral, cognitive, and system approaches. The choice of methods appears to be made by a combination of the therapist's theoretical orientation and the specificities of the adolescents being treated.

In the psychoanalytically oriented framework, the approaches of Freud, Bion, and Kohut have been adapted to the group protocol. By and large, the developmental, interactive, and cohesive processes dictate whether individual or group address is used.



## Identity Group Psychotherapy

[Rachman \(1989\)](#) proposes a theoretical model based on the resolution of the adolescent's identity crisis in the group context and uses a blend of creative introspection, free thought, verbal and fantasy experimentation, and active techniques. The latter include role play, psychodrama, and dream interpretation, as well as specially devised scenarios to permit adequate self-disclosure and working through of problems.

## Interactional Psychodynamic Adolescent Group Psychotherapy

It is proposed that confrontation, empathy, and interpretation are the therapeutic triad underlying this approach, and that all three stem from a common source ([Azima, 1989](#)). Confrontation accentuates the verbal enunciation of thoughts and feelings, whereas empathy involves the experiential process incorporating the other person's feelings and thoughts. It is postulated that the peers in the group are representative of varying confrontational and empathic styles. Some personalities are in need of a more confrontational approach, whereas others need longer nurturance and silent understanding. Interpretation occurs at the point in the therapeutic process when there has been sufficient empathic confrontation and clarification to uncover and give meaning to the underlying unconscious conflicts, and it should occur in synchrony with the individual and the group process.

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### CASE ILLUSTRATION

John was a 15½-year-old with marked narcissistic and grandiose features. For many sessions, he boasted that he could live on his own, that he did not want to be in school, and that he had many friends. The other five group members could hardly get a word in, as he pontificated and analyzed everyone. Gradually, certain members began to confront and question him and to express their annoyance. He soon revealed that his mother had divorced his alcoholic, abusive father and later married an older, quiet man. This man could not tolerate the patient, and he was moved into a small apartment with one of his brothers, who soon left, and he continued there alone. As these facts and feelings came out, he was amazed to regain the empathy of many group members. The therapist in the early stages had assumed an empathic, understanding approach with John and only gradually began to confront the narcissistic defenses. Although the patient was willing to interpret everybody else's problems, he could not accept other people intruding into his inner life. Many sessions later, a pretty adolescent to whom John was clearly attracted told him in a direct, confronting manner that he would have a hard time making friends, especially with girls, because he was not truly interested in them, and that he was sure to make others very angry by his know-it-all manner. John was stunned, averted his gaze, bent over, and remained silent. He slowly lifted his head, and holding back his tears, he said, "I think you are right, and that's what I am afraid of."

### Comment

It may be necessary to confront the silence of adolescent members very early, in an understanding way. The psychodynamic significance of intrapsychic and interpersonal communications and interactions remains the cornerstone of the interactional psychodynamic group psychotherapy approach. As in all psychotherapies, the goal is for the adolescent to develop self-understanding, independence, self-esteem, and interpersonal competence.

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The preceding clinical example was taken from an outpatient, open-ended, heterogeneous group with an average patient attendance of 2 years. In this model, the goal is the development of autonomy and independence. Parents are seen only at intake and at the end of each year's group, with the adolescents' consent. Confidentiality is highly protected in this model, and this in turn promotes faster divulgence of material. This approach is facilitated in a country (such as Canada) that provides funds so that parents do not have to pay the bills. In addition, in the Canadian system of health care, any adolescent older than 14 years of age can request treatment without the parents' knowledge. Outpatient clinics and private practice group psychotherapy often involve a combination of psychopharmacology, combined individual and group therapy, and parallel or conjoint parent or family therapy. In long-term treatment necessitated by regressions or traumatic events, a combined network approach has been used effectively ([Richmond, 1989](#)).

## Inpatient and Residential Treatment Groups

Inpatient and residential treatment groups form an integral part of most adolescent units and residential treatment centers ([Chase, 1991](#); Kleiger and Helmig, 2000; [Stein and Kymissis, 1989](#)). The group format varies indefinitely according to the degree of pathology, intellectual level, and longevity of the group, and number of absences of group members. Inpatient psychotherapy groups ([Kymissis, 1996](#)) are advantageous in that they can focus on ongoing resistances and acting out in the group and the hospital network. Conformity and compliance as to weekend passes, attendance at meetings, and taking of medication are strengthened in the group context. The handling of confidentiality in inpatient groups is a delicate issue, and it seems wise to explain at the outset the team's sharing of information. In ward situations where adolescents are assigned to certain staff members, there often is conflict between the patients and different staff teams.

## APPLICATIONS FOR SPECIAL POPULATIONS (ADOLESCENTS)

### Depression

Parentally bereaved adolescents were treated in a large discussion group format using other parentally bereaved adolescents as therapeutic assistants to help reduce the resistance to talking and giving feedback ([Levy and Zelman, 1996](#)). A 7-week antidepressant, antisuicide group was conducted with seven teenage women using a collaborative feminist and narrative approach to externalize, empower, support and define the depression ([Johnson, 1994](#)). [Clarke et al. \(1995\)](#) assessed a prevention program targeted for 150 adolescents at risk for future depressive disorder. A randomized 15-session trial of group cognitive intervention was compared with a usual case-control group. Survival analysis indicated a 12-month advantage for the prevention program and a decrease in depressive indices. In a further study, [Clarke et al. \(1999\)](#) compared maintenance cognitive behavioral with acute CBT groups with booster sessions. One hundred twenty-three adolescents with major depression or dysthymia were assigned to one of three 8-week acute conditions: adolescent group CBT (16 2-hour sessions), separate parent group, or a wait-list control. Subsequently, the members completing the CBT groups were randomly reassigned to one of three conditions for the 24-month follow-up period, namely, assessments every 4 months with booster sessions, assessments only every 4 months, or assessments every 12 months. Results indicated that acute CBT groups yielded higher depression recovery rates (66.7%) than the wait list (48.1%) and greater reductions in self-reported depression. Outcomes for the adolescent-only and adolescent plus parent conditions were not significantly different. Rates of recurrence in the 2-year follow-up were lower than in treated adult depression. The booster sessions did not reduce the rate of recurrence in the follow-up period but appeared to accelerate recovery among the subjects who were still depressed at the end of the acute phase. The authors concluded that CBT was an effective intervention for adolescent depression. It is of some interest that the parallel parent group did not add significantly to the reduction in depression. This confirms this author's belief that for adolescents, separate treatment that protects confidentiality and allows separation from parents may be the treatment of choice, whereas young children profit more from parallel, integrated modalities.

[Fine et al. \(1991\)](#) reported on the comparison of two forms of short-term group therapy for 66 outpatient adolescents clinically diagnosed as depressed. Subjects were randomly assigned to either a therapeutic support group or a social skills group. Posttreatment, adolescents in the therapeutic support group showed a greater decrease in depressive symptoms and significant increases in self-concept. At the 9-month follow-up, adolescents in the therapeutic support groups maintained their improvements, but adolescents in the social skills group had now caught up in their improvement between the posttreatment group and follow-up assessments. The authors postulated that the original gains made in the therapeutic support group were necessary to alleviate the depression before members were able to profit from the problem-solving strategies taught in the social skills groups. The manic defenses in the mourning process were described for an adolescent group, using a Kleinian analytic framework. The group as a whole focused on the process and the dynamics of the relationship between the adolescents, staff, and family ([Toder-Golden, 1999](#)).

### Self-Mutilation and Self-Destructive Behavior

Self-mutilation and self-destructive behavior have received scant attention in the group literature for children and adolescence. [Sansone et al. \(1996\)](#) have described an integrated psychotherapeutic approach in the management of self-destructive behavior in eating-disordered patients with borderline personality disorder. A group psychotherapy approach was found useful for a reasonable resolution over time of self-destructive behavior.

[Hartman \(1996\)](#) discusses deliberate self-cutting by adolescents in psychiatric inpatient units. The author suggests that the interpersonal aspects of the cutting are neglected, with undue attention given to the individual patient, who may be acting out the group's conflicts and discontents as well as protesting against inadequate staff supervision.

### Social Phobia

Cognitive-behavioral group therapy for social phobia in female adolescents was described by [Albano et al. in 1995](#). The results of a pilot study ([Hayward et al., 2000](#)) compared the outcome results for 35 girls with social phobia at high risk for major depression, 12 of whom were assigned to a treatment group and 23 to a

nontreatment group. Of the 11 subjects who completed treatment, there was a significant improvement and reduction in the symptoms of social anxiety and depression. However, at the 1-year follow-up there were no significant differences by treatment condition, but there was suggestive evidence that the treatment of social phobia lowers the risk for relapse of major depression among the subjects with a history of major depression. Combining the decline scores for social phobia and depression produced more robust treatment effects for the 1-year follow-up. The results indicate that there was a moderate short-term effect of group CBT for female adolescents, and that a decrease in social phobia also may reduce the criteria for major depression. This latter finding suggests the usefulness of group-based prevention programs for shy, socially incompetent children. The question of adjuvant pharmacotherapy for social phobia was raised because there have been positive findings with its use in adult social phobia ([Heimberg et al., 1998](#)).

### Anger, Violence, and Conduct Disorder

Groups for the control of anger, violence, and conduct and criminal disorder have increased in response to the rise in rebellious, destructive behavior in society. The following studies are included because of their innovative clinical and research approaches.

A structured group for undersocialized, acting-out adolescents that used a pretherapy training program and an initial therapy contract showed positive clinical outcomes ([Corder, 1996](#)).

Cognitive-behavioral anger management groups showed improved arousal control, cognitive restructuring, and prosocial skills ([Feindler and Scalley, 1998](#)). A CBT group of 11 adolescents showed statistically significant changes in aggressive behavior, attentional problems, self-esteem, anxiety, depression, and somatic complaints, as shown on posttreatment scores on the Youth Self-Report, the Achenbach Child Behavior Checklist, and the Piers-Harris Children's Self-Concept Scales ([Kastner, 1998](#)). A brief anger management therapy group of 10 to 12 sessions showed improved functioning in 25 adolescents in hospitalized groups, compared with 25 in control groups ([Snyder et al., 1999](#)).

A teen abuse group using a CBT model targeted socioeconomically depressed black and white adolescents. The 10-week program was part of a Master's-level social worker's training module. The approach appeared clinically promising. [Carlin \(1996\)](#) describes a large group treatment of 25 severely disturbed, conduct-disordered adolescents. Needs for effective leadership, acceptance of the members' cultural differences, and insurance of maximum safety influenced more positive interpersonal relations. [Byrnes et al. \(1999\)](#) compared reductions in criminality in three different formats—group, individual, and family therapy—in a sample of 532 adolescents in a residential and day-treatment program over a 4-year period. The major finding were (a) the number of hours in group therapy explained the greatest variance in the reduction of criminal charges, followed closely by hours in individual therapy, whereas hours in family therapy was not a significant predictor; and (b) residential treatment was associated with greater reductions in adult correctional commitments than was day treatment.

The findings that group therapies are effective treatment for delinquent adolescents confirm the early findings of [Bratter \(1989\)](#), [Friedman and Glickman \(1986\)](#), and [Raubolt \(1983\)](#).

### Trauma and Abuse

Trauma groups for a variety of disorders have been described for children and adolescents who have become the victims of natural disasters, sexual abuse, violence, and atrocities of war. Classifications of posttraumatic disorders in the third (revised) and fourth editions of the *Diagnostic and Statistical Manual of Mental Disorders* emphasize significant cognitive, behavioral, and physiologic somatic effects after the trauma. A variety of group interventions, including supportive, CBT strategies have been used to reduce posttraumatic stress disorder symptoms to expose the trauma and to work toward improved coping skills ([Foy et al., 2000](#); [Kopala and Keitel, 1998](#)). [Glodich and Allen \(1998\)](#) reviewed the group therapy literature on preventing trauma reenactment in adolescents. Group CBT and psychoeducational strategies are considered important ways of interrupting the pernicious cycle of reenactment and further risk-taking behavior. [Everly et al. \(1999\)](#) compiled a meta-analysis of the effectiveness of psychological debriefing with vicarious trauma. An outline study of 41 sexually abused adolescent girls (13 to 18 years of age) were divided into small, structured groups. Each of the seven treatment sessions consisted of a didactic presentation, an art activity, and the development of a positive associational cue. Posttest assessment revealed improved adaptive functioning and skill mastery.

Female adolescent survivors of sexual abuse responded to a goal-oriented therapy group ([Backos and Pagon, 1999](#); [Furniss et al., 1988](#)). Adolescent girls facing the loss of their parents through acquired immunodeficiency syndrome were treated in a 2-year psychoanalytic group. [Cohen \(1996\)](#) analyzed the transference-countertransference in this angry, depressed group of girls. It is likely that through the countertransference, the therapist was able to empathize with the adolescents' plight and work through a treatment plan. [Henry \(1996\)](#) explored human immunodeficiency virus-related risk taking in a psychodynamic group for adolescents.

### Alcohol and Substance Abuse

The number of groups for adolescents addicted to alcohol and drugs has increased to meet treatment and prevention needs. A variety of models exist, including multimodal programs ([Bratter, 1989](#); [Friedman and Glickman, 1986](#)) in residential and outpatient clinics ([Bogdaniak and Piercy 1987](#); [Gonet, 1998](#); [Nastasi, 1998](#)). The use of multifamily play groups for families in addiction recovery was effective in promoting parent-child communication, the development of a nonblaming attitude, and an understanding of children's reactions to their addiction ([Cwiakala and Mordock, 1997](#)). A therapeutic community drug treatment program that studied 938 adolescents (15 to 17 years of age) who were admitted to residential treatment for substance abuse revealed that one-third of the sample showed histories of sexual abuse. A Cox regression analysis showed that a history of sexual abuse is related to earlier onset of alcohol and illicit drug use. It was suggested that drug use may function to ameliorate feelings of depression and poor self-esteem that accompany childhood abuse ([Hawke et al., 2000](#)).

[Kaminer and colleagues \(1998\)](#), using manual-guided interventions, measured the treatment process in CBT and interactional group therapies for adolescent substance abusers. In a 15-month follow-up of a pilot study, [Kaminer and Burlison \(1999\)](#) reported on the comparison of 32 dually diagnosed adolescents, randomized into 2 short-term outpatient group psychotherapy groups, one using CBT and the other an interactional treatment (IT). At the 3-month follow-up, no patient treatment matching effects were shown. However, adolescents in the CBT group showed a significant reduction in severity of substance abuse compared with those assigned to the IT group. At the 15 month follow-up, there were no differential improvements as a function of therapy type. However, subjects maintained significant treatment gains in the substance abuse, family function, and psychiatric status domains of the Teen-Addiction Severity Index, and both CBT and IT were associated with similar long-term gains. This study is the only one to date that demonstrates no superiority for short-term group CBT compared with short-term group IT.

[Pressman and Brook \(1999\)](#) have described a multiple group psychotherapy approach with adolescents with psychiatric and substance abuse comorbidity, treated in an inpatient psychiatric setting. The involvement of a multidisciplinary team using an integrative approach showed promising results.

A five-phase group model for outpatient adolescents who abuse substances was described by Spitz and Spitz (1955). It includes the following phases (a) evaluation and orientation, (b) entry into the group, (c) establishing a working climate, (d) a middle or working stage, and (e) transition out of the group. The authors conclude that group therapy is the treatment of choice.

### Eating Disorders

[Azima \(1992\)](#) described an intensive group psychotherapeutic interactional model for outpatients seen in a heterogeneous group. Parental involvement was indicated for the younger but not the older teen group. A variety of techniques and activities, including drama therapy and guided imagery, are used in short- and long-term eating disorder groups ([Moreno, 1998](#); [Wurr and Pope-Carter, 1998](#)).

[Mitchell and collaborators \(1990\)](#) compared the efficacy of antidepressant drug therapy with structural, manual-guided group therapy for a total of 12 weeks. The overall finding was that the addition of the antidepressant to the group psychotherapy did not significantly improve the eating disorder, but did ameliorate the features of depression and anxiety. At 6-month follow-up ([Pyle et al., 1990](#)), 30% of the sample had relapsed; however, group psychotherapy alone or combined with drug therapy showed lower relapse rates than treatment with medication alone. It also was found that neither attendance at the maintenance group sessions nor imipramine maintenance was associated with a better outcome.

[Leung et al. \(1999\)](#) evaluated 10-week group CBT with 20 women diagnosed with anorexia nervosa, aged 17 to 58 years. At posttreatment, group CBT was found to be ineffective in symptom reduction, and basic core beliefs were irrelevant to outcome. The authors concluded that group CBT in the current short form is insufficient



to induce changes, and suggested further group treatments to address the issues of poor motivation, lack of insight, and ambivalence toward treatment.

A clinical study of an expressive group therapy eating disorders program reported positive behavioral change in this adolescent group, but there was no research to support these impressions ([Shander and Orbanic, 1995](#)).

It can only be concluded that eating disorders, especially anorexia nervosa, are resistant to change with short-term formats. Further research is needed to assess multimodal programs, including motivational preparation before group treatment. The links of eating disorders to sexual abuse and borderline and self-destructive behavior need to be noted ([Sansone et al., 1996](#)).

### Other Disorders and Issues

The subject of *learning-disabled adolescents* overlaps with the previous children's section in this review. Residential, outpatient, and school programs offer specialized groups often integrated with other academic modules. Investigators agree that both the learning disability and social/emotional problems must be attended to in the group format. ([Coché and Fisher, 1989](#); [Mishna and Muskat, 1998](#)). Computers and videos are helpful adjuncts to the group treatment ([Cox and Lothstein, 1989](#); [Gardano, 1994](#)).

[Bernstein et al. \(2000\)](#) investigated the use of imipramine plus CBT in the treatment of 63 *school-refusing* adolescents with comorbid anxiety and major depressive disorders. The findings were that imipramine plus CBT is significantly more efficacious than placebo plus CBT in improving school attendance and decreasing symptoms of depression in school-refusing adolescents.

*Parent and family group therapy* are combined more frequently in the treatment of younger children; parallel and combined groups appear indicated with acutely ill, traumatized adolescents, and where a return to living in the family is indicated.

The use of *multifamily group therapy* has been recommended in the treatment of dually diagnosed adolescents ([Kymissis et al., 1995](#)), and with abusive and neglectful caregivers ([Meezan and O'Keefe, 1998](#)).

A *father-adolescent son group* was described by [Ginsberg \(1996\)](#). The goal of these groups was the developments of insight, closer communication, and understanding. A combined experimental/psychoeducational format was used as part of a group therapy program called the *Parent-Adolescent Relationship Development Program*.

A multimodal group program for *pregnant and parenting* adolescents involved collaboration with parents, school, and community ([Stoiber et al., 1998](#)).

### Culture

The influence of *culture and society* on therapy groups has been the subject of increasing investigation ([Serrano and Hou, 1995](#)). Culture-focused group therapy has been reported as enhancing bonding with both the shy and introverted adolescent, as well as dealing with identity issues in gang-motivated youth ([Vargas and DiPilato, 1999](#)).

Psychotherapy groups were carried out with youth experiencing war (Schneider and Cohen, 1996) and refugee trauma ([Brumen-Budanko, 1998](#); [Foigel, 1997](#)). [Azima \(2002\)](#) has reported on the use of groups with immigrant and refugee children and their families, and has outlined a training model for mental health professionals.

## THE GROUP PSYCHOTHERAPIST: FUNCTIONS, REALITY ISSUES, AND COUNTERTRANSFERENCE

Aside from the obvious managerial duties of selecting, composing, and deciding on time and place, the major focus of the group therapist is to forge a *therapeutic alliance* appropriate to the age level of the patient. It must be made clear to the group that the common goal is the understanding and solving of painful thoughts and behavior. Increasingly, patients are being prepared, before and in the initial group sessions, to understand the functioning of the mind and the ability to analyze the self, and to be responsible for uncovering and talking about conflicts hidden in their inner worlds. A parallel task for the therapist is to orient the group members to be aware of their behavior with each other, to share thoughts and feelings, and to offer suggestions about how to deal with these problems.

Functioning in the group context puts greater stress on the therapist, especially with children who act out physically and regress rapidly ([Azima, 1986](#)). With adolescents, there is the accompanying disrespect and rebellion against authority figures ([Azima, 1973](#)). The reports of homogeneous groups for young patients with bulimia, anorexia, diabetes, migraine, thalassemia, and cancer indicate improvement in motivation and compliance with prescribed diets, exercise, and the use of medication.

A leader must solve the quandary of how to be a competent therapist and a respected authority figure, and not an admonishing disciplinarian. The reality is that therapists working with adolescents may experience sudden eruptions of anger at and fear of some members, as well as show undue sympathy and overprotectiveness to others. Consider the following examples: A sudden brawl breaks out; a member swears at and insults the therapist; an adolescent produces a gun or a knife; one of the group members breaks into the therapist's car. Should the therapist's disturbed reactions to any of these episodes be classified as countertransference, or realistic? It is clear that more punitive rage can be activated in the group therapist working with children and adolescents than with adults. Furthermore, the scoffing, belittling attitudes of adolescents often cause narcissistic injury to the doctor's status.

There is general agreement that today's child and adolescent group therapists have become more actively involved, less permissive, more spontaneous in their play, more confronting, and in general less distant in their relationship with group members. With adolescents, the therapist tries to assume an emotional/cognitive role model midway between the adolescent and the parent. Therapists with overclose identification with adolescents are put at risk of collusion, passivity, or acting out, whereas therapists with too distant an identification are rendered vulnerable to possible rebellion against parental figures. A good practice with adolescents is to assume an attitude of controlled curiosity and sophisticated ignorance, especially in the early stages of the group. An overintellectual approach on the part of the therapist is likely to produce silence, fear, withdrawal, and withholding in the group members.

Adding to the therapist's countertransference is the pressure from parents for the therapist to see the child from the parents' point of view. The therapist must be realistically empathic and not overjudgmental. In the clinic or day-hospital setting, the therapist often is caught in a tug of war between team members who side with or against the parent, teacher, or judge.

In many cases, the therapist's reaction should not be classified as countertransference unless there is evidence of ongoing behavior patterns that block or are destructive to the group process. Therapists who do not do well with this age group usually are unable to deal with the acting out, overplayfulness, and disregard for the status of authority figures. But it is precisely these defensive resistances that provide the first insight into the group members' problems. Therapists who have been raised in a strict, obedient fashion, with strong moral and ethical standards, may be more vulnerable to the adolescent age group. Fearing loss of control of their own anger, they become overly silent and permissive in their leadership.

Group psychotherapists who do not overcentralize their position are more likely to be perceptive of the interactive psychodynamics and to reflect on the variety of positive and negative feelings and thoughts that corroborate or differ from their own. The interactive interpersonal variations become a therapeutic tool for the therapist and in many instances dilute or eliminate countertransference responses by clarifying and comparing the responses of other group members.

Some helpful qualities of the group therapist working with children and adolescents include comfort in a group, spontaneity, flexibility, playful creativity, and the ability to set adequate limits as a rational, empathic role model. Continuing clinical research in the area of child and adolescent group psychotherapy will, it is hoped, clarify and identify which therapeutic models are most effective with specified age groups, intelligence levels, and diagnoses.

## CONTRAINDICATIONS

The question of contraindications to group psychotherapy for children depends not so much on the diagnoses, symptoms, or deviant behavior, but on the total

composition of the group, the competence of the staff, the availability of support staff, and whether the treatment is carried out in residential or outpatient settings.

Special problems and hazards are presented by children who are explosive, cruel and vindictive, sexually acting out, overly autistic, seclusive, and depressed. Children with physical stigmata often are scapegoated. When children are enuretic or encopretic, entry into the group usually is postponed until adequate controls are developed. Children of very low intelligence do poorly in verbal psychotherapeutic groups but profit from socialization, compliance, and support groups. Homogeneous groups in residential treatments report better outcomes with retarded and delinquent populations.

Some children entering day-hospital programs initially may not be ready for or capable of entering a psychotherapy group until they have adequate controls and are capable of listening to others and expressing themselves. Younger children usually must be involved in multimodal therapeutic school programs with a gradual transition to triadic therapies with another child or parent before group psychotherapy is possible. Short preparatory groups are necessary for children with poor frustration tolerance.

For adolescents, the basic contraindications outlined previously apply as well. For outpatient treatment of adolescents 15 to 18 years of age, contraindications include acute psychosis, heavy medication, drug addiction, and delinquent acting out. Again, it appears that motivation, psychological mindedness, and attraction to the norms of the adolescent peer group are the more critical variables. In addition, an alliance must be articulated that sets the therapeutic goals for the group.

The selection of the specific therapeutic modality for children and adolescents should be made in a more sequential fashion, considering the developmental stage, the type of problem, and the degree of intrapsychic or interpersonal deficit. It may be that one child may need individual play therapy first, whereas another requires integration into a group. In addition, the combination of individual, family, or group becomes realistic only if time and expense are not factors. It is likely that some patients are held in one fixed therapeutic modality too long before another is tried.

This review shows that clinicians have been increasingly challenged to do groups with children who previously have been considered contraindicated (e.g., children with autism or Asperger's syndrome, the developmentally and intellectually handicapped, and the traumatized and abused child). Expressive therapies and structural play activities have been introduced for the nonverbal or less verbal child.

## CLINICAL AND RESEARCH TRENDS

Group as a therapeutic modality has been increasingly confirmed by both clinical and research investigation, in a wide variety of areas. Clinics as well as inpatient, residential, private practice, and community programs have expanded rapidly. Applications to psychiatric, medical, neurologic, community, and social issues have grown at an unprecedented pace.

The trend is toward short-term, cognitive-behavioral, interactive models often using manualized guides. These approaches are much more conducive to applying statistical research designs. The goal is to find ways of carrying out research with long-term psychodynamic groups and specifying criteria for change. There is acknowledgment that some short-term models are not effective with very resistant disorders (e.g. eating disorders, severe depression, and substance abuse). In such cases, multimodal models integrating group therapies with pharmacotherapy may be indicated. Partnership in contributing to government policy is needed for broader-based intervention and prevention for the major psychiatric disorders, the socioeconomically deprived, and for alcohol, drug, and cigarette consumption. The involvement of the media has contributed positively in addressing mental health issues. The expansion of information on the Internet and the growth of chat groups has moved us into the area of prevention and group therapy on-line.

A predictive trend is for the use of parent-infant/child groups for healthy development. Schools are becoming increasingly involved in doing groups with the underachievers, the aggressive bullies, and the creative thinkers, as well as research studies.

Increasingly, research has expanded and answered the past paucity ([Azima and Dies, 1989](#); [Azima, 1996](#); [Dies and Riester, 1986](#)). Meta-analysis confirms the efficacy and efficiency of group psychotherapy for children and adolescents ([Hoag and Burlingame, 1997](#)).

Clarification is needed as to the inclusion or exclusion of parents and family in treatment. For adolescents at the stage of separation, family therapy may be contraindicated. Studies that have found little therapeutic input with family therapy require replication. For day therapy centers, parent involvement appears a necessity. At follow-up, parents rarely are included in booster sessions, and the decline in their participation likely has a negative impact on the results.

Further investigation of the interactive process of the peers in the group matrix continues to be neglected, partly because of the lack of satisfactory group measures, and the cost of these investigations.

Current and future outcome research will likely be easier to carry out with the introduction of more efficient and sophisticated instrumentation. The field lacks long-term follow-up studies. Outcome results could help specify what length of therapy, short or long term, and what style of therapy, cognitive-behavioral, interactional, or psychodynamic, is indicated for specific disorders. Where results show little clinical effectiveness for the group intervention, other therapeutic modalities should be considered (e.g. individual or pharmacotherapy) in combination or sequentially. The current trend is toward inpatient multimodal models, involving school and community. The efficacy of outcome is now considered not only in terms of self-esteem, pleasure, and improvement in functioning in the home, school, and work, but in terms of cost of the delivery of mental health care. Here, group therapy has the advantage in that more children can be treated at less cost. It is clear that research supports group therapy effectiveness, and children and adolescents are decidedly excellent candidates for this interpersonal treatment modality.

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The term *family therapy* refers to interventions that attempt to alter interactions among family members and thereby improve the functioning of the family as a unit and the functioning of individual family members. Family therapy interrupts interactional patterns that cause distress between individuals and, correspondingly, within individuals. Family therapy eventually addresses the concerns of all family members, but most commonly begins by focusing on the clinical needs of a symptomatic child.

Family therapy denotes a conceptual framework guiding interventions and a specific psychosocial intervention. For a generation, family therapy has been guided by the concept of the family as a system. In this view, psychopathology resides in the system, not in individuals, and interventions are designed to change family relationships because symptoms are seen as regulating family relationships. Symptoms in a child are necessary for the system, and behavioral dysfunctions are seen as dysfunctions of the entire system. The influence of the systemic approach, although generative of numerous hypotheses and new ways of looking at the problems of children, has lessened. This chapter emphasizes that although symptoms can *maintain* interactional problems, these interactional problems also *generate* problems for children. Family therapy that changes relationships ultimately changes the internal lives of individuals, particularly those of developing children.

As an intervention modality, family therapy is more accurately termed *family treatment* because this denotes different ways in which clinicians can work with families—with the entire system, with dyadic subgroups, and with individual family members. Any intervention that specifically improves the functioning of the family can legitimately be seen as an aspect of family treatment. Traditionally, family therapy implied that all family members were present for treatment. For many practitioners, eclectic family therapy approaches that encompass behavioral, educational, and psychological interventions are seen as family treatments. Further, specific interventions focusing on parental dysfunctions and facilitating parental support can legitimately be seen as a subset of family interventions. Because parents perform executive functions for the family, increased parental stability will have a salutary effect on all aspects of family functioning. *Family therapy* and *family treatment* are used interchangeably in this chapter.

Family therapy uses key elements of the other psychosocial therapies. Family therapy uses a behavioral emphasis in that clinicians observe behaviors and design plans to alter them by intervening in the interactional patterns associated with problematic behaviors. This often is done in session through therapist directives and out of session through homework assignments. Family therapy uses concepts of individual therapy such as developing an alliance with the family, assessing familial resistance, and considering therapist countertransference to the family's behavior. Perhaps most clearly, family therapy shares characteristics of group therapy. Individuals in the family perform functions for the family as a whole, but these functions are not just derived from family sessions. Roles assigned by biological relationship of parent to child and sibling to sibling, as well as the shared history of each of the family members, have an impact outside the consulting room (Josephson, 2000a).

Any contemporary effort to define family therapy must recognize a relatively recent phenomenon: there is increasingly less consensus on what actually constitutes normal family experience. Early systemic family therapy was based on the concept of the family as a stable nuclear unit with extended family support. Many of the transitional experiences of the contemporary family are receiving increased attention and these include, but are not limited to, stepfamilies, single-parent families, and serial cohabitation (Barnes, 1994). Achieving consensus on a definition of family therapy is now more complicated because of this apparent shift in the definition of the family.

### FAMILY INFLUENCES ON PSYCHOPATHOLOGY

It is self-evident that experiences in the family comprise an important, perhaps major, influence on children's emotional development and in the causation of psychological and psychiatric disorders. This intuitive position is substantiated by significant empirical evidence (Garnezy and Masten, 1994; Rutter, 1991). There should be little debate about the need for a systematic evaluation of family functioning in any child or adolescent being assessed for psychiatric disorder. Research in

psychopathology reveals that family discord, disruption, and disorganization are strongly associated with psychiatric disorder ( Jenkins, 1994). Psychiatric risks appear to arise more from specific interpersonal relationships rather than general family milieu characteristics. This is supported by behavior genetic analyses that separate the effects of shared and nonshared environmental effects ( Plomin and Daniels, 1987), and by detailed clinical studies ( Buchanan et al., 1991). In spite of this evidence indicating the family's importance in the etiology of children's problems, the field of family therapy has had to grapple with views in child and adolescent psychiatry that suggest that family interaction never causes individual psychopathology but merely responds to it ( Koplewicz, 1996; Sargent, 1997). These views appear influenced by historical factors, not the least of which were the inaccurate attributions of the etiologic role of familial and parental dysfunction in major mental disorders ( Fromm-Reichmann, 1948). The unfortunate blaming of parents appears to have given way to the field's reluctance to implicate familial problems as etiologically relevant to child and adolescent psychiatric disorders.

This chapter emphasizes the bidirectional nature of the influence of families. If families nurture and assist children, which they surely do, they also can hinder the developmental and clinical status of children. Family therapy practiced at its highest levels empathically relates to the family and vigorously attempts to discern the role the family plays in the disorders under investigation and treatment. Family therapy recognizes that parents and families shape children, and children shape parents and families ( Leventhal and Conroy, 1991; Rutter and Cox, 1985). Family therapy must empathically relate to family difficulties and, at the same time, urge families to change patterns of interaction where this is indicated.

## HISTORY

The history of family therapy throughout the past century is fascinating and convoluted, with numerous reviews of this history available ( Broderick and Schrader, 1991; Ravenscroft, 1996; Sholevar, 1997). There are several historical themes that illuminate the current status of family therapy in the treatment of child and adolescent psychiatric disorders. Ironically, much of the early family therapy literature did not emphasize children or adolescents. Without children, there is no family. Family therapy often emphasized systemic principles that frequently did not take into account the differential effects of the age of a family's children and related aspects of developmental psychopathology. Clinicians treated the family system as a game of checkers, when the analogy of chess was much closer to the clinical reality. To further the chess analogy, the presence of individual psychopathology meant certain family members were restricted in some of their moves. This relative avoidance of children in family therapy has shown encouraging signs of changing ( Combrinck-Graham, 1989; Zilbach, 1986).

It is ironic that the systemic approach minimized the role of children because most observers agree that the beginnings of family therapy occurred in the early 1900s as child guidance clinics treated the problems of children in a family context. The orientation of these clinics at the time was influenced by the psychoanalytic ideal of separate, confidential, individual treatment. Even though Freud had pointed out the interplay of family forces in intrapsychic development, the primary clinical intervention focused on the mind of the child. In these clinics, the psychiatrist treated the child and the parents were seen by the social worker. This approach reflected the primacy of the individual approach, a situation that continued through the first half of the century. Even though clinicians were beginning to report relationships between parental dysfunctions and conduct disorder ( Johnson, 1949) and schizophrenia ( Fromm-Reichmann, 1948), most psychiatrists still continued to see family interviews and conflicts as ancillary to the treatment of the child's internal conflicts. John Bowlby (1949) stated that he found family interviews useful "before treatment is inaugurated." This statement by an influential psychiatrist simultaneously encouraged clinicians to think of family influence yet underscored that the definitive treatment was with the individual.

## Pioneers

Many of the early pioneers of family therapy became involved in family therapy because of their disenchantment with psychoanalysis or their opposition to the hegemony of the medical model, or both. Nathan Ackerman, arguably the father of family therapy, was a child psychiatrist and psychoanalyst. He was typical of the analytically trained early family therapy leaders who believed their individual treatments were less effective because they were undermined by families who had difficulty adjusting to therapeutically induced changes in their children. Ackerman saw that individual dynamics appeared to arise from similar dynamics in parents. His commitment to thinking about families, coupled with his influential position in the psychiatric community, did much to win acceptance for family therapy. In 1960 he founded the Ackerman Family Institute in New York City.

At the same time, modifications of psychoanalytic theory began to emphasize social forces: Heinz Hartmann's adaptive ego psychology, Harry Stack Sullivan's interpersonal psychiatry, and Eric Erikson's epigenetic developmentalism. Concomitantly, psychoanalytic object relations theory was being developed by Melanie Klein, D. W. Winnicott, and others in England. These clinicians and theorists took into consideration the interactional nature and relational matrix that appeared to give rise to intrapsychic process. In doing so, they indirectly fostered the development of family therapy. Parallel activity between psychosocially aware, individually oriented therapists and family therapists continued into the 1950s and 1960s. As an example, empirical research by Margaret Mahler and her colleagues on the separation-individuation process furthered the concept that development does not occur apart from a social matrix. Mahler's seminal work supports family therapy interventions, yet it rarely is mentioned in family therapy literature ( Mahler et al., 1975).

## Radical Environmentalism and Family System Theory

The next era in the development of family therapy was revolutionary. In the 1950s, 1960s, and 1970s, the family was defined as a system, and for many clinicians the only way to intervene with a family was through systems therapy. This perspective dominated family therapy for a generation, and its impact is still being felt. The biologist, Ludwig Von Bertalanffy (1968), had significant recurring impact as perhaps the major theoretician of general systems theory.

Similar to the development of psychoanalytic concepts after their introduction by Freud, the concept of the system soon was developed further by a number of different clinicians. Their different emphases became subsumed under various schools of family therapy ( Table 84.1). Carl Whitaker, known for his unique, idiosyncratic style and creative clinical abilities, founded the *experiential family therapy* school (Whitaker and Keith, 1981). Whitaker used an antihistorical, antitheoretical, here-and-now approach that emphasized emotional expression and self-actualization.

**Table 84.1. Varieties of Family Therapy**

*Strategic family therapy* emphasized communications between family members and used paradoxical techniques. Gregory Bateson, Jay Haley, and Don Jackson were early leaders in this group that described the "double-bind" communication ( Bateson et al., 1956). In 1958, Jackson established the Mental Research Institute, which fostered much generativity in strategic therapy. Finally, *structural family therapy* was developed by Salvador Minuchin, initially derived from his work with poor families during the 1960s (Minuchin et al., 1967). In contrast to many early family therapists who worked with middle class families, Minuchin's direct, pragmatic approaches in a difficult-to-treat population soon captured the imagination of many in the field. Other clinicians who maintained interest in developmental theory and analytical processes while working with families included John Bell (1961), Murray Bowen (1966), and Ivan Boszormenyi-Nagy (Boszormenyi-Nagy and Ulrich, 1987).

This was an exciting era because early systems proponents believed, not unlike earlier proponents of psychoanalysis, that mental illness could be cured through this new approach. Along with this radical view came a rigid adherence to the approaches of the systemic perspective that became conflictual for the field. For example, it was not uncommon for a family to present for a psychotherapy session without one member and have the session canceled because it was believed that the entire family had to be present for the process to be effective. Systems therapists were trying to make the point that the family is a system and it is impossible to deal with



the entire system with members absent.

Systems therapists moved away from individual psychodynamic approaches, yet remained vigorous environmentalists. The here-and-now focus of family systems treatment was frequently demonstrated through live clinical interviews in formal and informal educational settings. A generation of family therapists was spawned through attending workshops and observing the techniques of charismatic figures. The advent of video technology allowed practitioners to demonstrate their work, which added visual learning to the traditional process note method of psychotherapy education. Contrastingly, direct observation appeared to encourage further the emphasis on techniques and the dramatization of the change process. It was not uncommon for clinicians to teach that strategic interventions in a “stuck system” could produce results within one session.

Family therapy techniques became the order of the day. Some were creative and generative of new hypotheses of family functioning. As an example, disruptive children in a family would be asked, in a *paradoxical intervention*, to become increasingly disruptive at the first sign of a parental disagreement, and by this action would keep the parents escalating to open conflict. By prescribing the hidden function of such systemic problems, the therapeutic process rendered them explicit and voluntary. If a family complied with the paradoxical directive, these elements of mental functioning were brought under conscious control. If the family resisted accepting the directive, the dysfunctional structure changed and the problem resolved. Developing newer and newer techniques began to drive the field, a field that lacked a unified theory of family functioning. A generation of family therapists mixed elements of systems theory with a collection of techniques, as the field of family therapy became technique driven.

Given this trend, it was not surprising that polarization began to occur between family therapists and certainly between family therapy and the field of child and adolescent psychiatry (McDermott and Char, 1974). Child and adolescent psychiatrists and family systems therapists interacted less frequently. Ravenscroft (1996) has described the separatist climate of therapists who practiced outside the mainstream of psychiatry, including child and adolescent psychiatry. The attitudes taken were frequently “anti” the following: medical model, designated patient, individual symptoms, differential diagnosis and differential therapeutics, and individual therapy, especially psychoanalysis. Systems therapists eschewed history and the explanation of specific etiologic factors, often derisively referring to these linear explanations as archaic and antisystemic. The developmental needs and perspectives of children were not a focus of systems therapy. By ignoring individual psychology and broader social networks, this narrow, rigid systemic view was similar to earlier developments in psychoanalysis where, for example, all behavior was seen as unconsciously derived and, therefore, interpretable. The pervasiveness of these ideas led some family therapists to bemoan the loss of the “self in the system” (Nichols, 1987).

### Rapprochement and Integration

Numerous advances in psychiatry in the 1980s and 1990s, as well as specific research on developmental psychopathology, led to the integration and rapprochement of family therapy with child and adolescent psychiatry. The current status of family treatment, and the orientation of this chapter, is that a family therapy approach that integrates systems, individual psychodynamic, and behavioral concepts most effectively treats the psychiatric disorders of children and adolescents. Such family treatment is cognizant of biological vulnerabilities and is flexible enough to include parent training and education, as well as individual therapy with children, adolescents, and parents, and to work with the broader social networks that are involved with disturbed children. Integrated family treatments typically include aspects of several different schools of family therapy. Family therapy approaches differ with specific psychiatric disorders, and clinicians increasingly tailor approaches to the unique characteristics of each disorder (Table 84.2).

**Table 84.2. Clinical Issues in the Family Treatment of Specific psychiatric Disorders**

A confluence of several areas supports the integration of family treatments with other interventions, particularly interventions with individual children and parents (Josephson, 2000a).

1. Child development studies (Emde, 1983; Main and Goldwyn, 1984; Stern, 1985; Zeanah and Zeanah, 1989) emphasize that the relational family context generates the inner mental world of a child, also termed *internal working models of relationships*.
2. Clinical psychiatry emphasizes an interactive mosaic of risk and protective factors (Kagan, 1988). Child psychiatry uses “goodness of fit” to describe biological vulnerability interacting with environmental demands to determine clinical outcome (Thomas and Chess, 1980). Biopsychosocial psychiatry uses a stress-diathesis model (Engel, 1980; Rosenthal, 1970) in that the stressfulness of an event depends on the unique biological and psychological characteristics of the individual experiencing the event. In this model, the family may be a risk factor or a protective factor.
3. Family researchers (Reiss, 1989) emphasize factors that reside in the individual as well as factors that reside in the system.
4. Psychodynamic psychiatry continues to include interactive concepts in its theoretical base. Object relations theory is based on the internalized object relations derived from life experience (Scharff and Scharff, 1987), and self psychology describes parents as “self objects,” providing mirroring and idealizing functions necessary for healthy child development (Kohut, 1971).
5. Outcomes of specific medical disorders are increasingly seen as influenced by family processes (Reiss et al., 1986). This fact is underscored with the development of a new journal, *Family Systems Medicine*, devoted to this topic.

### Current Trends

Even as many forces have led to an integration of family treatment with other therapeutic approaches, dissatisfaction with systems approaches has been associated with a new development in family therapy, the use of *narrative therapy* as an organizing construct (Freedman and Combs, 1996). This movement is encouraging but harbors a potentially divisive element in the trend toward integrating therapies. On the one hand, this approach appears to be a return to the developmental perspective, that each individual life is lived in stages and in each stage, one's life story unfolds. Respect for the individual perspective of each person in the family is engendered by the clinician's careful, empathic listening responses. This change seems a clear response to the technicism and hierarchical approaches of many systemic family therapies, wherein the clinician issued various change directives to the family. Although there is a similarity to a historically based, developmental perspective, there also is a distinct difference. This trend in family therapy is heavily influenced by social constructivism, the “postmodern” idea that there are no essential truths except the ones individuals construct for themselves. As a result, the therapist conducts family therapy without *a priori* assumptions regarding healthy family life and normal child development, but rather joins the family through interpreting their experience.

The problem in integrating this approach to child and adolescent psychiatry is obvious. The child psychiatrist attempts to identify generalizable, if not universal, principles of families and child development based on clinical and research perspectives. The narrative therapist brings no such structure, or perspective, to therapy and attempts through “therapeutic conversations” to help the family construct a new story or a current reality. Although not explicitly antimicrobial, this approach appears to run the risk of marginalizing family therapy. In its attempt to regain the subjective, it dismisses concepts important to the experience of child and adolescent psychiatrists. The emphasis on the family's creation of reality makes the approach difficult to research as well.

Encouragingly, some clinicians see the possibility of rapprochement here as well (Malone, 2001). The medical model was misunderstood by many systems therapists who did not recognize that the idea of recursive feedback loops is consistent with a medical model. As blood pressure drops, the heart rate increases to maintain constant perfusion throughout the circulatory system. Similarly, the notion that an individual child, or parent, has a specific disorder does not exclude the importance

of family narrative and individual experience. Subjective experience can be maintained while empathically eliciting and categorizing symptoms that individuals and families are experiencing. By decreasing the emphasis on pathology, by denying generalizable principles of individual or systemic functioning, and by emphasizing “conversations” as a core element of treatment, the family therapy as envisioned by narrative therapists again runs the risk of being placed outside the mainstream of medicine and psychiatry.

Narrative family therapy will contribute most significantly to child and adolescent psychiatry by simultaneously enhancing the reemphasis on the patient's story while not dismissing core concepts in medicine and psychiatry. Narrative therapy is strongly tied to a specific, “postmodern” philosophy and, as such, is vulnerable to cultural shifts.

Contemporary psychiatry emphasizes biological mechanisms of disorder, often coming close to *biological reductionism*. A natural correlate of this trend is a deemphasis on family contributions to psychopathology. Although families may not always *cause* psychopathology, they can increase the *risk* for disorder. Family factors do not differ from biological and other psychological and social factors that increase risk for disorder, but rarely are powerful enough to be the sole determinants of disorders. Risk factors, such as genetic vulnerability to depression (biological), inability to trust others (psychological), and unemployment (social), *predispose* individuals to disorders but do not *cause* them. The tendency of contemporary psychiatry to translate biological risk into biological cause is a significant challenge for family therapy. As a result, families increasingly pursue pharmacologic interventions for many child behavior problems ( [Sprengr and Josephson, 1998](#)). Reimbursement systems that cover only the brief, infrequent clinical sessions of pharmacologic treatments further exacerbate the challenge to the practice of family therapy.

## AN INTEGRATED PERSPECTIVE ON FAMILY THERAPY

The contemporary approach to family therapy in the treatment of child and adolescent psychiatric disorders emphasizes integration. Systems theory has had a significant impact by offering valuable new perspectives on family treatments. The pendulum has now swung to reassert the importance of individual history and narrative. The integrated approach must consider systemic, psychodynamic/developmental, and behavioral perspectives. There is some difficulty in finding a term for the family therapy approach that deals with the relationship between family processes and intrapsychic development without the term being unwieldy. For example, Malone has used the term *psychoanalytic object relations attachment theory* (Malone, 1991). To facilitate readability, *psychodynamic* and *developmental* are used interchangeably in the remainder of this chapter to denote that particular family therapy perspective.

In the systems view, individuals in the family are interdependent. No change occurs in a family system without other things changing accordingly and, as change proceeds, the family acts to maintain equilibrium or homeostasis. Symptoms are not seen as residing in the child or adolescent but as serving a purpose, or function, for the family system. A child's physical symptoms, which result in his father canceling a business trip, appear to strengthen a tenuous attachment to this father and, at the same time, decrease the child's anxiety about ongoing marital conflict related to his father's frequent absences. Each family member is presumed to act in a way that would oppose symptomatic improvement in the child. The family interaction becomes a shared coping mechanism and, by this mechanism, members avoid distressing affects associated with change. This perspective often gives the clinician the ability to anticipate what events would transpire if a child's symptoms were ameliorated. Therapeutic interventions attempt to manage tension inherent in change and to prevent families from reverting to old patterns of interaction. In this instance, the family may achieve a new equilibrium and the child's symptoms remit as father adjusts his work demands and the marriage bond is renegotiated. Family therapy approaches that possess some of the characteristics of a systemic view are the structural approach and the strategic approach.

The integrated view of family treatment also emphasizes the unique life experience and individual development of each family member. The emphasis on life experience integrates the concepts of developmental and psychodynamic psychiatry with some of the current notions of narrative in family therapy. This perspective also suggests mechanisms of transmission. It presumes that a child's life experience in family relationships becomes internalized as a psychological structure. In adulthood, these internalizations prepare the way for choice of a partner and new family formation. When a parent's developmental experiences are negative, such as sexual abuse, parental harshness, or parental indulgence, they predispose the parents to initiating pathologic family interaction. To some extent, these developmental experiences predict how a parent negotiates the transitions of the family life cycle in the next generation. The family's passage through the life cycle and the interactions that are associated with this passage contribute to the definition of the inner world of the developing child. Family treatments that have elements of this approach are intergenerational family therapy ( [Bowen, 1978](#)), contextual family therapy ( [Boszormenyi-Nagy and Ulrich, 1991](#)), and object relations family therapy ( [Scharff and Scharff, 1987](#)).

Finally, the integrated approach to family therapy must include an understanding of behavioral elements based on social learning theory. This eminently practical approach teaches parents how to apply environmental contingencies such as positive and negative reinforcement, punishment, and extinguishing responses, all of which shape children's behavior. Remarkably, many parents do not know how to apply basic behavioral management techniques. These techniques can be taught directly and through their application, parents can see dramatic changes in their child's behavior. Behavioral approaches to family treatment include functional family therapy ( [Barton and Alexander, 1981](#)), parent management training ( [Forehand and McMahon, 1981](#)), and behavioral family therapy ( [Griest and Wells, 1983](#)).

An integrated approach to family therapy does not diminish the uniqueness of each school. The definition of schools of family therapy has been useful in the advancement of the field, and there may be some training advantages in learning one specific approach before practicing eclecticism. In our current state of knowledge, one school has not been shown to be more efficacious than another. The general orientation of each school is summarized (see [Table 84.1](#)), but this chapter does not review each school in depth, apart from a brief discussion regarding the most useful techniques of several of the schools.

The integrated approach to family therapy draws support from three areas: clinical practice, research in developmental psychopathology, and contemporary psychiatry's interactive model of symptom development.

### Clinical Practice

Several individuals have been attempting the integration of individual/psychodynamic and systems perspectives in clinical practice for a number of years ( [Kantor, 1985](#); [Malone, 1983](#); [Pinsof, 1995](#); [Scharff and Scharff, 1987](#); [Slipp, 1984](#)). Two common clinical problems illustrate the importance of simultaneously considering these two perspectives.

In a family with an adolescent with anorexia nervosa, excessive attention to the child's eating serves the function of diverting attention from a marriage that lacks intimacy, and serves to *maintain* the eating disorder. As this interaction proceeds, however, the excessive attention and solicitous parenting toward the adolescent begin to *generate* problems in her. Specifically, her sense of specialness, entitlement, and dependency, all derived from the experience of parental figures watching her every move, are maladaptive in social situations. As this young girl begins to negotiate adolescent relationships, she finds others are not nearly as solicitous and attentive as her parents. The experience of weight loss conveys a needed sense of identity, control, and self-efficacy. In this sense, family interactions not only have maintained her eating problem but have contributed to her own internal problems.

In like manner, adolescents who exhibit self-harming behavior draw concerned family members toward themselves. The systemic consequence of self-harm also is accompanied by the need for an assessment of the affective and perceptive state of the individual adolescent. Self-harming behavior affects others, yet also is a clear indication of inadequate individual coping with distressing, painful affects.

### Research Supporting Integration

A number of family researchers and child development researchers have provided empirical support for integrating systemic and psychodynamic/developmental perspectives. [Reiss \(1989\)](#) conceptualizes family continuity in terms of the *representea* family and the *practicing* family. According to this categorization, in the represented family not only are relationships represented by inner working models of self, but the coherence and stability of the relationships themselves are located in, and conserved over time, by such internal structures. In the practicing family, the coherence and stability of family relationships do not reside in individuals but rather in the coordinated practices of the family as a whole. At least four specific lines of research are concerned with family factors in the mechanisms of intergenerational transmission of psychopathology. Several studies ( [Belsky and Pensky, 1988](#); [Main, 1984](#)) have demonstrated that the transmission of patterns across generations of abuse and child maltreatment are mediated by the parents' internal working models. This research does not consider how a parent's internal working model becomes translated into actual abusive relationships with his or her child. To understand this aspect of transmission, the practicing family perspective must be considered. Studies on alcoholism ( [Steinglass et al., 1987](#)) indicate that its transmission across generations is influenced to the extent the alcoholic individual's drinking behavior is included in family rituals. The “coercive family” engages in a pattern of coordinated family sequences that, according to [Patterson \(1982\)](#), maintains conduct disorder in boys. Finally, [Goldstein \(1987\)](#) has confirmed with adolescent patients what adult studies have shown: a strong association



between the presence of high expressed emotion and the development of schizophrenia spectrum disorders.

Other studies documenting the relationship of individual psychological development to life experience include those of Emde and colleagues ( [Klinnert, 1986](#)), who describe the concept of “social referencing.” In these studies, infant and toddler behavior is shaped by the behavioral responses of parents. Similarly, [Anders \(1989\)](#) has described the process of development as requiring the regulatory functions of caretakers to proceed smoothly.

### Family Therapy, Neurosciences, and Integration

Although biological reductionism seems to be on the rise, the stress-diathesis model ( [Rosenthal, 1970](#)) and the biopsychosocial model ( [Engel, 1980](#)) allow for an integration of family, developmental, and biological factors. Psychiatric disorders currently are viewed as the result of a mosaic of biological, psychological, and social risk and protective factors. Not all families with interactional difficulties are associated with child and adolescent psychiatric disorders (e.g., overprotectiveness is not always associated with an anxiety disorder). On the other hand, many individuals at biological risk for disorder do not develop the disorder in question (e.g., monozygotic twins are not uncommonly discordant for schizophrenia). This mosaic of risk factors suggests that psychiatrists can make a diagnostic statement regarding an individual child, yet appreciate the importance of family interaction. The psychiatric mainstream has recognized that psychiatric disorders can cause disturbed family relationships, but disturbed relationships can be associated with the onset of child and adolescent psychiatric disorders ( [American Psychiatric Association, 1994](#), pp. 681–690). An integrated family therapy model that integrates the findings of biological and developmental research is the model that best serves the field because it will be the model most likely accepted by practicing child and adolescent psychiatrists.

## CLINICAL PROCESSES: ASSESSMENT

Effective family therapy is based on a thorough assessment of the family, which provides data for the clinical formulation. It is the formulation that delineates the family's role, if any, in the onset and maintenance of psychopathology and the relative effect of individual child psychopathology on the family. Although numerous sources are devoted to general family assessment ( [Nichols and Everett, 1986](#)) and family therapy techniques ( [Minuchin and Fishman, 1981](#)), the following delineates the relevant clinical processes involved in assessing and treating a family of a child and adolescent with a psychiatric disorder. Consistent with the integrative approach of this chapter, these processes are those that have the consensus of the field and have been developed by multiple contributors. The following section identifies the goals and objectives of family assessment, the clinical data of assessment, and the typical sequences of family treatment, and provides a clinical example to illustrate treatment principles.

### The Family Interview

The assessment of the family is based primarily on the clinical interview. Rating scales and self-report measures can contribute increased detail to an assessment but typically are used for research ( [Fredman and Sherman, 1987](#)). The family interview should obtain history of the immediate clinical problem, or presenting complaint, and history relevant to general, pervasive family functioning. The interview should observe interactions between family members and assess the thoughts and emotions of individual members in the family. The interview must gather enough data to develop a formulation of family problems that allows the clinician to determine if, and how, these family factors relate to the clinical disorder. The data of the family interview serve the goal of developing a formulation and, thereby, a rational intervention strategy. Interventions may directly attempt to alter family relationships or solicit family support for other specific interventions. An essential goal of the family interview is to develop an alliance with each family member that facilitates future contact.

In addition to general goals, the family interview should achieve specific objectives ( [Josephson and Moncher, 1998c](#)). A well conducted family interview (a) indicates areas of family interaction requiring behavioral intervention; (b) identifies marital and individual impediments to systemic and behavioral interventions; (c) reveals which areas of a child or adolescent's development have been affected by family interaction; (d) identifies problems and areas of concern of other family members; (e) suggests which areas of delayed parental development have predisposed to family interactional problems; and (f) helps determine whether the family is compensating for a child's disorder or is contributing to the child's disorder.

#### PREPARING FOR THE FAMILY INTERVIEW

Before the interview, the clinician should review all available clinical data, determine the referral source, identify who has recognized the need for change, and understand whether other agencies and professionals are, or have been, involved with the family. The most common presentation requiring a family interview is a symptomatic child, but on occasion a family may present with an identified family problem and request a family intervention.

There are two basic approaches to the initial interview. Some clinicians request that the entire family be present for the first session and focus this session on family interactional factors, the meaning of symptoms for the family system, and how the symptoms are maintained by the family system. Other clinicians adopt a more eclectic, flexible approach and do not request that the entire family be present initially. This approach tends to make families less defensive because the clinician uses separate interviews with child and parents to gather extensive history before observing the family as a unit. When parents are interviewed alone, important facts about the child's development and symptoms emerge. An adequate diagnostic appraisal requires the gathering of factual information about the child and about the family. Individual interviews with parents tend to be superior for this purpose. The detection of family psychopathology is adequately detected through individual interviews with parents and child, yet such interviews are less effective in the assessment of how pathology arises and is maintained ( [Jenkins, 1994](#)).

Individual parental interviews emphasize traditional elements of the psychiatric evaluation such as transference, countertransference, and resistance, as they relate to parents. They also allow the clinician to assess parental temperament and personality so that the clinical data can be placed in context ( [Greenspan, 1991](#)). Parents also provide their perceptions regarding discipline techniques and communication in the family.

The clinician must determine who will participate in the family interview. All first-degree biological relatives who reside with the identified patient should attend the initial family interview. This interview also should include those who have influential regular interactions with the identified patient, such as grandparents who have daily contact or parenting responsibilities. It may be necessary to have several meetings with such individuals to observe fully patterns of interaction. Children of divorced parents should be interviewed with the biological parent who has regular physical custody and, where relevant, a new partner. Wherever possible, separate interviews with the noncustodial biological parent are indicated to provide a complete perspective on the child's problems. It is not uncommon that some family members fail to attend when requested to do so. When this occurs, it frequently is due to some family interactional problems that are contributing to the presenting complaint.

#### STAGES OF THE INTERVIEW

There is some consensus about the specific stages in the family interview: a social stage, a problem stage, an observation stage, and a summation stage ( [Goldner, 1987](#); [Haley, 1976](#); [Josephson and Moncher, 1998a, 1998c](#)).

As in any meeting of strangers, the beginning of family therapy requires a greeting and the introduction of each person, tailoring these interactions to the developmental level of each child present. This *social stage* quickly gives way to the *problem stage*. Although most of the participants have some idea of the problems that have led to presentation to the clinic, the initial inquiry into these problems begins with the parents, acknowledging their executive function in the family. Subsequently, the clinician explores the problem with each family member. It is not uncommon to leave the identified patient, child or adolescent, until the end to convey support and decrease defensiveness.

It is important to gather a history of acute events and a sequential account of particularly troubling interactions that are associated with the clinical presentation. The process of therapy actually begins with this aspect of assessment. As the interviewer elicits information from family members, the style and structure of questions introduce new information into the family system. This has been termed *interventive interviewing* ( [Tomm, 1987, 1988](#)). Another relevant interview technique is *circular questioning*. By this method, the clinician does not directly confront symptomatic individuals about their behavior, but explores patterns of communication within the family as problems are sequentially discussed with each family member. Each participant in the interview is invited to express his or her perspective on the behaviors and interactions of others. This type of interviewing often produces new material for families to consider, which can be threatening and, at the same time, very relevant.

The *observation* stage occurs throughout the interview and, because relationships are better understood by observation than by report, is an essential element of the assessment. This stage begins by observing choices of seating arrangement. At any point in the family interview, as a family member is sharing his or her perspective

on family problems, the family may begin to interact in ways that demonstrate the presenting complaint or other problems. Once an important family interaction is set in motion, the assessment of this interaction takes precedence over further historical data gathering, which can be reinitiated after the spontaneous interaction has subsided. The observation stage forces the interviewer to decide whether to continue to focus on the presenting problem of a child, gather associated aspects of family history, or focus on immediate interactions that have interrupted the flow of the interview. The clinician should pursue the most pressing issue for the family, being ever mindful of the presenting complaint.

When a child's dependent behavior is part of the presenting complaint and the clinician observes the father answering questions for the child and the child looking to the mother for direction, the clinician has the benefit of living with the problem as opposed to merely hearing it described. Such observations have great clinical utility in that, once they are comfortable, families tend to behave in the clinic as they do in natural settings. It is important in the observation stage to be every mindful of the possibility that in broadening the interview to general aspects of family life, family members can become defensive at this apparent intrusion by the interviewer. Because one of the key goals is to develop an alliance with the family and have them return to be involved in the treatment of their child, it is a sensitive and important decision when to probe less directly into family life.

There are significant challenges for the child and adolescent psychiatrist in family interviewing compared with individual interviewing. The basic challenge in conducting a family interview involves simultaneously gathering the facts of history and observing family interaction. It requires a certain degree of clinical skill to deal simultaneously with the apparent lack of organization in the presentation of clinical material, distracting noise and spontaneous discussion by various family members, and the concerns of multiple individuals.

Finally, the *summation stage* includes the clinician's brief comments regarding which changes appear to be needed, typically starting with elimination of the presenting problem. This stage may describe the need for other assessments of the individual child and further family interviewing, including family members not present initially. The clinician should clarify with the family whether their ongoing presence is necessary merely to support their child or whether there is evidence of family interactional problems that need to be addressed to benefit the child.

Although the many schools of family therapy and approaches to treatment offer unique perspectives on family therapy, there is an emerging consensus that the data on which interventions are based can be summarized in four categories: family history, family structure, family communication, and family regulatory functions. [Davidson et al. \(2001\)](#) have reviewed the content to be covered, providing a clinician's guide to relevant lines of questioning in assessing these four areas.

### Data of the Assessment: Family History

The data of history can be further categorized into four areas: (a) the family interactional history related to the presenting complaint and, if different, the history of pervasive family functioning; (b) parents' developmental history; (c) the history of the marital relationship; and (d) the history of the family as a unit (i.e., family life cycle). The approach to gathering family history differs according to the experience of the parents. Parents who have more than one child draw on a comparative, developmental database unavailable to first-time parents.

#### INTERACTIONAL HISTORY

It is not uncommon for the history of a child's presenting problems to be associated with specific interactional sequences. For instance, aggression, self-harm, and refusal to eat may arise after interaction between family members. The acute interactional history is very useful in understanding the precipitation of clinical problems. Clinicians must determine if the descriptions of family interactions associated with acute events are similar to family interactions that have occurred on a chronic basis. The interactional history helps distinguish between adjustment problems and those chronic interactions that have a more enduring impact on child development.

#### PARENT HISTORY

Whereas the onset of children defines a family, family history is informed by an understanding of the developmental histories of each of the parents and the history of the marital relationship. The clinician asks, "What type of person is this parent?" A systematic developmental history of each parent, derived from the descriptions of family-of-origin experiences, informs an understanding of parental strengths and weaknesses and parental personality functioning that mediates parental role functioning. Most parents' adaptive and maladaptive parenting strategies have been influenced significantly by how they were parented. Formal psychiatric and medical history is a standard part of this history.

#### MARITAL HISTORY

The marital history is a logical continuation of the individual parent history. The choice of partner significantly reveals aspects of individual functioning, both conscious and unconscious, that predispose to family health or family difficulty (e.g., the dependent woman who marries a tyrannical, controlling man). The clinician asks, "What drew these two individuals together?" The family's successful negotiation of life cycle tasks is the main task of the marital unit, the executive unit of the family ([Wamboldt and Reiss, 1989](#)).

#### HISTORY OF THE FAMILY AS A UNIT

Effective family interviewing requires knowledge of the stages of the family life cycle ([Carter and McGoldrick, 1999](#); [Combrinck-Graham, 1985](#); [Walsh, 1993](#)). Families with infants are dealing with issues of nurturance and attachment; those with toddlers are confronting issues of limit setting and the draining demands of constant observation; those with school-age children are addressing issues of socialization and achievement; and families with adolescents deal with the dilemmas of the imminent independence of their child and the accompanying evidence that she is not quite ready for complete emancipation. Finally, families deal with the complete emancipation of all their children, with couples facing renegotiating their relationship in middle age. Mastering stages of the life cycle includes mastering anticipated challenges, such as children entering formal schooling, and unanticipated challenges, such as a child's life-threatening illness. A unique aspect of the history of the family life cycle is a search for enduring, unique family beliefs and recurring themes in family life. Themes that endure in the family unit (e.g., the men in the family will assume leadership roles in the community) may be associated with puzzling patterns of family interaction that have existed from generation to generation. The family unit history often is schematically summarized in the genogram ([McGoldrick and Gerson, 1989](#); [Fig. 84.1](#)).

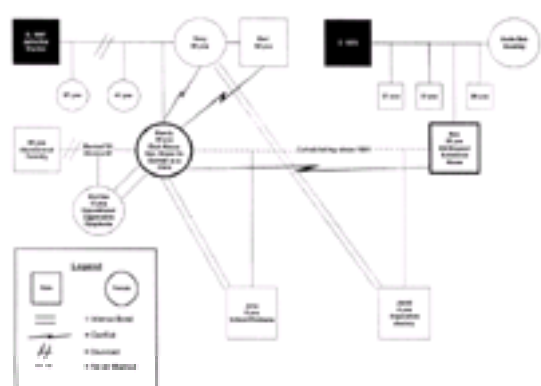


Figure 84.1. The Family Genogram as of 1999.

### Data of the Assessment: Family Structure

Family structure refers to the typical organizational and transactional patterns and hierarchies that exist between individuals in the family ([Minuchin, 1974](#)). The family interview, through historical data and observation, produces information from which the clinician can assess family structure. The important components of family



structure are adaptability, cohesion, and boundaries.

#### *ADAPTABILITY*

The healthy family has a flexible structure with stable transactional patterns that shift when circumstances dictate the need for change. Clinical families may be too chaotic, with interactional patterns and individual family roles constantly changing, or too rigid, with the family unable to change typical ways of interacting, even as life circumstances demand such changes.

#### *COHESION*

The healthy family demonstrates a balance between connectedness and separateness, whereas clinical families may be too emotionally close (i.e., enmeshed) or too disengaged.

#### *BOUNDARIES*

The healthy family has emotional boundaries between subsystems (e.g., parent–child) that are permeable but clear (e.g., an adolescent teasing father while father remains an authority figure), whereas clinical families often have boundaries that are rigid, diffuse, or misaligned (e.g., a child serving as the primary source of emotional support for a parent).

### **Data of the Assessment: Communication**

Family communication refers to verbal and behavioral interactions by which family members impart information to each other about their individual needs and their perceptions of others in the family. Components of family communication are clarity, emotional expression, and problem solving.

#### *CLARITY*

Healthy families communicate clearly and consistently with affect that is congruent to the message conveyed. Clinical families tend to communicate ambiguously about both minor transactions and those of major importance.

#### *EMOTIONAL EXPRESSION*

Healthy families communicate with an open expression of affect congruent with the message conveyed, whereas clinical families may either block the expression of feelings, express affect incongruent with life experience, or both.

#### *PROBLEM SOLVING*

Healthy families agree that problems exist, negotiate differences in conflicts, and are able to use new information. Clinical families tend to have multiple individual perceptions of the problem, are unable to sacrifice toward common family goals, and are unable or unwilling to perform the tasks necessary to assist family coping. Clinical families are ineffective at problem solving and have a decision maker who is either a poor communicator, authoritarian, or indecisive.

### **Data of the Assessment: Family Regulatory Function**

The physical, social, and emotional development of children evolves in an interactional context that facilitates the mastery of developmental tasks. The family is perhaps the most powerful regulator of the developmental process. Regulation has been described as implying an equilibrium or balance between inhibiting and facilitating interactions between caretaker and child ( [Anders, 1989](#)). Families that appropriately regulate a child's development are attuned to developmental needs and adapt their behaviors accordingly. These needs vary according to a child's developmental stage and temperament, as well as a family's particular life situation. In some situations, for example, children need a limit or restriction, and in other contexts they need encouragement of autonomous functioning. Children may need to be protected and, at other times, may need to fend for themselves. Families who are in clinical difficulty often have several patterns of regulatory dysfunction, exacerbated by skews between the different approaches of mothers and fathers ( [Josephson and Moncher, 1998c](#)).

## **CLINICAL PROCESSES: FAMILY TREATMENT**

Once family interviews are completed, the data gathered in the four domains—history, structure, communication, and regulatory function—reveal what family factors are related to clinical problems, what factors serve as a general context for the problems, and what factors are unrelated to the problems. This formulation serves as the basis for family intervention. With some symptomatic children, the family is viewed as etiologically important in the onset and evolution of the child's problems. Here, the clinician sensitively explores and elucidates the role of family conflict in the child's disorder. At other times, family interaction is seen as a response to illness or disorder in the child. In such cases, the clinician educates and empathically supports the family as they endure difficult circumstances. Complex family situations may involve both approaches. Although there is no widely accepted classification of families, the clinician through these efforts at formulation gains a sense of the type of family with which he or she is dealing and tailors the approach to family treatment accordingly (see [Table 84.2](#)).

In contemporary child and adolescent psychiatry, it is rare that family therapy is initiated at the onset of a clinical encounter. It is much more common for an evolution of interventions with the family to take place, from more simple to complex ( [Josephson and Moncher, 1998b](#); [Ravenscroft, 1996](#)). Systemic and dynamic/developmental formulations lead to a sequential integrated approach to family therapy. The question is not whether to do family therapy, but when to do so. The family always must be involved in the treatment of a child or adolescent psychiatric disorder. When to involve the family directly and which specific elements of family treatment to use become the critical determinations for the clinician.

The following description presents a typical clinical process in which family therapy becomes embedded. The process described includes phases *before* the formal family therapy and *after* a family intervention may take place. Each phase described often unfolds chronologically, but not necessarily so. This general process includes a number of different features of many schools. Specific formulations of clinical problems and their related approaches may be of use in certain clinical situations, leading to some variation in this pattern.

### **Immediate Problem Solving**

Clinical disorders present as the gradual culmination of pervasive, ongoing problems that lead to referral or present acutely as the onset of crisis. Families present with problems that require immediate interventions, often behavioral and pharmacologic. To stabilize a crisis, a child may need to be removed from the immediate family environment, including psychiatric hospitalization for emergency evaluation and treatment of life-threatening situations. Clinicians may need to use community resources such as shelters for runaway children or emergency foster care. In less acute situations, families are given brief, problem-focused advice on child management to decrease family distress.

### **Parent Education**

After stabilization and immediate problem solving, education is the focus of the next stage of family intervention. Here, the clinician offers directives about the management of child and adolescent behavior that are explicit, easily understood, and quickly implemented by many parents. Parent management training and education often suffice to interrupt behavioral problems, particularly when the problems are mild or parents are merely inexperienced. Parents often benefit by learning how to set limits, learning to foster a child's independence, and acquiring relevant facts about child development. When education is only partially effective, or completely ineffective, the clinician typically has observed parental psychopathology or marital conflict underlying the inability of parents to respond to direct educational efforts. The clinician begins to develop questions—Why can't these parents set limits? What prevents them from nurturing?—that prepare for intervention in family interaction.

## Intervention in Family Process

At this point, the point of traditional family therapy, the clinician sees the entire family and notes patterns of interaction that are maintaining the clinical problem. Conjoint family therapy refers to sessions in which all family members are treated by a single therapist or cotherapists. Concurrent family therapy refers to family therapy and individual therapy conducted at the same time, usually with different clinicians. A final modality, the multiple family group, refers to meetings of several families, all of whom have an individual family member with similar problems (e.g., the disruptive child) or developmental challenges (e.g., the emancipating adolescent). In each of these varieties of intervention, family therapy sessions are held regularly, with variations in frequency, and can last as long as 2 hours.

The clinician challenges patterns of interaction using techniques such as those developed by the various schools of family therapy (see [Table 84.1](#)). Predictably, as the field has advanced since the time when technicism predominated, the most useful interventions have found their appropriate place in the family therapist's practice. This is not unlike the historical development of psychoanalysis, which had many of its most useful constructs—the unconscious, resistance, and transference—appropriated by other forms of psychotherapy. Now, the phenomenon of transference is an accepted aspect of general psychotherapeutic practice. In like manner, each family therapy school has contributed specific interventions that now are part of general family therapy practice. There are numerous, extensive descriptions of these interventions ([Gurman and Kniskern, 1991](#); [Minuchin, 1974](#)).

Family therapy interventions share many of the basic therapeutic tenets of individual psychotherapy. These include developing an alliance with the family, working through family resistance, and recognizing a type of “family transference” to the therapist. In addition, factors common to all successful psychosocial interventions are congruent with the techniques of family therapy: the healing context; a confiding relationship with a helper; a plausible causal explanation; the provision of optimism; and the provision of success experiences ([Frank, 1973](#)). Once the clinical formulation is clear in the clinician's mind, any number of different techniques can address problems in family functioning. This is an essential point: When the clinician discerns what needs to occur in a family, he or she can quite rationally determine how to do it.

As a general rule, structural and strategic approaches are most relevant for “here-and-now” problems, whereas intergenerational models address more chronic, pervasive family issues. Specific formulations for certain disorders can be very helpful in developing intervention sequences. When [Minuchin et al. \(1975\)](#) described core features of the “psychosomatic family” as enmeshment, rigidity, overprotectiveness, and lack of conflict resolution, they also implied ways to intervene with these families. In like manner, [Johnson's \(1949\)](#) classic description of “superego lacuna,” as it applied to some families with a conduct-disordered youth, was generative of new therapeutic approaches. Such special formulations are the exception, not the rule, yet when they exist, their striking presentations suggest specific targets for family therapy interventions.

*Structural family therapy* interventions include reframing, family enactment, and realigning boundaries. *Reframing* is an intervention by which the therapist attempts to give a positive, adaptive connotation to problematic behaviors. As the therapist provides an alternative way to perceive a behavior, he or she has the same intent of the individual therapist who clarifies and interprets. For example, a family therapist hears of a wife's anger and apparent disinterest in her husband's business trips. The therapist states, to the husband, “Your wife sure has an interesting way of saying ‘I love you’ and ‘I want to be with you.’” In family *enactment*, the family therapist promotes the replication of a family problem to understand its interactional and structural aspects. By directing a dependent adolescent to make decisions regarding his or her summer plans, the therapist can observe pathologic responses such as the overinvolvement of a controlling parent. At times, the therapist does not need to promote an enactment of a problem but merely pursue a detailed discussion of several interactional family problems that have occurred since the last session. In *realigning boundaries*, the therapist shores up boundaries that are diffuse and permeable or attempts to diminish rigid boundaries. For example, a family consultation reveals both parents are having difficulty controlling their three children. Their egalitarian beliefs include the ideal that listening to children is very important. They often conduct family votes on various issues. The family therapist educates the parents that they are in the executive position in the family and that, although they need to listen to their children, they do not need to have family boundaries so permeable that the children decide, by their majority vote, how to run the family. The direct nature of strategic interventions has immediate learning potential for families and can provide dramatic change experiences. The family therapist with a structural orientation often resembles a “traffic cop”—encouraging some to speak, directing others to stop speaking, getting participants to sit in different parts of the room, and changing chairs with parents.

Strategic family therapy interventions include paradoxical intervention and circular questioning. In a *paradoxical intervention*, the clinician makes a demand contrary to expectation, such as the request to exaggerate a symptomatic family pattern. In the family of a young girl with anorexia nervosa, the clinician encourages the mother to spend more time with her daughter and to help her daughter more often, while encouraging the father to remove himself from family life and develop his own pursuits. Prescribing the covert function of these family interactions renders them explicit and voluntary. If the family complies with the clinician, the interactions are brought under conscious control, antecedents to the overprotectiveness are explored, and a desire for liberation from this behavioral pattern is verbalized, usually by all involved. If the family resists the directive, the dysfunctional interaction changes. This technique may be less successful in the family therapy where members are cognitively or emotionally unable to process subtlety, and may be less useful in treating families with young children.

In *circular questioning*, the therapist does not directly confront the symptomatic individual but explores patterns of family communication. The clinician does not challenge the adolescent boy about his aggressive, noncompliant behavior. The family therapist elaborates the problems through interaction with the boy's mother, who describes its effects on her, her concerns of its effects on other children, and her anger at her husband's apparent unwillingness to attempt to control their son.

In *extended family intervention*, intergenerational processes in the broader kinship are the focus rather than events in the immediate nuclear family. Although it often is not practical to involve these extended family members on a regular basis, a well timed consultation can open up past interactional experience that has prevented the individuation of parents who are having trouble facilitating their children's emancipation.

Object relations intervention approaches deal frequently with projective identification. The family therapist notes a family member projecting parts of the self into another person, followed by identification with, and reaction to, these perceived attributes. This process is problematic enough in marital conflict, but is extremely toxic to children, whose sense of self is still developing and dependent on external validation. For example, the father of a teenage boy is extremely upset when his son becomes noncompliant and begins to experiment with behaviors that challenge the father's moral code. The father believes the son is going to develop sexual and drug-related problems, yet his rigid parenting seems to be provoking his son to engage in the forbidden activities. Later in therapy, the father, the son of a minister, reveals that he had impregnated a girl when he was a teen and had a significant alcohol problem in young adult life. Here, the family therapist intervenes through clarification and interpretation of the family interaction, and facilitates the father's recognition that his behavior toward his son is fueled by his own life experience and related internalized object relations.

*Narrative therapy* attempts to *externalize the problem* ([White, 1990](#)). This intervention involves two sets of questions. The first set of questions asks family members to describe the influence of the problem on their lives and relationships. In the second set of questions, the therapist asks the same individuals to describe their own influence on the “life of the problem.” Through this “influence questioning,” the problem is separated from the person and neither the family member nor the relationship between family members is blamed. Through externalizing the problem, family members gain some distance from problems that have been shaping their lives.

Altering family interactions may eliminate the problem and provide opportunities for improved adaptation in the family. Yet, these interventions often are resisted and unsuccessful. In these instances, individual family members often have their own reasons for resisting change.

## Considering the Individual Perspective

Intervention in family interaction patterns frequently are unsuccessful because of the individual motivations of family members. It seems that problematic family interactions often prevent individual intrapsychic conflicts from being expressed and, correspondingly, being treated. For example, in trying to interrupt an overly close mother–adolescent daughter dyad, the family therapist becomes aware of the mother's helplessness and sense of affective of distress at the thought of “losing her daughter.” Preexisting developmental factors that placed her at risk to overprotect her daughter begin to be exposed, and this exposure is resisted. The adolescent's dependency and selflessness seem the effects of mother–daughter symbiosis. The mother's conflict cannot be addressed while the family interactive processes of overenmeshment and overinvolvement is continuing. As family therapy attempts to help the daughter individuate, the mother's intense affects associated with this intervention place her at risk for becoming overinvolved, once again, in her daughter's life. By considering the internalized world of specific family members and using individual interviews to explore these worlds, the family therapist can intervene effectively with such resistance.

Family treatment can thus lead to individual interventions with either parent or the symptomatic child and can prepare for a marital intervention. It is important that this



process naturally unfold because there can be complications. For example, a clinician must not be drawn into “scapegoating” an individual family member through individual therapy and its implication of internalized pathology. Work with an individual family member may suggest the individual is the primary problem and could maintain family pathology rather than interrupt it.

## Marital Therapy

In a natural progression of family interventions and individual work, couples often consider their marital relationship some time after initial family interventions. Parents more easily reflect on their own contributions to clinical problems when their child's disorder is stable. Except in cases of imminent marital disruption, this stage usually occurs later in family treatment and indicates a deepening and maturing of the therapeutic process. Past issues surrounding the choice of a partner, and specific problems in the family life cycle, come into perspective for couples who are considering the difficulties of their child.

## Intervention with Siblings

Problematic family interactions typically affect more than one child. Symptomatic expression can be modified by a child's intelligence, temperament, or birth order, among other factors. As intervention in family process stabilizes one child, the needs of other children often become manifest. The clinician must assess whether the onset of symptoms in a sibling is a diverting mechanism away from another problem, such as father's alcoholism (systems view), or a statement of the sibling's individual vulnerability, such as low self-esteem (dynamic/developmental view).

## Termination

Some problems, which are phase specific, respond well to parent management training and brief family therapy interventions. In these instances, cases can be terminated. With multiproblem families, family interactions need periodic, ongoing monitoring. As children move through various stages of the life cycle, families may need to have renewed contact with a clinician who knows them well. The duration of family treatment varies from brief interventions for specific problems, to longer-term treatments with intermittent clinical contacts around different problems, often posed by different family members.

This sequential, integrated approach to family treatment is supplemented with other supports and interventions. Specific socialization difficulties often respond to group therapy. Educational supports often are necessary for children who have emotional difficulties, and parents who collaborate with schools maximize the chances of educational success for their children. Children with disruptive behavior disorders frequently are involved with the courts. This involvement, although always stressful, often contributes to the limit-setting efforts of parents who are having difficulty in this area. Families who are attempting to control their children can have their efforts strengthened when the behavioral consequences for breaking the law are applied by a legal agency.

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### CASE ILLUSTRATION<sup>1</sup>

Merrilee, a 15-year-old girl, was referred by her maternal grandmother, with whom she was living. She had been expelled from school, become increasingly oppositional, and experienced depressive symptoms with vague suicidal ideation. Merrilee would return to her mother's home for periods of time, only to get into conflicts with Mandy, her mother, and Bob, her mother's live-in boyfriend of 8 years' duration. Merrilee's father and Mandy had divorced when Merrilee was 3 years old (Fig. 84.1).

The initial *formulation* of family problems included a mother dependent on her daughter for affection, an immature “stepfather” with limited vocational skills and low self-esteem, and a maternal grandmother viewed by the mother as intrusive. Merrilee's adolescent behavior disturbance seemed the culmination of persistent developmental problems. Her two younger half-siblings, Bob's sons, aged 6 and 4 years, also were suspected of having difficulty.

Merrilee was hospitalized to ensure safety, complete a diagnostic evaluation, and initiate pharmacotherapy. *stabilized the crisis*, and resulted in a thorough assessment of familial strengths and liabilities. Contacts with community resources—mental health agency, court, and school—helped prepare for outpatient treatment. Merrilee was noncommunicative in individual and group therapies, but did comply with the behavioral expectations of the unit. She was discharged with noticeably improved affect, although she remained defiant.

*Parent education* in behavioral management techniques was an early focus in the outpatient course of treatment that followed hospitalization. Bob and Mandy were taught how to provide Merrilee with consistent discipline, and the family therapist discussed how a nurturant, nonpunitive stance could facilitate Merrilee's acceptance of limits. Mandy related to her daughter as a sister would, indulging her when consistent discipline was indicated. Efforts to get Mandy and Bob to work together to set limits on Merrilee were unsuccessful because she accurately perceived that Bob had no sanctioned role in the family. Merrilee's tyrannical, aggressive behavior toward her 6-year-old half-brother necessitated *intervention in family process*.

Merrilee's rejection of Bob's authority revealed problems in family structure. Bob was not supported by Mandy, and Merrilee took advantage of this fact, often commenting, “You are not my father.” Mandy frequently joined her daughter in berating Bob, exacerbating family tension. This structural problem made effective behavioral control of Merrilee impossible. It appeared that commitment in the couple's relationship was tenuous and that perhaps Mandy was continuing the relationship only because of the income she received from Bob's Social Security disability benefits.

A family intervention attempted to strengthen the parents' relationship and decrease the control of the grandmother, who not only allowed Merrilee to stay in her home but was caring for Jacob, the 4-year-old half-brother. Efforts were made to have both Jacob and Merrilee return home, with Bob and Mandy working together to provide nurture and discipline. Parent education and intervention in family process were frustrated by the *sig* *individual needs of parents*.

Mandy was a self-preoccupied woman who had experienced a severe burn when she was a young child. Some facial disfigurement and a father who spent all of her developmental years incarcerated seemed important factors in her low self-esteem. Mandy's choice of husbands further reflected a low self-concept. Merrilee's father, Mandy's first husband, abandoned the family, and Bob, 8 years her junior, had cognitive impairments that were associated with employment difficulties. Individual sessions with each parent revealed their feelings of being overwhelmed with raising three children. The role of grandmother took on a new perspective. Individual contacts with the grandmother revealed her to be less intrusive and more the family safety net, providing support and structure for a couple that desperately needed it. Merrilee refused individual therapy but received some support from a group intervention. Merrilee became increasingly aggressive toward John, the 6-year-old. It became apparent that the reunification of the family, with all three children in the home, taxed the parents as individuals and their relationship.

Bob fled from the responsibilities of parenting while Mandy undermined Bob, refusing to marry him. This inadequate parental coalition precluded effective parenting. Family process interventions, thus, became secondary to an evaluation of Bob and Mandy's relationship and its future. Little hope was offered for the children's stability without clarification of their relationship. Several episodes occurred in which Bob impulsively left the home or was asked by Mandy *Marital therapy* reviewed these conflicts and their antecedents, with the goal of enhancing Bob's and Mandy's skills in resolving conflict.

School personnel initiated contacts with the family therapists (a cotherapy team) to *iden* *problems with sibling*, John and Jacob. John was seen as an aggressive, noncompliant boy, whereas Jacob was seen as distractible, preoccupied, and impulsive. They appeared to be in competition with each other for the affections of teachers. Individual assessments and psychotherapy were offered to the boys, with Bob and Mandy receiving further, developmentally oriented parent management training.

The treatment of this disorganized family was fraught with difficulty, and the process described had several recursive loops. Well into treatment, Merrilee required rehospitalization for aggressive behavior and experienced legal detention for destruction of property. The family received in-home, behavioral parent training for several months. Ultimately, Merrilee could not be integrated into her mother's home, and when residential placement was unavailable, she was permanently placed with her grandmother. This boundary clarification seemed to help Mandy and Bob to focus on the needs of John and Jacob, who remained with them. John's and Jacob's behavior improved in the school setting, although in individual sessions they both manifested indications of insecurity related to their father's impermanence in the family. The parents made some progress with their individual conflicts but continued to have difficulty with a long-term commitment in their relationship and experienced another physical separation.

The continuing availability of the family therapists, who identified the needs of individual family members and the need for family system alterations, served an organizing function for this multiproblem family. When the therapists identified *theadjunctive supports outside clinical session*, they coordinated family therapy with the activities of other agencies.

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## CLINICAL ISSUES

### Indications for Family Therapy

Family treatment, in some form, always is indicated in treating children, and usually indicated in treating adolescents ( [McDermott, 1981](#)). The family is the developmental context of each child's life and can powerfully impede a child's development or dramatically aid it. Although families always must be involved, the specific target of intervention—individual parent or child, marital unit, or entire group—varies. With some disorders, the family is viewed as etiologically important in the onset and evolution of disorder (e.g., conduct disorder). Here, the clinician empathically explores family conflict and educates the family about their role in the child's disorder. With other disorders (e.g., autism), family interactions and responses are seen as the response to illness in the child. In these instances, the therapist empathically supports the family and provides sources of referral for further support. [Table 84.3](#) offers some guidance to the clinician in making this difficult, yet important, distinction.

Family health is suggested when some of the following characteristics are present. The more characteristics are evident, the more confidence the family therapist can have that observed family interactions are the cause of a child's disorder and therapy should emphatically support the family.
1. Parents understand developmental norms.
2. Parents are free of major psychiatric disorders.
3. Parents are committed to their children's well-being.
4. Parents have been exposed to their child's needs.
5. The family has members (includes grandparent) who have mastered developmental tasks.
6. The family does not have a child with demonstrable organic deficit.
7. Parents are mutually supportive.
8. Child's behavior is not demonstrably sensitive to environmental change (e.g., conflict with family and other family).
9. Parents have demonstrated effectiveness with other children and settings (i.e., discipline).
10. The identified child has (a) differential responses to the family environment compared with other environments (e.g., "center of home," "corner of home," "room"),
11. Mental disorders occur in other siblings.

### Table 84.3. A Clinical Guide to Determining the Directional Effects of Family Influence

Empathic exploration of family issues is indicated when parental psychopathology complicates parenting efforts; there is empirical and clinical support for the efficacy of family approaches; a clinical problem presents as an interactional problem (e.g., physical conflict between son and father); or a child fails to respond to repeated medication trials.

Empathic support of families is indicated in the treatment of medical disorders such as diabetes, psychiatric disorders with a strong biological component such as schizophrenia, and neurologic disorders such as mental retardation and autism. The overwhelming stresses faced by families of such ill children require the mobilization of individual and community support. Education, respite care for children, and linkage with support agencies are all important adjuncts to family treatment.

#### Contraindications to Family Therapy

The main areas of contraindication involve legal and ethical issues. In family treatment it is contraindicated to:

1. Repeatedly involve individuals who are not regularly involved in a child's life.
2. Continue to work jointly with parents who are divorced. Ongoing work with each parent and new family unit should occur, but in separate sessions. This decreases confusion for the young child and minimizes the chances of reawakening old conflicts in the adolescent.
3. Conduct family therapy that increases the potential for harm to family members. The family therapist may find that intense affects emerge during the exploration of family conflicts. Careful monitoring of these affects is imperative and, at times, a cessation of this type of uncovering work is indicated.

#### Complications

Family therapy can have dramatic effects relatively quickly. When progress does not occur, the clinician should be alert to several of the common impediments to successful treatment.

1. Minimal father involvement. Mothers often bring children by themselves for treatment. Fathers either resist the process of intervention or are physically unavailable. When fathers do get involved in treatment, progress still may be difficult, but the chances of success are increased.
2. Lack of clinician objectivity. Family problems mobilize strong countertransference responses. Affect-laden areas such as child-rearing practices, divorce, human sexuality, and gender role may be areas that clinicians are dealing with in their own lives. Unconscious clinician attitudes may negatively affect treatment.
3. Inattention to nonnuclear family relationships. Some relationships may have significant potential to be a resource for the child (e.g., a mother's boyfriend), whereas others may be counteracting the positive effects of therapy (e.g., an intrusive grandparent). These factors must be accounted for in planning family therapy.
4. Lack of appreciation of the cultural context of the family. Norms of expected family development and child development vary according to culture and socioeconomic level. Treatment approaches should be adjusted accordingly.
5. An inaccurate formulation of the family's role in the child's disorder. Progress is frustrated when families are inappropriately held responsible for a child's biologically based disorder. On the other hand, progress is delayed when a family receives support when its conflicts should be explored and change expected. When the latter is operative, it often is complicated by excessive reliance on medication interventions ( [Sprengr and Josephson, 1998](#)).

#### Ethical Issues

Most ethical issues in family therapy are related to issues of confidentiality and the clinician–patient relationship ( [Jensen et al., 1989](#)). In family therapy, the therapist relates simultaneously to other members of the family, in addition to the identified patient. The family therapist can comfortably discuss, with any family member, material that previously emerged in family sessions. Dilemmas arise when information that a family member shares with the therapist, in the absence of certain family members, has relevance to those members. In this instance, the therapist must encourage the individual to bring this information into future family sessions. With discerning clinical judgment and permission of the individual, the clinician may initiate sharing the information with the family.

Family therapy initially may focus on an identified patient, but it is not uncommon that as therapy progresses, the concerns, and perhaps disorders, of other family members become a focus for treatment. The therapist is at risk of coming into conflict with the needs of other family members as therapy proceeds. Family members who have not met each other's needs may become less invested in therapy when one member appears to be benefiting most directly from the clinician's intervention. The family therapist must be aware of this issue and make a deliberate attempt to relate to all family members. As the family therapist develops a therapeutic alliance with the whole family, all will accrue benefit. The clinician should make it clear that over time, each family member will be heard and each perspective will be taken into account, even though some sessions may seem to be weighted more toward one member's self-interest than another's.

#### Developmental Considerations

Family therapy differs dramatically according to the constellation of ages presented by the family in treatment. The younger the child, the more likely it is that the nature of the parent–child relationship will determine presentation. Excluding definite organic disease (e.g., autism), caregiving practices are the prime determinant of presentation in infants and toddlers. Parental availability and capability to nurture are important considerations. With toddler and preschool children, parents must be consistent and have significant energy to respond to their consistent demands and provide the structure they require. As the child enters formal schooling, families must facilitate success and competence in academic endeavors. Mastery of these endeavors is heightened through consistent, structuring efforts of an available parent. It is at this stage that parents usually recognize the strengths of their children and try to maximize them, as well as minimize weaknesses. Adolescent children present numerous dilemmas because they often request independence without demonstrating the competence to handle such independence. Emotions involved with physical and geographic moving away from the family are significant for both the parent and adolescent. The increased contemporary potential for negative consequences of high-risk behaviors involving drugs, motor vehicles, and sexuality brings a sense of urgency to many family therapy approaches to adolescent disorders.

#### Cultural Issues

In the family therapy literature, there is an increased exploration of the relationship of family dynamics to social class. Some of the relevant issues include the phenomenology of class-specific family dynamics, cultural differences in normative family processes and values, consequences of social class prejudice and discrimination, and clinician–patient interactional difficulties when social class and cultural differences exist ( [Canino and Inclan, 2001](#)). Families dropping out of therapy may be due to therapist lack of cultural awareness or therapist countertransference problems. It is not uncommon for clinicians to have difficulty accepting family styles and interactions that are not part of their own cultural tradition (e.g., the egalitarian therapist confronting a conservative, controlling father).

The family's cultural background influences beliefs about normative family structure, communication style, and child development. With respect to family structure, cultures differ regarding the appropriate level of involvement of the extended family. In some cultures, it is not uncommon for the extended family to be significantly involved in family life, including extended family members living in the same domicile. In contrast, most North American families are typecast as nuclear without intimate extended family involvements, with blended families and single-parent families becoming increasingly common variants. Despite differences in family structure, families in each culture must meet the many developmental needs of children.

Culture influences the range of emotional expression acceptable in typical family communication. For example, family emotional outbursts may appear out of control to some ethnic groups, but may be the norm for others. In like fashion, the clinician may believe a family to show blunted and defensive expression of affect when a narrow range of emotional expression is common in the cultural background of the patient's family. Whatever the stylistic difference, however, clarity and competent problem solving are necessary elements of successful communication of families in all cultures.



## WORLD VIEW

Many family beliefs and values with respect to child rearing are directly influenced by a family's religion, world view, or philosophy of life ( [Josephson, 2000b](#)). It is important for the clinician to understand the characteristics of a family's world view, an underappreciated component of cultural influence. If this world view is distinctly different from that of the clinician, it is important to have the family explain these aspects of their culture to the clinician, who should respond in an open manner. Joining with the family in a discussion of their world view can clarify some diagnostic issues and potentially increase treatment alliance. On occasion, family therapy may need to be conducted with the aid of someone from the family's culture or religion for the clinician to understand clearly the family milieu that is influencing the developing child.

## ALTERED FORMS OF FAMILY STRUCTURE

In contemporary culture, altered forms of family structure are increasingly common. This includes same-sex parents raising children and multiracial adoptions, with blended families and single-parent families being the two that most frequently affect clinical practice. Blended families refer to a family reconstitution, most commonly after a divorce or the death of a parent. These situations share similarities in that one biological parent is actively removed from day-to-day contact with the child. The key difference is that the divorced parent usually is involved with his or her child's development in an ongoing, albeit intermittent, basis, whereas the absence of a parent through death is an unalterable loss. Consequently, the acceptance of the reality of parental absence is more straightforward, even though the circumstances of death can affect its impact on the family.

When a child's biological parent forms a new union with a stepparent, the family therapist must consider the numerous challenges such families face in realigning their relationships. It is important for the family therapist to understand how the children were prepared for their parent's remarriage, as well as whether the biological parent and the stepparent reviewed the unique needs of each child involved in the transition. Did the biological parent make clear what was expected of his or her new partner? Did the stepparent have the opportunity to express concerns he or she had about the new union and its attendant parenting tasks?

Family therapy of the children of divorce must consider that children, consciously and unconsciously, often long for reunification of the biological parents. This can continue many years after a remarriage and the formation of the new, blended family. Such a persistent desire often explains hostility toward the stepparent that frustrates the development of family harmony.

When the blending of families results in children of two previous families becoming step-siblings, the complexity of interactions increases. Intense affects can be generated by such things as favoritism between a biological parent and a biological child and frank competition between step-siblings and biological siblings for the affection of parents. Practical matters such as arranging visitation schedules and dealing with child support often take a significant amount of family time, particularly if they are associated with ongoing conflict. ( [Visher and Visher, 1988](#)).

Single parents include those previously married and those widowed, as well as those never married. The family therapist must be aware that because the demands of single parenting are significant, there often are surrogate caretakers and other adults who are involved with children. The family therapist must make every effort to inquire whether such individuals exert a significant influence on the child being treated and, if so, they should be included as part of the assessment. It is important for the therapist to consider the single parent's resources, time and financial, the number of children in the household, and the sex of the children. There are unique challenges for the single parent associated with raising a same-sex child and an opposite-sex child ( [Josephson, 1998c](#)).

## Training

There remains debate regarding the appropriate approach to family therapy training for child and adolescent psychiatrists. This debate is embedded in the larger question of how much and what type of psychotherapy training a child and adolescent psychiatrist should receive. An information explosion has inundated child and adolescent psychiatry curricula with significant amounts of new information, typically in the area of neurobiology. This is occurring at the same time that family fragmentation and other social difficulties are stressing children increasingly ( [Achenbach and Howell, 1993](#)).

The classic approach to family therapy training focuses on the trainee learning one approach, or school, thoroughly as a foundation for the subsequent incorporation of new knowledge. The educational premise is that through learning one family therapy approach, the family therapist internalizes the systemic perspective. This approach is likely more effective than residents trying to master several schools of therapy and collecting knowledge of various techniques but acquiring limited conceptual integration. On the other hand, competing schools' claims for uniqueness have given way to consensus on the important areas of family assessment and intervention. It now seems clear that family therapy needs to integrate both systemic and psychodynamic perspectives. The next generation of training in family therapy will likely be more eclectic and pragmatic, and more easily assimilated by child and adolescent psychiatry residents. The ongoing challenge will remain, as it does for any psychosocial treatment, to continue to have family treatment as a vital part of the child and adolescent psychiatrist's therapeutic armamentarium.

## RESEARCH AND FAMILY THERAPY

[Gurman et al. \(1986\)](#) made the direct pronouncement, based on their monumental review of the marital and family therapy literature, that "family therapy no longer needs to justify itself on empirical grounds." Since then, however, there have been further reviews that have indicated gradual and substantive research advances in demonstrating positive family therapy outcomes ( [Asen et al., 1991](#); [Combrinck-Graham, 1990](#); [Diamond, 2001](#); [Diamond et al., 1996](#)).

There remain significant challenges in that many studies have been difficult to assess because they use heterogeneous patient samples, including patients with different diagnoses and at different developmental levels. In addition, studies of family therapy outcome continue to be more frequent in the population of adult psychiatric patients than in the child and adolescent population.

Not surprisingly, the marginalization of family therapy from the field of psychiatry affected family therapy research. The scientific method clashed with the systems approach of many early family therapists. The systems view, that pathology was maintained by recursive feedback loops and did not reside in the patient, rendered traditional methods of research inappropriate for assessing the family. As an example, research that focused on measures of individual patient outcome was discouraged. Over the last several decades, however, new developments in developmental psychopathology research and increased eclecticism in clinical practice, including the use of behavioral and educational approaches in family therapy, have opened up research avenues. Research in family treatment now assesses behavioral approaches, educational approaches, as well as systemic and psychodynamic approaches. As a result, it is increasingly acceptable to use individual patient characteristics as research outcome measures.

There is general consensus that family therapy approaches have been shown to be effective for treating schizophrenia (i.e. adolescent patients), conduct disorder, and substance abuse. There are some data to support its effectiveness in treating eating disorders, although this has been controversial. The extant research literature in the family therapy of externalizing disorders significantly outweighs the literature on the family treatment of internalizing disorders. The family treatment of internalizing disorders, such as anxiety disorders and depressive disorders, shows promise in that family risk factors are quite prominent in these disorders. This discussion of research follows the approach of [Diamond et al. \(1996\)](#), who discussed family risk factors for disorder as a prelude to reviewing the research on outcomes of family treatments for specific disorders.

The rationale for discussion of family risk factors is important. Given the difficulties in family therapy outcome and process research, it is important that epidemiologic research can first establish that family risk factors are, indeed, associated with disorder. Designing family interventions to diminish risk factors is consistent with medical research in disorders of unknown etiology but with known risk factors (e.g., cardiovascular disease). Although the risk factor for a pathologic family process (e.g., parental harshness) may be a focus for family intervention, this does not imply the process is specific to a particular disorder. In addition, it is incumbent on the clinician to maintain a bidirectional perspective, considering that an apparent family risk factor may be a cause, or effect, of the disorder in question.

## Schizophrenia

The interaction patterns of negative attributions about a patient's illness and high levels of parental overinvolvement, hostility, and criticism have been shown to increase rates of relapse of adult schizophrenic patients ( [Vaughn and Leff, 1976](#)) and to predict adolescent onset of schizophrenia ( [Doane et al., 1981](#)). This line of research, originally focusing on adult psychiatric patients in Britain ( [Brown et al., 1972](#)), was followed up by American research by Goldstein et al. in 1978 that

showed that a reduction of expressed emotion during treatment reduced the likelihood of relapse in adolescent patients.

At this point, several psychoeducational approaches have been well studied and shown to be successful. These programs focus on reducing expressed emotion and increasing positive family interaction and coping. Numerous intervention trials in adolescents as well as adults ( [Diamond, 1996](#)) have demonstrated that medication and family intervention provide better prophylaxis against rehospitalization than medication alone. Although questions remain regarding the contribution of family environment to this disorder, there is no longer a question whether family treatment for this population is effective.

### **Conduct Disorder**

Conduct disorder is the child and adolescent psychiatric condition that has the clearest documentation of family risk factors. Numerous reviews ( [Diamond et al., 1996](#); [Kazdin, 1987](#)) have shown that such factors as harsh and inconsistent discipline, marital discord, single parenthood, and parental psychopathology (e.g., substance abuse) have all been linked to conduct disorder. One widely discussed family interactional pattern is that of [Patterson \(1982\)](#), who described the cycle of reciprocal, coercive processes in families that reinforce aggressive child behavior and inconsistent parenting responses.

Parent management training, which teaches parents to be more consistent with their behavioral contingencies, more clear in their communication of behavioral expectations, and more available to nurture their children, has shown positive results. Follow-up studies have found parent management training to have long-term benefits ([McMahon, 1994](#)). Several studies of functional family therapy ([Alexander, 1988](#)) have shown that improvement in family interaction is associated with treatment gains and lower relapse rates in juvenile delinquents. Functional family therapy sees conduct disorder symptoms as maladaptive, yet legitimate in their overall functional significance. In this approach, therapists attempt to “relevance” behavior as a way of decreasing defensiveness and blame by reframing the child's conduct problems as a way of getting his or her needs met. Several variations of family-based treatments of conduct disorder have been shown to be effective, including augmentation with individual and group treatments. Individual treatments have been shown to be more effective when used in conjunction with marital and family interventions ([Dadds et al., 1987](#)).

Family treatments of conduct disorder recently have gone beyond family to include other systems. [Henggeler et al. \(1998\)](#) have extensively studied multisystemic therapy (MST), an intensive family- and community-based treatment that addresses multiple systemic determinants of serious antisocial behavior. The intervention is based on the premise that, in addition to family, conduct-disordered adolescents are involved in a complex network of interconnected systems that includes peers, school, and neighborhood. MST uses a home-based model of service delivery that attempts to improve parenting skills and increase parental emotional resources needed to raise teenage children. Correspondingly, the intervention attempts to empower youth to cope with serious social problems in their neighborhoods. The MST program has demonstrated the reduction in long-term rate of criminal offending in serious juvenile offenders, reduced rates of out-of-home placement, improvement in family functioning, and, most recently, reduction of days in hospital when hospitalization is needed.

### **Attention Deficit/Hyperactivity Disorder**

Family risk factors for attention deficit/hyperactivity disorder (ADHD) often are confounded by the significant comorbid presentation of conduct problems and oppositional defiant disorder. When a child with ADHD has symptoms of oppositional defiant disorder, families typically have greater conflict, anger, and rebelliousness, along with more aversive and negative communications ([Barkley et al., 1992](#)).

Most studies of treatment outcome in the ADHD population focus on pharmacotherapy and behavioral treatments. The National Institutes of Health Consensus Development Conference ([National Institutes of Health, 2000](#)) indicated that pharmacotherapy treatments of ADHD demonstrated better outcome than behavioral approaches. There was some suggestion that combined behavioral and pharmacotherapy approaches had some advantages over sole approaches, but the advantages were deemed modest. This study recommended the need for longer-term studies, beyond 14 months, to assess outcome better. Family therapy has not been formally studied in this population, yet, clearly, any implementation of behavioral paradigms must involve parents. One longitudinal study ([Satterfield et al., 1987](#)) demonstrated that stimulant medication in combination with long-term, multimodal psychosocial treatment had better outcome, with respect to the presence of conduct problems, than did long-term medication treatment alone. These results were maintained at a 9-year follow-up assessment.

### **Substance Abuse**

Adolescent substance abuse is associated with chronic family conflict, parental substance abuse, inadequate parent–child attachment, and coercive, inconsistent parenting ([Liddle and Dakof, 1994](#)). These factors are similar to the family risk factors for conduct disorder, which is not surprising in light of the significant comorbidity of the two conditions.

In widely quoted work, [Stanton and Todd \(1982\)](#) effectively applied structural and strategic family therapy to patients receiving methadone. Since that work, numerous studies ([Diamond et al., 1996](#)) have demonstrated the superiority of brief family treatments over individual and group treatments for reducing drug use. Family treatments had lower attrition rates, and it also has been demonstrated ([Szapocznik et al., 1988](#)) that family treatment approaches significantly increased the likelihood of adolescents attending the first treatment session. A significant development in the empirical assessment of family-based treatments in 1992 was the establishment of the Center For Research on Adolescent Drug Abuse at Temple University (CRADA), which was dedicated to family-based treatment research.

### **Depression**

Four family factors have been repeatedly associated with the onset of a depressive disorder in children and adolescents. These include neglect and poor parent–child attachment, hostility between parent and child, parental psychopathology, and ineffective parenting practices. Although these factors obviously are not specific, and associated with other psychopathology, they are highly predictive of depression ([Beardslee et al., 1996](#); [Sexson et al., 2001](#)).

Treatment studies of depressed youth continue to be relatively sparse. Few have targeted family relationships as primary treatment foci ([Brent et al., 1997](#)). This appears likely to change because several innovative approaches in the family therapy for the treatment of depressed adolescents are evolving ([Diamond and Siqueland, 1995](#)).

### **Anxiety Disorders**

Family risk factors for anxiety disorders are not as clearly delineated, and treatment studies on anxiety also are lacking. Current studies suggest that a parent with an anxiety disorder increases the likelihood that his or her offspring will also have a similar disorder. Parental anxieties toward fear-producing situations appear to be communicated to the child in explicit or implicit ways ([King and Noshpitz, 1991](#)). An epidemiologic study suggests that family difficulties in the areas of role performance, values, and norms were identified in the families of school-refusing, anxious children ([Bernstein et al., 1990](#)). Family risk factors appear to covary for both anxious and depressed children.

Family-based treatment research is focused on parents administering cognitively based interventions for the treatment of fears and general anxiety. Although cognitive–behavioral treatment approaches have gained ascendance, one study suggests that when family intervention is coupled with cognitive–behavioral treatment, treatment effectiveness is enhanced ([Barrett et al, 1993](#)).

### **Eating Disorders: Anorexia Nervosa and Bulimia Nervosa**

[Humphrey \(1994\)](#) has differentiated subgroups of families in which a child has an eating disorder. A review of these studies suggests that families with a bulimic child, or a child with both bulimic and anorectic symptoms, show more hostility, chaos, isolation, and impulse disorders. Families of children who have only the symptoms of anorexia nervosa demonstrate less externalizing behaviors, yet show evidence of rigidity, dependency, and emotional restrictiveness.

Family therapy has, for some time, been seen as a core component in the treatment of eating disorders ([Lange et al., 1993](#)). This tradition was stimulated by research on the use of structural family therapy by [Minuchin and colleagues \(1975\)](#) in the treatment of families of anorectic patients and other “psychosomatic families.” Subsequent studies have not confirmed a specific anorexigenic family structure ([Ravenscroft, 1996](#)). There continues to be evidence, however, that family factors do influence the emergence and maintenance of eating disorders and that a flexible treatment approach, using family treatment with other modalities, is clearly indicated and likely effective. [Russell et al. \(1987\)](#) found, and confirmed in 5-year follow-up ([Russell et al., 1994](#)), that family therapy produced better maintenance of weight gains and improved menstrual functioning for adolescents when clinical problems had developed within 3 years of the initiation of treatment. Russell et al. found that



older patients improved more from individual therapy compared with family therapy. The assessment and treatment of family interaction continues to be important for patients with eating disorders, particularly younger patients.

## Metaanalytic Reviews

[Diamond \(1996\)](#) has reported on three metaanalytic studies of family therapy. Metaanalysis is a statistical method by which results from several different studies are standardized and then combined to yield an average effect size. The three studies reported by Diamond suggested that family-based therapies were as effective as other models of intervention. This review suggested that more valid estimates of efficacy can be derived only from larger samples of well designed studies. Several problems exist in this methodology, including the overrepresentation of externalizing disorders in family metaanalytic studies.

## Family Treatment Process Research

Since 1990, family process research has made modest, but steady, advances. In contrast to outcome studies that determine the results of an overall treatment intervention, process research goes into greater detail on how specific processes of therapy contribute to outcome. A key concern of process research is the study of patterns of patient–therapist interaction that lead to intrasession change. This methodology attempts to operationalize and validate basic techniques of family therapy. Techniques studied have included assessing the effects of cross-generational coalitions between parent and child; reframing; contributing to a more positive attributional set about a problem; and paradoxical interventions. Clearly, mapping in session therapy sequences and tying them to outcome represents an advance in the ability to understand the mechanisms of change in family therapy ([Diamond, 1996](#)).

In summary, there is an evolving empirical tradition in family treatments. Outcome studies suggest positive results when family therapy is used in conduct disorders, schizophrenia, and substance abuse, with encouraging findings in eating disorders. Based on family risk research, there appears to be potential in using family therapy approaches for the internalizing disorders. Research in family therapy will benefit from (a) replications of previous studies; (b) more studies in the area of internalizing disorders such as anxiety and depression; (c) treatment models developed from basic psychopathology and process research; (d) increased development of reliable, valid, and treatment-sensitive family assessment tools; (e) assessment of treatment models in community (i.e. “real world”) settings; and (f) focus on parental psychopathology and marital conflict ([Diamond, 2001](#); [Diamond et al., 1996](#)).

## THE FUTURE

The future of family therapy remains bright, while at the same time the field faces numerous challenges. Family therapy will always maintain an intuitive advantage compared with other psychosocial therapies. Simply, without families children would not exist. Parents create children and either facilitate their journey into adulthood or frustrate this natural developmental passage. As our science proceeds and child and adolescent psychiatrists can discern when, and how, to intervene more effectively with families, children will benefit. Yet, there remain limitations to implementing family therapy effectively in today’s child and adolescent psychiatry.

Systems-oriented clinicians still are reluctant to accept current psychiatric diagnostic nomenclature. Although the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* includes Axis IV, delineating psychosocial problems, this has not satisfied many family therapists. In the absence of a relational disorders classification, the existing nomenclature still describes disorders in an individual, contrary to the basic systems postulate that symptoms do not reside in individuals but result from family context. There is hope that this controversy will recede as integrated approaches to treatment emphasize the compatibility of individual dynamic, developmental, and systems approaches.

The explosive development of the neurosciences has broadened the diagnostic and treatment capability of the contemporary child and adolescent psychiatrist. Contrarily, its emphasis has obscured the clinical reality that most disorders result from a combination of biological, psychological, and social vulnerabilities. Psychosocial variables can protect children from expressing disorders with biological contributions, whereas psychosocial stresses can foster the clinical expression of a biological vulnerability. The clinical correlate of this fact is that families have strengths and weaknesses with broad implications for family therapy. Many psychiatric training programs are having difficulty conveying this broad picture of psychiatry.

If the revolution in neurosciences was not enough, an economic revolution in health care has added to the challenges of the family-oriented clinician. Many third-party payors resist reimbursing for family therapy because it is seen as psychosocial support and not a defined medical treatment. The changes in health care delivery greatly influence the characteristics of psychiatric treatment and the roles of the respective mental health disciplines. It is not uncommon for the child and adolescent psychiatrist to be reimbursed solely for providing biological treatments and the nonmedical therapist for providing psychosocial treatments, such as family therapy.

Within a generation, the definition of the family has shifted to the point where it is often a matter of contentious debate. It has become difficult to enter into a rational discourse about the family, even if it includes factual data, without offending some constituency. Are there better ways to raise children than others? Are some forms of family structure healthier than others? These straightforward questions can become inflammatory and difficult to discuss, even in some academic settings. What does seem clear is that the developmental needs of the children have not changed. The family of the future, however defined, must continue to meet these developmental needs.

## CONCLUSION

Any brief review of family therapy in child and adolescent psychiatry only scratches the surface. This chapter has reviewed historical antecedents, current status of the field, a review of basic clinical processes and issues, a discussion of the disorders that appear to be helped most directly by family therapy, and a summary of future challenges. There is much yet to be learned. Working with the family of the child or adolescent psychiatric patient is of critical relevance. Child and adolescent psychiatrists remain mental health practitioners with a broad perspective on working with individuals at all stages of development and at all levels of the biopsychosocial continuum. The family is at the heart of a developmental, biopsychosocial perspective that remains the core of our field.

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\*Case illustration reprinted with permission from Josephson A: Family therapy. In: Sadock B, Sadock V (eds): *Comprehensive Textbook of Psychiatry*, 7th ed. Philadelphia, Lippincott Williams & Wilkins, 2000, pp. 2821–2831.

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## 85 PARENT WORK

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The purpose of this chapter is to describe psychotherapeutic work with parents as a means of helping the child who is being seen in psychiatric outpatient settings. The discussion begins with the rationale for engaging parents in the child's treatment, proceeds to issues to be considered in the assessment evaluation processes, and reports on primarily evidence-based interventions with parents relevant to the child's difficulties. Parent work is described here, using multiple program examples, as an integrated model of therapy that uses many approaches, including cognitive-behavioral and psychodynamic methods, as determined by the needs of the child and family.

Parent work rests on a model that is child centered and family focused ([Stroul and Friedman, 1996](#)). This implies that the types and combination of services provided must be driven by the needs of the child and his or her family. It also means a commitment to adapting the services to suit the child and family, not the other way around. Also implicit is providing services to the child within the context of the family and maintaining the integrity of the family wherever possible. Children must not be served by a bifurcated system, one that separates them from the significant adults in their life. In most cases, parents are primary caregivers, and according to the system of care philosophy they must be supported, assisted, and involved in every decision regarding service delivery ([Stroul and Friedman, 1996](#)). Professionals and families need to work together in relationships of respect and mutual support in all aspects of planning, program development, service delivery, and evaluation. Families need to have the lead voice, as well as choice, in decisions regarding treatment plans for their children. Our charge is to operationalize this inclusive model in both training and practice.

[Jones \(1999\)](#) tracked the evolution of the child guidance movement from its roots in the Juvenile Psychopathic Institute in the last decade of the 19th century to its expansion in the 1920s and 1930s and into the present. This movement was characterized by an ecologic perspective that focused on the role of the family and society in the child's development. However, Jones proposes that along with this growth, problems emerged that propelled the field of child mental health into a problematic situation, namely an erosion of the team concept of psychiatrist, social worker, and psychologist in which psychotherapy was in the domain of any of the three disciplines. Jones argued that the role of society and family in the practice of child mental health and such factors as culture, economics, and education have not received enough attention. The lack of attention in training programs given to working with parents also has been emphasized by [Jellinek \(1986\)](#). The early philosophy of the child guidance movement was reintroduced in September 2000 when the Surgeon General proposed a paradigm shift—in effect, a return to an ecologic approach in the delivery of child mental health services ([U.S. Public Health Service, 1999](#)). A federal initiative, The Child and Adolescent Service System Program (CASSP), has, in fact, for over a decade tried to implement such a model in the service system ([Stroul and Friedman, 1996](#)). We believe it is important to integrate both the medical model, with its contribution to diagnostic understanding of a problem, with the ecologic model, which supports the team approach and recognizes the multiple factors in a child's life.

The same social factors that gave impetus to the child guidance model in the 1920s, when parents were in a quandary over how to deal with their “flapper”-influenced children, is mirrored today as our society confronts a mobile lifestyle and varied family structure with few precedents to offer guidance, support, or preparation. The rationale for working with parents should be obvious. However, the diminishment of the team approach and the ecologic model alluded to earlier, as well as the limitations set by managed care, have created barriers to engaging parents in child treatment.

### RATIONALE FOR PARENT WORK

Poor parenting can create, maintain, and exacerbate child behavioral problems through a number of negative patterns (see also [Chapter 86](#)) and negative reinforcement. [Patterson \(1982\)](#) described a “cycle of reciprocal coercion” whereby parents inadvertently encourage bad behavior. In turn, difficult children can place heavy burdens on parents and families.

Child behavior and parents' relationships with their children are partially rooted in the affective life and in the parents' unconscious issues and conflicts ([Freud, 1960](#)). Because of the tendency of humans to replicate behavior patterns unconsciously over time, the parent-child relationship often becomes the arena in which parents reenact with their children aspects of relationships with their own parents ([Fraiberg, 1954](#)). In theory, once the parents' intrapsychic issues and conflicts are identified, worked through, and resolved at some higher level of integration, the child should be less burdened with the parent's emotional legacies.

Hence, parent work that is psychodynamically informed relies primarily on principles that encourage parents to understand the ways in which they have repeated their own childhood experiences in patterns of behavior as adults, and especially where they have consciously or unconsciously reenacted these experiences with their child. In practice, parent work rests on the assumption of the validity of psychodynamic, behavioral, and cognitive theories. Thus, prerequisites for doing parent work are skills in individual, marital, family, and group interventions based on these theories, as well as knowledge of child and adult development. Awareness of indications for psychoanalysis, psychopharmacologic therapy, and psychological testing also is required for purposes of referral. In addition, clinicians need to be familiar with services provided by schools, pediatricians, day care centers, and other community agencies that touch the lives of the child and his or her family.

### ASSESSMENT OF PARENTAL INFLUENCES

Before determining what type of intervention is needed, an assessment of the parent should be completed. This assessment should include (a) the nature of the parents' difficulties with the child, (b) individual attributes of each parent, and (c) motivation for treatment. A guideline for the assessment of the parent is shown in [Table 85.1](#).

<b>Reason for referral</b>
By whom, and for what reason
Parents' concerns, their "diagnosis," and their prediction of the outcome
<b>Family history</b>
Family constellation: identifying essential descriptive data regarding members of the household such as age, employment, cultural and religious affiliations, living conditions
Parent presentation: impressions, including relevant physical presentation, degree of comfort or anxiety in interview situation, affective presentation, relative contribution of each parent, agreement or disagreement in describing the child
Individual history of each parent
Marital history: Describe adjustment in current marriage/ previous marriages as significant
<b>Family style</b>
Summary of personal, social, environmental, academic, psychiatric, and medical history: relevant peer and family relationships, should include strengths and weaknesses
Discipline style of parents: How does the parents' (co)parental perspective compare with the child and siblings (if any) and with parents when they were child's age?
Developmental history: Significant history of pregnancy, labor and delivery, infancy, sleeping, eating, talking, motor development, language, play interests, attitudes and fears, aggression, relationships
Medical history/psychological testing
Medical/psychiatric history of child as relevant
Genetic consultation of family
Recommendations/disposition (indicate if recommendation accepted)

**Table 85.1. Parent Evaluation Outline**

The complete family history is indispensable for understanding the complexities of the child and his or her family. In the family constellation, it is important to know who are the people in the life of the child. Their age, cultural and religious influences, employment, education, socioeconomic status, and living conditions need to be explored.

The marital history and individual history of each parent are key components of the parent assessment. In taking a history, the clinician often learns that an adult's relationship with his or her child mirrors the one he or she had with a parental figure. The interviewer may find that the child and his or her siblings are replicating the marital conflict. During history taking, dynamics such as repetition compulsion and primitive defenses (e.g., projective identification), which are having a deleterious effect on the child, may be revealed. The parents' own histories influence how each of them cope with a child who is handicapped with pervasive developmental disorder, autism, Tourette's disorder, retardation, and other serious impairments (Lefley, 1987; McLoughlin, 1992). The family style, including the degree of affection and support available to family members, the degree of conflict and anger in the family, and how conflict and anger are managed, is crucial to assess. Whether the child is reacting to parental/family dynamics or bears his or her own serious mental illness, the individual history of each parent, the marital history, and the family style all are crucial elements in understanding the child in the context of the family.

As we have mentioned, possibly the most important issue to explore when evaluating parents is whether they appear to be projecting their own conscious or unconscious conflicts onto the child or are in some other way using the child as a vehicle through which they reenact an aspect of their own emotional histories or current life problems. If so, it must be further determined whether this conflict is limited to a relatively small area of the relationship or if it is all-inclusive. "Good enough" parents can identify and maintain parental, marital, and generational boundaries. Sometimes the parents' difficulties in maintaining such boundaries are part of a characterologic problem. In this case, individual psychotherapy may be indicated for the parent.

If the assessment determines that the parent is ready to continue and if the situation is a suitable one for outpatient clinic treatment, regular, usually weekly or semiweekly, appointments with both parent and child are scheduled. Optimally, although it is not always possible, the child should see his or her own therapist, while the parents work simultaneously with their therapist. The process that takes place is referred to as *parent work* rather than *parent guidance*, *parent training*, *counseling*, *parent psychotherapy*, or some other denominator because these other terms are restrictive and usually are subsets of the larger domain of the interventions used with parents.

## SPECTRUM OF INTERVENTIONS IN PARENT WORK

The services offered in the course of parent work after the evaluation are selected from a spectrum of potential interventions (Armbruster and Fallon, 1994). It is not unusual to use a psychodynamic approach with one set of parents, and to use a cognitive-behavioral approach with another, or both, during the course of a given treatment. Even among that group of parents with whom the psychodynamic model is appropriate, the clinician might determine that marital therapy (Scarf, 1987) is the best way of treating one couple, whereas in the case of another family, she or he might make the assessment that intensive psychodynamic psychotherapy with only one member of the marital dyad is the intervention that is most likely to be effective (Chethick, 1976, 1989; Fromm-Reichmann, 1950). The goal is to free the child from the conflict that has been displaced from the marital to the parent-child relationship. If, in the course of this work, an issue arises that suggests a more educative or informational approach, the work can shift from psychotherapy to parent guidance. In Hollis and Woods' (1981) topology, a dynamic intervention shifts to a didactic one. If parents do not have the capacity for reflection and their observing ego functioning is limited, a cognitive-behavioral approach may be the most useful. However, the cognitive-behavioral approach has been shown to be very successful for working with a variety of parents of children with externalizing behaviors (Kazdin, 1997a; McMahan, 1999). The parent work may rest with such approaches or return to a more insight-oriented intervention. The clinician also needs to interact with others in the child's environment, such as noncustodial parents, stepparents, relatives, teachers, and other school personnel.

The flexible, selective use of different, clinically indicated treatment modalities characterizes parent work. These modalities can vary in depth, intensity, and duration, depending on the internal or external situation of the parent at a particular time. This spectrum of intervention includes but is not limited to parent guidance, supportive therapy, and psychoanalysis. Marital, individual, group, and family therapy also may be used, depending on the clinical issue. At times, the parents may be seen exclusively or more frequently than the child. This is true particularly if parents have unresolved issues from their own childhood or mental health problems, or need help in structuring the home environment and disciplining the child.

To determine an appropriate treatment modality, the goal of the parent work always must be kept in mind: to help the child by helping the parent. The challenge of parent work is to facilitate parents' understanding of their relationship with their child and to help them achieve some resolution of their own conflicts manifested in their behavior, especially when these behaviors are specific to the interactions with the child (Armbruster and Fallon, 1994).

## TREATMENT CHOICES FOR SPECIFIC DISORDERS IN CHILDREN

### Profusion of Treatment Choices

There are over 200 different treatment approaches in child and adolescent therapy (Kazdin, 1997). However, as mentioned earlier, it is common for therapists to integrate different interventions in response to the needs of parents and their children at different stages in the treatment process (Armbruster and Fallon, 1994; Diamond, 1996; Sargent, 1997). Because it is unlikely that any single method will be able to solve the multifactorial problems that have emerged in today's social climate, parent work may be used in combination with individual, family, or group therapy. In the following text, examples of successful intervention for parents whose children present with a variety of disorders are discussed. Although many of the interventions presented demonstrate positive outcomes for child symptoms, before describing them there are some common limitations of research that need highlighting:

- Few studies investigate the impact of parent outcome on child treatment, and much needs to be learned about how such investigations should be done.
- Little is known about the processes by which change occurs. For example, when treatment groups are compared to wait-list control subjects, is change due to regularly meeting with a therapist, to specific intervention, or to the developmental process?
- Sampling bias in effectiveness studies may affect outcome where subjects are not randomly allocated and there often is a high rate of attrition before treatment begins.
- Positive outcomes may be partly due to more motivated parents remaining in treatment.
- Most studies have short-term or no follow-up to treatment because of the difficulty of finding long-term funding for projects. Therefore, it is not possible to determine the longer-term gains of intervention.
- Few efficacy studies demonstrate clinical significance or generalizability to real-world contexts.
- Studies rarely address which treatments do not work.

### Developmental Issues

In working with parents, it is critical that the developmental context of the child be at the forefront of helping the parent understand the child. Parents' expectations



often are unrealistic given the child's age. Understanding the child's developmental stages may mitigate a potentially complicated situation leading to parent–child discord. In such situations, the child may feel inappropriately pressured and develop poor self-esteem, and the parents may be chronically disappointed. These feelings are expressed by anger on both sides. Frequently, both didactic and insight approaches are useful in such situations because a parent may have been the recipient of unrealistic expectations and be perpetuating his or her experience. In our experience, the most effective treatment is when two clinicians are engaged, one for the child and one for the parent. However, as stated earlier, specialization and managed care have made this unrealistic in most settings. At present, one clinician usually sees both parent and child. However, in the case of the adolescent, the parent in most instances is seen by a separate therapist, except when family therapy is indicated.

### Cultural Context

In our increasingly diverse society, developmental phases vary in different cultures. Clinicians need to be sensitive to such issues and not attempt to impose the biases derived from their own experience and training onto families where cultural norms may be antithetical to the therapist's perceptions and knowledge base. This is particularly true for immigrant families from traditional societies, as well as for subcultures outside the American mainstream, in which child-rearing practices and parental expectations may be very different from the clinician's. In the past, waves of immigrant families tried to assimilate as quickly as possible. However, this is no longer true, and various cultures are encouraged to maintain their practices rather than relinquish them. For example, our emphasis on separation and individuation in adolescents is particularly perplexing for families where the extended family is housed in a single residence and where adult children are expected to live until married or even have their spouse move into the parental home. The same is true for corporal punishment, such as spanking. Rather than confront a family head on, the therapist might explore (a) the parent's upbringing, (b) the family's cultural norms, and (c) whether such punishment has been effective. Often this type of discipline has not corrected the problem and the clinician may be given an opportunity to segue into alternative disciplinary approaches. (Obviously, in the case of injury to the child the clinician as a mandated reporter must contact the appropriate state child protection agency.) In this era of diversity, it is particularly important that the cultural as well as socioeconomic contexts be considered and respected.

After this background, specific behavior and emotional disorders associated with parent treatments now are discussed.

### Behavioral Problems and Disorders

Family characteristics that are associated with conduct problems in children are similar to the characteristics that predict premature dropout or treatment failure. These characteristics include low income, low educational attainment, teenage pregnancy, isolation, high stress, single parenthood, parental psychopathology, criminal history, substance misuse, marital discord, and depression ([Webster-Stratton, 1998](#)). Children who are vulnerable to conduct problems typically have academic problems, poor social skills, and poor problem-solving skills ([Webster-Stratton, 1998](#)). Identification of these risk factors helps in designing preventative strategies in vulnerable populations.

Although parent behaviors alone do not cause child psychopathology, parents can reinforce and exacerbate overt conduct disturbances in their children through their maladaptive interactions ([Diamond, 1996](#)). Harmful parenting styles such as inconsistent discipline, physical abuse, excessive criticism, hostility, disengagement from school, and lack of stimulation are all correlated with behavioral problems in children ([Webster-Stratton, 1998](#)). In [Woodward and colleagues' \(1998\)](#) study, after controlling for conduct disorder (CD) and parental psychopathology, the strongest predictor of hyperactivity was disciplinary aggression by a parent.

Children with behavior problems place particular strains on parents ([Diamond, 1996](#)). For example, boys with hyperactivity were found to be less compliant and more off task, negative, attention seeking, talkative, and demanding of parents than control children ([Woodward et al., 1998](#)). Parents of children with overt conduct problems experience greater stress, see themselves as less skilled and knowledgeable, and are at risk for depression, other personal distress, and marital discord ([Anastopoulos et al., 1993](#)).

Most outpatient psychiatric clinic referrals are for externalized behavior such as attention deficit/hyperactivity disorder (ADHD), CD, and oppositional defiant disorder (ODD). Externalized behaviors are referred more often for treatment because children tend to be referred for treatment of behavior that is problematic for others as well as themselves ([Kazdin and Weisz, 1998](#)). As a result, work with parents that has received considerable attention over the past few decades has focused on teaching parents particular procedures that modify interactions with their child, with the goal of encouraging positive behavior and reducing deviant behavior of the child ([McMahon, 1999](#)). This cognitive–behavioral approach is referred to as *parent training* (PT), and it is used preeminently in the treatment of preadolescent children who exhibit overt conduct problems, the arena in which it has the most empirical support ([McMahon, 1999](#)).

Parent training programs use social learning principles to develop positive prosocial behaviors and decrease deviant behavior through positive reinforcement. The primary theoretical bases are operant conditioning ([Skinner, 1938](#)) and research of parent discipline practices ([Patterson, 1982](#)). There are numerous studies of parent management training programs (PMT) or PT using behavioral approaches for use with children with CD, ODD, and ADHD ([Kazdin, 1997a](#)). Examples include:

- A program for 34 children with ADHD aged 6 to 11 years ([Anastopoulos et al., 1993](#)). Children and parents in this program showed improvements in symptoms and functioning (reduced stress and increased self-esteem) in contrast to a wait-list control group that showed no improvement.
- A parenting program targeting behavioral problems in children 3 to 8 years of age ([Scott, 1998](#)). The program had three parts: teaching techniques to increase desired behavior; reducing unwanted behavior; and developing strategies for avoiding troublesome situations.
- [Webster-Stratton \(1989\)](#) conducted many studies to examine her PT program for children with CD. Variations of the program (for 3- to 8-year-olds) include videotape modeling and group discussion ([Webster-Stratton, 1989](#)). Positive outcomes were demonstrated in a study designed to strengthen the protective factors in a population vulnerable to CD. With a sample of 394 Head Start mothers, interventions in the experimental group focused on parenting competence, child social competence, and home–school connections. There were positive outcomes for the first and second measures, but not for the third. The study demonstrates that low-income parents can be engaged in and benefit from parenting programs ([Webster-Stratton, 1998](#)).
- [Pfiffner and McBurnett \(1997\)](#) measured the outcome of a parent generalization-training component, which was added to a social skills training (SST) program for 27 parents of children 8 to 10 years of age with ADHD. These parents were trained to support their children's transfer of social skills from the program to everyday use. A different group of similar parents received SST alone, and there was a wait-list control group. The results showed that both groups receiving SST improved compared with the control subjects, but that the PT component did not yield additional gains.

Although these studies are not conclusive, they demonstrate clinically significant improvements achieved by PMT and PT approaches on a wide range of measures, often bringing half of the sample into the normative range. Gains were maintained 1 to 3 years after treatment in most cases, and the impact was found to generalize to other areas such as sibling behavior and marital relationships ([Diamond, 1996](#); [Kazdin and Weisz, 1998](#)).

Studies also have attempted to delineate the important mechanisms of support for parents of conduct-disordered children. [Prinz and Miller \(1994\)](#) focused on opportunities for parents to discuss nonbehavior management problems such as job stress, health problems, family disputes, and other personal worries in a group setting. In a comparison of parent groups, those in the experimental group (“enhanced family therapy”) had a lower dropout rate, suggesting that the additional support may have helped them cope more effectively with the challenges of parenting a conduct-disordered child as well as other environmental stressors affecting the family. In globalizing this study, a need is identified for parents of children with CD to receive help with problems other than child management.

Another option in addressing this parental need is multisystemic therapy, which focuses on interrelated systems and how they affect each other. This form of therapy has been used with delinquent youths, resulting in reduced delinquency, decreased emotional and behavioral problems, and improved family functioning ([Henggeler et al., 1998](#)).

[Scott \(1998\)](#) advocates a combination of PT and emotional support for parents of children with behavioral problems because these families are more likely to be economically disadvantaged and in need of assistance with related financial, employment, and housing problems. He stresses that didactic work is unlikely to achieve positive outcomes without addressing other needs of parents, and general counseling by itself may not produce significant changes in behavior.

A prevention study in London evaluated the efficacy of increased parent support by front-line welfare workers. [Davis and Spurr \(1998\)](#) used health visitors and clinical medical officers to provide counseling to 55 parents of preschool children in a deprived area. The outcomes were compared with a control group that received standard community services. Parents who had received additional counseling on top of normal services showed increased self-esteem, decreased stress and emotional difficulties, more positive attitudes toward their children, improvements in their home environment, and decreased child behavior problems. However, many of the families referred never engaged with the service, so there may be sampling bias.

[Dadds and McHugh \(1992\)](#) designed a study aiming to improve parenting and child conduct behavior symptoms. Twenty-two single parents were assigned to one of two groups: The experimental group received parent training plus “ally support training” (where a named person agrees to offer support) and the control group received only parent training. Although having an ally did not produce significant results, the importance of support was demonstrated by the fact that maternal perception of a high level of available social support from friends was the best predictor of positive response to treatment.

Before improvements in parenting can be made through parent interventions, the engagement of the family in treatment is the first task of clinicians. A study of suicidal adolescents ([King et al., 1997](#)) showed low compliance with treatments involving parent guidance and family therapy compared with pharmacologic treatment and individual therapy. The difference in compliance was explained by the practical difficulty of mobilizing a family, resistance to the idea that the family is implicated in the problem, and perceptions about the relative effectiveness of different treatments. The study found that the inclusion of a family educational component assists in the engagement of parents in treatment.

In conclusion, engaging parents of behaviorally disordered children often is critical to addressing the child's difficulties and improving his or her functioning. Often the child is responding to problematic issues in the family. However, a variety of interventions exist, and it is the clinician's responsibility to access the most effective in a particular case.

As the following discussion of parent interventions for disorders other than behavioral ones illustrates, similarly effective results have occurred when parents have been actively enlisted in the treatment.

### **Mood Disorders**

A study on parental intervention for mood-disordered children used a 90-minute psychoeducational workshop to discuss mood disorder symptoms; etiology, course, and prognosis; and treatment and family factors that affect outcome ([Fristad et al., 1998](#)). The Understanding Mood Disorders Checklist was administered before and immediately after the workshop. Results indicated that parents' knowledge of mood disorders increased significantly after the workshop. This increase was greater for the fathers, whose lower baseline knowledge before the workshop progressed so that it equaled that of the mother. Further, the Expressed Emotion Adjective Checklist was administered before and 4 months after the workshop. This revealed improved expressed emotion scores in parents, who also reported decreased negative and increased positive emotions. Again, fathers' scores were raised to levels similar to those of mothers' after the workshop. The authors suggested that these two results potentially could improve the home environment by providing consistency in parenting. In this case, educating parents about mood disorders was helpful; parents were less vulnerable to perceiving their child's mood as a reflection of their poor interaction with their child and were less likely to blame themselves or feel diminished as parent. When parents feel inadequate, a negative cycle of interaction often is created between parents and child, with deleterious effects for both.

Interpersonal psychotherapy for depressed adolescents (IPT-A) is a brief, time-limited intervention based on the premise that no matter what the underlying cause, the depression is “inextricably intertwined” with interpersonal relationships ([Mufson et al., 1993](#), p. 3). IPT-A, which has been shown to be effective in various empirical studies for treating adolescent depression, brings into treatment the parents and siblings of the adolescent for either support or for direct intervention to change family patterns, which in turn often are replicated in relationships outside the family ([Mufson et al., 1993](#)).

### **Dissociative Disorder**

Parental involvement in the study of treatment of children with dissociative disorder was the most significant factor in a positive outcome ([Silberg and Walters, 1996](#)). Factors associated with treatment outcomes in 34 children (3 to 14 years of age) with dissociative identity disorder who were treated in two geographically different settings by the same three-phase treatment approach were examined. Dissociative children living with dissociative-disordered parents had outcomes just as positive as those in nondissociative outcomes.

### **Anorexia Nervosa**

A recent study was carried out to compare the efficacy of behavioral family systems therapy (parents are seen conjointly with the child) with that of ego-oriented individual therapy (parents are seen collaterally) as treatment for adolescents with anorexia nervosa ([Robin et al., 1999](#)). The results showed that although both treatments were effective, the behavioral family systems therapy produced a faster return to health. The results indicate the importance of parental involvement in the treatment of young adolescents with anorexia nervosa, but does not require all therapy to include conjoint child and family sessions. This intervention emphasized the premise that anorexia nervosa needed to be seen in the context of family, not simply focused on the child.

### **Pediatric Traumatic Brain Injury**

Behavioral dysfunction is a commonly reported outcome of pediatric traumatic brain injury (Knight et al., 1991 in [Kinsella et al., 1999](#)). This study assessed the behavioral outcome of a sample of children with traumatic brain injury up to 2 years after the initial incident. One finding of the study was that acute emotional reaction of the parents to the injury was predictive of child behavioral outcome (although not after 2 years), which suggests that the parents' coping resources may affect child behavior after the injury. The authors believe this emphasizes the role of the family in the child's response and the importance of supportive interventions. Again, evidence of the ecologic perspective emerges, along with the need to intervene in the context of the family system.

### **Autism**

When the design for behavior modification in the treatment of autism included parents as members of the treatment team, children showed higher IQ scores and rates of class placement in school, and the effects were maintained for a mean of 5 years (Lovaas, 1987 in [Kazdin and Weisz, 1998](#)). Here, again, the inclusion of parents in the child's treatment appears as a key factor in improved outcome.

In summary, the engagement of parents is a critical component of a multifaceted approach in the treatment of children with a variety of diagnoses. We have described an armamentarium of interventions for specific diagnoses; it is the clinician's task to assess the efficacy of the treatment for the particular child and family. Although diagnoses are an important aspect in the determination of interventions, consideration also must be given to parents. Given all the variables that exist in the ecology of the family, the child, his or her parents, and perhaps others, the clinician's training, skill, and experience are deciding factors in selecting the intervention in working with the parents that will have the most positive impact on the child. In the assessment of the parents, it is critical to identify both strengths and deficits, for the intervention selected may rely more on the parents' positive attributes than on their weaknesses.

## **TREATMENT CHOICES WHEN PARENTS HAVE SPECIFIC DISORDERS**

### **Pathogenesis**

Parents with their own psychiatric diagnoses introduce another level of complexity in both assessment and treatment. Children of psychiatrically ill as well as substance-abusing parents have an increased risk of psychopathology ([Andrews et al., 1990](#); [Beardslee et al., 1998](#); [Cummings and Davies, 1994](#); [Gondoli and Silverberg, 1997](#); [Hill and Muka, 1996](#); [Inoff-Germaine et al., 1997](#); [Klimes-Dougan et al., 1999](#); [Kuperman et al., 1999](#); [Lundy et al., 1997](#); [Warner et al., 1999](#); [Williams, 1998](#)). The transmission of mental illness from parent to child depends on a variety of mediating factors that are biological, psychosocial, and environmental. Although early studies examined the impact of maternal disorders on the development of the infant, current evidence emphasizes the risk to children and adolescents of all ages resulting from mental illness in either or both parents (biological or custodial) ([Hill and Muka, 1996](#); [Lundy et al., 1997](#)). Increasing evidence indicates that major depression runs in families and that children of depressed parents are at risk for this illness ([Beardslee et al., 1983](#); [Hammen et al., 1990](#); [Orvaschel, 1990](#); [Weissman et al., 1984, 1987](#)). Family, twin, and adoptive studies have indicated that there is a genetic connection between affective disorders in parents and their children ([Beardslee et al., 1998](#); [Cadoret, 1978](#); [Torgerson, 1986](#)). However, the link is stronger for bipolar than unipolar depression, and neither case undermines the important role of environmental factors in transmission ([Beardslee et al., 1998](#)).

[Beardslee and colleagues' \(1998\)](#) review of studies revealed that a child with an affectively ill parent has a 40% chance of experiencing an episode of major depression by the age of 20 years and is more likely to exhibit general difficulties in functioning, increased guilt, interpersonal difficulties, and attachment problems.



According to [Weissman and colleagues \(1987\)](#), children of depressed parents are at threefold increased risk for school problems and suicidal behavior, and have an increased prevalence rate for substance abuse and poor social functioning.

Conflict between parents has been identified as an important linking mechanism between parental and child psychopathology. Studies support the associations between high marital conflict and poor adjustment in toddlers and distressed families predicting child behavior problems ([Abidin et al., 1992](#); [Armbruster et al., 1996](#); Reid and Crisafulli, 1992). A “disturbed family system” will “likely take an early toll on the child”; conversely, problematic children often increase stress within families ([Donenberg and Baker, 1993](#)). [Cummings and Davies \(1994\)](#) observe that psychiatric illness may cause or be caused by marital problems, or that external stressors such as poverty may precipitate both. Children may be affected both by the observation of conflict and by impaired child management practices. Further, marital problems may decrease the parents' ability to provide the support that may have helped the child cope with the parental disorder ([Beardslee et al., 1998](#)). [Armbruster and colleagues \(1996\)](#) found marital conflict rather than divorce had a negative impact on children's behavior.

### **Impact of Parental Mental Illness on Children**

Associating with someone with mental illness is stressful for most people. However, having a parent whose illness may prevent him or her from providing nurturing, stability, structure, and consistency is particularly difficult for a child. Children may blame themselves for the parent's behavior and feel that they caused their situation, and as a result may suffer guilt and a poor self-image. Children need predictability; a mentally ill parent's erratic and emotionally labile behavior may result in extremely poor parenting skills, all of which may have a deleterious impact on the child's development. Studies suggest that the risk for children increases proportionately with the duration of the parent's disorder, the number of episodes, and their severity ([Hammen et al., 1990](#) in [Beardslee et al., 1998](#)).

The following studies illustrate the impact of psychiatrically impaired parents on children.

[Williams' \(1998\)](#) study of adult daughters describes the emotional trauma resulting from their experience as children of mentally ill mothers. The subjects remembered their mother's “disappearance” on admission to the hospital, parentification, and deficits in social skills. Common themes raised were hatred of themselves and their mothers, lack of support from their extended families, isolation, and envy of those with normal families. All children had current psychiatric diagnoses.

Another study of 20 mother–child dyads found a strong correlation between maternal depression and behaviors in children ([Lundy et al., 1997](#)). A link between ineffective discipline and externalizing behaviors in 6- to 11-year-olds was identified, and children with both internalized and externalized disorders showed fewer positive behaviors when their mothers were depressed.

[Inoff-Germaine et al. \(1997\)](#) examined how parental affective illness was related to family, dyadic, and individual functioning. Compared with 18 control families, the family functioning in the 41 experimental families with affective illness was limited in terms of organization, communication, and affect regulation. The mood and behavior of the children also were affected, and families with a unipolar parent functioned better than those with a bipolar parent.

### **Resilience, Prevention, and Treatment**

Despite the risks to children who grow up in families where a parent has a psychiatric diagnosis, many children fare well, indicating that a genetic predisposition to mental illness is not the only factor in determining child psychopathology, and that those interventions that address parenting behavior may help prevent more chronic disorders.

In response to an Institute of Medicine mandate ([Leckman et al., 1995](#)) encouraging research on the treatment and prevention of psychopathology in children at risk owing to parent mental illness, a study in Boston attempted to promote resilient traits in non-ill children ([Beardslee et al., 1998](#)). The researchers targeted eighteen 15-year-old children whose parents had an affective illness and intervened to teach parents how to increase their children's understanding of their illness. The children of parents who responded positively to the study reported better outcomes in terms of depressive symptoms and global functioning.

[Free et al. \(1996\)](#) examined the impact of psychotherapy for depressed mothers on affective language with their children. The study is based on the premise that affective language can produce a confusing environment where children are drawn into their mother's sadness and in which their empathy is exploited. The authors predicted that psychotherapy would help depressed mothers to be more responsive to their children, teach them about emotions, and offset the risk of developing difficulties. Three groups were used for the study: depressed mothers receiving no treatment, depressed mothers receiving treatment, and a control group of nondepressed mothers. The sample totaled 84 mothers. Depressed mothers who had received psychotherapy were able to interpret emotional expression with more accuracy (particularly negative emotions) than the no-treatment group. The authors concluded that depressed mothers who receive treatment are more accurate in their interpretation of emotional expression and may pass the same skills on to their children.

Positive outcomes of group therapy also were reported for the four adult daughters of psychiatrically ill mothers ([Williams, 1998](#)). After 10 sessions, all were able to cease individual therapy despite having presented with psychological problems. At a 12-month follow-up, all the women reported improvements in parenting of their own children in mood and self-perception.

In summary, psychiatrically ill parents are a major risk factor for their children's mental health. Early identification and treatment are important for both the child and his or her family. Improvement in the parent–child relationship can occur if the parent is emotionally available to understand the impact of his or her illness on the child and can be helped to communicate this to the child, as well as learn improved parent management skills. The child must be helped to understand that he or she did not cause the impairment, be educated about the illness, and be given guidance and support in how to cope with such issues. However, despite the burden on child and family, research suggests that effective interventions with positive outcomes do exist. Removal from a family may occur in extreme cases, and the clinician often is called on to provide an opinion or testimony.

### **Substance-Abusing Parents**

According to statistics, there are 28 million children of alcoholics in America, and almost 11 million are younger than 18 years of age. In addition, there are an unknown number of children of other substance abusers ([Adger et al., 1999](#); [Johnson and Leff, 1999](#)). The relationship between parental substance abuse and subsequent alcohol-related and other problems in children has been extensively documented. However, it is difficult to isolate substance abuse as the change variable because of the high prevalence of other stressors, including comorbidity with a psychiatric disorder ([Hill and Muka, 1996](#); [Kuperman et al., 1999](#)). In a study of 76 children, 8 to 18 years of age, of alcoholic parents (with no psychiatric diagnosis) and a control group, the children of alcoholic parents displayed more psychiatric diagnoses than the control children ([Hill and Muka, 1996](#)). The risk increased when the child was living with a biological mother and a custodial father who were alcoholics.

As with the psychiatric disorders discussed previously, there are biological, psychosocial, and environmental factors mediating substance abuse ([Johnson and Leff, 1999](#)). Familial and parenting factors include disruptions such as lack of family rituals and frequent conflict, poor home management, lack of communication skills, ineffective parenting strategies, physical violence, decreased family organization and cohesion, isolation and stress, and frequent family moves ([Johnson and Leff, 1999](#)). The results of these risk factors on the children of substance-abusing parents are adjustment problems, psychopathology, lack of awareness, insight, and empathy, anxiety, depression, low self-esteem, impaired emotional development, and externalizing behavior problems, including delinquency. In a study by [Kuperman and colleagues \(1999\)](#), poor parenting by adults with alcohol dependence and antisocial personality disorder was associated with conduct disorder, alcohol abuse, and cannabis abuse in children.

The recognition of the impact of alcoholic parents on children has increased the attention given to parent- and family-directed interventions ([Loveland-Cherry et al., 1999](#)). Some studies have found encouraging results with family and parental interventions as a means to prevent alcohol use and misuse, as well as a preventative measure to limit the effect alcoholic parents have on the development of their children. A study by [Loveland-Cherry and colleagues \(1999\)](#) used a parental intervention that aimed to (a) increase protective factors (factors that protect adolescents from alcohol use), such as a cohesive, supportive family environment, clear rules for expected behavior, and parental monitoring and knowledge of the predictors and consequences of alcohol use; and (b) decrease risk factors such as permissive or inconsistent parental discipline as well as parental alcohol use and approval of adolescent alcohol use. The family intervention focused on general parenting skills and family functioning as well as on factors specific to alcohol use and misuse. The study showed promising results for the efficacy of this family intervention as a universal prevention program to decrease initiation of alcohol use and subsequent misuse for most adolescents who do not report prior drinking. Additional interventions are needed for those adolescents who already have used alcohol.

Another study (Nye et al., 1991) implemented a family-based intervention program for the prevention of conduct problems among preschool-age sons of alcoholic fathers, in an attempt to change the potential risk structure. The study also examined the interplay between parent expectations and commitment to treatment in predicting change in child behavior and parenting style. Parent expectations at pretreatment were found to have influenced their early investment in the program, thus predicting treatment outcomes. Parent and therapist satisfaction ratings during treatment were associated with one another and with expectations that the program would continue to promote changes in the child. Parent investment was a particularly important influence on outcome because increased investment throughout the program was associated with improvement in child behavior and decreased authoritative parenting at termination.

For substance-abusing parents with children, the research again notes that interventions provided in the context of the family may auger well for good outcome. Many therapists will not treat individuals with substance-abusing problems unless they are involved in a 12-step program and regularly attend an Alcoholics Anonymous group. Often, support groups for such children, such as Al-Anon, are helpful in reducing a sense of isolation and blame, as well as offering coping strategies. As with psychiatrically ill parents, in extreme cases the child may have to be removed.

## OUTCOME RESEARCH

Numerous studies have indicated that parental involvement in the child's treatment is an important indicator of good outcome ( Hudson and Harrison, 1986; Rosenstock and Vincent, 1979; Tittler et al., 1982). Various issues that arise in outcome are discussed ( Table 85.2 and Table 85.3).

Reference	Issue	Outcome
Ulan and Langer, 1983; DeWitt, 1983	Assessment of problem and mutual goals between therapist and parent	Continuance in treatment
Cole and Magnusen, 1967; McAdoo and Gagne, 1972; Rosenstock et al., 1981; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	High expectations for child and parent	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance

Table 85.2. Predictors of Positive Treatment Outcome

Reference	Issue	Outcome
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance
Ulan and Langer, 1983; DeWitt, 1983; Gager et al., 1983; Rosenstock and Stephenson, 1988; Armbruster and Kazdin, 1994	Parental involvement in treatment and compliance	Increased compliance

Table 85.3. Predictors of Negative Treatment Outcome

## Naturalistic versus Research Settings

General indicators of successful outcomes include improvement in child behavior, improvement in parental functioning, and maintenance of treatment gains ( Kazdin and Weisz, 1998). Most treatment outcome studies in outpatient children's mental health services have taken place in research clinics. Findings from research studies have been consistently positive. For example, of 150 child therapy outcome studies reviewed by Weisz and Weiss (1993), cognitive-behavioral interventions showed more positive outcomes compared with nonbehavioral treatments. Randomized, controlled trials have indicated improvements in the quality of parenting; success in bringing half the children with diagnosable disorders into the nonclinical range; and maintenance of gains 3 to 10 years later ( Scott, 1998). There remain questions about the magnitude and scope of change and durability of outcome effects, and the applicability of these treatments to naturalistic settings still is unclear ( Kazdin, 1997a; Kazdin and Weisz, 1998). In contrast, there are only a handful of published outcome studies based in "naturalistic" settings, with mixed and far less positive results. Weisz and Weiss (1993) address this problem with the "good news bad news joke," which states that "the good news is that child psychotherapy works; the bad news is, not in real life" (p. 96).

Contrasting procedures between research and "real world" clinics may explain these observed differences in treatment outcome. Werry and Andrews (1996) point out that "there is little resemblance between what researchers study and what practitioners do" (p. 879). Differences in procedures between the research and the naturalistic settings include the following: subject recruitment, parental involvement in treatment, stringent treatment protocols, exclusionary criteria, manualized treatment, and completion of measures by both therapist and patients ( Kazdin, 1991; Kendall and Southam-Gerow, 1995). Such procedures are often anathema to both clients and clinicians in naturalistic settings and likely result in different clinicians and different subject populations being seen at research- versus non-research-based clinics.

In the adult literature, Seligman (1995) describes this dialectic by distinguishing between "efficacy" studies in research clinics and "effectiveness" studies in naturalistic settings. Under tightly controlled conditions with sophisticated methodology, efficacy studies conducted in research clinics differ from effectiveness studies, which assess "how patients fare under the actual conditions of treatment in the field" ( Seligman, 1995, p. 966). Although both approaches contribute to our knowledge of treatment outcome, effectiveness studies may hold the greater potential to teach us about the majority of clients and clinicians who participate in the "real world" of mental health.

## Parent Attributes that Affect Outcome

There are a number of parent and practice characteristics that can contribute to selection of treatment intervention and are predictive of different outcomes in parent work. These include ethnicity, socioeconomic status, the parent's psychiatric history, parent-therapist relationships, flexible scheduling, and access and availability of preventative services. Some characteristics in parents and children have been shown to predict poor outcomes across a range of therapeutic approaches. Parent characteristics include denial of problems, refusal to cooperate, personality disorder, psychopathology, and a parental history of physical and sexual abuse. Child characteristics include severe antisocial behavior, hyperactivity, and learning difficulties. Other factors are related to social environment; disorganized, poor living conditions; lack of social support; and a hostile partner ( Scott, 1998). Preventative services to vulnerable families tend to be community based, taking resources into neighborhoods to make it easier for poor families to access services ( Davis and Spurr, 1998; Webster-Stratton, 1998).

Researchers have agreed that parental motivation and positive attitude toward treatment appear to be "crucial factors" and the "only reliable finding" in predicting continuance in a child guidance clinic ( Armbruster and Kazdin, 1994; Cole and Magnusen, 1967; Gager-Piacun, 1985; Gould et al., 1985; McAdoo and Roeske, 1973; Novick et al., 1981; Pekarik and Stephenson, 1988). Motivation is a key ingredient in the assessment of parents, as discussed earlier. In the interventions described earlier, motivation also was critical for compliance with the study protocols.



## Practice Attributes that Affect Outcome

Agreement on the problem and mutual goals between therapist and parent appears to augur well for treatment ( [Dechillo, 1993](#); [Lake and Levinger, 1960](#)). Improving communication between therapist and parents and understanding parental expectations, perceptions, and attitudes also have been identified as crucial to treatment continuance ( [Singh et al., 1982](#)). "In general, professionals seem reluctant to accept the concept of improvement" without their intervention ( [Madger and Werry, 1966](#), p. 716). In children's mental health, this resistance to the "parents' point of view" ( [Madger and Werry, 1966](#)) may be described as clinic centrism ( [Armbruster and Kazdin, 1994](#)), observer bias ( [Magder and Werry, 1966](#)), staff bias ( [Mirin and Namerow, 1991](#)), and blame ( [Early and Poertner, 1993](#)). Parents "vote with their feet" ( [Mirin and Namerow, 1991](#)) when the match with the clinician is not comfortable and the offered service does not meet their needs. [Chess and Thomas' \(1986\)](#) concept of "goodness of fit" is useful to consider in this context. Examining the match between the proffered service and the child and family, as well as the tension between patients' goals and expectations and those of clinicians ( [Armbruster and Kazdin, 1994](#)), is particularly important in parent work. Kazdin and colleagues have reported that when child treatment includes the parents, there are gains in both child and parent functioning ( [Kazdin, 1985](#); [Kazdin et al., 1992](#)). The recent MTA Collaborative Multi-modal Treatment Study of Children found that the combination of interventions that intensively engaged parents was more successful than the other interventions used ( [Wells et al., 2000](#)). Hence, findings underscore the importance of families and of working with them to support effective child treatment ( [Campbell et al., 1993](#); [Hibbs et al., 1993](#); [Yama et al., 1993](#)).

## Attrition and Its Relationship to Outcome

Fifty to 60% of children referred for therapy commonly drop out prematurely ( [Armbruster and Kazdin, 1994](#)). Dropping out limits both the benefits of treatment and, by introducing a sampling bias, the effectiveness of outcome studies ( [Prinz and Miller, 1994](#)). There are well established characteristics of families who drop out of treatment: socioeconomic disadvantage, minority status, high stress levels, family dysfunction, difficult living circumstances, parental psychopathology, and severity of child problems ( [Armbruster and Kazdin, 1994](#)).

[Kazdin \(1997b\)](#) demonstrated, in a 4-year, prospective study, that there are other barriers associated with treatment that are separate from the characteristics listed previously that increase the risk for dropping out. These are practical obstacles (e.g., transportation), perception of treatment as demanding or irrelevant, and poor relationship or alliance with therapist. Parents often enter into parent work with low self-esteem and little confidence in their parenting ability. If they are then made to feel blamed, criticized, and attacked, the likelihood of dropping out of treatment is increased ( [Sargent, 1997](#)). Alternatively, building a strong alliance between the therapist and parent with an attitude of working collaboratively facilitates a sense of responsibility and urgency on the part of the parent. The cooperation of parents is essential to the successful outcome of child therapy, and behavioral treatments require reinforcement from parents.

To reduce dropout from therapy, [Scott \(1998\)](#) recommends visiting the family in the home, trying to fit in with the daily routine of the family, and providing child-minding services for other children. [Prinz and Miller \(1994\)](#) also recommend providing transport, offering therapy at out-of-office hours, and demonstrating sensitivity to cultural issues. [Kazdin's study \(1997b\)](#) has further implications for practice because it suggests that individualized interventions during the therapy process can reduce the likelihood of early termination. Examples of these interventions are discussing parent expectations, building a stronger alliance with enhanced communication between therapist and parent, and addressing other life stressors ( [Prinz and Miller, 1994](#); [Singh et al., 1982](#)).

For the researcher, attrition harms the design and obfuscates the findings; for the clinician, attrition is an indication of patient noncompliance or dissatisfaction. Some studies have taken an alternative approach by acknowledging the parents' perspective as it relates to attrition. Parental dissatisfaction with the services includes lack of improvement in their children's behavior, dislike of the interpersonal aspects of the evaluation, dislike of group treatment for themselves, and unfulfilled expectations ( [Ewalt et al., 1972](#); [Farley et al., 1975](#); [Singh et al., 1982](#)).

Clinic-centrism does not acknowledge that the patient's goals, as opposed to the therapist's, may have been met, and that dropout may not be treatment failure ( [Armbruster and Kazdin, 1994](#); [Tuckman and Lavell, 1959](#); [Viale-Val et al., 1984](#)). Nor should the assumption be made that dropouts harbor a negative view of what mental health services may offer. Clinicians may find it useful to acknowledge that the admission of a problem—for example, a parent calling a clinic—is the first step in the process of change, and for some persons this may be all that is necessary. [Farley et al. \(1975\)](#) found that dropouts did not have a negative view of interventions received but, to the contrary, 88% of them would recommend the services to others. These authors did not depict dropouts as "rejecters" of therapy, and with others suggest that mental health professionals, as well as patients, share the responsibility for dropout ( [Farley et al., 1975](#); [Kourney et al., 1990](#)).

## Cultural Issues Related to Outcome Research

The clinic-centric perspective also may be related to issues of cultural/ethnic compatibility associated with attrition among minority patients. African Americans in general have significantly higher rates of dropout from therapy and use fewer services than nonminorities ( [Armstrong et al., 1984](#); [Goodman and Seigal, 1978](#); [Sue, 1977](#); [Vail, 1978](#); [Vernon and Roberts, 1982](#)). The finding may not be due to minority status *per se*, but rather may reflect a mismatch of services or incompatibility of cultural/ethnic views, a "cultural incongruity" between those who receive and those who provide services ( [Cheung and Snowden, 1990](#), p. 284). Indeed, [Flaskerud \(1986\)](#) found that ethnic/racial differences between clinician and client, and location of the agency outside the ethnic/racial community, all predict dropout. Given differences in behavioral problems and parenting between ethnic groups, authors emphasize that PT programs should not be assumed to be universally effective ( [Florsheim et al., 1996](#); [Forehand et al., 1997](#)). However, [Ho and colleagues \(1999\)](#) found that PMT worked well with a Chinese population despite anticipation of cultural incompatibilities. Disruptive behavior was reduced and improvements in parent-child interactions were maintained at a 4-month follow up. Ho et al. highlight the danger of stereotyping, but generally found PMT to be consonant with Chinese cultural norms, given the emphasis on discipline, obedience, and parental responsibility and structured interventions. However, lack of data and "little contribution from rigorous, formal research" (p. 289) are major concerns in examining mental health service use patterns for minorities ( [Cheung and Snowden, 1990](#)). Because most of the existing studies are of adult patients, these concerns are even more acute for child research.

## SUMMARY

There is a consensus that work with parents increases the positive outcomes for children in treatment, and future models of the child psychiatric services are urged to pay "close attention to variables other than children's psychiatric symptom levels," such as parent characteristics ( [Jensen et al., 1990](#)) and other environmental factors. In helping children overcome various problems, multiple perspectives are critical. The research interventions described in this chapter point to the importance of parent involvement in achieving effective results for children. Although engagement with parents has not been especially emphasized in the training of child mental health professionals, as noted at the outset, the Surgeon General in the first National Conference on Child Mental Health in September 2000 proposed a paradigm shift to an ecologic model that actively includes the parents and family in the treatment of the child ( [U.S. Public Health Service, 1999](#)). Policy makers, administrators, and others are seeking a new way of thinking about the relationship between families and service providers at both the individual family and the policy level. They have arrived at a philosophy that emphasizes the interdependence of all participants and the need to engage in interventions as allies in the challenge to improve the lives of children. Intrinsic to this philosophy is the recognition of the importance of moving from a hierarchical to a team model. Family members rarely are accorded the same respect as clinicians, policy makers, researchers, or administrators. Yet, in most situations they are the "experts" in regard to their children. When professionals begin to recognize what family members already know—that families are crucial participants in the ecosystem in which children grow—the challenge is reframed. Once everyone fully acknowledges and respects the membership parents already have, the task becomes one of how to develop and maintain connections between professional and familial members. Their inclusion as active partners in providing services for their children is necessary, responsible, and effective.

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# 86 PSYCHODYNAMIC PARENT PSYCHOTHERAPY: TREATING DISORDERS IN PARENTING AND IN THE PARENT–CHILD RELATIONSHIP

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Most parents are “good enough,” although all parents at times lose empathy and do things that fail to meet their children's needs. However, many parents are not “good enough,” because they cause too much harm to one or more of their children as a result of the characteristic ways in which they think, feel, and behave. Although parents with parenting disorders often feel unreasonable anger and disappointment toward their children, these parents also are very much in love with those same children. Parents who are hurtful, even most of those who engage at times in overt abuse and neglect, sincerely want to be able to treat their children well. In addition, parents who are themselves victims of external forces and powerful mental experiences that are out of their control are still responsible for the hurtful behavior. Such paradoxes are the hallmark of parents' inner struggles to manage unwanted thoughts and feelings without acknowledging them, and they also reflect parents' often incompatible wishes to reduce their own pain without abandoning their obligation to foster the well-being of their own children.

Psychodynamic explorations of the inner lives of hurtful parents reveal in almost every case a disorder of the parents' enduring patterns of thoughts and feelings about the particular child they are hurting. The concept of the parental mental portrait of the child is introduced in this chapter as a framework for a psychodynamic understanding of the enduring patterns of parental thoughts and feelings that characterize every parent–child relationship. The concept of an unhealthy parental mental portrait of the child is at the center of the clinical approach recommended here, because it is the approach that best enables clinicians to secure enduring improvements in the parent–child relationship that are profound enough to promote the long-term well-being, autonomy, and healthy mental life of the child who is being hurt, the overriding goal of all parent therapy.

Many parenting interventions are not adequate to treat significant disorders of the parent–child relationship vigorously enough to improve the parental mental portrait of the child. First, when a disorder of the parental mental portrait of the child exists, there may or may not be hurtful child-rearing practices, but there is always a disruption in the constructive emotional tone and mutuality of the parent–child interactions. Consequently, parenting interventions designed to help parents to select socially appropriate child care practices are not sufficient, because the negative emotional tone and the lack of mutuality in the parent–child interactions almost always persist even after appropriate child-rearing practices have been adopted. Second, by the time parents seek help for parenting disorders, the destructive mental patterns that characterize the disorder of the mental portrait of the child have usually become self-sustaining. Thus, parenting interventions that aim primarily to alleviate the familial, social, economic, political, or biological factors that helped to create and sustain the disorder of the parental mental portrait of the child are often no longer sufficient to help a parent to change it. Third, most of the thoughts and feelings that characterize a disorder of the parental mental portrait are unconscious. Therefore, interventions that primarily help parents to improve their conscious knowledge of child development, children's needs, appropriate child care practices, and positive caregiving attitudes are also insufficient. Psychodynamic parent psychotherapy is often the best treatment option because it is a clinical approach, perhaps the only one in child psychiatry, that has as its primary goal relatively profound and enduring improvements in the content and regulation of the self-sustaining unconscious mental patterns that pressure parents to hurt their children repeatedly.

Effective diagnosis and treatment of parenting disorders are extremely difficult because of the extraordinarily powerful and disruptive psychological reactions that are stimulated in therapists when they are working closely with patients who are hurting their own children. It is most upsetting to clinicians to come to terms with the knowledge that their patients are good enough ordinary adults, not very different from the therapists themselves, and those same patients are also hurtful parents who make choices that are not compatible with their moral commitment to support the well-being of their own children, a commitment that is one of the most universally held and most important moral standards of our culture.

Engaging in a professional caregiving relationship with such parents forces therapists to think about their own very difficult unresolved moral issues and the hard choices they face every day that affect the spiritual center and meaning of their own lives. Even very experienced psychodynamic psychotherapists frequently fail to manage their psychological reactions to these difficult moral issues very well. As a result, therapists employ various unconscious and conscious defensive psychological strategies to avoid full awareness of the true nature of the moral dimension of the parents' problems.

Such defensive thinking disrupts the therapeutic stance profoundly and has a devastating impact on effective diagnosis and management of parenting disorders in the following ways. Therapists who work within a conventional psychoanalytically informed clinical approach may simply overlook the serious nature of the parenting problem. Other clinicians who work within a more medical clinical approach may fail to address the parenting problem because they allow themselves to set aside the long-term primary treatment goal of achieving lasting improvements in the parent–child relationship that promote the child's long-term mental health, well-being, and autonomy and instead adopt an exclusively narrow, short-term treatment approach. Such a narrow treatment approach is usually focused on improving the child's behavioral adjustment and symptoms, but sometimes it is focused on treating acute problems of the parent that are not directly related to parenting. The narrow treatment focus facilitates the therapists' own motivation to divert attention from the parenting disorder, the inner life of the parents, and especially the most troubling aspects of the parents' inner life, the thoughts and feelings associated with the moral dimension of the parenting disorder.

However, even when they identify a serious parenting disorder and sustain their attention to the inner life of the parent, therapists may still defensively ignore the most disturbing aspects of the true nature of the parent's responsibility for making ethical decisions that are harmful to the child. By engaging in unsystematic, unrealistic, dichotomous moral reasoning, therapists can protect themselves by granting a false blanket exoneration to hurtful parents or by dismissing the parents as very different from the therapists themselves or by exaggerating the patients' immorality and demonizing them. In addition, therapists may protect themselves with the self-deluding pretense of abstaining from any ethical judgment of the parent, by assuming a neutral therapeutic posture that is almost always a rationalization for the experience of exaggerated blaming or blanket exoneration that has been suppressed.

Such defensive moral reasoning is the most important cause of failure of even the best clinicians to diagnose and treat parenting disorders effectively ( [Siskind, 1997](#)). Of course, when clinicians do not identify or attend to significant parenting disorders, they cannot recommend parenting psychotherapy when the children and their parents need it. When they embrace a view of their patients based on exaggerated blaming or blanket exoneration, they undermine the therapeutic stance. When the therapeutic stance is undermined, clinicians cannot make sound clinical judgments, nor can they sustain the empathy, respect, and fidelity to both parent and child that are required to support the professional therapeutic relationship.

Strong psychological reactions, denial, and defensive moral reasoning are impossible for parent psychotherapists to avoid completely. Conversely, however, it is almost impossible for clinicians to sustain such destructive defenses if they discipline themselves to hold onto dual empathy for the parent and the child and an

appropriately complex, comprehensive understanding of the parents, the child, the parent–child relationship, and the psychotherapy. Therefore, the primary goal of this chapter is to suggest that clinicians prevent serious treatment problems by disciplining themselves to do the following: (a) formulate the clinical parenting disorder with the parental mental portrait at the center, by taking into account the complex dynamic interactions of the full range of biological, psychological, social, and moral factors; (b) achieve, monitor, and sustain dual empathy for parent and child; (c) explicitly assess and attribute to the parent a balanced and realistic determination of ethical responsibility for decisions that hurt the child; (d) sustain the primary long-term clinical goal of advocating for long-term improvement in the mental life of the child; (e) protect the determining role of individualized clinical judgment that is based on the primary goal of parent treatment in association with other appropriate values including the unique personal values of the child and family; and (f) adopt a body of clinical theory and scientific knowledge that is appropriate to encompass the moral dimension of professional medical care, that is, a body of knowledge and theory about profound suffering as well as severe pain, free choice as well as biological and environmental determinants, and the values that imbue the lives of individual patients with dignity and meaning, as well as the values that support the achievement of competence, mastery, adaptation, social success, and survival.

The chapter begins with a discussion of the concept of the parental mental portrait of a child, including the origin, pathogenesis, and impact of disorders of mental portrait and a comprehensive framework for formulating parenting disorders. A description of the powerful psychological reactions of therapists to the moral dimension of parenting disorders is followed by a presentation of additional practice guidelines designed expressly to help therapists to manage those responses better. The chapter concludes with some selected practical suggestions about how best to begin and perform parent psychotherapy and encouragement for clinicians to evaluate all the children they see clinically for defects in the parent–child relationship and to recommend psychodynamic parent therapy much more often than they do now. The presentation is directed toward clinicians who are already trained as competent psychodynamic therapists and who have an opportunity to work under conditions that are compatible with the performance of relationship-based psychotherapies.

## PARENTAL MENTAL PORTRAIT OF THE CHILD

The concept of the parental mental portrait of the child is introduced here as a framework for the psychodynamic understanding of the enduring patterns of parental thoughts and feelings about a particular child. The mental portrait concept is the central element in a modal formulation designed to help clinicians think about parenting problems in a way that is most likely to achieve the primary therapeutic goal of enhancing a child's long-term well-being, autonomy, and healthy mental life. In the following section, the concept of the parental mental portrait is defined, and then disorders of the mental portrait of the child are described, including their origin, their pathogenesis, and their relationship with parenting disorders, including the role of parental choice in their expression and the impact they have on the parent–child relationship and the development of the child.

### Defining the Parental Mental Portrait

All parents sustain a unique mental portrait of each of their children. The *parental mental portrait* of the child is a psychodynamic understanding of the enduring features of a parent's inner life that is focused on the thoughts and feelings associated with a particular child. It is an understanding that is informed by certain useful clinical concepts selected primarily from the interpersonal ([Gill, 1983](#)) and object relations ([Kernberg, 1982](#)) aspects of psychoanalytic theory. The mental portrait of the child refers primarily to a parent's enduring, global synthesis of all the remembered thoughts and feelings that are closely associated in that parent's mind with the subjective experience of a particular child ([Feraholt, 1976](#); [Feraholt et al., 1985](#)). These remembered thoughts and feelings, primarily unconscious, are more or less well organized, often hierarchically, to form a global mental synthesis that serves to represent a particular child in the mind of the parent. The global synthesis is best thought of as an enduring dynamic mental system, not simply a static composite or “average” memory. The mental portrait has both an enduring, current, structural dimension and a historical, developmental dimension.

The parental mental portrait includes what is commonly called the meaning of the child to the parent and is informed by what others have written about as the mental representation ([Kernberg, 1980](#); [Sutherland, 1980](#)), narrative story line ([Schafer, 1992](#)), working model ([Greenson, 1960](#); [Stern, 1985](#); [Zeanah et al., 1989](#)), and cognitive and developmental schema ([Bruner, 1990](#); [Piaget, 1953](#)). Because the mental portrait guides the perception, selection, and interpretation of new information and remembered thoughts and feelings about the child, it has a major impact on all the parent's behavior in relation to that child.

The mental portrait model for formulating parenting disorders is not limited to the psychoanalytic notions that constitute the mental portrait concept itself. The model for formulating parenting disorders is a theoretically pluralistic model, but not a syncretic or eclectic model; it depends on concepts drawn from a variety of theories. This variety of concepts is needed to take into account the many factors that may be important to treatment planning for any particular clinical case of an individual child in a family with parenting disorders. The important factors usually include the biological and psychological state of each family member, the nature of the family unit, and the social institutions, economic forces, and political forces that influence parents. However, the model presented in this chapter is unusual because it emphasizes the conscious value-based choices of the parent; this model includes the moral dimension in addition to the biological, social, and psychological dimensions of the clinical situation.

To encompass the moral dimension of the clinical situation, the model presented here includes the language and concepts that are required to organize data about the relevant values, life plans, ideals, and standards of the particular parents, as well as those of their families, religions, and ethical traditions. These personal and cultural factors are the ideals that guide important parental choices. This model also requires concepts, methods, and data that are suited to describe, in a way that is clinically useful, the moral courage and competence of a particular parent to uphold his or her own ideals by meeting the standards of conduct associated with them and by resisting strong impulses to betray ideals to seek gratification or avoid pain. The model of formulation also emphasizes the degree of autonomy of therapists and factors that inform their value-based choices. It includes the necessary language, concepts, and methods to make individualized clinical judgments that integrate a professional commitment to the ethics of traditional medicine in a Western rights-based democracy with respect for the particular values of each individual parent.

### Disorders of the Parental Mental Portrait

Because all parents lose empathy and hurt their children at times, disorders are distinguished from “good enough” parenting based on clinical judgments of the severity, pervasiveness, and chronicity of the disturbances in the parenting relationship. However, less severe disturbances in parenting are also identified as clinical problems deserving treatment if they are judged to be detrimental to a child with particular vulnerabilities. Disorders of parental experience of the child are characterized by insufficient empathy, denigration, or idealization, overinvolvement or remoteness, and a poor match with the actual characteristics of the child. The discrepancy between the parent's view of the child and reality stems from distorted perceptions, misinterpretations, confusions, overly simplistic interpretations, and even unconscious fabrications about the child. These cognitive distortions are associated with ego mechanisms of defense and are discussed later.

When there is a very severe defect in regulation within the portrait, parents unconsciously experience the child in a prejudiced way, almost all the time. They attribute exaggeratedly evil designs to the child, but sometimes their attributions involve unrealistic perfection ([Miller, 1981](#)). In the most serious cases, a parent may consciously experience a child as if he or she were someone or even something else (e.g., father, mother, sister, demon, monster, genius, angel, or god). Some parents, even when there is no other overt psychiatric disorder, have no doubt that their rigid, black-and-white characterizations of the child are completely true. The parental prejudices of disturbed parents portray the child in terms of a range of undesirable caricatures or stereotypes, including versions of the following: the coldly calculating, self-aggrandizing villain; the wild, unsocialized primitive; the irresponsible, lazy, manipulative tramp; the tragic, fragile, suffering invalid; and the generous, powerful, brilliantly intelligent, creative, invulnerable leader. The case of Jason M. (see later) illustrates such a defect in a parental portrait so severe that it could not be sufficiently repaired.

More commonly, the defective portrait manifests in more subtle ways. The parent repeatedly experiences brief episodes of exaggerated affect and inappropriate ideation and affect that occur in the course of difficult interactions with the child. Most of the time, the handicapped parent may sustain a reasonable attitude toward the child, often affectionate, but always with defects in empathy, that is, a failure to understand the child's point of view. The prejudices are not consciously held, but they can be elicited in the course of an intensive psychiatric interview. The second illustrative case in this chapter, Douglas T., describes a less severe defect in parenting that is responsive to treatment.

### Development of the Parental Mental Portrait

The mental portrait begins with the parent's fantasies before meeting the child. In the course of an adult's “healthy” development as a parent, the mental portrait of a child is repeatedly reworked to achieve higher levels of organization and complexity, best to protect and support the developing competence and autonomy of the child. Such ongoing development of the mental portrait is best viewed by parent therapists in terms of two interactive processes: (a) the process by which the components of the mental portrait are repeatedly reconciled with perceptions of the real child who is changing and (b) the process by which the parent's mental representations of the child, initially tightly entangled with the parent's self-representations, are gradually separated in the parent's mind from those representations of



the parent's self.

These two developmental processes in the development of the mental portrait depend on a sequence of parental experiences of loss, grieving, and restoration, which are understood best using concepts borrowed from the study of mourning after the death of a loved one ([Engel, 1962](#)). When, in the course of development, the parental mental image of the child is challenged by the parent's perceptions of the real child, the parent suffers a psychological loss that disrupts the integrity of that parent's experience of self. If the mourning processes are significantly obstructed or distorted, the mental portrait will be compromised. Thus, the same etiologic factors are present in both pathologic deviations in grieving and in defective development of the mental portrait.

### **Etiology of an Unhealthy Mental Portrait**

Viewed from the perspective of the mourning that the parent must achieve in the development of the healthy mental portrait, the causes of deviant, defective development of the portrait include the factors that interfere with mourning. Extreme ambivalence toward the child is a common cause. Ambivalence may develop because a healthy child comes at a very bad time, there is a painful, physically damaging pregnancy and birth, or care of the child is very difficult. Ambivalence may develop if a normal child fails to meet unrealistically high parental expectations, such as the capacity to repair a failing marriage, to achieve a reunion between a parent and a grandparent, to fulfill the parent's unmet needs for parental care, or to solve the parents' problems in social adjustment. Ambivalence may also result from the overwhelming intensity of the psychological loss associated with an unhealthy child, as may occur if a child is born deformed or handicapped severely or if a child almost dies during gestation, at birth, in an accident, or from a potentially fatal illness.

Many different biological, psychological, social, economic, and political factors can interfere with a parent's capacity to mourn a loss and in this way contribute to the development of defects in parenting. They include the following: psychiatric problems in the parent, such as parental depression and personality problems such as pathologic narcissism; memories of unresolved past losses after the death of a loved one, disrupted love relationships, or childhood neglect and abuse; lack of preparation for various difficult experiences of parenthood, including ignorance and misinformation about pregnancy, labor, delivery, child development, or child care; lack of emotional support from loved ones after the loss because of marital problems or social isolation; and compounding losses from additional stressors, including physical illnesses, the death or separation from a loved one, marital discord, family disorders, social isolation, unemployment, poverty, sexism, and racism. The interaction of various factors at any one time may trigger disturbances in the development of the mental portrait of one child even in a parent who sustains healthy portraits of other children.

### **Pathogenesis of an Unhealthy Mental Portrait**

Almost all the mental processes central to a defective mental portrait transiently distort the mental life of every parent. Again, problems are distinguished from "good enough" situations by degree, pervasiveness, and balance. In the intrapsychic sphere, problems in the mental portrait concern both content and regulation. Most commonly, there is a failure to manage adequately the boundary between the mental representation of the child, that of the parent's self, and that of each of the parent's own parents, although representations of other loved ones may sometimes play a role, too. Many psychodynamic concepts can help the therapist to generate a useful intrapsychic understanding of the nature of particular aspects of the psychological processes that underlie the defective regulation. The ego mechanisms of defense are indispensable to such psychodynamic formulations, especially projection, projective identification, and a set of related concepts that encompass both the intersubjective and the interpersonal perspectives on the parent-child relationship ([Bollas, 1987](#); [Bollas, 1992](#); [Masson, 1984](#); [Miller, 1981](#); [Miller, 1991](#); [Sperling, 1974](#); [Sutherland, 1980](#)). However, more comprehensive notions are also helpful in some cases, such as the child in the parent's mind as "self-object," "symbiotic object," "unacceptable self," and even "sexual fetish" ([Schreier, 1992](#)).

Two clinical concepts—identification and reactivation—are especially useful in describing intrapsychic dynamics. A healthy parent "identifies" strongly with a very young child. Various processes of internalization lead to intense and irrational identifications that are useful when they are properly regulated, because they help to sustain sufficient commitment and interest in the child. However, a pervasive, fixed, and irrational identification can be at the center of defective regulation when the mental representation of the parent's self and that of the child tend to become stuck in the fused state, stuck in a detached state, or to alternate erratically from extreme detachment to extreme fusion. In a state of extreme fusion, characteristics of the child that are significantly different from the parent are overlooked; in a state of extreme detachment, the identifications needed to sustain an essential intuitive empathic knowledge of the child are impossible.

A healthy parent also experiences the eruption of repressed memories of childhood in the context of his or her identifications with a child, because that child is at the same age, in the same stage of development, or is having experiences that are similar to those the parent had when the memory was created. This process, which is referred to as *reactivation*, is often a constructive impetus for continuing personality development in the parent. However, if the reactivated memories are too disturbing, then reactivation may contribute to defective regulation by causing tumultuous emotional responses or eliciting primitive defensive operations, especially denial and distortion of those aspects of the child that stimulated the intolerable reactivation ([Coleman et al., 1953](#)).

Some related concepts, including projective identification, extractive identification ([Bollas, 1987](#); [Bollas, 1992](#)), and introjective identification, are also useful in understanding certain aspects of the defective mental portrait. Although historically connected to terms describing intrapsychic processes such as *projective identification* ([Klein, 1946](#)), these concepts have been expanded in current psychoanalytic theory so they can be used to bridge the intrapsychic and interpersonal spheres ([Tansey and Burke, 1989](#)). In the *current* discussion of the parent-child relationship, these bridging concepts refer to an interactive psychological process by which the parent places an attribute from his or her unconscious mental portrait directly into the mental life of the child, specifically into the child's unconscious self-representation.

These interactive processes, like the intrapsychic processes referred to earlier in this section, are ubiquitous in the relationship of a child to a parent and are usually compatible with healthy personality development. However, projective or extractive identifications can interfere with healthy development and are important aspects of defective regulation in an unhealthy mental portrait associated with parenting disorders. They may contribute to the development of the child's experience of a false self, when too many important aspects of the child are removed from his or her authentic self-portrait and too many incompatible traits are placed into the child by the parent. In addition, particular unhealthy personality traits (e.g., defects in conscience—superego lacunae) may be unconsciously put into the child by a parent.

It is sometimes useful for the parent therapist to view the psychological processes of a defective portrait from the perspective of the family unit as a whole. At such times, concepts of group dynamics that apply only within the interpersonal sphere, such as delineations, designations, and scapegoating ([Ackerman, 1958](#); [Stierlin, 1977](#); [Vogel and Bell, 1960](#); [Zinner and Shapiro, 1972](#)), are most useful. When group dynamics are in focus, the individual intrapsychic mechanisms and bridging psychological mechanisms mentioned earlier are subsumed within the interpersonal group concepts that the therapist is using at that time.

### **Conscious Aspects, Including, Choice, Guilt, and Responsibility**

Some disturbed parents, even those who are socially well adjusted and free of overt psychiatric disorder, intentionally do very harmful things to a child in a vindictive or cynical frame of mind, at times with premeditation and planning. Most hurtful parents do not consciously intend to hurt their children. However, even parents who are basically well intentioned are never completely oblivious to hurtful actions; they are repeatedly aware, even if only transiently and peripherally, that something is wrong with the way they are thinking and feeling about the child as well as the way they are treating the child. Most hurtful parents lie to everyone including their doctors about what they know about their own parenting problems, before they are engaged in a treatment. However, in the course of describing to a trusted therapist in detail many specific interactions, they usually reveal at the least that they did realize, at times, that they were doing things to make a bad situation worse, neglecting to do things that they knew could make it better. These hurtful choices include suppressing appropriate or indulging inappropriate emotions, noticing that people outside the web of the colluding family suspected a parenting problem, or making only the minimal effort to change even when professionals identified the seriousness of the parenting disorder. Very disturbed parents often have elaborate rationalizations for their abusive behavior and may even experience a dissociative state during the short time that they are actually doing something awful to their child. However, even parents driven by the most powerful psychological drives are aware, to some degree, at some times, that something is very wrong. They almost all report, in the course of treatment, that they consciously decided to hide their actions even from those they trusted most, although they maintain paradoxically that they believed at the time that there was nothing wrong with what they were doing to their children.

Therefore, most parents with a defective mental portrait of a child decide many times to deceive themselves, to deceive their loved ones, and not to ask for help. These decisions are made although these parents have the freedom to act differently, if they exerted enough effort. Such parents decide to continue to hurt their children and indulge their desire to avoid various uncomfortable experiences that would result from revealing the problem to others to seek help. Such uncomfortable experiences include the following: (a) guilt, shame, and other psychological pain; (b) deprivation associated with giving up some important gratification; (c) shame associated with social rejection; and (d) tangible loss associated with economic and legal consequences. Such decisions to continue to hurt their children and not to seek help are, in fact, choices for which parents are responsible in the context of a variety of extenuating circumstances. When parents think they are vulnerable to

such self-accusation, they often try to escape from guilt, more or less consciously, by adopting a simplistic, one-dimensional image of themselves such as innocent victim, martyr, or handicapped person. To embrace such a caricature of themselves in relation to their children, parents must also adopt reciprocally extreme conscious characterizations of their children, such as abusing villain or “cross to bear,” thus increasing the child’s vulnerability to being mistreated.

### Consequences for the Parent–Child Relationship

A parent's conscious attitudes and feelings are communicated to the child in good part through planned, easily observable actions that include child-rearing practices. Overt assault, sexual abuse, or gross neglect is almost always associated with a severe defect in the parental mental portrait. However, the converse is not true; that is, serious defects in parenting are usually associated with child-rearing practices that are socially appropriate. In fact, a disorder of parenting often presents to the clinician as a problem in the child who is being raised in what appears to be an appropriately loving, concerned, stable, and well-adjusted family.

Although there may be no easily observed deviant child-rearing practices, unconscious thoughts and feelings, such as loss of empathy, rejection, idealization, sexualization, and other disturbed aspects of the parent's inner life, will still harm the child. Unconscious thoughts and feelings influence the child primarily through nonverbal communication and the emotional tone surrounding words and overt actions, that is, unintentional, “microscopic” aspects of the parent's behavior. These microcommunications include the subtext of the parent's verbal communications, the timing of intended actions, and body language. The subtext, or latent content, of verbal communication is expressed through tone of voice (pitch, melody, rhythm, and stress), sound patterns of selected words, slips, jokes, evoked images, connotations of words, and associative sequences. The timing of actions often communicates meaning in relation to the context of the action and state of reception in the child as a biological organism and psychological being. Body language communicates unconscious thoughts and feelings through facial expressions, patterns of direct eye gaze, posture, gesture, proximity modulation, style of movement in the presence of the child, and ways of touching, avoiding touching, or responding to being touched.

### Consequences for the Child

Even transient and mild disorders in the mental portrait may cause considerable mental anguish, but when there is an enduring and pervasive disorder, even a very resilient child will experience profound mental suffering associated with pervasive and enduring personality disturbances. A defective portrait may also induce the onset of a psychiatric disorder, even when a biological origin is primary, and the defective portrait will almost always worsen the course of a psychiatric disorder in the child. Similarly, defects in the parental mental portrait can precipitate and exacerbate many other medical and developmental problems. In fact, a disorder of parenting often presents only as a complaint about a child with developmental delay, disruptive behavior, mood disorder, or somatic symptom.

Although the deleterious impact of the defective parental mental portrait on a child's personality development can be understood in numerous ways, four are very useful: (a) every extremely painful experience that seriously disrupts the child's experience of safety and trust in the parent–child relationship has a profound and enduring traumatic impact; (b) the dysphoric experiences within parent–child interactions have an incremental impact; (c) the unique learning experiences associated with internalization in love relationships, including projective identification and extractive identification, impose specific unconscious parental expectations on the personality of the child; and (d) the unique learning experiences associated with the child's perception of parental self-indulgence and betrayal of parental fidelity to work very hard to do no harm and to promote the child's well-being disrupt the child's moral development.

It is tempting to speculate that, in some cases involving very bad parenting of a vulnerable infant, global disturbances of brain function may be associated with deviations in postnatal structural development of the brain as well, deviations that could have long-term psychiatric, educational, and psychophysiological implications. Such speculation is controversial among many research scientists, but it is supported by considerable indirect evidence. For example, disturbances in the parental mental portrait can interfere with a child's central nervous system functioning profoundly enough to stop growth and to precipitate asthma, diabetes, projectile vomiting, or dehydrating diarrhea. Further support comes from various data derived from pediatrics and animal investigations, which document deviations in postnatal brain development associated with permanent significant abnormalities of perceptual and synthetic psychological functioning as a result of early and prolonged sensory deprivation or perceptual challenges. Data associating child abuse, psychiatric disturbance, and neurobehavioral deviations in violent criminals are also suggestive, although it must not be forgotten that biologically damaged children are more likely to behave in ways that precipitate mistreatment by their caregivers ([Hoffer, 1995](#); [Perry, 1995](#); [Radic et al., 1994](#); [Singer 1986](#)).

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#### CASE ILLUSTRATION

This case illustrates the devastating impact of a severe disorder of a parental mental portrait on a child in a family where child-rearing practices were by conventional standards “good enough.” The child suffered with severe psychiatric disorders, personality deviations, and physical problems as a result of serious ongoing disorders in the emotional tone of the interactions with his parents, even while overt, easily observable child-rearing practices were, for the most part, appropriate. Countertransference problems were responsible for the delay in diagnosing the parenting defect. The treatment reflects child advocacy and dual empathy for both the child and the parents. The father's defective mental portrait resolved, and the boy's situation was improved, but the mother's psychological disability was so severe that she could not tolerate the psychodynamic parent psychotherapy. She was unable to grieve her own losses even in the context of intensive outpatient psychotherapy, and the defective mental portrait of her son could not be repaired enough for them to continue to live together.

##### Case Presentation

Jason, age 7, was admitted to a combined medical and psychiatric research hospital ward for psychiatric evaluation and studies of his growth hormone regulation. He weighed 22 pounds and was 3 feet tall. (National averages for second graders are 50 pounds and 4 feet.) Before his admission to the hospital, Jason was spending long periods alone in his room, doing nothing but sitting on his bed, motionless and mute. He also had inconsolable screaming spells for no apparent reason, ate garbage, drank from toilet bowls, and ate his own vomitus. He rarely spoke at home, and he usually acted unfriendly, angry, and demanding with his family.

As an infant, Jason had cried incessantly, rejected comfort, recoiled when held, ate so much that he vomited, and slept only during the day. In his second year he clung to his mother and displayed frequent strong tempers with head banging and breath holding. During his third year, he became indifferent and listless, stopped growing, and developed the behavior problems that still were prominent 4 years later when he was hospitalized at age 7.

Jason's parents were middle-class, educated, intelligent adults without any formally diagnosed, psychiatric disorder. They were “good enough” parents to their other children, who were growing adequately and were well adjusted at home and at school. Before treatment, both mother and father accepted this bizarre situation almost without complaint. Supported by their pediatrician, they believed, without any supporting medical evidence, that Jason was brain damaged from birth and that, as a result, he could not help being self-centered, demanding, and unaffectionate. They did not physically abuse, sexually molest, or neglect Jason. After consulting their pediatrician, they had used child-rearing practices that were reasonable, considering their son's extreme behavior problems. In fact, they spent a lot of time trying to teach him self-help skills and appropriate social behavior.

##### Treatment Course

Once in the hospital, Jason was seen in individual psychotherapy, and his parents were seen in parent psychotherapy. Jason lived in a pediatric hospital environment with very intensive nursing, a school program, and a “child life” program. Jason's abnormal growth hormone metabolism corrected itself (without medication) in days, and he began to grow at the rate of 1 cm per month, with proportionate weight gain. His social adjustment was excellent from the outset, his school work improved, and he was free of all his previous bizarre behavioral symptoms. There was no evidence of psychosis or brain damage of any kind. Although his depression lifted enough to allow his body to function more normally, Jason's play therapy continued to reveal extensive abnormalities of his inner life, including intense depression, rage, and deviant personality development.

Jason's father at first was perplexed when his son improved so rapidly in hospital, but in treatment he gradually recognized the family problems, accepted a psychosomatic formulation of the growth failure, and became interested in improving family relationships by better understanding unconscious mental life. He began to like Jason and to notice the boy's feelings. His mental portrait of his son rapidly improved once the family patterns were identified and modified. Jason's mother, conversely, became depressed, angry, and anxious as her son improved. She could no longer displace and project her impulses onto her child. She desperately tried to insist that he was not really changing and even denied his measurable growth. As her anxiety escalated, she accused her husband and the medical staff of turning against her and lying about Jason's progress. She threatened to end the marriage. When she was not very angry, she was depressed, with suicidal thoughts. Jason's mother's mental portrait of her son was inflexible; she was not strong enough to allow it to change with the psychiatric care that was available to her.

It became evident as their history unfolded in treatment that the parents' detached, dehumanized view of their son as a brain-damaged retarded child had caused them to ignore Jason's loneliness and despair and their own sadness and anger. When Jason no longer behaved like a brain-damaged child, Jason's father changed his impression of his son, but Jason's mother revealed a fixed “negative portrait” of Jason in her mind that she could neither change nor erase. She could see only his hidden anger and selfishness and completely mistrusted his sincere expressions of affection, sadness, and fear. She visited less and less often, and was always angry when she did. Gradually, as treatment progressed, she relinquished her conscious conviction that Jason was possessed with evil; her overt anger diminished, and she complained of feeling that there was a “transparent wall” between herself and her son. Yet, the discomfort they experienced with each other remained intolerable.

Mrs. M. confided that when Jason was still an infant she had developed a completely negative view of him and had been desperately frightened that he would “destroy” all she had obtained in life. She was self-critical and deeply depressed at the time, a significant factor in the origin of her parenting disorder. She believed that her depression was caused by her infant son's “anger” and “disappointment” with her. In our conversations, she began to express her rage and to talk about fantasies in which she violently assaulted her son; she attributed supernatural powers to him and used terms appropriate for a demon or an animal. As it turned out, Mrs. M.'s portrait of her son was modeled on similar sketches from her childhood, in which she worked constantly but unsuccessfully for the approval and affection of her own depressed and bitter mother.

##### Comment

The outcomes of extremely serious cases, like this one, are variable. In some instances, it is possible to help parents as disturbed as Mrs. M. to alter significantly the character of the mental portrait of the child. In this case, however, Jason's mother was not able to relinquish her negative mental portrait of her son because it was required to stabilize her sense of self. Jason's mother and father decided, for reasons too complex to describe here, that Jason should live with supervised foster parents. The decision was experienced with great sadness by everyone, but it was accomplished in an atmosphere of honesty. Jason remained symptom free and well adjusted while living in a foster home where he came to be loved. Working in his own individual psychodynamic psychotherapy, Jason gradually began to understand that his mother's rejection was real and not imaginary, and that it was primarily the result of her own psychological problems and not because he was a bad or inadequate person.

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## PSYCHOLOGICAL REACTIONS OF PARENT THERAPISTS



## **Pain Associated with Dual Empathy for Both the Hurt Child and the Hurtful Parent**

The painful psychological reactions of a therapist doing parent therapy stem from many sources. It is painful to identify with a parent who is suffering. It is even more painful to identify with a child who is suffering, a problem faced in all child psychotherapy. Identifications with the child are especially challenging, because they reactivate the therapist's early memories of being a hurt child, memories that are, in turn, associated with the superstitions, magical thinking, and unbearable global emotional states that are similar to fragmenting terror, homicidal rage, and hopeless despair. In addition, parents with character problems subject therapists to intense projective identifications that encourage empathic failures. All these factors are difficult for psychodynamic therapists, but not extraordinarily so in parent psychotherapy. However, other extraordinarily powerful and disruptive psychological reactions are inevitably elicited.

Such reactions stem primarily from three sources that force therapists to contemplate difficult, often unresolved, moral judgments about themselves, judgments that are central to the meaning of their lives. First, identifications with the hurtful parent force a therapist to contemplate memories of choices they have made that have hurt others in love relationships. Although there are important differences among individuals, every therapist has painful memories of hurting some loved one. No therapist, however mature, could ever become fully invulnerable to the painful guilt and shame associated with such memories. For therapists who are themselves parents, memories of hurting their own children are particularly difficult to revisit.

Second, identifications with the child force a therapist to contemplate the extraordinarily confusing experience of a child who is being mistreated, humiliated, and betrayed by a parent who also loves that child and who is basically committed to being a good parent. Such an experience for the child is complicated by a context in which the dominant parent and the entire social world of the child, sometimes including a misguided parent therapist, collude to "silence" the child by denying that the child's experience of the parent's betrayal is even in part valid and by insisting that the child accept a false interpretation of reality that depicts the parent as only doing as much as possible to be helpful. Thus, the child experiences as real both his or her own valid perceptions of the parent's decisions to hurt and also the incompatible misinterpretations that are imposed by that child's dominant social reality. Similarly, the child experiences his own rage and despair as both a valid response to the parental betrayal and a malignant response to trustworthy caregiving.

Third, dual empathy for both the hurtful parent and the hurt child forces a therapist to think about the everyday indulgence in human evil by us all. Most hurtful parents are ordinary, good enough people, not too different from therapists, who, without being subject to extreme pressure or coercion, do choose to betray their fidelity to the well-being of their own children, one of the most unusually important moral commitments in any society. Therapists who contemplate the immorality associated with good enough ordinary people hurting their own children are vulnerable to terror that is associated with attending to the high potential for unethical acts in our neighbors and ourselves. Such contemplation also stimulates the guilt that is associated with being reminded of the responsibility we all bear for the ways we choose to tolerate the degrading mistreatment of other human beings in our own extended families and communities, not to mention that of the millions of people in remote, underdeveloped areas of our world. Every therapist is vulnerable to such intense terror and guilt when reminded that his or her daily life is embedded in a society that routinely harms and degrades classes of people and fails to do all that could be done to help those who are being hurt by others.

## **Therapists' Denial and Defective Moral Reasoning as Defense**

When such extraordinary powerful psychological reactions are not managed well, problems arise that disrupt effective diagnosis and treatment. Therapists look away from the serious nature of the parenting problem either by using denial or by adopting an exclusively narrow, short-term focus that allows therapists to divert their attention from the parenting disorder, the inner life of the parent, and the moral dimensions of the clinical problems. In addition, therapists may ignore the complexity of the parents' ethical infidelity, by engaging in unsystematic, dichotomous moral reasoning that results in exaggerated moral blaming of the parent, blanket exoneration, or the self-deluding pretense of abstaining from any ethical judgment.

Therapists engaging in exaggerated blaming often think of the parent in a caricatured fashion, as a villain. Only a total villain deserves unqualified blame, because he or she does not try to love the child. For a villain, nurturing child-rearing practices are employed only to avoid social and legal censure. Characterizing a parent as a villain protects the therapist from painful identifications with a hurtful parent who is a good enough ordinary person, loves the child, but is in part ethically responsible for decisions that betray that parent's commitment to protect the well-being of their own child. Exaggerated blaming also protects the therapist from some of the most painful aspects of identifications with a child who is being hurt by a parent. It simplifies the child's experience to fit the character type common in children's folktales, by ignoring that the child is not just being mistreated but is being betrayed by a primary caregiver who is loved and feared, who determines the validity of child's own experiences, and who also loves that child and wants to treat the child well.

Blanket exoneration of the hurtful parent is usually achieved by the therapist who chooses to believe that the parent is trying as hard as possible to love and treat the child well but, compelled by forces beyond human control, is completely powerless to be less hurtful. In such instances, the therapist adopts a simplistic view of the parent as a martyr to the burdens of caring for a child that no parent could manage successfully or to a set of circumstances that would leave any parent too handicapped to care for a child that other more fortunate parents could enjoy. Blanket exoneration can also be achieved if a therapist relies exclusively on the concepts, methods, and findings of natural materialist science. Using such natural science concepts, parenting problems can be characterized only as patterns of thoughts, feelings, and behavior that are solely determined by natural forces interacting within a complex biopsychosocial system. Such false exoneration of the parent also protect the therapist from painful identification with the hurtful parent who is a good enough ordinary person, loves the child, but is in part ethically responsible for decisions that betray that parent's commitment to protect the child's well-being.

In the most extreme instances of blaming, therapists are not able reliably to suppress their contempt for the villain parent who persists in hurting the seemingly innocent child. In the most extreme instances of parental exoneration, therapists experience conscious contempt for the bad child or pity for the totally incompetent parent. However, such blaming and exoneration are sustained by experienced therapists most often because they are masked and therefore unexamined. They are masked by the false conviction that the therapist should abstain from ethical judgment of the patient to be "professional." Such a prohibition against moral judgment is associated with a misleading standard that is often mistakenly referred to as "scientific objectivity," "therapeutic neutrality," or a "nonjudgmental therapeutic stance." ([Langs, 1982](#); [Schafer, 1983](#))

All these common defensive strategies are almost impossible to sustain unless therapists allow themselves to accept an overly simplified psychological understanding of the clinical problem, the child, the parent, the parent-child relationship, and the therapeutic process. Such overly simplified psychological thinking cannot be sustained, in turn, without being buttressed by inappropriate clinical theory and clinical science. The inappropriate clinical theory that most therapists use to sustain these defensive strategies involves the rote application of established standardized methods of disease identification and management, methods that exclude the complex psychological meaning of the child to the parent and the moral dimensions of parental choice and responsibility. The inappropriate clinical science most therapists use to sustain their defensive strategies involves the exclusive misapplication of the language, concepts, and methods of natural science to formulate clinically defined defects in the mental lives of parents.

A clinical formulation that is based exclusively on the knowledge of determinist natural science automatically excludes the parent's agency on which the moral dimension of parental responsibility depends. The parent is likely to be characterized simply as ignorant, socially disadvantaged, incompetent, character disordered, or psychiatrically disordered. The child is characterized simply as genetically vulnerable, temperamentally difficult, or psychiatrically disturbed. The proper treatment is characterized simply as the reliable application of specific empirically validated standardized interventions to modify the specific symptoms or signs of identified disorders.

In addition, the defensive strategies, denial, and dichotomous thinking about the moral dimension of parenting problems are almost impossible to sustain if the therapist can maintain dual empathy for both parent and child or if the therapist can hold onto a focus on the long-term primary goal of parenting assessment and treatment, which is the long-term well-being and healthy mental life for the child. To engage in denial of the parenting problem, the therapist must suppress empathy for the child. Simplistic blaming of the parents requires that therapists suppress empathy for the parents. Such empathic failures are self-evident. However, empathic failure associated with unqualified exoneration of the parent who is not taking responsibility for the hurtful behavior is less easy to comprehend. A comprehensive consideration of the various types of empathy and of the components of the empathic process is needed. In contrast to instances of blaming, a therapist who simplistically exonerates a parent may retain empathy for many aspects of the parent's life experience outside the hurtful interactions with their child. In addition, an exonerating therapist may even maintain a capacity for some components of empathy for a parent's experience within the parent-child relationship, for example, intuitively sharing a parent's conscious experience of anger toward a child who refuses to be comforted or a child who chooses to destroy property and hurt other people.

However, the therapist who has completely exonerated a parent as a result of defensive moral reasoning will not be able to achieve empathy that encompasses the

complexities and paradoxes that make up the unique conscious and unconscious subjective experiences of a hurtful parent who both loves and hates and who is both victim of powerful forces and responsible perpetrator of mistreatment. For example, a therapist who simplistically exonerates parents can comprehend the frustration of parents who cannot comfort a child they love, but that therapist will not be able to share important aspects of the guilt, shame, and helplessness that accompany the rage and terror that parents unconsciously experience for a crying child who cannot be comforted.

### **Practical Consequences of Therapists' Denial and Defensive Moral Reasoning**

The practical problems that stem from therapists' denial of parenting problems and defective moral reasoning disrupt all critical phases of the work with parents. First, the clinical assessment may be inadequate. Although the most glaring abnormalities of parenting behavior may be noted, much more common problems in the emotional atmosphere of the parent-child relationship are usually overlooked. Similarly, clinicians may ignore most parenting problems altogether to focus exclusively on the child's psychiatric disorder and on genetically determined etiologic neurobiological factors.

Second, the formulation of the clinical problem may be inadequate. In a defensive frame of mind, when clinicians formulate ongoing problems in parenting behavior, they are prone to ignore the central role of unconscious parental thoughts and feelings about the child. They tend to understand the inappropriate parenting behavior simply as the result of the parents' ignorance about the child's development or ignorance about the child's psychiatric disorder or as a direct result of the child's difficult behavior and symptoms, parental psychiatric disorders, physical illnesses, marital problems, family problems, or environmental stressors. Based on overly simplified understandings of the problem that exclude the most critical aspects of the hurtful parent's mental life, clinicians most often recommend only individual treatment for the child with no treatment for the parent or only child guidance, psychoeducation, environmental manipulations, respite, or case management, when psychodynamic parent psychotherapy is required. Even when the parents' mental life is included in the formulation, the central defect in parenthood is often ignored, and as a result individual psychotherapy, marital therapy, or family therapy is recommended when psychodynamic parent psychotherapy would be much more effective.

Third, when clinicians engage in denial or defensive moral reasoning, the implementation of the psychodynamic psychotherapy is often ineffective. In the course of a parent treatment, the parent psychotherapist may lose empathy for the parent, lose empathy for the child, assume a rigid prescriptive treatment style, or allow the parent to avoid the thoughts and feelings associated with the parent-child relationship by inappropriately drifting toward other spheres of the parent's inner life.

### **Guidelines to Help Manage Therapists' Reactions**

The approach to the management of therapists' strong psychological reactions recommended here relies heavily on the use of practice guidelines. Effective personal psychotherapy is essential, but it cannot possibly completely insulate therapists from the strong psychological reactions discussed here. Reducing the therapist's activity, increasing vigilance about following the ground rules, and making conscious efforts to be morally neutral limit some of the detrimental consequences of these strong reactions, but they do not adequately protect the therapy. However, the six principal guidelines suggested in this chapter are required to sustain the therapeutic frame of mind required for psychodynamic parent psychotherapy. In addition, case consultations and peer supervision are often necessary. Consultation and supervision are most helpful if they involve psychodynamic therapists who are experienced in working with children and parents, have been striving already to work in a fashion that addresses the moral dimensions of the parenting problems and the treatment, and are familiar with the six primary guidelines presented here.

Current psychoanalytic theoreticians clarify the role of the therapist's conscious judgment and choice in the empathic process. Systematic professional approaches to conscious self-scrutiny are used primarily to detect and to rectify various countertransference distortions and impediments. In addition, disciplined self-scrutiny is used to prepare the way for a more complete kind of empathy, in which a therapist actively and consciously projects his or her consciousness into the inner world of the parent, as an experiment in thinking and feeling, to grasp intuitively a more complete sense of the experience of that parent ( [Buie, 1981](#); [Flax, 1994](#); [Margolis, 1989](#)).

The six guidelines recommended in this chapter are expressions of unspoken norms of psychodynamic psychotherapy. Although they are not explicitly articulated in the clinical theory that guides most psychodynamic psychotherapists, many of them have been deeply ingrained in the training of psychotherapists through clinical mentoring. These guidelines are meant to supplement, not supplant, the use of established guidelines of psychodynamic psychotherapy, standardized, "evidence-based" (e.g., DSM-IV) classification of psychiatric disorder and consensus parameters of intervention for identified psychiatric disorders. The approach advocated here is built on a moral foundation that includes a commitment to each child's inner life and autonomy, the particular meaning of the child to the parent, the unique meaning to each parent of specific aspects of his or her history and circumstances, and the parent's ethical responsibility to uphold fidelity to the child's autonomy and best interests. These aspects of the clinical situation can hardly be addressed at all if therapists rely solely on the language, concepts, and methods of evidentiary-based medicine. Similarly, such aspects cannot be adequately addressed, even when a therapist is able to make wise use of theory and technique from both the evidence-based approach and psychoanalysis. Hence, the guidelines suggested here are essential supplements to established practice.

#### ***GUIDELINE 1: THE PARENTAL MENTAL PORTRAIT OF THE CHILD***

A full evaluation of parenting should always be organized in accordance with a model with the parental mental portrait of the child at its center, because this model best helps the therapist to implement the primary goal of the treatment, that is, lasting improvements in the parent-child relationship that promote the long-term well-being, autonomy, and healthy mental life of the child. The parental mental portrait is a psychodynamically informed description of enduring patterns of thoughts and feelings, primarily unconscious thoughts and feelings, about a particular child. The parental mental portrait model for the formulation of parenting disorders has the parental mental portrait at its center. The model embeds the parental mental portrait in the context of a dynamic interactive systemic profile of that parent's particular psychological, biological, social, economic, and political circumstances and gives priority to the unique characteristics of the child and the parent-child relationship. In addition, the formulation of the portrait should reflect a developmental perspective informed by a thorough psychological history of the parent, the child, and their relationship in the family unit. Most important, the formulation of any parenting problems should always include an understanding of the parent's conscious decisions about how to think, feel, and act in relation to the problems experienced as a result of the disorder of the mental portrait. It should include the variety of competing and often incommensurate values that are relevant to those decisions and also the parent's effort and capacity to make wise judgments and to act with courage and discipline to implement them. Clinicians should reject as incomplete any formulation of the parenting problem that undervalues the importance of the parent's unconscious thoughts and feelings, ecological circumstances, developmental processes, or psychological history. Similarly, clinicians should reject any formulation of a parenting problem that does not explicitly address the parent's ethical responsibility adequately.

#### ***GUIDELINE 2: SUSTAINING DUAL EMPATHY FOR BOTH PARENT AND CHILD***

Parent psychotherapists should consciously discipline themselves to achieve, monitor, and sustain dual empathy for both the hurtful parent and the hurt child, throughout the diagnostic and treatment phase of the work, because dual empathy is an essential condition of successful parent psychotherapy. Empathy for the child is required to resist the tendency to deny the parenting problem, to achieve a full appreciation of the clinical problem, and to prevent defensive drift of the therapeutic focus to other pressing concerns. Empathy for the parent, which includes an appreciation of the parent's ethical responsibility for choices that are harmful to the child, is required to make accurate judgments about the parent and to sustain the professional healing relationship required to manage the suffering associated with the exploration of unconscious mental processes.

#### ***GUIDELINE 3: ASSESSMENT AND ATTRIBUTION OF ETHICAL RESPONSIBILITY***

The parent therapist should make a carefully reasoned assessment of the responsibility of a parent for making decisions to act in ways that perpetrate harmful behavior toward the child or fail to seek or accept help. Deliberate, sound assessments of the parent's ethical responsibility, which are essential to successful psychotherapy, are distinguished here from defensive moral judgments that disrupt diagnosis and treatment. Such a professional assessment of responsibility is needed for many reasons. It is required to help the therapist avoid inadvertent defensive moral reasoning. Therapists also need to assess parental responsibility to help parents both to forgive themselves for their own irresponsible behavior and to assume full responsibility for their choices.

The experience of forgiving oneself, taking responsibility, accepting appropriate guilt and shame, and making appropriate internal changes and reparations relieves excessive guilt, shame, and other kinds of psychological suffering that are associated with moral self-deception. The experience of forgiving oneself relieves suffering by liberating parents to experience the child more realistically, to analyze negative dystonic thoughts and feelings associated with the child, to mourn the psychological losses associated with parenting, to identify the hurtful behavior accurately, and to pursue their own value-based life goals in parenting and in other areas of their adult lives.

Parents also need to help their children, other family members, and friends to understand and forgive them for the hurtful behavior. However, children also hurt



parents in a disturbed parent–child relationship, and parents need to forgive their children and to help them to forgive themselves because children, like parents, are responsible for their own hurtful behavior. Although especially important in psychodynamic parent psychotherapy, an honest assessment of various immoral behaviors is needed to help almost all psychotherapy patients understand better the role of freedom and responsibility in their efforts to pursue their own life goals and values.

Therapists should use a nuanced, informed, and flexible assessment of parental responsibility. The assessment should be based primarily on values implicit in child psychiatry as it is practiced in our secular, pluralistic, free society, but also on the personal values of the therapist and those of the parents, their family, religion, and ethnic culture. The assessment of parental responsibility is very difficult to do constructively. Such an assessment requires systematic consideration of many different factors, but even with an extensive profile of data, it is still very difficult to judge accurately how courageous and hard working a parent has been at various times and in various contexts.

Parents are often more desperate than they know and than anyone around them can tell. In view of these complexities, a therapist's assessment always should be tentative and performed in a spirit of generosity, empathy, and respect. Responsibility for hurting the child should be estimated initially and reevaluated often during the course of treatment. In the course of ongoing psychotherapy, reassessments should be performed collaboratively with increasing leadership by the parents. However, helping parents to acknowledge ethical responsibility requires caution, respect, tact, and empathic support, because relinquishing their own caricatured self-image of victim, martyr, or incompetent person can be extremely dangerous for some parents at certain times in their lives.

#### *GUIDELINE 4: PRIMARY ADVOCACY FOR THE MENTAL LIFE OF THE CHILD*

Parent therapists should explicitly adopt and advocate for changes in the parent–child relationship that promote the optimal inner life and long-term well-being of the unique individual child as the primary goal of the parent treatment, although the parent is the patient engaged directly in the therapy. Explicit commitment to the long-term well-being of the child as primary is required by therapists to manage their own reactions to the moral failings of the parents who are their therapy patients and to resolve ethical conflicts about treatment goals, conflicts that arise when the needs of the children are in conflict with needs or preferences of their parents. Similarly, but not unique to parent psychotherapy, an explicit commitment to the child's long-term well-being, inner life, and autonomy is required to protect the children's needs when they are in conflict with the needs of insurance companies, hospitals, outpatient clinics, or the financial needs of the professional. In the presence of these very real conflicts, it is often difficult and painful for clinicians to keep the child's unique needs primary.

The commitment to the child's long-term well-being is expressed in an explicit advocacy for the development of healthy qualities in the child's mental life. A value-based view of an optimal “healthy” inner life for that child ( [O'Rourke et al., 1992](#) ) is defined by accepted norms of our liberal democratic society, Western medical traditions, and a legacy of centuries of experience in caregiving. Such norms are implicit in the aspects of child psychiatry, pediatrics, and child development that are focused on achieving enduring changes in the mental life of children. Similarly, an implicit understanding of a “healthy” parental mental portrait is one that supports best the development of a healthy inner life for a child.

In practice, the clinician should discuss the explicit value-based child advocacy position during the initial evaluation to build the initial treatment alliance with a parent and to guide the decision to recommend psychodynamic parent psychotherapy in the first place. Many very valuable clinical interventions for children or parents are not primarily committed to enduring improvements in the child's inner life. Although the goals of relieving symptoms, managing disorders, and improving social maladjustment are usually complimentary to the unique value-based personal goals for the individual patient, they are sometimes incompatible for a particular child, with a particular history, at a particular time, in a particular circumstance. In circumstances in which there is a parenting disorder, interventions that successfully help the child to achieve good social adjustment to a parent–child relationship that remains psychologically destructive to the child are inconsistent with a primary commitment to support the development of healthy personality, including self-determination, self-respect, and freedom from inner torment. Similarly, successful interventions to relieve symptoms and maladjustment most often are associated with improvements in the child's inner life, but sometimes the child's inner life remains unhealthy, although that child has become relatively asymptomatic. In such cases, giving priority solely to the child's social adjustment leaves the child's personality development unprotected.

Therefore, clinicians should accept treatments for a child that are directed solely toward symptom relief, disease management, and improved social adjustment only when they best serve the particular child's inner life and long-term well-being. Short-term relief of pain and improved adjustment in family, school, and other important life contexts are always subordinate goals to a healthy long-term quality of inner life for a child. Parent therapists should advocate for psychodynamic parent psychotherapy on behalf of the child whenever the potential benefits to the child outweigh the costs and risks for the child, even if parents prefer a less expensive or less painful approach.

The advocacy position provides a place to stand from which a parent therapist can consciously and systematically review the costs and benefits of a parent entering into parent psychotherapy. Such a value-based position is required to decide properly when to encourage parents to endure pain, to accept personal risk, or to assume expenses on behalf of their child. The explicit advocacy position also helps clinicians to decide when they must vigorously oppose strong financial and administrative incentives from insurance company managed-care or school systems to do only the most inexpensive, focused treatments. ( [Hoffnung and Ferholt, 1997](#) ) Similarly, during the course of a treatment in progress, a commitment to the inner life of the child and his or her long-term well-being helps therapists to sustain a focus on the parenting problem and especially on the parental mental portrait of the child and the emotional tone of the parent–child interactions. For example, the advocacy position helps parent therapists to resist parental pressures to support the parent's social adjustment, the parent's fulfillment in another love relationship, or other aspects of the parent's life goals in ways that conflict with the long-term well-being of that parent's child.

#### *GUIDELINE 5: PROTECTING CLINICAL JUDGMENT BASED ON APPROPRIATE VALUES*

Clinicians should diagnose each parent's clinical problems and determine all aspects of individual patient care by using expert clinical judgment. The primary goal of the treatment, which is the long-term well-being and autonomy of each unique child, serves as the primary guiding principle for all clinical judgments. However, many other values are taken into account, as are a wide range of data, formulated from various perspectives.

Good clinical judgment requires an appropriate assessment of the clinical situation, including reliable data about the personal characteristics of the parent, child, parent–child relationship, and family that is gathered in the context of a traditional, professional, caregiving relationship. Such good clinical judgment also requires an appropriate formulation of the clinical data, a model to guide the synthesis and organization of the data, that is informed by the best available knowledge and theories in the natural and various human sciences. Such a formulation should encompass various accounts of the clinical situation as viewed from diverse perspectives and levels of organizational complexity, including biological, psychological, behavioral, interpersonal, family, social, cultural, spiritual, and moral dimensions.

Good clinical judgment must be based on the consideration of many different alternative actions including those drawn from a diversity of professional orientations, including, descriptive, psychoanalytic, cognitive, learning, existential, interpersonal, and family systems perspectives, while taking into account the best current biobehavioral technology and the available resources. Algorithms for the management of psychiatric disorders cannot be used to determine treatment decisions, but they are useful as one important factor to consider when making individualized clinical judgments. Clinical judgment is informed by calculations of the likely therapeutic outcomes of such actions by taking into account the unique circumstances of the patient and the likely losses, risks, and economic costs of those same actions.

The cost-to-benefit and risk-to-benefit determination for any action or inaction that is being considered not only should be based on the primary goal of treatment, but also should take into account several other sources of competing and often incommensurate values ( [Sider, 1984](#) ). Such values include the unique life goals of the parent in treatment, values appropriate for the particular child, values of any other parent, those of other important members of the family, explicit medical ethics, appropriate professional medical mental health values goals and standards, and the psychiatrist's personal values ( [Hundert, 1987](#) ).

The expert clinical judgments that are used to determine all aspects of care should be made by professionals of the parent's choice in collaboration with the well-informed, relatively autonomous, uncoerced parent in treatment, often including consultation with both parents and sometimes the child and other family members. Such collaborative judgment requires an adequate professional caregiving relationship characterized by fidelity, privacy, empathy, respect, and compassion.

All clinical judgments about care must be held to the standards of professional practice, which are designed to serve the well-being of the individual patient and to do no harm. Professional standards that guide professional judgment are only valid when they are established, maintained, and implemented by a profession that is relatively independent of commercial pressures from large employers, government bureaucracies, the vicissitudes of politics, and the self-interest of the professionals. A full discussion of the training, supervision, support, and conditions of practice that are required to ensure and protect adequate standards of clinical judgment is not

feasible here.

#### **GUIDELINE 6: ADOPTING APPROPRIATE CLINICAL THEORY AND CLINICAL SCIENCE**

The misapplication of concepts and methods appropriate for the study of the material world to the way in which clinicians think about diagnosis and treatment undermines the primary practice guidelines that clinicians need to manage the moral dimension of work with hurtful parents. To sustain these guidelines, parent psychotherapists need explicitly to endorse an approach to science that can support a body of clinical theory that is appropriate to guide clinical judgment, to protect dual empathy, and to provide a credible basis on which to defend the values, goals, standards, guidelines, and conditions of practice required to protect psychodynamic psychotherapy from both the powerful internal psychological reactions to the moral dimension of parenting problems that undermine it and the powerful external commercial forces that threaten to eliminate it ([Ferholt et al., 1986](#); [Hunter et al., 1991](#)).

First, clinicians must retain a strong conviction that valid empirical science is not limited to the methods appropriate to study of the biological and other aspects of the natural world, but it also includes the concepts, methods, and data that are uniquely suited for the rational, systematic, reliable, and valid study of important human phenomena. Such phenomena include consciousness, autonomy, self-definition, identity, moral responsibility, love relationships, and rational judgment based on complex systems of human values ([Berlin, 1954](#); [Eddington, 1957](#); [Ferholt et al., 1986](#); [Harré, 1972](#); [Koch, 1959–1963](#); [Krutch, 1953](#); [Manicus and Secord, 1983](#); [Searle, 1993](#); [Spence, 1982](#); [von Bertalanffy, 1968](#)).

Second, while admitting the serious limitations of the extensive body of scientific research on professional caregiving, parent therapists must achieve the self-confidence and philosophical sophistication required to reject the misapplication of the language and methods of biology, economics, commerce, or industrial management to the study of clinical caregiving. They must also be able to resist efforts to eliminate the language, concepts, standards, guidelines, and practice conditions required to support professional caregiving and to refute the false view that dismisses clinical judgment based on individualized caregiving values as the unscientific art of medicine, an art that requires little professional expertise because it is based solely on folk wisdom, intuition, and basic human kindness. In addition, therapists must be able to resist the dismissal of individualized judgment-based professional caregiving based on the false claim that it is less essential than standardized evidentiary-based disease management.

Third, parent therapists must insist that clinical theory guiding individual patient care, although informed by behavioral and biological data generated in controlled clinical trials, must not be derived from the direct application of natural science knowledge. This is because many of the uniquely human experiences that are most important to clinical care include complex psychological variables that are not determined by the same types of predictable causal relationships that characterize the laws and theories of the biological and behavioral sciences. The foundation of good clinical theory must rest on human science that gives priority to the more thickly descriptive empirical methods required to study of the most important clinical phenomena that are not readily reducible to easily measured biological or behavioral variables without compromising their validity.

For example, complex mental data and reliable scientific empirical methods that are not experimental in design are essential to the rational systematic study of the complex, conscious and unconscious, dynamic, mental life of parents and children, freedom of choice, moral dilemmas, love relationships, family culture, mind–body interactions, and individualized patient care, including various relationship-based psychodynamic psychotherapies. In fact, they are the only scientific methods that offer valid knowledge suitable to help clinicians to determine treatment goals and to apply the knowledge and technology of natural science in a manner that properly reflects the unique values, needs, and situation of each individual patient.

Fourth, parent therapists should employ clinical theory that pluralistically, rather than eclectically or syncretically, combines many sources of knowledge from both the natural sciences and human sciences ([Havens, 1987](#)). A full discussion of the best way for clinicians actually to use explicit clinical theory, scientific knowledge, and technology in clinical practice is not feasible here. Briefly, good practice requires a professional healing relationship and value-based clinical judgment, both of which, in turn, depend on a good therapeutic clinical stance. The psychotherapeutic clinical stance is the frame of mind or, as Schafer puts it “a version of a therapist's personality or second self,” that is shaped especially for a particular psychotherapeutic role. The clinical stance depends on both the personal qualities and the training of the therapist, and it is focused by a small group of basic clinical concepts and guidelines. A parent therapist regularly brings such clinical concepts and guidelines to mind to generate and sustain the best therapeutic stance, even under pressure.

#### **PRACTICAL SUGGESTIONS FOR THE INITIAL PHASES OF TREATMENT**

The clinical evaluation of a child should take place over several sessions, spaced over several weeks, if possible. In addition to the history of the child and family, it should include direct observations of the child alone and the emotional tone of the parent–child relationship and other family interactions. Once a parenting disorder is identified, the psychiatrist should consider recommending psychodynamic parent psychotherapy even when a parenting disorder is not the reason the evaluation was initiated. For example, psychodynamic parent therapy may be the best recommendation for some adults who present to therapists with a chief complaint of marital problems, or even psychiatric symptoms or their own, especially during pregnancy, the postpartum period, or during their child's adolescence.

Usually, both parents should be involved in the treatment (conjointly or individually). Too often, fathers still are neglected in or excluded from treatment ([Ferholt and Gurwitt, 1982](#)). Occasional sessions including the children and their parents and sessions of the whole family are useful, but the children should most often be seen in concurrent individual therapies of their own, even if they are participating in family sessions, are not obviously disturbed, or are very young.

The same therapist may be able to perform a treatment for both the child and the parents; however, it is often best for two therapists to work in close collaboration. The collaborative arrangement is often required when the child needs very intensive and private treatment. Such an arrangement may also be essential to avoid intense unmanageable transference reactions in the parent, which are dangerous to the child and the therapist, if the child is an adolescent who is misbehaving or acting defiant in the family or when either an adolescent or a parent has a severe character disorder or a tendency to be dishonest and vengeful. Further, when it is clear that a parent has serious psychopathology, it is helpful for the parent therapist to insist at the outset on individual therapy for the vulnerable parent, in addition to and concurrently with the parent therapy. Serious clinical crises do occur regularly in these treatments, and some are serious enough to cause concern about murder, suicide, divorce, a major psychiatric episode in a family member, extrusion of the child (or one of the parents) from the family, or a lawsuit, grievance proceeding, or other form of attack on the therapist by a parent.

#### **PRACTICAL SUGGESTIONS FOR ONGOING TREATMENT**

A therapist's self-knowledge about his or her own psychological reactions to hurtful parents can be used to help parents to sustain self-esteem, to enhance empathy for their victimized children, and to learn better strategies to manage their parenting defects. By using an intersubjective formulation of the therapeutic relationship ([Bollas, 1987](#); [Little, 1981](#); [Searles, 1979](#); [Tansey and Burke, 1989](#)), carefully selected countertransference disclosures can be especially helpful. However, the intersubjective approach to psychodynamic formulation and transference analysis should not be allowed to become the primary technique in this work.

Therapists should deliberately strive to enhance every parent's self-respect by expressing appreciation for the parent's courage and discipline in the therapy and by helping the parent to identify the stressors impinging on him or her and the pain associated with them. In some cases, therapists should educate parents to help them develop a parenting stance guided by the same ethical and psychological tenets underlying the basic guidelines of parent psychotherapy. Such a parenting stance can be applied constructively to family decision-making and to a parent's conduct in interactions with a child.

For example, when a child feels hurt and acts sullen or angry in response to a parent's behavior, it is not useful for the parent to view the event simply as a matter of the parent's behaving inappropriately or the child's being overly sensitive. Rather, it is most useful to think of the interaction in terms of shared responsibility. Often in such instances, a parent should learn ways to acknowledge responsibility for behaving in a way that hurt his or her child, even if the parent's behavior is conventionally appropriate and would not have hurt most children, or even this child at other times. Acknowledging responsibility for hurting the child may even be part of the best course for a parent who has acted only in response to angry, rude, or destructive behavior from the child. Parents are advised to extend themselves to their children in this way to accommodate the vulnerability of children who feel insecure about themselves or unsafe with that parent and in the world. Many children are too guilt prone to think or talk about their responsibility for a hard interaction until after their parents first concede some regret for hurting them. Although these are simple truths, applying them over and over again in the context of the trials and tribulations of every day family life is extremely difficult.

In some cases, the therapist must also teach parents to avoid circumstances that elicit their own destructive states of mind, to recognize destructive mental states early when they inadvertently recur, and to manage them with discipline. Ironically, this often requires that the parent recruit the help of others, including, at times, the



child who may have provoked the very state of mind that the parent is trying to manage.

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#### CASE ILLUSTRATION

Although the discussion in this chapter focuses on the more severe disorders of parenting that are most difficult to treat, many cases that require psychodynamic parent psychotherapy are less difficult. This case illustrates the course of parent treatment relatively free of countertransference disturbance. Dual empathy is sustained in good part because the therapist has adopted the clinical schemes purposed in this chapter. This case also demonstrates the characteristics of a more subtle disorder in the emotional tone of the parent-child relationship than that in Jason M.'s family.

#### Case History

Douglas, age 7 years, in the second grade, lived with a 3-year-old sister, an infant brother, and his parents, who were both college educated, professionally trained, and financially secure. Over the past year, he had gradually developed an oppositional defiant disorder with anxiety symptoms. Douglas had early indications of poor neurobehavioral self-regulation, and as he grew older, he was unusually intolerant of frustrations and transitions and prone to brief periods of exaggerated developmental anxieties, unusual fears, and withdrawal associated with sadness and self-criticism. Conversely, Douglas also showed evidence of intrinsic resilience and acquired competence.

Douglas's psychiatric disorder developed in association with parenting problems and marital discord at the time his parents were forced to cope with severe environmental stressors. Both parents were competent and well motivated; they wanted to address the family problems before they asked Douglas to be in treatment. Thus, the initial child psychiatry intervention selected in this case was parent psychotherapy, performed during weekly, 50-minute sessions, almost always attended by both parents.

#### Therapy

A trusting therapeutic relationship was built with both parents during the first months in the course of gathering data, discussing formulation and diagnosis, seeking solutions to pressing management problems, and providing a cognitive framework for sustaining empathy and understanding for Douglas. This was a kind of psychoanalytically informed psychoeducation offering a clinical theory of parenting to help create a better mental stance in the parents when they were interacting with their son. The parent counseling also focused on the organization of family life, the collaboration of the parents, and the practical management of difficult situations involving Douglas.

The parents came to feel less frightened, confused, and self-critical about their parenting, improved the schedule and behavioral rules within the household, criticized each other less, and arranged for the father to spend more time with his son and to take more leadership in discipline. They learned to anticipate difficult periods for their son, to avoid some, to prepare him better for others, and to respond more constructively when he did become very upset.

They also learned an unconventionally generous way to respond to their son's oppositional defiant behavior, based on an interactive formulation that interprets his defiant behavior as a result of his one-sided interpretation of their actions, not as a problem with the development of his conscience or his capacity for impulse control, sustained attention, social skills, or moral discipline. They used an unconventional way of sharing responsibility for their son's misbehavior while sustaining a clear-cut although attenuated set of limits and expectations associated with unnegotiable, enforceable, reasonable consequences for transgressions. Over time, Douglas began to seem more comfortable and to behave better.

The nature and procedure of psychodynamic psychotherapy were explained, and gradually the conversations in the therapeutic sessions shifted focus to feelings the parents had for each other, those they each had when Douglas behaved in ways that hurt them, and those associated with their early memories about Douglas. The father's negative feelings, once exposed, were easily modified. Douglas's mother, on the other hand, discovered intense feelings of frustration, deprivation, guilt, shame, and anxiety in association with a tendency to slip into a frame of mind dominated by an ego-alien prejudice that reflected a negative mental portrait of her son. In this primarily unconscious frame of mind, the mother viewed Douglas as a self-centered, ungrateful, manipulative, dishonest, immoral person who was dominating, exploiting, and abusing her because she was not good enough for him no matter how hard she tried to make him happy. He dominated her by provoking her guilt, threatening to hurt her, and threatening to abandon her.

A crisis in the mother developed as she experienced an onslaught of very uncomfortable thoughts and feelings about interactions with her parents, now and in her difficult childhood. With a great deal of support from her husband at home and in the sessions, a strong relationship with the therapist, the help of several dear old friends, and a brief course of antidepressant medication, she managed to sustain herself during a period of intense grieving associated with uncovering very painful repressed memories of early experiences and a reconsideration of her current experiences with her parents. As the conversations about her past were elaborated, the therapist guided them to retain a focus on Douglas, and the connections between the feelings and thoughts she had about her mother and her son emerged without interpretation.

Over the time this work was being done in therapy, dramatic improvements occurred in the tone of the interactions between Douglas and both parents, his oppositional defiant disorder was resolved, his dysphoria was reduced significantly, and he became more expansive in his life outside the family. After 18 months (approximately 70 sessions), the parents were ready to stop the conjoint parent psychotherapy, and Douglas's mother decided to seek her own individual psychotherapy. Douglas did not require medication or individual psychotherapy.

#### Comment

The psychiatric disorder in this child may well have been ameliorated by any one of a variety of interventions, or it may have even dissipated without professional intervention. Thus, the parent-child interactions would have improved as well; however, it is argued in this chapter that without psychodynamic parent psychotherapy the improvements in the parent-child relationships would have, almost certainly, been more narrow, superficial, and transient, and the child's personality development and long-term well-being would have been compromised.

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## CONCLUSIONS

In conclusion, a disorder of a parent's mental portrait of a child is always painful and destructive. If it is severe and persistent, it can create a tormented inner life for the child, can destroy the qualities of personality we value in this culture, and can contribute to the creation of an adult prone to crush the inner lives of others. A serious parenting problem also adversely affects the course of a psychiatric disorder in the child, no matter how prominent the neurobiological substrate. Child psychiatrists and other mental health professionals responsible for children should (a) be proficient at discerning the presence of parenting disorders even in the absence of deviant child-rearing practices, (b) be competent at performing psychodynamic parent psychotherapy, (c) engage parents in this kind of treatment more often, and (d) advocate for changes in the health care system that will restore the powers, resources, and time that professional child psychiatrists need to restore, maintain, and implement standards of care that include the assessment of the inner lives of parents and appropriate diagnosis and treatment when defects in parenting are identified.

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## 87 PSYCHOTHERAPIES: A CRITICAL OVERVIEW

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### About this Review

#### Some Fundamental Questions about Psychotherapy

##### 1. Can Psychotherapy be Validly Researched?

##### 2. Does Psychotherapy Work?

##### 3. If It Works, Just How Good Is It?

##### 4. Is Psychotherapy Better than Placebo?

##### 5. Is One Kind of Psychotherapy Better than Another?

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#### Summary and Conclusion

#### Chapter References

The power of one person to comfort, to teach, or to influence others is a universal part of human experience or, in short, has incontrovertible face validity. Psychotherapy is merely an attempt to capture this power within a healing context, to systematize it so it becomes transmissible (and researchable), and to regulate it in a helping professional relationship for the protection of the consumer and therapist alike. In this section on treatment, some chapters address the various kinds of psychotherapy used with children and adolescents. In general, these chapters focus on the theory, practice, and clinical indications of particular methods. Beneficence of outcome and an established place in the therapeutic armamentarium and in teaching programs in child and adolescent psychiatry are largely assumed.

In this review, the field of psychotherapy is examined as a whole and is subjected to critical scrutiny using the assumptions and value systems accepted in medicine and other helping professions; that is, the best way to establish efficacy and safety and to advance theory is through the scientific method, based on the null hypothesis or, as Bertrand Russell put it, not doubt, but dogmatic doubt.

However, the scientific basis of medicine is rather recent and was made possible only by enormous growth first in the physical sciences and then in the biological sciences in the 19th and 20th centuries. Yet medicine is as old as the first civilizations, all of which defined certain individuals as healers and expected them to care for—and cure—the sick and the dying, whether they had the knowledge to do so or not. Given the lack of effectiveness and downright noxiousness of most medical remedies until the 20th century, the wonder is that medicine survived at all as a profession. Cynics could say that this is mostly the result of the patient's fear of death and need to believe in a savior and the result of the skill of the medical profession in disguising its impotence and chicanery, as portrayed by Racine, George Eliot, and Mark Twain, among others. However, also important in the continued survival of the medical profession is what is portrayed in the famous, if sentimental, 19th-century painting *The Doctor* (in the Tate Gallery in London), in which late at night, a doctor sits, thoughtful and troubled, at the bedside of a sick child. It is said that the painter, Sir Luke Fildes, executed this work in gratitude for the most attentive care given to his child. (The fact that the child died does not seem to have diminished this gratitude!) The physician was perceived as trying all remedies, but more important, as being there when needed and showing care and concern.

Medicine is thus equally a humanistic and a scientific profession, and the spectacular growth in technologic knowledge has been matched, it seems, by increasing public concern about the diminution in personalized, humanistic care. In addition to its own value system, medicine is influenced by the political, economic, and cultural ecosystem that shapes its form and dictates its execution. It is thus necessary to temper any critique of what doctors do with due consideration for all these factors besides the scientific.

Psychotherapy has some distinctive features that differentiate it from many other medical treatments. First, its practice is not limited by statute to licensed physicians, and there are those who will argue that the rules of medicine thus do not apply. However, it can be said equally that physicians who practice psychotherapy are still so obligated, and that should be one of the discernible differences between psychiatrists and nonmedical professionals. Second, although medicine did originally depend on charisma and caste to establish validity of treatment, this has gradually faded considerably in favor of hard data from well-designed clinical trials (evidence-based medicine). For a variety of reasons, some of which reflect the sheer complexity of the subject although others are less worthy, traditional child psychotherapy was and is still largely dependent on charismatic leaders and innovators rather than on data ( [Werry, 1989](#)).

## ABOUT THIS REVIEW

Since the previous edition of this textbook was published in 1996, there has been increased interest in the issue of psychotherapy research with children. In 1998, two important journals devoted issues to descriptions of current status, problems, and outcomes in psychotherapy research: the *Journal of Abnormal Child Psychology* and the *Journal of Clinical Child Psychology*. One aspect of this interest has been definitions of psychotherapy, which are increasingly attempting to include issues of patients' collaboration with the therapist and the context of therapy. An example is [Brent and Kolko's \(1998\)](#) definition that psychotherapy is a modality of treatment in which the therapist and patient work together to ameliorate psychopathologic conditions and functional impairment through focus on the therapeutic relationship, patient attitudes, thoughts, affect and behavior, and social context and development. This definition remains independent of the technique or underpinning theory, whether psychodynamic, client-centered, behavioral, individual, play, group, family, brief and long term, and so on.

What is striking is the generic nature of this definition, which may appear simplistic to practitioners of particular modalities of psychotherapy. However, there is both a utility and a good reason for keeping the definitions broad. First, with a few exceptions, research has demonstrated that much of the therapeutic power stems from a set of variables common to all kinds of psychotherapy, however different they may seem. Second, many kinds of child psychotherapy are insufficiently explicated to examine their putative differences from each other. Third, there are insufficient data to examine all kinds of child psychotherapy individually, and aggregation of studies, despite all the problems therein, is necessary to obtain sufficient statistical power. Finally, those who think that their kind of psychotherapy is superior, despite insufficient data to support their contention, may be motivated to do the studies necessary to prove the point.

Unless otherwise stated, in this chapter psychotherapy is described as applied to children, with *child* taken to include infants, children, and adolescents. This is not an exhaustive review of psychotherapy. Detailed analysis of individual studies is not undertaken because these already exist ( [Barnett et al., 1991](#); [Casey and Berman, 1985](#); [Faubert and Long, 1991](#); [Hazelrigg et al., 1987](#); [Henggeler et al., 1993](#); [Kazdin, 1991, 1993](#); [Kovacs and Paulauskas, 1986](#); [Ollendick, 1986](#); [Shirk and Russell, 1992](#); [Target and Fonagy 1996](#); [Tramontana, 1980](#); [Weiss et al., 1999](#); [Weisz and Weiss, 1993](#); [Weisz et al., 1987](#); [Weisz et al., 1992](#); [Weisz et al., 1995](#) ). Rather, some basic questions are posed, and some of these reviews are used in response. The answers are cast in terms of their implications for research training and practice in child psychiatry.

## SOME FUNDAMENTAL QUESTIONS ABOUT PSYCHOTHERAPY

Our overview of 1996 in the earlier edition of this text posed a list of 12 questions that are, in our opinion, fundamental to improving treatments in this area. Since this first overview, some further issues have arisen. However, it is striking that many of these questions remain of relevance and regrettably still unanswered several years.

1. Can psychotherapy be validly researched?
2. Does psychotherapy work?
3. If it works, just how good is it?
4. Is psychotherapy better than placebo?
5. Is one kind of psychotherapy better than another?
6. Can psychotherapy be taught?
7. Is more psychotherapy better than less? Or what is the dose effect for psychotherapy?

8. Is psychotherapy better for some children than for others? How does the child's context (e.g., family, social, ethnic) influence the effectiveness of psychotherapy?
9. Is psychotherapy safer than biological therapies?
10. Is psychotherapy acceptable to consumers?
11. Is psychotherapy cost effective?

## 1. CAN PSYCHOTHERAPY BE VALIDLY RESEARCHED?

As all the cited reviews show, one of the more persistent problems in evaluating psychotherapy is the lack of sufficient good research to answer the questions under discussion. There are those who believe that psychotherapy is not measurable because it concerns the elemental nature of human beings not only within the individual, but also as interacting with others such as parents or therapists. Indeed, this argument was used to dismiss the findings of some of the first attempts to evaluate psychotherapy such as the American Institute of Psychoanalysis study of the 1960s. Much of the good research on traditional individual psychotherapy with children (excluding behavior therapy) was done in a flurry of optimism between 1963 and 1973 ([Barnett et al., 1991](#)), although since the mid-1990s there has been a resurgence of interest in the area, perhaps driven by the funding issues that force clinicians to produce data to support their approaches ([Kazdin, 2000](#), [Salzer et al., 1999](#), [Weisz et al., 1995](#)).

None doubts that the task of researching psychotherapy is extremely complex and, as with applied research in humans, very difficult to pursue, but that should be a challenge, not a deterrent. Science operates by removing only one skin of the infinitely layered onion of Nature at a time, and although the task will never be complete, the success of science in our everyday lives is there for all to see. Is any research about psychotherapy better than nothing? Clearly, if the result is meaningless nonsense, or worse, misleading sense, the answer must be no. However, the rules for good psychotherapy research were set out many years ago ([Heinecke and Strassman, 1975](#)), and methods by which to enact these rules, especially those derived from adult psychotherapy, have advanced considerably since then ([Barnett et al., 1991](#); [Kazdin, 1991](#); [Kazdin, 2000](#); [Shirk and Russell, 1992](#)). Add to this advances in the technology of measurement in psychiatry as a whole and in adult psychotherapy ([Bergin and Garfield, 1994](#); [Kazdin, 1991](#)) and in other areas of child psychiatry (e.g., psychopharmacology), and there is now a formidable body of knowledge to facilitate this process. In particular, Kazdin's book is full of practical suggestions for research that must surely end the argument that psychotherapy cannot be properly researched once and for all ([Kazdin, 2000](#)).

In any case, a reviewer does not get the sense from serious writers on psychotherapy (apart from gurus) that the dearth of good research in most forms of psychotherapy except behavior therapy results from a lack of tools to do the job ([Barnett et al., 1991](#); [Shirk and Russell, 1992](#); [Shirk and Russell, 1995](#)). What appears to be lacking is the will. There are those who would defend child psychotherapy, especially its traditional forms, on the grounds that it has not been adequately studied ([Shirk and Russell, 1992](#)), however, such research as there is with children suggests that the findings do not differ qualitatively from the much vaster, more robust, and more active field of psychotherapy research in adults ([Kazdin, 1991](#); [Kazdin, 1993](#); [Kazdin, 2000](#)). Thus, it is possible to draw on this fund of knowledge ([Andrews, 1993](#)) to make tentative statements for child psychotherapy when child data are inadequate. Further, the issues of methodologic complexity and shortcomings in studies have been overstated. It has been shown ([Weiss and Weisz, 1990](#); [Weisz et al., 1995](#)) that even when the effects of methodologic flaws are allowed for in metaanalyses, substantial effects of psychotherapy are still discernible.

There is one serious criticism that clinicians may legitimately level at research in psychotherapy—to meet the requirements of rigor (and publication within the time span imperative to success in promotion, reputation, and grantsmanship!), there is little resemblance between what researchers study (i.e., efficacy of treatment) and what practitioners do (i.e., treatment effectiveness) ([Kazdin, 1991](#); [Ritvo and Papiłsky, 1999](#); [Weisz et al., 1992](#)). For example, Kazdin lists several major differences such as subject recruitment, locus, duration and theoretical orientation of treatment, and involvement of parents ([Kazdin, 1991](#)). There is also the issue of adequate follow-up because delayed (sleeping) effects have been demonstrated ([Kazdin, 1993](#)). The answer to this problem seems to be clear—there is a need for practitioners to research themselves or actively to promote and assist in the process, rather than have unfriendly and uncomprehending researchers speaking for what they do. If psychotherapists fail to do this, then they have no right to complain that research rarely reflects the real world of clinical practice. How to bridge this gap is discussed in detail by [Weisz et al. \(1995\)](#).

## 2. DOES PSYCHOTHERAPY WORK?

There are now more than a thousand controlled studies that look at different treatments for children and adolescents ([Kazdin 2000](#)). With the exception of one reviewer ([Levitt, 1957](#); [Levitt, 1963](#)), all others, especially the metaanalyses ([Barnett et al., 1991](#); [Lonigan et al., 1998](#); [Weisz et al., 1987](#); [Weisz et al., 1992](#); [Weisz et al., 1995](#)) involving hundreds of studies, conclude that, *independent of the kind of psychotherapy, treatment is better than no treatment*. Some have been concerned that this comforting convergence of results conceals serious problems of method, which either inflate the efficacy ([Shirk and Russell, 1992](#); [Shirk and Russell, 1995](#)) or raise doubts about the validity of conclusions ([Barnett et al., 1991](#); [Heinecke and Strassman, 1975](#); [Klein, 2000](#)). Although these concerns do have some legitimacy, as already noted they have been overstated, especially when there are sufficient studies for a metaanalysis ([Weiss and Weisz, 1990](#); [Weisz et al., 1995](#)). Thus, it seems quite in order to say that the evidence does favor efficacy and thus that the practitioner can claim that psychotherapy is a *legitimate treatment*, although, as discussed later, this statement is subject to caveats regarding for whom, for how long, and for which kinds of psychotherapy.

## 3. IF IT WORKS, JUST HOW GOOD IS IT?

If, as just concluded, psychotherapy is better than no psychotherapy, just how often is it so and to what degree? In their metaanalysis of more than 100 studies, [Weisz et al. \(1987\)](#) found the effect size was 0.79; that is, 79% of children were better off treated than not—a very substantial proportion. Only 6% of children were made worse. In clinical practice, then, this means that the odds that psychotherapy will help are very high, provided, of course, that approximately the same criteria for selecting patients used in most studies are applied, which they are not ([Kazdin, 1991](#)), although the algebraic summation of these differences is probably zero, because some would favor and others disadvantage practice. This may explain why [Weisz et al. \(1987\)](#) did not find much difference between genuine clinic and “analog” studies (e.g., recruited subjects).

However, the actual *amount* of benefit produced is less impressive, accounting for less than 20% of the outcome variance ([Weiss and Weisz, 1990](#)). This finding suggests that the *average effect* of psychotherapy is rather small, although most practitioners would probably consider a 20% improvement as realistic and valuable. In addition, as discussed in more detail later under cost:benefit ratio, the measures used may not do justice to the flow on effects of psychotherapy beyond mere clinical improvement.

As the question of efficacy continues to be debated, interest grows in the role of theory. Kazdin (1999) argues that theory has been almost entirely neglected, the consequence being that there is little to guide us about how treatment works and how to refine it. This theme has been expressed by others as well ([Ritvo and Papiłsky, 1999](#); [Weisz and Hawley, 1998](#).) A further issue has been the lack of developmental focus of much research to guide such areas as timing of outcome measures or even planning and executing of research ([Eyberg et al., 1998](#)).

## 4. IS PSYCHOTHERAPY BETTER THAN PLACEBO?

In his review of adult psychotherapy, [Andrews \(1993\)](#) points out that, although psychotherapy has been demonstrated to be better than no treatment, few studies try to find out whether psychotherapy is better than “placebo” or, in short, has effects not solely dependent on expectancy, ordinary human caring, status of the therapist, and what Andrews calls “good clinical care.” Even in adult psychotherapy, this question appears still to remain unanswerable ([Andrews, 1993](#)). Of all the psychotherapies, behavior therapy has by far the greatest amount of evidence to suggest that its effect extends beyond mere placebo ([Andrews, 1993](#)), although there are those who would query the clinical relevance of some of its effects.

Nearly all the studies of child psychotherapy compare its effects against no treatment or alternative forms of psychotherapy, thus making estimate of the placebo effect impossible. Kazdin ([1991, 2000](#)) and [Fonagy \(1997\)](#) point out that it is often practically difficult to design effective placebo-type procedures. For example, how does the researcher ensure that therapists have the same commitment and enthusiasm to the placebo as practicing therapists have to their treatment? What are the ethical and legal implications of common placebo conditions such as random assignment to treatment or no treatment? These issues may be complex but cannot be avoided: as differences between placebo and psychotherapy emerge, then second-stage research to identify the variables responsible for the superiority becomes economically and ethically justifiable. However, as Kazdin writes, “random assignment is preferred but it ought not to be worshipped” ([Kazdin, 2000](#)). The state of evidence in the area is such that almost all questions remain open or doubtful, and findings from even well-formulated naturalistic studies could be of considerable use.



## 5. IS ONE KIND OF PSYCHOTHERAPY BETTER THAN ANOTHER?

In their review, [Casey and Berman \(1985\)](#) conclude that there is no difference among therapies, although protagonists for traditional psychotherapy seem to be unaware that data were sufficient to compare only behavioral and client-centered therapy. [Weisz et al. \(1987\)](#) criticize the way that Casey and Berman's evaluative methods disadvantage behavioral studies, add more studies, and conclude in their metaanalysis that behavior therapy is clearly superior to other therapy in children. Similar conclusions are reached by [Andrews \(1993\)](#) for adult psychotherapy, for which he estimates the size of the superiority to average at least half a standard deviation. In rebuttal, Shirk and Russell ([1992, 1995](#)) conclude that it is not possible to say that behavior therapy is superior to psychodynamic psychotherapy in children because the studies of the latter have been so flawed as to make them invalid. [Kazdin \(1991\)](#) also believes that the issue is undecided because of lack of data in most methods other than behavioral. However, [Weiss and Weisz \(1995\)](#) find little support for this hypothesis using metaanalysis. Furthermore, Weisz et al. (1999) have examined nine studies of traditional child therapy and have found an average effect size of only 0.01. None of these studies included randomized design: when such a study of 160 children was completed, the effect size shrank to  $-0.08$ .

Thus, whereas the answer is still not clear, there is some indication that behavior therapy may be superior to other forms of psychotherapy (although only by half a standard deviation, which may not be a great deal in terms of clinical effect). This is particularly true if the symptom focus of behavior therapy is accepted as a legitimate focus of all psychotherapy. Other forms of psychotherapy do themselves a disservice in their persistent reluctance to conduct research because most of the apparent superiority of behavior therapy resides not only in more data but also in the rather narrower and therefore more measurable targets ([Kazdin, 1991; Weisz et al., 1995](#)).

Since the last edition of this book was published in 1996, there have been both an expanding study of some modes of psychotherapy and an increased likelihood that specific problem-domains rather than general symptoms will be selected for study. It is now possible to state that a few treatments are empirically supported, for example, systematic desensitization for anxiety, fears, and phobias, cognitive-behavioral therapy for depression, classroom contingency management for attention deficit disorder with hyperactivity, and parent management training for conduct disorder ([Kazdin, 2000](#)). Kazdin notes the extreme brevity of the list (12 treatments in all, a marked contrast to the 551 psychotherapies listed in an appendix in his book) and points out the ongoing dominance of behavioral and cognitive-behavioral methods.

## 6. CAN PSYCHOTHERAPY BE TAUGHT?

Training is taken as essential for the practice of any kind of psychotherapy, and some modes, notably psychoanalysis, go to extraordinary lengths in training. However, is there any evidence that these training programs improve efficacy and efficiency of therapy? Research on training in child psychotherapy has suggested that it has little discernible effect on therapist efficacy ([Shirk and Phillips, 1991](#)), and in fact, the evidence suggests that training may have negative effects ([Weisz et al., 1987](#)). Again, in the carefully controlled Newcastle study ([Kolvin et al., 1988](#)), it was found that untrained teacher aides were more effective than psychologists. A similar result was found by [Weisz et al. \(1995\)](#). However, most of the studies examining this issue are seriously flawed in that training was often confounded with other critical variables, such as method of psychotherapy. Also against the value of training, graduates of training programs often view their training as seriously defective, although this may be not so much a problem with training itself but with the way in which it is done. Finally, there is lack of apparent difference in efficacy of therapists from differently oriented training programs ([Shirk and Phillips, 1991](#)). There are, however, persistent indications that some persons make better therapists than others ([Bergin and Garfield, 1994; Shirk and Phillips, 1991](#)). Moreover, the type of problem treated may interact with training in a complex way ([Weisz et al., 1995](#)).

Collectively, these uncomfortable facts suggest that whatever is needed to do psychotherapy may well be acquired by the trainee before or after training is undertaken—because the critical ingredient in psychotherapy may not be so much theoretical framework or technique, but some nonspecific personal capacity to relate to other human beings, melded into the combination of other humane or common sense things that go to make up Andrews' "good clinical care." If this be so, it is hardly surprising that a training program has little impact on a trainee who has been learning human relationships for most of the life span and will continue to do so. [Shirk and Phillips \(1991\)](#) point out that because of the lack of any real effort to relate research findings to the practice of psychotherapy, training lacks the kind of explicit goals that could improve its efficacy. Even cognitive-behavioral therapy, the one type that seems to have clearly explicated training methods (often in manuals), does not seem to have looked at the efficacy of training.

## 7. IS MORE PSYCHOTHERAPY BETTER THAN LESS?

The average duration of treatment varies greatly across psychotherapies, practitioners, and researchers. It is therefore reasonable to ask whether more is better than less. Although analogies are hazardous, pharmacotherapy accepts the concept of an optimum dose—too low and it is ineffective, too high and it becomes poisonous. Research on dose effects of psychotherapy in adults ([Andrews, 1993; Koss and Butcher, 1986; Orlinsky and Howard, 1986](#)) is limited, most of it is flawed to some degree, and there are almost no comparative data on really long-term treatment (2 years or more). However, the general conclusion is that the dose-response relationship is logarithmic. Most of the gain occurs within the first 10 to 20 sessions, the size of the effect is enhanced by therapist's and patient's knowing that therapy is time limited, and the effect may even diminish in some patients with longer periods of therapy ([Koss and Butcher, 1986](#)).

There are few data in child psychotherapy on dose effects ([Andrade et al., 2000; Kazdin, 1993; Salzer et al., 1999](#)), but what data there are do not support the idea that more is better ([Casey and Berman, 1985](#)). There is also the disconcerting fact that dosage is dictated largely by patients rather than by therapists and by what underwriters will pay rather than by clinical considerations ([Andrews, 1993](#)). If psychotherapists want to convince underwriters that more is better, they will have to do some urgent research in these days when the push is to reduce health care costs ([Krupnick and Pincus, 1992](#)). The adult literature ([Orlinsky and Howard, 1986](#)) and that pertaining to children ([Fonagy and Target, 1994](#)) suggest that such research is worth pursuing.

## 8. IS PSYCHOTHERAPY BETTER FOR SOME CHILDREN THAN FOR OTHERS?

One of the more dramatic changes in child psychiatry since 1980 has been the rise of disorder-based classification. Pharmacotherapy has disorder-specific indications, and it seems plausible that diagnosis would affect psychotherapy, too; indeed, this seems to be the case in adults ([Andrews, 1993](#)). Child psychotherapy seems to have been slow to catch up with these advances, but it is now doing so to some extent. The list of empirically supported treatments for particular disorders is small, as already noted ([Kazdin, 2000](#)). This is clearly work that needs pursuing.

Other variables that ought to affect the efficiency and the type of psychotherapy needed (and are common clinical issues), but that are only just beginning to be addressed, are comorbidity, concurrent pharmacotherapy, age, socioeconomic class, family variables, and culture ([Armbruster and Kazdin, 1994; Fauber and Long, 1991; Fonagy and Target, 1994; Kazdin, 1991; Kazdin, 1993; Kazdin, 2000; Tharp, 1991; Trainontana, 1980; Weisz et al., 1987](#)). For example, [Weisz et al. \(1995\)](#) found that adolescent girls may do better, a finding suggesting that more attention to such variables is required. Practitioners often complain that these are the factors that influence treatment to the greatest degree, yet very little work examines such issues.

## 9. IS PSYCHOTHERAPY SAFER THAN BIOLOGICAL THERAPIES?

It is also often assumed that psychotherapies are preferable to pharmacotherapy because they are less "invasive" and can therefore present no risk to life or limb. This may be true at a physiologic level, but psychotherapy is clearly not free of side effects of stress and emotional upheaval during treatment and may make an unknown percentage of patients worse ([Andrews, 1993](#)), although this number appears quite small in children ([Weisz et al., 1987](#)). In addition, up to 50% of child patients never complete treatment to the satisfaction of the therapist ([Armbruster and Kazdin, 1994](#)). The causes of this are complex and could include rapid response but may help to conceal adverse outcomes. Practitioners of pharmacotherapy recognize the need to monitor closely side and adverse effects. The lack of data on side and adverse effects of child psychotherapy clearly needs addressing, because it reflects practice based on unproved assumptions of safety and efficacy.

## 10. IS PSYCHOTHERAPY ACCEPTABLE TO CONSUMERS?

The findings that many children are considered unsuitable for psychotherapy, that up to 50% of those selected may drop out of treatment ([Armbruster and Kazdin, 1994](#)), and that some patients and families report negative experiences ([Andrews, 1993; Weisz et al., 1987](#)) suggest a gap of unknown size between consumers and therapists. The causes are complex but deserve careful study because acceptability to young people and families is likely to be an important variable in efficacy. Conversely, [Weiss et al. \(1999\)](#) found that even though little support was shown for the effectiveness of traditional child psychotherapy, parents of children receiving it

reported higher levels of satisfaction compared with parents of control children who received academic tutoring.

Finally, almost no data exist about the views of children and adolescents regarding their treatment. This lack raises the issue of outcome measures. How are decisions made about the kinds of outcomes that will be examined? As [Fonagy \(1997\)](#) says, “whose outcome is it anyway?” Outcome measures have often lacked practical utility, seldom moving far beyond whether children continue to be symptomatic. Clearly, there is a much bigger world out there.

## 11. IS PSYCHOTHERAPY COST EFFECTIVE?

This issue is reviewed in detail for adult psychotherapy by [Krupnick and Pincus \(1992\)](#). They conclude that, despite an impressive body of research attesting to the power of psychotherapy to alleviate destructive and painful illness, data needed to assess cost effectiveness are lacking. Although underwriters and administrators seem to think otherwise, this is a most complex issue, too much so to discuss here. (Interested readers are referred to the cited article and its reference list.) However, by way of example, one of the bigger problems is the failure of a substantial body of outcome research to use measures critical to the cost:benefit equation of the impact of treatment on functions such as education, creativity, work, social obligations, health, and social costs, rather than the usual estimates of improvements in illness-related variables, such as depressive or anxiety symptoms. Again, because of even greater paucity of data, the view with children is even more opaque. Although it seems agreed that psychotherapy is effective with children to some, if rather modest, degree ([Weiss and Weisz, 1990](#)), no answer can be given on whether it is cost effective. Conversely, the long-term costs of *not* treating children are equally unknown but are probably enormous ([Kazdin, 1993](#)). This suggests that good studies using the principles outlined by [Krupnick and Pincus \(1992\)](#) are likely to show the worth of psychotherapy in terms acceptable to the businesses that increasingly control both public and private resources for health care. It is worth noting, too, that it is not just a question of psychotherapy showing its worth—it will need to be shown that other methods such as social reforms or government policies will not do much better for considerably more children.

Although the cost:benefit ratio of child psychotherapy is unknown, some indicators suggest that cost efficiency could be improved a great deal. First, it does not make economic sense to use highly trained expensive staff such as psychiatrists to do what the research suggests can be done as well by less trained staff ([Kolvin et al., 1988](#); [Weisz et al., 1995](#)). However, there are a few signs that, in some areas, better trained staff may do better. Moreover, supposedly “untrained” staff in most studies have very experienced supervisors ([Weisz et al., 1987](#)). It may be more a matter of moving to the executive role in a team than discontinuing involvement with psychotherapy altogether. It is likely that [Andrews' \(1993\)](#) concept of good clinical care can be delegated, but management plans, treatment manuals, and some kinds of specific or complex treatments will, rather like complex surgery, still have to be done by experienced therapists.

Second, it seems likely that most of the benefit from psychotherapy occurs in less than 10 to 20 sessions and is facilitated by knowledge in both parties that treatment will be time limited ([Koss and Butcher, 1986](#)). Therefore, any longer-term therapy should be reserved for exceptional cases. For example, the finding of a crude logarithmic relationship between duration of therapy and effect ([Orlinsky and Howard, 1986](#)) means that the law of initial values applies; that is, when the disability or suffering is very large, longer treatment, though ever diminishing in effect, may still produce sizable changes simply because there is so far to go. However, the decision to extend treatment should be based on demonstration that psychotherapy is effective and, if so, for which particular disorders.

Third, behavior therapy seems more efficient than other psychotherapies, and it seems reasonable to make this the treatment of first choice (after good clinical caring). In addition, behavior therapy is remarkable for its emphasis on delegation of therapy by explication of targets, process, and outcome, thus reducing costs further. Until research can show the advantages of pursuing other forms of treatment and for which patients (as indeed seems most likely it could), it is hard to justify them, in the first instance.

Fourth, it is customary in some forms of therapy (e.g., family therapy, group therapy) to use cotherapists, and there may be further discussions among cotherapists and supervisors after each session. Although this is laudable in terms of peer quality assurance, it doubles or triples the cost of the treatment. Of course, this is offset if the alternative would be multiple, individual treatments. Research to see whether cotherapists and supervisors improve efficiency to the degree that they increase costs is sorely needed.

Fifth, in some disorders, other treatments such as biological therapies are likely to be far more cost effective. For example, methylphenidate produces much larger effects in the core symptoms of attention deficit hyperactivity disorder than do psychotherapies of any kind ([Barkley et al., 1993](#)), although there remain many problems that medication does not touch.

## SUMMARY AND CONCLUSION

The most significant finding is the lack of good data by which to make critical decisions about the teaching and practice of psychotherapies, especially those that dominate in child psychiatry. Paradoxically, in view of their continued popularity, the traditional psychotherapies have been little studied since the early 1980s ([Barnett et al., 1991](#)). Family therapy fares a little better ([Henggeler et al., 1993](#)) in that there seems to be more research activity, although not nearly enough has been addressed to outcome. No other method comes close to behavior therapy in amount and quality of research ([Kazdin, 1993](#); [Kazdin, 2000](#); [Weisz et al., 1987](#); [Weisz et al., 1995](#)).

Such data as there are reveal some disquieting findings that are at odds with the certainty with which psychotherapy is taught, practiced, and marketed, and this makes it seem, at times, more like a religion than a professional activity ([Weiss et al., 1989](#)). For example, serious doubts exist about the specificity of different treatments (except behavior therapy), the value of training, the high cost of psychotherapy, and socioeconomic and cultural limitations in application. Inexplicably, despite these doubts, traditional and family therapies continue to flourish and form a centerpiece of most training programs in child psychiatry.

In child psychiatry as a whole, there is currently a sense of excitement, an intellectual renaissance. Although fueled largely by biological psychiatry including taxonomy, epidemiology, molecular biology, and psychopharmacology, behavioral scientists are increasingly lending their specialized research and theoretical knowledge to round out this medical subspecialty, as any issue of the *Journal of the American Academy of Child and Adolescent Psychiatry* makes clear. Amid this intellectual ferment, psychotherapy still depends too much on tradition, charisma, extended preceptorship, assertion, and presumption of beneficence. In this respect, it resembles medicine of a bygone era, out of step with the 21st century. We hope that the sleeping giant is beginning to awake, as the generally impressive articles and reviews cited here show, although these are almost exclusively written by nonmedical behavioral scientists. As [Kazdin \(2000\)](#) notes, it is now possible to compile a list of empirically supported treatment of children. However, the list is short and does not include any traditional methods of child therapy, which still lags behind the intellectual activity in adult psychotherapy.

There is good reason to teach and practice behavior therapy, including cognitive-behavioral therapy, because it has a clear advantage in efficacy and in volume of supporting data, and its approach is generally more transmissible and heuristic. However, child psychiatrists need to overcome their idea that this is something that only psychologists do and move on to master, teach, practice, and supervise it.

Teaching and practice of forms of psychotherapy other than behavioral are less defensible unless they are perceived for the moment as simply part of good clinical caring, in which case, it is unlikely that prolonged training, high fees, or long-term or intensive treatment can be justified. The finding that many practitioners currently earn their living from long-term or traditional psychotherapies does not mean that child psychiatry training and practice can be exempt from the painful radical restructuring that is a feature of the economic world. As long as child psychiatry belongs to and wishes to avail itself of the privileges of being part of medicine, it must adhere to the fundamental ethical values that no treatment can be assumed to be effective and safe—it must be demonstrated to be so. The 21st century will be that of evidence-based medicine, and child psychiatry cannot afford to be left behind averring instead of proving.

The strongest justification for continuation of nonbehavioral psychotherapies is in research and in care of the severely ill, in whom proven treatments have failed. Even then, these treatments should ordinarily be delegated to less highly paid personnel to reduce cost—until such time, if ever, it is clearly demonstrated that child psychiatrists can do it better.

As discussed in the introduction, psychotherapy may be justified as an essential part of medicine's other arm—humanistic concern and care for the sick and their relatives ([Pardes, 1990](#)). Psychiatry must maintain its current prerogative among medical specialties of being able to take time to listen to children and their families and thus to exercise the traditional medical activity of humane clinical caring ([Pardes, 1990](#)). In view of the lack of distinctive effects in different kinds of psychotherapy, it seems likely that many practitioners of psychotherapy, when faced with a distressed patient, abandon most of the theoretical orthodoxy they were taught in favor of what seems sensible, caring, and ethical. Those practitioners who conceptualize children's suffering, handicaps, and maladaptations within traditional frameworks have no need to change if it enables them to deliver this most important element of medical care. However, there is a substantial difference between such a position and promulgating, teaching, or practicing most psychotherapies as proven treatments for child psychopathology, especially within child



psychiatry, which is supposed to show medicine's skepticism toward treatment and proper regard for the cost of health care.

Looking at the dramatic remedicalization of psychiatry since the early 1980s (Wilson, 1993) and the main thrust of current research, it seems clear that the role of the child psychiatrist in the 21st century increasingly will resemble that of other physicians (i.e., performing diagnostic assessments encompassing medical, psychological, social, and cultural issues; participating in multidisciplinary case conferences; delegating administration of all except especially skilled treatments) and thus will differ substantially from that of many teachers in the past, who spent much of their time on psychotherapy. Unless it can be shown that psychotherapy greatly enhances effectiveness in diagnosis and delivery of good clinical caring, there seems little justification for the predominance of nonbehavioral psychotherapy in child psychiatry training. Because skill in psychotherapy and clinical caring seems more related to personal qualities than to training, it may make more sense to concentrate on better selection of medical students and psychiatric trainees.

The time has come for psychotherapy to undergo a painful personal analysis and confront the nagging perennial questions about psychotherapy, by using the tools of science: What do we know about psychotherapy that is derived from research, not assertion or tradition? How can this knowledge be used to determine what sort of psychotherapy should be given, to which patients, for how long, at what cost, and by whom? How should psychotherapy be taught, if at all? What is needed is more research, more intellectual curiosity, and more excitement and less complacency, less dogma, less training, and less unexamined clinical practice.

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# 88 CHILD AND ADOLESCENT PARTIAL HOSPITALIZATION AND AMBULATORY BEHAVIORAL HEALTH SERVICES

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## DEFINITION

*Partial hospitalization* has been used historically as an umbrella term describing many varieties of alternative care. With the advent of managed care and health care reform, less costly and less restrictive treatment modalities are in increasing demand. To provide definition for these alternatives, which stretch along the continuum of psychiatric care between inpatient and outpatient office visits, three levels of ambulatory behavioral health care have been outlined ( [Kiser et al., 1993](#)). In this new environment, partial hospital programs (PHPs) are providing short-term, crisis stabilization services as an alternative to or as a step down from acute inpatient care. Day treatment programs (DTPs) are serving the needs of children and adolescents with moderate to severe disorders who require interventions focused on improved level of functioning, skill building, and disease management. Intensive outpatient programs (IOPs) or partial day programs function as intermediate or step-up programs from outpatient psychotherapy. These programs offer treatment for up to 3 hours per day and often accommodate children and adolescents after a half-day or full day at school ( [Robinson, et al., 1999](#)).

In the behavioral health delivery system, these services represent a specific segment along the continuum of care and offer more intensity than outpatient services without the iatrogenic effects of hospitalization. Ambulatory behavioral health care services strive to provide intense, highly structured treatment. They do so by using a variety of therapeutic modalities: individual, group, and family therapy, as well as medical and nursing services. This multimodal treatment requires an interdisciplinary staff typically drawn from psychiatry, psychology, social work, educational or vocational therapy, occupational or recreational therapy, and nursing. A working definition of ambulatory behavioral health care follows:

Ambulatory behavioral health care is a time-limited, active treatment offering therapeutically intensive, coordinated, and structured clinical services that incorporate the benefits of a therapeutic milieu within a program or within the home and community ( [Kiser et al., 1993b](#)).

## HISTORY

The history of ambulatory behavioral health care for children and adolescents is brief. The first PHPs were for adults in Russia (late 1930s), Canada (late 1940s), and England (early 1950s). The Menninger Clinic (Topeka, KS) introduced the concept to the United States when it opened its unit in the mid-1950s. (The closure of Menninger's partial hospitalization services in 2000 marked the end of an era.) In 1963, the Community Mental Health Center Act mandated comprehensive services including partial hospital care for children and adolescents. This movement had three main origins: deinstitutionalization, interest in the role of family and community, and movement to cost-effective treatment alternatives.

Throughout the late 1990s and into the present, the modality has been severely underused ( [Novello, 1979](#); [Prevost, 1981](#)). With only 2% of those in need of treatment receiving services in PHPs ( [Krizay, 1989](#)) and with more than 46% of programs operating at less than 75% capacity [ [Association for Ambulatory Behavioral Healthcare \(AABH\), Overview of the Partial Hospitalization and Ambulatory Care Industry, 2001](#) ], underuse is a major problem for the survival of many programs. Three clinical issues contribute to underuse of partial hospitalization. First, for providers, it is difficult to serve moderately to severely disturbed children and adolescents in an open system with less restrictiveness and a less structured environment. Second, for families, it requires major commitments to family therapy, to transportation, and to keeping a difficult child at home. Third, for referral sources more accustomed to and better trained to treat severely dysfunctional patients in inpatient programs, it necessitates a shift in the style and conceptualization of clinical practice. Fourth, reimbursement policies serve as a major impediment to wider use of PHPs. Traditionally, most policies with mental health care benefits had better coverage for inpatient than outpatient services and no coverage for PHPs ( [Leibenluft and Leibenluft, 1988](#); [Novello, 1979](#); [Weithorn, 1988](#)).

Now, with an emphasis on cost containment, managed care focuses on eliminating unnecessary inpatient services with efforts directed toward development of systems favoring outpatient practices ( [Hoge et al., 1992](#)). Although early indications from managed care pointed to a desire to ensure both quality and efficiency, fiscal management frequently translates to either rationing care or providing the cheapest services for the shortest length of time possible regardless of clinical decision-making and effectiveness data ( [VandenBos, 1990](#)). Cost-containment pressures are driving the development and use of less intensive options, such as IOPs or multimodal outpatient treatments. Approvals for care at the ambulatory level are sometimes based on cost without adequate consideration of the child's or adolescent's treatment needs.

## THEORETICAL ISSUES

The continuum of ambulatory mental health services is founded on six essential principles:

1. Services are designed for [children and adolescents] who present with a psychiatric and/or chemical dependency diagnosis and the need for treatment more intensive than outpatient office visits and less restrictive than 24-hour care.
2. Services provide a coordinated array of active therapies determined by an individualized treatment plan and based upon a comprehensive evaluation of patient needs.
3. Patients are treated in a manner that simulates real-life experience with the least amount of disruption to normal daily functioning [allowing for optimal transfer of therapeutic benefits to the natural environment].
4. Services require active involvement of the [child/adolescent and the family].
5. Services are available on a consistent basis, augmented with 24-hour crisis backup.
6. Services are cost efficient ( [Kiser et al., 1993b](#)).

From these six essential principles, two basic philosophies of care provide the core for child and adolescent ambulatory behavioral health services. First, these services provide treatment to patients requiring intensive therapeutic intervention with the least amount of disruption to their normal daily functioning. This requires ambulatory services to define the limits between when patients can be treated safely in an outpatient setting and when they require the restrictiveness of hospitalization. Thus, ambulatory behavioral health services are designed to maintain a balance between treatment in the least restrictive environment and risk management.

Second, ambulatory behavioral health care encourages providers to take advantage of the "open system" inherent in the modality. This philosophy emphasizes the use of family supports and strengths, as well as community agencies and programs. Two major sequelae result from this philosophy of care. Ambulatory services invite the patient and the patient family to maintain a higher level of functioning than treatment settings that remove the patient from the home. Philosophically, this

translates into a program of therapeutic interventions designed to maintain power within the parental and familial subsystem and to view that subsystem as competent in providing care for the child. Interventions are structured to alter dysfunctional interactional patterns rather than remove the child from these interactions. Translated into program policy, ambulatory behavioral health programs only use techniques and treatment strategies that a family can also implement at home.

Additionally, ambulatory programs promote reliance on community support systems and programs whenever possible. Benefits of this approach include limiting the development of dependence on the treatment team, expansion of and appropriate use of resources, decreased lengths of stay, and improved adjustment after discharge (Herz, 1982). This unique combination of providing “security and structure while simultaneously promoting patient responsibility and autonomy” (Hoge et al., 1988) may be the particular advantage of this modality.

The philosophies underlying ambulatory behavioral health care can be accomplished in programs with a variety of theoretical orientations. Behaviorally oriented programs use principles of learning theory. The goals of a behavioral program are, consequently, to teach desirable behaviors and to extinguish undesirable ones. For example, most PHPs, although not operating from a behavioral orientation, use some form of level system for determining the consequences of patients' behavior (Kiser et al., 1986). Another approach uses a psychoeducational orientation with the classroom as the basic structure for program design. “Students,” rather than “patients,” usually receive psychotherapy in conjunction with classroom instruction. Programs using systems theory emphasize the theoretical proposition that many different areas of a child's life influence functioning: biological, intrapsychic, family, peers, school, work, neighbors, religious affiliation, and others. Within a systems orientation, programs are challenged to assess problems and to intervene on multilevel basis (Kiser and Pruitt, 1991). Many of the programs operated in conjunction with inpatient services within general hospitals are based on a medical model in which all patients receive psychiatric diagnoses and treatment is prescribed by a child and adolescent psychiatrist (Novello, 1979).

These orientations differ in the manner in which they view the child, his or her problems, and the treatment program. Although many theoretical orientations are possible, within an organization, the model used should be consistent across the continuum of care. Conflicting models established among components of care may cause confusion for the staff as well as the patient and family.

## GOALS OF TREATMENT

The clinical functions of ambulatory behavioral health care differ according to the level of care offered (Glasscote et al., 1977; Kiser et al., 1993b). Partial hospitalization and other intensive nonresidential treatment options function to provide short-term crisis intervention and stabilization of symptoms as an alternative treatment to hospitalization or an intensive transition setting to shorten a hospital stay. Less intensive ambulatory modalities provide support, maintenance, symptom reduction, and skill-building to prevent relapse, longer-term hospitalization, or residential care. Finally, multimodal outpatient services provide a step up for patients for whom one or two office visits per week may be insufficient. Another goal or function of ambulatory behavioral health services can be extensive evaluation involving observation, identification of problem areas, diagnosis, and formulation of treatment plans (Casarino et al., 1982).

## CLINICAL ISSUES

### Indications

The continuum of ambulatory behavioral health care represents three levels of care, each of which targets populations with differing needs. The important tenet is that admission decisions are based on matching interventions and services with the needs of the patient in terms of intensity, structure, and accessibility, for example.

Treatment organizations define their target population by establishing admission criteria. From this programmatic standpoint, evaluation to determine appropriateness for ambulatory behavioral levels of care involves the following six factors: (a) psychiatric signs and symptoms, (b) level of impulse control, (c) level of functional impairment, (d) parental support, (e) physical health, and (f) ability to pay. Specific criteria for the three levels of ambulatory behavioral health care are presented in Table 88.1.

Class	Level 1	Level 2	Level 3
Psychiatric signs and symptoms	Current DSM diagnosis, acute to distressing symptoms related to acute or chronic condition	Current DSM diagnosis, moderate to severe symptoms related to acute or chronic condition	Current DSM diagnosis, moderate symptoms related to acute or chronic condition
Level of impulse control	Unstable control of impulses	Unstable control of impulses	Good control of impulses
Level of functional impairment	Severe difficulty functioning in multiple areas including school, activities, community, and home	Severe difficulty functioning in one or more areas including school, activities, community, and home	Moderate difficulty functioning in at least one area including school, activities, community, and home
Parental support	None to provide adequate control and support of program during meetings and weekends with close monitoring and frequent contact with staff	None to provide adequate control and support of program during meetings and weekends with monitoring and assistance from staff	None to provide adequate control and support of home during meetings and weekends with monitoring and assistance from staff
Physical health	No requirement for 24-hour medical care	No requirement for 24-hour medical care	No requirement for 24-hour medical care
Ability to pay	Economic resources available to support care of this level	Economic resources available to support care of this level	Economic resources available to support care of this level

Table 88.1. Admission Criteria for Child and Adolescent Ambulatory Behavioral Health Care

As pressures increase to manage children and adolescents with unstable, crisis presentations in less restrictive settings, it is critical for providers to develop decision-making tools for patient placement that take into account risk management. For child and adolescent providers, family and community support and structure become major factors in this decision-making process. A decision matrix, assessing both level of impulse control and parental support and structure, is a valuable tool for assessing appropriateness for admission (Fig. 88.1) (Kiser et al., 1991). Level of impulse control can be viewed along a continuum, with some patients demonstrating no problems and other patients requiring a structured, locked treatment environment because of severe problems with behavior control.

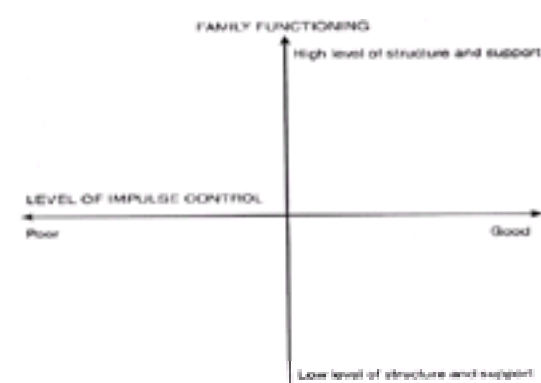


Figure 88.1. Decision matrix: risk management assessment. (From Kiser LJ, Heston JD, Millsap PA, et al.: Testing the limits: Special treatment procedures for child and adolescent partial hospitalization. *Int J Partial Hosp* 7:39,1991, with permission.)

On the other axis, the continuum of family functioning (support and commitment to treatment) is evaluated. It is expected that families will work with the treatment team to encourage their child to comply with program limits. Families who are unable or unwilling to participate in this manner are less likely to provide the daily support and structure needed by the patient, who is therefore unlikely to derive significant benefit from treatment in an ambulatory behavioral health care setting.



## PROGRAM OF THERAPEUTIC ACTIVITIES

The clinical components, that is, each patient's daily activities, are based on an individual treatment plan developed by the treatment team ( [Block et al., 1991](#); [Kiser et al., 1986](#)). Continuous monitoring of the patient's response to treatment dictates alterations in this plan. Therapeutic progress is monitored frequently by the multidisciplinary staff, and periodically, a treatment update is prepared and the patient's case is reviewed by the entire treatment team. Progress to date on problems is reviewed, new problems are discussed, and progress toward transition to a less intensive level of care or discharge is monitored.

A distinguishing characteristic of ambulatory behavioral health care is the intensity of diverse clinical components that most often include individual, group, family, and medical management. As with all treatment with children and adolescents, it is important that the structure and content of the therapy be appropriate for the patients' developmental levels.

### INDIVIDUAL THERAPY

Individual therapy is offered by most ambulatory behavioral health providers in the United States (AABH, Overview of the Partial Hospitalization and Ambulatory Care Industry, 1998; [Kiser et al., 1986](#)), usually on a weekly or twice-weekly basis, with a mean greater than 2 hours per week (AABH, 2001). Like individual therapy in other settings, it provides the patient with an opportunity to develop a close, interpersonal relationship with an adult other than a parent and an opportunity to work on intrapsychic problems.

Individual therapy in ambulatory behavioral settings differs from outpatient individual psychotherapy because of the wealth of information available to the therapist through contacts with or observations of the patient in other parts of treatment. An example of this occurs in a partial hospital setting when a depressed child denies withdrawal from peers but is frequently observed sitting alone during milieu periods. Differences between individual psychotherapy in ambulatory and inpatient settings are also significant and are mainly the result of the openness of the ambulatory system. Individual therapy in ambulatory behavioral health care can use material from daily encounters with parents, siblings, and peers to address dysfunction in these areas.

### GROUP THERAPY

Most ambulatory behavioral health services rely on group psychotherapies as a mainstay of their clinical programming. Group psychotherapy plays an important role in ambulatory behavioral health settings by allowing peers to develop the ability to deal with problems through the expression of feelings and experiences in a safe environment. This therapy also provides an opportunity for work on group dynamics, including cohesion, roles, and norms. Skill building is another important function of group psychotherapy. As with individual therapy, therapists in ambulatory behavioral health care have direct knowledge about patient functioning in multiple areas, thus allowing immediate and specific feedback to the group members.

### FAMILY THERAPY

The use of family therapies by many programs is consistent with the research demonstrating that family structure is predictive of successful completion of treatment and follow-up ([Prentice-Dunn et al., 1981](#)). Family involvement in treatment may take a variety of forms including traditional family therapy, multifamily groups, parent education classes, parent support groups, marital therapy, and in-home services.

Family therapy provides an opportunity to focus on family dynamics to facilitate change in the home environment. Within ambulatory behavioral health care, this therapy allows intensive work on family conflict and family structure on a day-to-day basis. Because patients remain part of the family while participating in treatment, new skills can be practiced and evaluated in a continuous manner. Another important benefit of family therapy in ambulatory behavioral health care is the ability to establish consistent rules and limits for the child both at home and in treatment. Work with the family and community helps to extend the therapeutic benefits of treatment into the patient's overall familial and social milieu. Finally, one powerful difference between family treatment in this modality and that of an inpatient unit is the emphasis on maintaining parental authority. For example, in partial hospitalization, parents are involved in every level of decision making and problem solving. Parents are, in effect, viewed as cotherapists.

### THERAPEUTIC ACTIVITIES

The extent of therapeutic activities such as expressive activities, daily living skills, diversional activities and the availability of a programmatic therapeutic milieu depend on the structure and intensity of the services offered. However, at all levels of ambulatory behavioral health care, providers emphasize development of an active, therapeutic milieu. In ambulatory behavioral health services that do not incorporate a programmatic milieu, the treatment plan prescribes active interventions to extend milieu-based therapeutic benefits to the home, workplace or school, and community, as needed. Recreation, movement, art, occupational, and milieu therapies, as well as community-based activities, provide staff and peer support for appropriate behavior in social settings. Patients learn constructive ways to use leisure time in activities that foster teamwork, cooperation, and task commitment. Frequent involvement in community events is consistent with the ambulatory behavioral health care belief that children and adolescents should remain active members of the community ( [Kiser and Pruitt, 1997](#)).

### EDUCATION

Because much of a child's life and often many of a child's problems revolve around school and related activities, intensive treatment programs for children and adolescents must grapple with ways to meet academic needs and difficulties. Ambulatory services meet the needs for educational services in very different ways. Intensive, full day programs may offer a school component daily ( [Block et al., 1991](#)). Acute programs with lengths of stay of less than 2 weeks may provide educational experiences based on a "homebound" model. Finally, less intensive ambulatory services, such as after-school programs or IOPs, may simply focus on enhancing school performance by offering tutoring or study skills and consultation with classroom teachers. Regardless of the level of care offered, ambulatory behavioral health care providers use the treatment environment to aid children in dealing appropriately with the frustrations encountered in the school setting, including dealing with authority and assuming responsibility for work. They also understand the importance of schooling in the lives of children and adolescents and thus view active school liaison as a major component of treatment. Obtaining fiscal support for the educational portion of treatment is an area of concern; cooperative arrangements with local education authorities are often necessary, thus requiring programs to meet several sets of standards and answer to several local and state agencies.

Provision of intensive mental health services integrated within the school setting is a growing trend in the provision of delivery of ambulatory behavioral health care to children. Programs of this nature are often joint projects of community mental health and the school system. Often these services are classroom based with lengths of stay that more closely resemble school grading periods. Individualized, multimodal treatment is offered on site delivered by an integrated, multidisciplinary team of educational and mental health professionals ( [Matzner, 1998](#); [Milin et al., 2000](#)).

### MEDICAL MANAGEMENT

The extent of medical involvement in the management of patients in ambulatory behavioral health care varies depending on the intensity and theoretical background of the program. Medical involvement within ambulatory behavioral health care ranges from medical supervision for patients with unstable, crisis needs to medical consultation for patients with low levels of risk and only moderate symptom presentations.

Trends are toward emphasizing aspects of the medical model in the most intensive ambulatory services such as PHPs. As various forces work toward decreasing rates of hospitalization for children and adolescents, more severely disturbed patients and those with combined medical and psychiatric disorders will be referred to PHPs. In such programs, psychiatrists function as attending physicians and members of the treatment team. Under this model, the physician can be seen as the team leader, orchestrating the treatment provided. The use of [Engel's \(1980\)](#) biopsychosocial model of medical practice is particularly congruent with the philosophy of ambulatory behavioral health care. Physician involvement is not limited to the specifics of medical management, but it includes substantial input into program administration and direction. The cost disadvantage may be counteracted by the preference of insurance companies and Medicaid to reimburse for medically directed services.

Thus, within programs offering acute symptom reduction and crisis stabilization, medical management includes an assessment of the patient's general medical and behavioral health status. Both medical and psychiatric diagnoses are assigned. This approach may involve direct physical examination done by a physician

associated with the program or, in those programs that are more community based, consultation and liaison with the patient's primary care physician or pediatrician. It is the responsibility of nurses and physicians to identify specific health problems that may be important factors in the overall functioning of the patient. These problems may be treated directly or referred to a community physician. Referrals often serve to educate and guide families in accessing needed health care services and to use these resources effectively.

Pharmacotherapy, another aspect of medical management, may be practiced within the structure of ambulatory behavioral health care with advantages over other therapeutic settings. As on an inpatient unit, observations from multiple viewpoints are available to determine specific indications for medications. The ability to monitor new medications or changes in regimens is augmented by daily contact with the patient and information about the patient's status in many different activities. This use of medication within a structured environment is frequently seen as an advantage over medication management developed on an outpatient basis. Moreover, in distinction to an inpatient hospitalization, the family is able to provide and to learn to provide ongoing feedback about medication response, side effects, compliance, or other concerns about medication.

In those programs that are designed as IOPs, psychiatrists may be contracted to function as consultants to provide specific, limited services. These services typically include initial psychiatric evaluation and diagnosis as well as monitoring pharmacotherapy. The psychiatrist may not be a part of the treatment team and is usually not involved in the day-to-day care of the patients or administration of the program. Although the advantages of this model are financial and logistical, and although they save the program the cost of a full-time specialist in a profession that is relatively undersupplied, the main disadvantage is the lack of physician input into the overall treatment of patients.

Finally, as managed care grows and continues to influence psychiatric care, physicians may be expected to help programs, patients, and their families to negotiate appropriate financial coverage for treatment. This assistance may come in clarifying diagnoses and in outlining treatment options. Frequently physicians in ambulatory behavioral health care are called on to participate in physician-to-physician reviews of cases that have been tagged as questionable by the managed-care company. The effective psychiatrist will be called on to document rationale for treatment in ambulatory behavioral health care and should be familiar with medical necessity criteria and effective arguments for obtaining treatment appropriate to the specific needs of each patient.

### Special Populations

Proponents of ambulatory behavioral health care suggest that it is a viable treatment mode for many special populations (e.g., juvenile offenders, patients with eating disorders, alcohol and drug abusers), as well as for nonpsychiatric populations (e.g., those with head trauma or epilepsy). Several reports of PHPs designed for adolescent offenders or those with adolescent antisocial behavior have appeared in the literature ( [Byrnes et al., 1999](#); [Comer, 1985](#); [Gaylor, 1979](#); [Kolko, 1995](#); [Matzner et al., 1998](#); [Myers et al., 2000](#); [Rey et al., 1998](#)). Treatment emphasis for offenders appears to be intensive group therapy and social, recreational, and special educational services, with generally positive outcomes noted for these teens, who are very difficult to treat.

Another special population described in the literature is preschoolers. Special emphasis in preschool PHPs and therapeutic nurseries for young children is placed on play, communication and language, socialization, skill development, and parent-child interaction. Young children benefiting from treatment in a therapeutic preschool include those with developmental delays, victims of physical and sexual abuse, and children with other severe emotional or behavioral disturbances ( [Rogers and Lewis, 1989](#)).

Health care reform and the interest in development of cost-effective treatments have resulted in programs that address the psychosocial as well as physical needs of chronically ill pediatric patients ( [Gavin et al., 1996](#)). Certain patients with eating disorders, sickle cell disease, asthma, diabetes, and other disorders may benefit from specialized treatment in ambulatory settings that incorporates health care professionals from appropriate pediatric specialties.

### Limitations and Complications

As various theoretical, ethical, economic, and other factors discussed earlier continue to discourage the use of hospitalization, ambulatory providers will be challenged to manage patients whose behavioral symptoms and impulsivity have escalated to the point that special treatment procedures are indicated. Policies and procedures for dealing with patients displaying aggressive, acting-out behaviors, noncompliance with program rules, and suicidal or runaway ideation are a necessary part of programming ( [Kiser et al., 1991](#)). Special treatment procedures designed for use in child and adolescent ambulatory settings must adequately address the issue of safety and must involve the family and the community, while maintaining the patient in the least restrictive environment.

Management of acutely and severely disturbed patients necessitates policies that address seclusion, physical holding, and restraints. Often PHPs simply do not offer these services because staff members believe that these procedures are appropriately used only in inpatient units. Other child and adolescent PHPs use quiet rooms and physical holding policies that do not violate the mandate of the program, in other words, procedures that can also be used by parents or guardians in the home environment. The quiet room and physical holding are used sparingly with adolescents; immediate transfer to a secure facility may be indicated for an aggressive or acutely psychotic, disruptive adolescent. Manual and chemical restraints are typically not used in PHPs or other ambulatory settings.

Just as programs must develop policies to help patients to control their behavior that are compatible with the program's theoretical basis and intensity of treatment, they must address similar issues with other problems both within the treatment setting and at home. For example, PHPs may be able to treat patients with significant suicidal ideation if these programs are able to maintain appropriate levels of observation and to plan for continued supervision after program hours using family therapy, telephone contact, beeper services, or other interventions. Runaway patients may similarly be placed in ambulatory treatment given policies for structure after hours and ability for liaison with community resources such as runaway homes or other community emergency shelters. Thus, intensive programs that are designed to serve as alternatives to inpatient treatment are able to treat severely disturbed patients using policies that address program structure, family resources, and community involvement.

Because of the intensity of programming, the developmental level and the severity of illness of patients treated, and the lack of restrictiveness, most ambulatory behavioral health care services operate with very small staff:patient ratios. Obviously, the level of staffing required depends on the intensity of care offered and the degree of disturbance within the target population. Within PHPs, a typical ratio is one staff person to fewer than four patients ( [Kiser et al., 1993a](#)), with a recommended range from 1:3 through 1:6 ( [Block et al., 1991](#)). For after-school programs or IOPs, staff:patient ratios of 1:8 to 1:10 are probably sufficient.

Even with small staff:patient ratios, the high intensity of the programs results in staff stress and burnout ( [Kiser et al., 1986](#); [Novello, 1979](#)). A fuller explanation no doubt lies in the philosophy of the approach: maintaining severely affected patients in the least restrictive environment.

### CRISIS MANAGEMENT AND HOSPITAL LIAISON

Ambulatory behavioral health care services that attempt to shorten or eliminate hospitalization must have adequate policies and procedures to handle emergencies that prompt immediate admission to treatment and the inevitable crises that develop during treatment. Although general principles are described earlier in the discussion of limitations and challenges, specific details of crisis management should be identified by each ambulatory behavioral health service, depending on the level of care offered.

At the most intensive level, providers of ambulatory care should have an integrated emergency system that allows immediate access to current clinical and treatment information. The following recommendations meet these crisis management needs ( [Kiser et al., 1986](#)): (a) an on-call child and adolescent psychiatrist available 24 hours per day, 365 days per year; (b) additional on-call services, to help families to resolve conflicts that do not require hospitalization or major changes in treatment plans, that are staffed primarily by partial hospital professionals; and (c) an established affiliation with both pediatric and psychiatric inpatient units. In the case of hospital-based programs, this should be addressed in policies regarding transfer between PHPs and inpatient units. In free-standing PHPs, formal affiliations with hospitals must be made so admissions can occur quickly and safely for patients with acute emergencies.

Less intense ambulatory programs must also develop emergency plans. These may include policies to admit patients to the PHP within a number of days and consultation with a psychiatrist to assist with decisions regarding emergency management and possible hospitalization. Coverage of crisis situations while a child or adolescent is in care can be handled by a 24-hour crisis call service. To provide a summary of ambulatory behavioral health care's indications and limitations, the advantages and disadvantages of the modality are presented in [Table 88.2](#).



Advantages	Disadvantages
1. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	1. Partial hospitalization programs are often expensive.
2. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	2. Partial hospitalization programs are often expensive.
3. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	3. Partial hospitalization programs are often expensive.
4. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	4. Partial hospitalization programs are often expensive.
5. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	5. Partial hospitalization programs are often expensive.
6. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	6. Partial hospitalization programs are often expensive.
7. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	7. Partial hospitalization programs are often expensive.
8. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	8. Partial hospitalization programs are often expensive.
9. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	9. Partial hospitalization programs are often expensive.
10. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	10. Partial hospitalization programs are often expensive.
11. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	11. Partial hospitalization programs are often expensive.
12. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	12. Partial hospitalization programs are often expensive.
13. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	13. Partial hospitalization programs are often expensive.
14. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	14. Partial hospitalization programs are often expensive.
15. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	15. Partial hospitalization programs are often expensive.
16. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	16. Partial hospitalization programs are often expensive.
17. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	17. Partial hospitalization programs are often expensive.
18. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	18. Partial hospitalization programs are often expensive.
19. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	19. Partial hospitalization programs are often expensive.
20. Partial hospitalization programs provide a continuum of care for children and adolescents with serious mental illness who do not require 24-hour care.	20. Partial hospitalization programs are often expensive.

**Table 88.2. Advantages and Disadvantages of Ambulatory Behavioral Health Care for Children and Adolescents**

**Discharge and Aftercare Services**

As integrated systems of care for child and adolescent become more prevalent, planning transitions between levels of care often replaces discharge planning for ambulatory services. Regardless, transition or discharge planning remains an integral part of treatment planning, beginning during the initial intake and continuing throughout the treatment process as transition or discharge goals are formulated and clarified. During the course of treatment, progress toward transition or discharge is continually monitored by the treatment team with input from the patient's family.

The following principles govern the planning process: (a) patients receive treatment in the least restrictive environment that provides the structure and intensity necessary; (b) patients receive treatment for the shortest time possible, to reach maximum treatment benefit; and (c) planning is done in a fashion that maximizes a successful transition to the patient's new setting (either more or less structured). Thus, transition or discharge planning involves attention to issues of termination, liaison with community resources (e.g., inpatient settings, school settings), and follow-up to encourage compliance with transition recommendations.

CASE ILLUSTRATION

The case of Tammy, a 16-year-old, African-American girl admitted to a PHP after a suicide attempt, is presented to illustrate the use of partial hospitalization in emergency care. Tammy was first seen in a pediatric emergency room on a Saturday morning by a psychiatry resident who determined that, although she had continued suicide ideation, she did not have a specific plan and could be treated without hospitalization. Over the weekend, the resident contacted Tammy and her family twice by phone and arranged for her to be admitted to a PHP on Monday morning. During her first few days in the program, she was monitored closely by the staff, and her suicide potential was evaluated at the end of each day by the medical director. During the first week, her suicide ideation resolved in response to the support and structure of the program. At the end of her second week in the PHP, she was stabilized, she started taking antidepressant medication, and she made the transition to outpatient family and group therapy after she and her parents stated their commitment to follow-up. The use of partial hospitalization prevented a psychiatric inpatient admission for this acutely symptomatic adolescent patient.

CASE ILLUSTRATION

Other aspects of ambulatory behavioral health care are highlighted in the case of Jamie, a 15-year-old boy who made the transition to a medium-intensity DTP. During a hospitalization for a life-threatening suicide attempt, Jamie's family's managed-care company decertified his admission and stated that he was no longer at acute risk of suicide. Because of continued serious depressive symptoms and chronic family dysfunction, he was determined to be inappropriate for routine outpatient management. He was admitted to a DTP with a strong family systems orientation. Over the course of his 8-week treatment, he was able to develop a therapeutic alliance with his individual therapist, and significant family restructuring was accomplished. The consulting child and adolescent psychiatrist was able to manage Jamie's medication and regularly monitor his suicide potential. At the end of 8 weeks, his depressive symptoms were decreased to the extent that he could make the transition to outpatient therapy and return to school successfully. This use of the ambulatory behavioral continuum allowed for prompt discharge from the hospital with continued consolidation of progress in a highly structured system.

CASE ILLUSTRATION

Finally, Eric provides an illustration of the ambulatory services offered within an integrated mental health and school setting. Eric, a 7-year-old African-American boy, was referred to a rural community mental health center for services. Eric presented with a family history of schizophrenia, extreme irritability, labile mood, noncompliance, tantrums, and physical violence toward his peers and adults. He had received multiple school suspensions and was at risk of expulsion. On intake to services, the clinician recommended participation in a newly established DTP housed at Eric's elementary school. The clinician also recommended individual therapy, family therapy, case management, psychiatric evaluation, and nursing services.

During Eric's 6-month participation in the DTP, Eric's academic program was infused with daily mental health services in the classroom setting. His daily goals included increasing compliance, decreasing anger outbursts, and decreasing physical aggression. He was able to improve peer relations while receiving immediate feedback and direct instruction on social skills by a master's level clinician, teacher, and teacher's assistant. Each staff member was able to apply behavior management principles consistently in his or her domain areas. Eric's parents actively participated in parent conferences as well as in family therapy sessions. After initial reluctance, they agreed to schedule a psychiatric evaluation, after which Eric was further stabilized on medication. Eric gradually made the transition to a regular classroom setting, where he is being maintained successfully. He continues with outpatient services.

**RESEARCH AND EVALUATION**

**Quality of Care**

Partial hospital and other nonresidential hospital alternatives share many common treatment goals and techniques and differ in important ways from both inpatient treatment and outpatient office visits. Accordingly, the measurement of quality in this modality presents some unique challenges. Using the underlying philosophies, the unique therapeutic aspects, the distinctive treatment procedures, and the historical problems and promises of the field, specific quality indicators can be formulated.

Several specific quality indicators of effectiveness for partial hospital and intensive outpatient services include valid and accurate admission criteria, standards of care, and low negative, critical treatment incidents. As an alternative treatment modality, it is incumbent on partial hospital and other ambulatory behavioral health services to establish admission criteria that reliably and validly identify patients who need intervention at a more intensive level than outpatient visits yet at a less restrictive level than 24-hour care. Having targeted an appropriate patient population, the standardization of services rendered and special treatment procedures need to ensure that the care offered provides the safety and security necessary to provide intensive treatment in a nonresidential setting.

Additional unique quality indicators include specific aspects of cost efficiency and patient satisfaction. Providing intensive treatment within a nonresidential environment mandates the use of outside resources and the reliance on strengths within the family and community for augmenting treatment, both of which translate into cost-efficient care. For ambulatory behavioral services, two specific areas of patient satisfaction become applicable: the patient's feelings regarding receiving treatment as an outpatient and the family's sense of contentment with their role in the treatment process.

Finally, for all ambulatory providers, viability is a critical factor, both at the level of the individual program and industry wide. Increased use, cost containment, internalization of quality, and establishment of an evidence base are integral to survival of the modality. It is incumbent on leaders in the field as well as managed care to improve patterns of use if alternative, intermediate forms of behavioral health care are to fulfill a role in the overall continuum of mental health services.

**Treatment Outcome**

Research on treatment effectiveness and outcome in psychiatry involves a complex constellation of factors, and major problems are encountered, including providing an objective, standardized definition of effectiveness and solving methodologic constraints such as difficulties of selection procedures and randomization ( Moskowitz, 1980). The scope of the research literature on the effectiveness of child and adolescent partial hospitalization is not yet broad enough to provide definitive information. Limited research has been done on treatment effectiveness or outcome with child and adolescent samples with only one or two controlled studies ( Burns et al., 1999). A summary of the studies conducted to date on adolescents in PHPs indicates generally positive outcomes, with gains in relationships and school performance (Corky and Zimet, 1987; Kettlewell et al., 1985). Decreased symptoms have been found after treatment in PHPs, and the effects are evident for up to 1 year after discharge (Grizenko et al., 1994; Kiser et al., 1996; Milin et al., 2000). Review of studies on treatment outcome with children suggests that 66% to 90% of patients demonstrate improvement and successful return to community-based schools. Studies suggest that family functioning (structure and stability) are major factors in improvement (Prentice-Dunn et al., 1981; Sack et al., 1987) whereas others conclude that younger children show greater benefits from treatment in partial hospital settings than do older patients (Blom et al., 1973; Prentice-Dunn et al., 1981). Baenen et al. (1986), after a review of outcome studies, report improvements in

behavioral, academic, and family role functioning after partial hospitalization with a variety of aftercare services needed. In a 5-year follow-up of severely disturbed children treated in a multimodal day program, improvements in global functioning, behavior, self-esteem, peer relations, and academic performance were maintained with parental cooperation of primary importance to positive outcomes (Grizenko, 1997). Poorer outcomes are associated with diagnoses and symptoms of disruptive behavior, family and patient history of mental health treatment, and family separations such as out-of-home placements (Kiser et al., 1996; Milin et al., 2000; Rey et al., 1998).

Movement to partial hospitalization away from traditional treatment modes would be alarming and irresponsible without treatment comparisons. It is essential to look at treatment effectiveness of partial hospital settings in comparison with effectiveness of other modalities. Again, published research has not been as extensive as desirable (Weir and Bidwell, 2000). The literature on child and adolescent populations contains four studies that compare treatment outcomes in partial hospitalization and residential settings (Byrnes et al., 1999; Goldfarb et al., 1966; Leone et al., 1986; Valesquez and Lyle, 1985). Overall, the findings suggest that partial hospitalization is equally effective with some patients on some variables, is more effective with some patients on some variables, and is less effective with some patients on some variables. Clearly, advocates of partial hospitalization must continue to compare outcomes from different treatment modalities and from various partial program models, as well as compare treatment outcomes of specific populations.

One of the major difficulties encountered in the area of outcome measurement is the lack of standardization of outcome variables and instruments. Six areas of outcome measurement are recommended for ambulatory behavioral health care, including cost of services, use of services after discharge, severity of symptoms, level of functioning, patient ecology (family and community support network), and satisfaction (American Association for Partial Hospitalization, 1994). Standardized assessment of these variables is a goal with the objective of providing industry-wide information regarding expected outcomes.

## CONCLUSION

Ambulatory behavioral health care represents an important segment of the continuum of psychiatric care available to children and adolescents with a wide array of disorders and allows these patients treatment in less restrictive settings than hospitals. In the current era of health care reform, this and other ambulatory mental health services will likely face increased demand. As policies encourage such options, it is necessary to be aware of differences among PHPs, DTPs, and IOPs, so patient needs can be matched with service variables such as theoretical orientation, targeted populations, types of treatments offered within the program, staffing patterns, and the degree of structure provided (Masters, 1997). Issues related to quality of care, therapeutic outcome, and cost effectiveness must also be considered. With careful review, it is possible to identify intensive PHPs that serve as alternatives to inpatient hospitalization, DTPs or IOPs that function as transitions between highly restrictive treatments and outpatient services, and programs that serve as outpatient alternatives.

Although partial hospitalization is an established concept and has a significant history, the field of ambulatory behavioral health care is in transition, as is health care in general. In the years to come, several questions will require answers as ambulatory behavioral health care providers face the broad challenges brought by health care reform. How will PHPs integrate with other children's services such as special education systems, juvenile courts, and state departments of child protection and human services? How will they fit into large health care organizations to provide prevention and related services? We are beginning to discover some solutions to these larger issues of integration and look to the future and the further development of these exciting modes of treatment.

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## 89 PSYCHIATRIC INPATIENT SERVICES

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### HISTORY

Just as child psychiatry as a specialty is only 50 years old, inpatient child psychiatry is virtually a fledgling treatment modality. Although the first few inpatient units were started in the 1920s and 1930s ([American Psychiatric Association, 1957](#); [Barker, 1974](#)), their treatment remained narrowly focused on children with postencephalic brain disorders, and their numbers remained small for nearly 4 decades. In the 1970s, a confluence of scientific, political, and fiscal factors produced a rapid rise in child psychiatric inpatient services.

This reached its apogee in the mid-1980s with a proliferation of child and adolescent units in both general and specialty psychiatric hospitals. The best of these services provided comprehensive, multimodal evaluation and treatment for several months' duration ([Harper and Geraty, 1985](#)). However, by the early 1990s, enormous fiscal and political pressures ([Woolston, 1993](#)) resulted in a halt to the expansion in the total number of inpatient beds as well as a dramatic reduction in the average length of stay ([National Association of Private Psychiatric Hospitals, 1992](#)). Despite the apparent dramatic reduction in length of stay, the rate of admission has not been similarly diminished ([Patrick et al., 1993](#); [Tsai et al., 1988](#)).

Current child psychiatric inpatient units are deeply embedded in the milieu of the hospital environment and therefore are quite distinct from other institutionally based treatments of emotionally and behaviorally disturbed children and adolescents. However, children and adolescents have been, and continue to be, treated in a variety of other nonpsychiatric, institutional settings, including juvenile detention, social welfare, and educational systems. To date, the choice of treatment settings has been determined more by politics and custom than by diagnostic need. Until the middle of the 20th century, serious mental illness in children was thought to be rare, and therefore psychiatric hospitalization was rare. Conversely, at the beginning of the 20th century, child welfare facilities such as orphanages and shelters for the homeless housed the largest group of institutionalized children. In 1935, the U.S. Congress authorized Aid to Families with Dependent Children, which provided federal welfare funds for home care. Concurrently, professionals and politicians came to believe that "normal" children did not require the expensive course of institutional programs, so many children in welfare facilities were deinstitutionalized.

Unfortunately, the juvenile detention system experienced a concurrent increase in children that completely offset the trend of deinstitutionalization in the child welfare system. Specifically, admissions to all types of juvenile correctional facilities increased ninefold from 1923 until 1979. This burgeoning use of the juvenile detention system was reversed by the passage of the Juvenile Justice and Delinquency Prevention Act in 1976 by the U.S. Congress. Between 1975 and 1979, the rate of detention for juvenile offenders decreased by 68% ([Weithorn, 1988](#)).

Two sets of legal decisions by the U.S. Supreme Court, one that supported the requirement for mental health coverage in insurance plans (*Massachusetts vs. Metropolitan*) and another that broadened the rights of parents to hospitalize their children voluntarily (*Parham vs. J.R.*), served to accelerate this trend. As a result of these various influences, in the mid-1970s, the rate of admission to psychiatric hospitals began to increase as the rate of institutionalization in the juvenile detention system began to decline. The more recent trend in the mid-1990s toward greatly reduced lengths of stay in psychiatric hospitals has apparently been offset by a corresponding increase in placements in residential treatment programs, juvenile detention, and group homes. This phenomenon of shifting children and adolescents from one institutional setting to another as a result of nontherapeutic or nonclinical forces has been described as *transinstitutionalization*. ([Weithorn, 1988](#)).

In the 1970s, the transinstitutional shifts not only resulted in a higher rate of psychiatric admissions for minors but also changed the specific type of psychiatric facility to which they were admitted. In 1970, the rate of children's admissions to private for-profit psychiatric hospitals was 9.3 per 100,000 children or one-fourth of the rate of admissions to federal, state, and county public hospitals (37.8 per 100,000). During the 1970s, admissions of minors to public hospitals declined by 30%, and admissions to private for-profit hospitals increased almost 100%, so in 1980, the rate of admissions of minors was about the same for both types of institutions. The rate of admissions of minors to private nonprofit general hospitals with inpatient services increased slightly from 63.3 per 100,000 in 1970 to 68.5 per 100,000 in 1980 ([U.S. Department of Health and Human Services, 1986](#)). In addition to reduced admission rates to public hospitals, the average length of stay dropped from 74 days in 1970 to 54 days in 1980. Lengths of stay remained constant in private for-profit psychiatric hospitals at 36 days and increased slightly in general hospitals from 9 to 14 days ([U.S. Department of Health and Human Services, 1986](#)).

In the late 1980s and early 1990s, three separate forces combined to reverse this trend of increased inpatient use, especially in the private psychiatric hospitals. First, the proportion of money spent on mental health services had become unacceptably distorted toward inpatient services. For example, in 1986, residential and inpatient mental treatment for minors used up 78% of the \$3.5 billion spent on all mental health services in the United States. Partial hospitalization and various outpatient services accounted for 22% or \$0.77 billion. For one specific insurance program, the Civilian Health and Medical Plan for the Uniformed Services (CHAMPUS), in 1989, the total mental health costs were \$613 billion, of which \$500 million went for inpatient psychiatric care in general and \$305 million for child and adolescent inpatient care in particular. Thus, nearly one-half (49.75%) of the entire CHAMPUS mental health costs for 1989 were accounted for by the hospital and residential treatment of children and adolescents ([U.S. House of Representatives Select Committee on Children, Youth and Families, 1992](#)). This rapid escalation of hospital costs combined with the national recession of the late 1980s to stimulate the development of cost-saving strategies such as managed care review and institutionally negotiated contracts with capitated reimbursements.

A second major brake on the rapid growth of hospitalization for minors was a nationally publicized series of criminal and unethical scandals that rocked private psychiatric hospitals ([U.S. House of Representatives Select Committee on Children, Youth and Families, 1992](#)). A third, and apparently much more positive, force has been the trend toward intensive, noninstitutional psychiatric treatments. Examples of this trend include the following: federal legislation such as the Children and Adolescent System Services Program ([Stroul and Friedman, 1986](#)); innovative state programs in Alaska, Vermont, and California; parent advocacy groups such as National Alliance of the Mentally Ill; and support from foundations from organizations such as the Robert Wood Johnson Foundation. All these various programs emphasize the need for multiple services (e.g., mental health, welfare, education, recreation, and housing) to be available in a seamless fashion to children and families. Rather than requiring the child and family to accommodate to the demands of various institutions, the programs seek to develop individualized integrated services customized to the needs of the child. These various reform-oriented groups have attempted to reconceptualize inpatient units as the most restrictive and rarely used component of an integrated system of care, rather than as the most accessible but disconnected fragment in a mental health nonsystem ([Foster, 1998](#)).

### DIMENSIONS OF PSYCHIATRIC SERVICES FOR CHILDREN

These extraordinarily rapid and constant changes in inpatient psychiatric treatment have been accompanied by numerous growing pains. The model (and perhaps modal) child and adolescent inpatient service of the mid-1980s was a 10- to 20-bed unit dedicated to comprehensive evaluation and treatment with a length of stay of several months ([Harper and Geraty, 1985](#); [Woolston, 1989](#)). More recently, beginning in the early 1990s, the average length of stay has dropped to several weeks. This change has resulted in a focus on crisis intervention and evaluation rather than on longer-term treatment. Such facilities are specialty units in general or larger psychiatric hospitals and are staffed with a multidisciplinary treatment team including child psychiatrists, pediatricians, psychologists, social workers, nurses, and teachers. High-technology diagnostic testing, including brain imaging, electroencephalography, and chromosomal analysis, is frequently employed ([Woolston and](#)



[Riddle, 1990](#)). Treatment is characterized by an orchestrated blend of milieu, family, pharmacologic, and individual therapies. For these and other reasons, children and adolescents may receive intensive evaluation and short-term treatment in hospital programs but then may be unable to progress to less restrictive and less expensive but longer-term treatments. This increasing intensity of treatment provided in inpatient units has tended to cut off such services from their historically related programs provided by educational, juvenile detention, and social welfare agencies. The very same medical, financial, and political forces that have promoted the growth of these inpatient units have created artificially rigid boundaries with these other related treatments. Typically, children may receive public or private insurance funding for a relatively short hospital treatment but are without benefits to cover intensive outpatient or residential treatment. Ironically, the shortening of hospital lengths of stay to several weeks frequently results in a more disruptive hospital experience because welfare agencies, schools, and outpatient treatment programs cannot respond to the newly identified needs of the child before discharge.

Although the psychiatric treatment of children and adolescents is sometimes conceived of as a relatively homogeneous treatment modality, considerable heterogeneity actually exists in inpatient facilities. Although inpatient units can be characterized by many different factors, the following six dimensions account for most of the variance: level of restrictiveness of treatment, length of stay, type of psychiatric disorder, developmental level of patient, hospital organization or affiliation, and philosophy of treatment. Although each of these dimensions is distinct, they are obviously interdependent. For example, children with severe, chronic disorders, such as mental retardation and autism, frequently need very long-term treatment. Their families usually do not have the financial resources to pay for such treatment, so the treatment facilities must rely on some form of public funding. A completely different constellation of factors is represented by adolescents with adjustment disorders. They are frequently treated in emergency or acute services funded by private insurance. Despite such clustering, these six dimensions provide a useful guide to understanding the current heterogeneity of services.

### **Level of Restrictiveness of Treatment**

Perhaps the fundamental dimension of inpatient child psychiatry is level of restrictiveness of treatment. By definition, inpatient treatment is the most restrictive modality, by virtue of its being an out-of-home, institutional setting. However, level of restrictiveness can vary considerably among psychiatric hospitals by such variables as whether admissions are voluntary or involuntary, the unit is locked or unlocked, participation by parents is encouraged or discouraged, and visits to the home community are facilitated or prevented.

This dimension of restrictiveness indicates part of the explanation for the rapid shifts among the population of mental hospital, welfare institutions, and juvenile detention facilities. All three sets of institutions, although supposedly treating children with quite different diagnostic descriptions, have in common the most restrictive treatment setting. Thus, from the point of view of funding or licensing agencies, hospitals, juvenile detention centers, and state institution shelters have virtually nothing in common. From the point of view of level of restrictiveness, all three facilities are nearly identical: They are highly restrictive, out-of-home, institutional placements.

### **Length of Stay**

Length of hospital stay is both reflective of other dimensions of a hospital program and a determinant of constraints on these very same dimensions. Attempts to apply rational prospective payment systems (e.g., diagnostic related group system) to inpatient child psychiatry have shown that the major determinant of length of stay is finding availability or explicit program limitations, rather than patient diagnosis ([Christ et al., 1989](#)). Traditional groupings of lengths of stay include emergency (3 days), acute (<4 weeks), short-term (1 to 3 months), intermediate (3 to 5 months), and long-term (>9 months). Although these divisions are somewhat arbitrary, they both reflect and organize treatment philosophy, psychiatric diagnosis, type of patient served, and institutional organization. For example, very short-term lengths of stay preclude comprehensive interventions because the necessary relationships cannot be formed and evaluations cannot be completed. In addition, therapies that require a definite time period, such as some medication trials or educational programming, are not feasible. However, such short-term lengths of stay may be minimally disruptive to the child's important social relationships. In contrast, hospital programs with very long lengths of stay can reasonably attempt such goals as character change and academic disability remediation, but they run the risk of deleterious institutionalization effects.

### **Psychiatric Disorder**

The typical psychiatric disorders of a specific inpatient population are determined by such factors as length of stay and institutional organization and, in turn, determine other variables, such as philosophy of treatment. The broad groupings of psychiatric disorders include adjustment disorders, mixed behavior or affective disorders, eating disorders, adolescent- or adult-onset psychosis, substance abuse, pervasive developmental disorders, childhood-onset psychosis, and mental retardation. Although the basic rudiments of diagnostic grouping exist, surprisingly little is known about the most basic aspects of the phenomenology of serious childhood-onset psychiatric disorders, including such aspects as natural history, epidemiology, etiology, and associated comorbidity ([Woolston et al., 1989](#)). The current state of ignorance about these fundamental issues places major constraints on specificity of treatment design as well as on intervention outcome evaluation.

### **Developmental Stage**

Hospital programs designed for the treatment of children and adolescents usually stratify their units according to the developmental age of their patients. Not surprisingly, hospital facilities follow the same developmental groups as do education programs. These include programs for children up to 3 years old (infant, toddler, and preschool), from 4 to 14 years old (elementary and middle school), and from 15 to 19 years old (high school and college). A program that is relatively homogeneous in developmental age automatically defines some of the constraints of psychiatric diagnosis, philosophy of treatment, and level of restrictiveness. For example, programs for adolescents typically treat patients with eating disorders, affective behavior disorders, substance abuse, and adolescent-onset psychotic disorders, whereas programs for young children focus on pervasive developmental disorders, disruptive behavior disorders, and, less frequently, childhood-onset eating disorders. Organization of elements of the treatment programming, such as the type of milieu and family therapy employed, is greatly influenced by the developmental stage of the patient.

### **Institutional Organization and Affiliation**

Another dimension defining hospital treatment is related to institutional organization or affiliation. One fundamental dichotomy is between publicly funded or affiliated hospitals and private hospitals. The publicly funded hospitals may be local, state, or federal, or some mixture of all three. Until recently, publicly funded hospitals have tended to be large psychiatric hospitals for severely and chronically disturbed children who frequently came from socially disadvantaged families. Because of the chronic nature of the psychiatric disturbance and relatively poor funding, these public hospitals tend to focus on benign environmental amelioration as a treatment philosophy. In these ways, some programs may more closely resemble residential treatment centers than inpatient services. The recent trends of deinstitutionalization and diminishing publicly funded hospitals have combined to result in shortened lengths of stay and decreased admission rates. Unfortunately, because few alternative mental health services for poor, chronically disturbed children have been implemented, the juvenile detention, social service, and education systems may well be forced to resume their previous roles in providing services. The other fundamental type of institutional organization group, nonpublicly funded hospitals, occurs as either nonprofit or for-profit hospitals. Typically, inpatient units in nonprofit private institutions are part of a general medical hospital that provides a full array of medical, surgical, and psychiatric services. These hospitals may be primary (local), secondary (state based), or tertiary (regional) facilities; each level has an attendant structure of resources and typical patient population. Psychiatric inpatient treatment in general hospitals is quite heterogeneous. Some general hospitals do not have discrete children's psychiatric units. Instead, patients are admitted to general medical or pediatric units in so-called "scattered beds."

Typically, children treated in these settings receive crisis intervention and then are discharged within 1 to 7 days. Conversely, discrete, dedicated child psychiatric inpatient units in general hospitals offer comprehensive evaluation and treatment services by a multidisciplinary treatment team. Average lengths of stay in these units range between 7 and 60 days. Although the median length of stay reported for psychiatric inpatients in general hospitals is 14 days, this number is probably a meaningless average between these two disparate groups in a bimodal distribution. Even within this special group of dedicated child inpatient units in general hospitals, there is considerable variation in philosophy of treatment, length of stay, and developmental or diagnostic characteristics of the patients.

For-profit private proprietary psychiatric hospitals have experienced tremendous growth since the early 1990s. In 1971, about 6,500 children and adolescents were admitted to proprietary psychiatric hospitals, whereas by 1980, about 17,000 children less than 18 years of age were admitted. By 1984, 23,000 adolescents (ages 13 to 17 years) alone were admitted to these hospitals, and by 1986, 38,000 teenagers were hospitalized ([Weithorn, 1988](#)). The explanation for this explosive growth of proprietary psychiatric hospital admissions is multifaceted, but it probably devolves on four main factors: diminished availability of juvenile detention and child welfare systems; increased funding provided by private insurance and Medicaid; increased social instability because of divorce, moves, and social disruption; and the relaxation of the statutory rights of minors by the Parham decision of the U.S. Supreme Court in 1979 ([Weithorn, 1988](#)). This rapid growth of proprietary psychiatric hospital admissions was accompanied by a storm of controversy and criticism that focused on excessive and inappropriate hospitalization of adolescents as a result

of unprofessional and unethical admission policies ([U.S. House of Representatives Select Committee on Children, Youth and Families, 1992](#); [Weithorn, 1988](#); [Woolston, 1993](#)). This rapid proprietary hospital growth and its attendant abuses have had certain consequences. Closer scrutiny by third-party payers and licensing agencies has occurred. This has resulted in numerous hospital closures and consolidations. In the 1990s, lengths of stay dropped dramatically in response to reduced insurance benefits and aggressive utilization review oversight by managed-care companies ([Patrick et al., 1993](#)).

## Philosophy and Goals of Treatment

The philosophy and goals of treatment espoused by various psychiatric inpatient services occur in a continuum ranging from custodial care to comprehensive evaluation and treatment of the child, family, and the important social environment. Although less ambitious treatment philosophies may reflect a callous or unethical approach to disturbed children, they may equally likely emerge from a realistic appraisal of fiscal, social, and clinical realities. Frequently, the level of intensity of the philosophy of treatment is highly correlated with the severity or chronicity of the psychiatric disturbance and the adequacy of funding. As the severity or chronicity of a disturbance increases and the level of funding decreases, the intensity of treatment diminishes.

### CUSTODIAL CARE

Custodial care is geared toward preventing harm from befalling the patient, whether from an inability to provide self-care or from injury. Custodial care is usually the dominant treatment philosophy only for the most severely disturbed psychotic and retarded children. Ironically, the only other setting in which custodial care is the prominent treatment philosophy is for acute, "social" admissions of relatively high-functioning children who need protection from their parents.

### ENVIRONMENTAL AMELIORATION

The next level of treatment philosophy is nonspecific, benign, environmental amelioration. Specifically, the institution provides good nutrition, safe and pleasant surroundings, medical care, appropriate recreational activities, and an adequate education. For example, this level of treatment is rarely the officially stated philosophy of a hospital program; it may be the *de facto* treatment goal because of inadequate staffing and treatment planning. For institutions such as child welfare agencies that provide services for children who are considered "normal," environmental amelioration may actually be an appropriate and desirable level of treatment.

### SELECTED EVALUATION AND TREATMENT

The next level of treatment is selected evaluation and treatment of some aspect of the child and his or her important social environment. For example, many traditional psychiatric hospitals and residential treatment centers focus on evaluating and treating the child's psychiatric and behavioral problems. These programs emphasize milieu and individual as well as special education programs and parental counseling therapies ([Harper and Geraty, 1985](#)).

Of all the specific treatment modalities currently employed, milieu therapy encompasses the broadest range and is most specific to this form of treatment philosophy. Milieu therapy has its roots in the writings of [Aichorn \(1935\)](#), [Bettelheim \(1950\)](#), [Redl and Wineman \(1952\)](#), and [Trieschman et al. \(1969\)](#). These authors emphasize the use of the child's "Life-Space Model," the notion that every aspect of the child's physical and social environment is important in therapeutic understanding and treatment. Although milieu therapy at first focused on an individual psychoanalytic understanding of the child, it has successively incorporated concepts of group, occupational, recreational, and behavioral therapies to create a multimodal therapeutic life experience. Although the specifics of each milieu program are different, depending on the psychiatric diagnoses and the developmental levels of the patient, the overall principles are similar ([Cotton, 1993](#)).

Individual therapies are considered to be all those interventions directed specifically toward the individual patient. They include individual psychodynamic psychotherapy, cognitive psychotherapy, individual behavior therapy, and pharmacotherapy. Although these therapies are employed in a similar manner and for the same indications as they are for nonhospitalized patients, modifications are necessary to account for greater frequency and intensity of treatment and greater severity of psychiatric disturbance in the patient.

Regardless of the specific treatment, inpatient psychiatry requires active coordination in implementing the program. This issue of creating a coherent, well-synthesized treatment program fashioned out of many different treatment elements and employing many different clinicians is a major challenge for hospital treatment. [Stanton and Schwartz \(1954\)](#) highlighted the frequently described phenomenon of significant clinical deterioration of patients when the coherence and consistency of the hospital program are destroyed by poor communication, divergent clinical opinions, and strongly held, emotionally charged differences among staff members.

Special education programs are an important, albeit much neglected, component of hospital programs. Perhaps most children hospitalized in psychiatric inpatient units have a significant cognitive (e.g., mental retardation or borderline intellectual functioning) or academic (e.g., specific developmental learning disability) disorder ([Woolston et al., 1989](#)). The identification of these disorders and the subsequent development of remedial teaching strategies can play an important part in a child's treatment. Unfortunately, wide regional variation occurs in the level of funding available for these educational services. Services may vary from 2 hours of tutoring in the child's bedroom to 8 hours of special education in a comprehensive school setting. As lengths of stay diminish below 4 weeks, linkages with the child's original school become more difficult, and providing a *bona fide* school curriculum becomes increasingly difficult.

In the service of maximizing the impact of these therapies on the child, those programs that use this level of treatment philosophy tend to disconnect the child from the important social environment, the family, and local school. Although family therapy or parent counseling may be offered, it occurs in parallel and quite separate from the active hospital treatment of the child. This level of treatment philosophy is heavily influenced by the traditional medical model of hospitalization: The child is sick; the hospital treatment is to cure the child's illness; when cured, the child can return home. Although this model works well for some acute illnesses, it is not appropriate for many chronic conditions, whether medical or psychiatric. Because such definite "fixing" of the child is not possible, changing the child's important social environment to be more suitable to the child's developmental needs may be as important as changing the child.

### COMPREHENSIVE EVALUATION AND TREATMENT

The highest level of treatment philosophy aims at comprehensive evaluation and treatment of the child, the child's important environment, and the interaction of the two. An appreciation of the complex and powerful effect of the evolving interaction between the child and his or her important social environment follows from understanding the concept of a transactional risk model ([Woolston, 1989](#)). The transactional risk model rests on the concept of "goodness of fit" proposed by [Chess and Thomas \(1984\)](#), as well as concepts of resilience proposed by such authors as [Gannezy \(1983\)](#), [Rutter \(1979\)](#), and [Werner and Smith \(1982\)](#).

Articulation of this transactional model is a relatively recent development in the inpatient treatment of children and awaits testing and development to determine its applications and problems. This model may provide a guide for both the orchestration of specific treatment modalities in an inpatient unit and the development of a network of services linking inpatient and outpatient programs to provide the most comprehensive and least restrictive treatment. Perhaps of greatest importance, this treatment philosophy attempts to address a major problem for hospital treatment: How can the gains achieved during the hospitalization be generalized and continued in less restrictive, more normative settings? In addition to "fixing" the child or, less frequently, "fixing" the family, this treatment philosophy attempts to ameliorate the interaction between the child and his or her environment so the child's developmental potential will be maximized.

In the current climate of several-week hospitalizations, a major focus is on developing a more comprehensive, more appropriate outpatient system of care. This development entails bringing the new information learned about the child and family to the professionals who provide social services, education, and mental health services. To accomplish this development of a new system of individualized care, the inpatient facility must have preestablished linkages with an array of services such as school systems, day treatment, in-home services, therapeutic foster care, protective services, medical providers, traditional outpatient services, housing resources, and recreational programs ([Bickman et al., 1996](#); [Foster, 1998](#); [Stroul and Friedman, 1986](#)).

One innovative approach has been intensive, home-based psychiatric services that begin as the child is discharged and continue until the child and family have an adequate system of outpatient care established ([Woolston et al., 1998](#)).

## FUTURE DIRECTIONS OF INPATIENT SERVICES FOR CHILDREN

The current status of inpatient child psychiatry is characterized by an exciting ferment of rapid development with a huge perceived need for treatment, but with a



nearly total absence of data about the mechanisms to meet this need (Manderscheid et al., 1993; Weller et al., 1995). In addition, the paucity of research on effectiveness and efficacy of inpatient treatment is remarkable given the large proportion of mental health resources devoted to this modality (Burns et al., 1999). As delineated in this chapter, psychiatric hospitalization is an enormously heterogeneous treatment modality. Before any assessment of the efficacy of hospital treatment, either absolute or in comparison with other treatments, can occur, the specifics of the hospitalization must be stated and spelled out in a manual, so fidelity can be ensured. Research has reported home-based, multisystemic therapy as an effective alternative to hospitalization. Although this research carefully described the experimental treatment, the hospital treatment had no such standardization (Henggeler et al., 1999). The further development of inpatient psychiatry will occur in response to the acquisition of more refined knowledge. Specifically, research is needed to increase understanding in several areas, including the following: basic phenomenology about childhood-onset psychiatric disorders and their association with psychosocial stressors (Gutterman, 1998); the development and refinement of specific treatment strategies; the use of biomedical diagnostic and treatment techniques; and the implementation of an integrated network of mental health services with an appropriate, nondiscriminating funding basis (Bickman et al., 1996; Foster, 1998; Stroul and Friedman, 1986).

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## 90 RESIDENTIAL TREATMENT

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### DEFINITION

*Residential treatment centers* (RTCs) are defined by the National Institute of Mental Health ( [Stroup et al., 1988](#)) as psychiatric organizations serving children and meeting the following criteria:

1. It is an organization, not licensed as a psychiatric hospital, whose primary purpose is the provision of individually planned programs of mental health treatment services in conjunction with residential care for its residents.
2. It has a clinical program within the organization that is directed by a psychiatrist, psychologist, social worker, or psychiatric nurse who has a master's or a doctoral degree.
3. It serves children and youth primarily less than 18 years of age.
4. The primary reason for the admission of 50% or more of the children and youth is mental illness, which can be classified by the codes set forth in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) and the tenth edition of the *International Classification of Diseases* (ICD-10), other than those codes for mental retardation, substance (drug)-related disorders, and alcoholism. )

### HISTORY AND ORIGINS

In 1729, the first U.S. orphanage was founded by Ursuline nuns in New Orleans to provide care for a group of children whose parents had died in a smallpox epidemic. This is the origin of the custodial, or residential, component of residential treatment in the United States. Religious institutions remained prominent in offering custodial care for many years. Recognition of the developmental differences characteristic of children gradually led to the formation of the specialties of pediatrics and child psychiatry and, in the 1880s, to the emergence of numerous special agencies for children. In the 1900s, mental health professionals, particularly psychoanalysts, child psychiatrists, psychologists, and social workers, began discovering and applying psychodynamic theories in the care and treatment of children. The main needs of the developing child were increasingly recognized, and comprehensive programs designed to meet those needs were planned. This program development constituted the first treatment component of residential treatment. Group living experiences, educational, physical health, and creative opportunities, and psychotherapy for children and their families, both individually and in groups, began to replace rigid custodial care, with its strong moral and discipline-oriented attitudes.

The field of residential treatment for children subsequently developed on an empirical basis to provide multiple models of treatment to suit different needs. The different models also reflected different theories of child development. Thus, psychoanalysis and learning theory initially provided significant foundations for two polar treatment approaches, between which various admixtures of the two could be found. Subsequently, other theoretical models were introduced, including a "goodness-of-fit" model and various cognitive and behavioral approaches.

A view that has gained ground conceptualizes the residential experience as a whole—group living, on-grounds schools, and psychotherapy—as constituting each child's therapy. Working under this view, all staff members are in full communication as they deal therapeutically with the children, their parents, and, in some cases, the whole family. Combined treatment modalities are used, including milieu therapy, family therapy, parent work, psychodynamic psychotherapy (individual, group), behavior therapy, pharmacotherapy, recreational therapy, art therapy, music therapy, and special education.

### STATISTICS, TRENDS, AND FUNDING

In 1990, approximately 68,729 emotionally disturbed children in the United States received residential treatment in 501 facilities representing a total bed capacity of 29,756, with a year-end census of 27,785. In 1998, approximately 78,520 emotionally disturbed children in the United States received residential treatment, with no significant increase in the number of facilities. Previous figures showed a general increase in growth in the 1970s and 1980s in the number of treatment beds available. There was a period of temporary decline in the early 1980s and a period of slight growth between 1986 and 1988 ( [Table 90.1](#)). During the 1990s, there was a renewed emphasis on reducing length of stay and placing children in the least restrictive treatment settings.

	1972	1976	1984	1986	1988	1990
Number of children	19,384	20,384	16,745	24,547	25,547	27,785
Number of facilities	340	375	322	457	440	501

Statistics from unpublished data National Institute of Mental Health, 1994.

**Table 90.1. Trends in Residential Treatment Bed Occupancy**

Statistics published by the National Institute of Mental Health in 1991 note the following trends in residential treatment. In 1988, California and Massachusetts had the highest number of RTCs, 48 and 38, respectively. Other states with 15 or more RTCs were Colorado, Illinois, Minnesota, Ohio, Pennsylvania, and Wisconsin.



Approximately 70% of RTCs had 50 or fewer beds. Twenty percent had 50 to 75 beds. Ten percent had more than 100 beds. More than 33% of children were in residential treatment facilities having more than 100 beds ( [Table 90.2](#)).

Staff Discipline	Number of Full-Time Equivalent Staff (1978-1984/1988)			Percent Distribution (%)		
	1978	1984	1988	1978	1984	1988
Psychiatrists	36	30 (+7%)	48 (+37%)	0.8	1.1	1.1
Psychologists	47	62 (+32%)	124 (+125%)	1.1	1.7	3.3
Social workers	176	233 (+32%)	477 (+169%)	4.0	5.8	12.7
Nurses	33	45 (+36%)	87 (+161%)	0.7	0.8	2.1
Other (A.A.)	147	173 (+18%)	374 (+155%)	3.3	2.5	8.6
Other (A.S.)	540	476 (-12%)	945 (+71%)	12.4	6.8	21.5
Administration and maintenance	579	574 (-1%)	1086 (+87%)	13.1	8.2	24.1

Full-time equivalent (FTE) staff in residential treatment centers. Reservations taken from the National Health and Medical Research Council, 1988 and 1990. All data are based on data from the National Health and Medical Research Council, 1988 and 1990. All data are based on data from the National Health and Medical Research Council, 1988 and 1990.

**Table 90.2. Number, Percent Change in Number, and Percent Distribution of Full-Time Equivalent Staff in Residential Treatment Centers, by Staff Discipline, United States, January 1978 and 1984 and December 1988**

In 1988, 28% of RTC patients were black and 10% were Hispanic. Mental illness was considered the major disability of 94% of RTC admissions. Only 1% to 2% were diagnosed as alcohol or drug abusers. Occupancy rates increased from 87% in 1977 to more than 95% in 1983 and remained at more than 95% throughout the 1980s and 1990s.

Indicators of higher turnover rates and shorter lengths of stay began to appear, and from 1986 to 1988 there was a dramatic increase in the number of RTCs that offered partial care and outpatient treatment services. In the 1990s, this trend continued. Directors of RTCs offering partial care and outpatient services reported that the severity of emotional and behavioral disturbances of children being referred to them increased markedly during the 1990s. Directors of RTCs also reported an increased need for psychiatric consultation because of increased use of psychotropic medications for children in all modes of treatment. Multiservice RTCs have also increased partial and outpatient services to children and families with substance abuse problems.

An estimated \$359.1 million was spent to maintain RTCs in 1977. By 1983, the figure had risen to \$573 million, an increase of 60%. In 1988, the figure rose to \$1.3 billion. However, in constant dollars, this represents an increase of less than 5%. In 1988, of funds available for RTCs, 70% came from state and local government sources, 5% came from federal funds, 10% from client fees, and 12% from all other sources. In 1999, the U.S. Surgeon General's report on mental health stated that RTCs were used by 8% of treated children while using nearly 25% of the national outlay on child mental health.

## CONTINUUM OF RESIDENTIAL GROUP CARE OF CHILDREN

Historically, children are placed in the continuum of group care through the channels of the social welfare, juvenile justice, and mental health service delivery systems. According to the National Survey of Residential Group Care in 1981, conducted by the University of Chicago, 3,914 facilities in the United States operated with a total daily census of 125,323 (Young et al., 1989). Children classified as being in facilities for emotionally disturbed children accounted for 16% of the total in 1981 ( [Table 90.2](#)). RTCs account for only a portion of the reporting facilities. Placement of children in different categories of facilities does not represent an accurate differentiation of children by diagnosis. The type of placement a child receives is also influenced by factors such as economic status, race, and geographic location.

## STAFF AND SETTING

The professional staff of an RTC includes child care workers, teachers, social workers, psychiatrists, pediatricians, nurses, and psychologists.

There were dramatic increases in the number of staff working in RTCs between 1984 and 1988. This increase followed a time of decline from 1978 to 1984. In 1978 RTCs employed 22,443 full-time equivalent staff. The number dropped 5% in 1984 to 21,211. Between 1984 and 1988, the number of full-time equivalent staff members working in RTCs nearly doubled, reaching 39,186. In 1988, there were only 103 full-time psychiatrists working in RTCs, with an additional 808 psychiatrists working part-time in RTCs. There were 370 full-time doctoral level psychologists and 472 part-time psychologists. Social workers with master's degrees (M.S.W.) comprise the largest number of professionally degreed staff, reaching 2,753 full-time and 328 part-time staff in 1988. Registered nurses with master's degrees numbered 163 full-time staff and 36 part-time staff in 1988. The percentage of distribution of full-time equivalent staff remained relatively stable from 1978 to 1988 ( [Table 90.2](#)).

The Joint Commission on Mental Health of Children made the following structural or setting recommendations in 1970:

In addition to space for therapy programs, there should be facilities for a first-rate school and a rich evening and weekend activity program and there should be ample space for play, both indoors and out. Facilities should be small, seldom exceeding 60 in capacity with 100 a maximum limit, and should make provision for children to live in small groups. The centers should be located near the families they serve and be readily accessible by public transportation. They should be located for ready access to special medical and educational services and to various community resources, including consultants. They should be open institutions wherever possible; locked buildings, wards, or rooms should only rarely be required. In designing residential programs, the guiding principle should be this: Children should be removed the least possible distance—in space, in time, and in the psychological texture of the experience—from their normal life setting ( [Joint Commission on Mental Health of Children, 1970](#)).

The actual design of the building should take into account the physical, cognitive, and emotional needs of children between 6 and 15 years of age. Private or shared bedrooms, separate showers and bath facilities for boys and girls, communal living rooms and dining rooms, safe and easy access to recreational facilities and classrooms, and therapists' offices close to the group living area are some of the requirements. Units serving specific age groups are preferred.

Most RTCs gradually increase in size; as they do so, the number of children in each residential unit tends to increase. At the same time, Marsden et al. (1970) find that the duration of treatment seems to shorten as this growth occurs. Older children tend to remain in residence for shorter periods of time than do younger children.

## INDICATIONS

The child's lack of age-appropriate internal controls and need for more consistent external controls, as well as the child's need for intensive specialized treatment, constitute basic indications for residential treatment. Such children may present with moderately severe symptoms of antisocial and aggressive behaviors; they may have psychotic symptoms, including loose associations and hallucinations; or they may exhibit precipitous, severe regressions. Bedwetting is a common symptom. Most, if not all, children referred for residential treatment have severe learning problems.

Genetic factors, organic brain damage, neurochemical dysfunctions, and adverse psychological experiences in the child, together with psychosis and other psychiatric disorders in the parents and an impoverished socioeconomic climate, are major causative factors. A history of parental deprivation, loss, seduction, incest, sexual and physical abuse, and physical injury is common, as is a history of multiple foster home placements.

Children and adolescents in residential treatment often have been exposed directly or indirectly to traumatizing physical and sexual abuse and have subsequently become aggressive, disorganized, and out of control. Milieu therapy using containment, structure, support, involvement, and validation is often helpful ( [Lawson, 1998](#)).

An unusually high prevalence of encopresis, especially in boys, has also been reported in children in residential treatment. In one study of 23 boys in residential treatment, nine were encopretic. Among these nine boys, seven had histories of sexual abuse, particularly sodomy, usually perpetrated by the father or father

surrogate ([Morrow et al., 1997](#)).

Using the classification system of the fourth edition of the DSM (DSM-IV) ([American Psychiatric Association, 1994](#)), the clinical diagnoses most often attached to the child include pervasive developmental disorders (not otherwise specified), attention deficit hyperactivity disorder, specific developmental disorders, conduct disorder, dysthymia, major depressive disorder, and anxiety disorders.

## REFERRAL, INTAKE, AND ADMISSION

Most children who are referred for residential treatment have been seen previously by one or more professional persons, such as a school psychologist, pediatrician, or members of a child guidance clinic, juvenile court, or state welfare agency. Unsuccessful previous attempts at outpatient treatment and foster home or other custodial placement often precede residential treatment. Consequently, considerable data are usually available to and are usually required by the intake staff of the RTC; however, residential centers differ in the evaluation and decision-making process used for selecting children for admission. Satisfactory studies comparing admission criteria and outcome correlations have not been done. The type of child referred to a particular institution is often idiosyncratic to that institution. The age range of the children varies from institution to institution, but most children are between 5 and 15 years of age. Boys are referred more frequently than girls.

An initial review of the data enables the intake staff to determine whether a particular child is likely to benefit from their particular treatment program. The next step usually involves interviews with the children and their parents by various staff members, such as a therapist, a group living worker, and a teacher. Psychological testing and neurologic examinations are performed when indicated, if these have not already been done. Children and their parents should be prepared for these interviews. A useful practice is to have the child spend a day at the center, both in class and in a residential unit.

During this admission and diagnostic process, a comprehensive profile of each child is established. There is at present no standard profile in use. Structured interview schedules are useful. A mental status examination of the child is essential.

The needs of each child, the skills of the staff, the balance of the patient population, and estimates of the prognosis are some of the variables weighed when children are accepted for admission. The parents may or may not be available for treatment. This entire evaluation and assessment process forms the basis on which the staff reaches a decision and makes a tentative treatment plan. A preplacement visit by the children and their families is usually arranged to help to prepare each child for admission.

## TOTAL STAFF COLLABORATION

Group cohesiveness among the staff improves performance. [Johnson \(1982\)](#) describes the measures that promote such cohesiveness: meetings to discuss team process issues; acquisition of leadership skills; clear lines of authority and role expectations; training programs; total team participation in decisions; and prompt expressions of support, concern, and empathy by the leadership. The cooperative attitude of administrative leaders sets the tone for residential treatment staff. [Haley \(1980\)](#) suggests that success in therapy may be determined in part by what happens among the professional staff members. Resolving communication problems, sharing perceptions and experiences, clarifying transference and countertransference issues, and providing a means by which staff can receive feedback on how well they are doing are matters that are as important for the staff as they are for the children.

## ADMINISTRATION

Administration of an RTC requires sound business practices in regard to defining the organization's purpose and mission, securing adequate financial and personal resources, clarifying lines of authority and accountability, and developing responsive programming. RTC administrators must be involved in statewide and national professional and political organizations, because changes in health care and social service policies require rapid adaptive changes in RTC programming and funding. Since the early 1990s, strong state and national consortiums of residential treatment facilities have been developed to play a vital role in assessing the changing needs of children, advocating for children's services, and sharing resources.

In addition to employing sound business practices, administrators of RTCs must understand the impact of severely disturbed children on the individual and group functioning of the staff. This has become more important in recent years as psychiatric hospital stays and other factors have led RTCs to admit even more seriously disturbed children, many of whom take psychotropic medication, than they had in years past. Administrative commitment to a supervisory process that acknowledges the impact of severely emotionally and behaviorally disturbed children on all agency staff members is essential. R. Howenstine (unpublished manuscript, 1984) suggests that a key administrative role in achieving staff collaboration is to conceptualize interdepartmental collaboration and conflict management as a vehicle for identifying, containing, and dealing with the aspects of the children's disorders that are internalized by the staff. [Brown \(1983\)](#) provides a theoretical and systematic approach to managing conflict at departmental interfaces from a management perspective and focuses on six major opportunities for change: (a) to alter perception, (b) to alter communication, (c) to alter behavior, (d) to alter the interface, (e) to alter one or both departments, and (f) to alter organizational context.

## CHILD CARE WORK

By far the largest amount of time in the child's life in a residential treatment setting is spent in group living. [Northrup \(1982\)](#) maintains that the child care worker in group living is usually responsible for the improvement in deeply disturbed children in residential treatment. These workers' use of "good enough parenting" allows them to serve as role models for the children. Child care workers also offer a structured environment that constitutes a therapeutic milieu. Tasks are defined within the limits of the child's abilities; incentives, such as increased privileges, encourage the child to progress rather than to regress. Every member of the staff is important; for example, the dietary staff may have a special role and meaning for some children for whom food and its associations have special significance, including feelings of security, deprivation, love, and aggression.

Children often select one or more staff members with whom to form a relationship through which they express, consciously and unconsciously, many of their feelings toward their parents. The child care staff should be able to recognize such transference reactions and to respond to them in a way that is different from the children's expectations based on previous or even current relationships with their parents. Sometimes, the child care workers know enough about a child to make an interpretation to the child when it seems appropriate—the so-called life-space interview ([Redl, 1959](#)). The literature on milieu therapy extends back to [Aichorn \(1935\)](#). The child care worker must also be aware of the countertransference problems stimulated by the child's behavior and, indeed, by other staff members in what is essentially a large, close, familylike community.

To maintain consistency and balance, the group living staff must communicate freely and regularly with one another and with the other professional and administrative staff members of the residential setting, particularly the child's teacher and therapist. The group living worker has perhaps the most potential among the staff members to produce therapeutic gains.

The child care staff members must recognize any tendency toward being the good, or bad, parent in response to the child's splitting behavior. This tendency in a staff member may become manifest as a pattern of blaming other staff members for the child's disruptive behavior. Similarly, the child care staff must recognize and avoid such individual and group countertransference reactions as sadomasochistic and punitive behavior toward a child.

A common milieu dilemma occurs when the child care worker has to differentiate the approaches needed for, say, the conduct-disordered or psychotically disturbed children in their care from an understandable tendency to want to apply a consistent approach to all children. Therapists and consultants need to be called on to educate child care staff regarding the developmental and psychodynamic needs of each child and how different techniques may be applied while maintaining unit cohesiveness and avoiding countertransference problems.

The structured setting should offer growth-promoting experiences and opportunities for facilitating and improving the adaptive behavior of the children. Particular deficiencies such as speech and language deficits, intellectual retardation, inadequate peer relationships, bedwetting, poor feeding habits, or attention deficits may be at the base of the children's poor academic performance in school and their unsocialized behavior, including temper tantrums, fighting, and withdrawal, and require particular attention.

Behavior therapy principles are also applied, particularly in group work with children. In the course of group therapy, behavior that is socially adaptive and appropriate may be awarded points. At the end of the session, all the children receive a prize of, say, candy, but the child who has the most points gets first choice. Behavior



therapy is a component of the total therapeutic effort of the residential center.

Unfortunately, many child care workers feel frustrated and confused, and they experience job dissatisfaction. [Rick and Charlesworth \(1982\)](#) suggest that this is partly because they do not have sanction to cross certain professional boundaries. Child care workers and other professionals—psychiatrists, psychologists, social workers, teachers—need to recognize the problem as a systems issue and should participate in education and accommodation aimed at realignment of professional roles. There is a corresponding need for a career development ladder for child care workers.

## PHYSICAL HEALTH

Many children referred to RTCs have physical health problems, including poor nutrition, dental problems, and frequent infections. Attention must therefore be paid to health care. Particular attention must be given to adequate hygiene, and standard universal safety precaution procedures should be followed. On-staff registered nurses are indispensable, along with a panel of physician consultants.

## SCHOOL PROBLEMS

Children in residential treatment frequently have severe learning disabilities, as well as disruptive behavior, and usually they cannot function in a regular community school. A thorough diagnostic assessment of a child's specific learning difficulties is required as a basis for a rational approach to the application of specific remedial measures ([Table 90.3](#)).

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1. Pediatric history: birth, development, personal, social, family, school, previous illnesses, life events
  2. Specific strengths: sports, creative arts
  3. Specific symptoms or skill delays or both: reading, writing, arithmetic, directionality, visual and auditory sequencing, memory, coordination, attention
  4. Associated behavior, personality trait or difficulties: adaptations, defenses, self-esteem, anxiety, depression
  5. Comprehensive pediatric physical examination and diagnostic consultations: visual, auditory, speech, and language evaluations
  6. Neurodevelopmental examination
  7. Psychiatric and psychological assessment
  8. Educational assessment
- 

**Table 90.3. Evaluation Process for Assessment of School Difficulties in the Child**

A special on-grounds school setting is usually required. Some of the characteristics of this on-grounds school include staff members who are skilled in special educational assessments, a low teacher:student ratio, specialized learning equipment including computers and word processors, and a curriculum designed to captivate the children's interest and motivate them to begin learning again.

The teacher is also often in the same relationship with the children as the group living worker and can offer them a structured environment, a model for identification, and a clarifying interpretation of their behavior, when appropriate. The teacher must be in free and regular communication with the other professional staff members, including the group living staff and the children's therapists. [Figure 90.1](#) shows some of the components of the educational process.



**Figure 90.1.** Components of the educational process. (From Lewis M: Residential treatment. In: Kaplan HI, Sadock BJ (eds.): *Comprehensive Textbook of Psychiatry*, vol 4. Baltimore, Williams & Wilkins, 1985, p. 1800, with permission.)

A major goal of the on-grounds school is to motivate the children to learn. This is done, in part, by providing multiple success experiences at whatever level the child is capable. While the children are in school, the school should have full responsibility for dealing with them. School staff should take advantage of what is known about the children as they attempt to improve the children's self-esteem, to motivate them to learn, and to provide opportunities for them to acquire adaptive skills. [Comer \(1980\)](#) notes that the child and adolescent psychiatrist can offer useful consultations to school staff members in this regard, and the child care workers and therapists can offer support to the teachers in understanding and managing a particular child. If the location of the RTC permits, the occasional child may attend a nearby public school, if indicated, while he or she remains in the residential program.

## THERAPY

Traditional modes of psychotherapy have a definite place in residential treatment. These modes include intensive individual psychotherapy with the child, group therapy with selected children, individual or group therapy or both for parents, and in some cases family therapy. However, several modifications need to be kept in mind.

The child also relates to the total staff of the setting and therefore needs to know that what transpires in the therapist's office is shared with all professional staff members. The therapist informs the child that what they discuss and do in individual therapy will not be revealed to other family members or to other children in the residential center but will in most instances be shared with professional staff members within the setting itself. Indeed, psychotherapeutic encounters often continue outside the setting of the therapist's office. Psychotherapy within a residential setting can hardly proceed without a full knowledge of the daily life of the child with peers and staff members in the group living situation and in the school. In addition, group living and school experiences can be made more meaningful if the therapist communicates freely and frequently with the child care worker and the teacher.

## PARENTS

Concomitant work with the parents is essential. The child usually has a strong tie with the parent, no matter how disturbed that parent may be. Sometimes the parent is idealized by the child but repeatedly fails the child. Sometimes the parent has an ambivalent or unrealistic expectation that the child will return home. In some instances, the parent must be helped to enable the child to live in another setting when that is in the best interest of the child. Most RTCs offer individual or group therapy with the parents, couples therapy, and, in some cases, conjoint family therapy.

The therapist who works with the child should also be the person who works closely with the family to help them deal with their feelings of guilt regarding placing the

child and with their tendencies to overcompensate for this placement by trying to indulge the child. Wild swings of ambivalence and reaction formation toward children before admission are sometimes replaced by hostility after they enter residential treatment.

Similarly, the parents need help when the children go home for a visit. The children's behavior often represents a demand for reparations in the form of constant demands for attention, sometimes virtually compelling their families to rearrange their schedules around the children's wishes. Children almost become tyrants during these visits and lead the parents to resent them because the parents are unable, without instructional help from the staff, to set limits. Children may also present a distorted picture of their parents to the staff as part of an unconscious attempt to ward off anxiety through splitting and projection.

### **Case Management**

All components of residential treatment must be effectively coordinated with regard to each specific child. The program should be carefully tailored to meet the needs of each specific child. Individualized treatment goals and objectives should be written with input from the therapist, teacher, child care worker, psychiatrist, psychologist, child, parent, referral source, and accountable funding source. Clinical social workers are specifically trained in the coordination of child and family services, as well as in individual psychodynamics and group dynamics. Clinical social workers increasingly play a case management role in residential treatment facilities, in addition to providing specific modes of therapy. In most RTCs, the clinician assigned to the child is also designated the case manager and is responsible for coordinating and integrating the work of the school, child care department, and clinical department, as well as that of the parent or guardian and accountable funding source. The case manager must have the responsibility and authority to ensure coordination and consistency of programming for designated children. In most RTCs, the clinician must also, of necessity, play a broader role in overall program management and the coordination of treatment teams. A particularly challenging problem must be faced by the clinician who serves as the case manager and therapist for the same child at the same time. The child and adolescent psychiatrist can serve a useful role as consultant to the case manager.

### **ROLE OF THE CHILD AND ADOLESCENT PSYCHIATRIST**

The presence of a fully qualified child and adolescent psychiatrist is essential for the staff of any RTC serving children and adolescents and their families. The exact administrative position may vary from that of chief administrator to medical director to psychiatric consultant and must be clearly understood by administrator and psychiatrist. Whatever the position and title, the psychiatrist should be asked to provide certain clinical, consultative, and educational services.

The need for the psychiatrist in RTCs has continued to increase in recent years because of changes that have occurred in the mental health delivery system and the resultant changes in the needs of the children and adolescents who are being admitted and treated. For example, relatively long-term inpatient psychiatric hospitalization has nearly disappeared in the United States. Many of the young patients who must be discharged under current criteria are not ready or able to be returned to their families even with the provision of the most extensive and sophisticated types of "wrap-around" services.

Thus, RTCs are receiving young patients with more serious, and less well-stabilized, psychiatric illnesses than in the past. To meet the challenge of this situation, the psychiatrist must be available to have significant input and responsibility in the intake process, in the communication with the referral source, in the initial evaluation and treatment planning, and, in particular, in the increasingly complex psychopharmacologic regimens the referring hospital may have initiated for many of the incoming young people.

As a corollary to the foregoing changes, there is an increasing need to be able to use referral to the short-term inpatient psychiatric hospital for emergency situations affecting the patient's safety or for severe regressive episodes that the RTC is not able to handle. Here again, the psychiatrist's role is crucial in helping the staff of the RTC make decisions in a given case, and in communication with the receiving hospital during the referral stage, after the patient has been admitted and during the planning for the return to the RTC.

The well-functioning RTC calls for a carefully orchestrated and multifaceted effort by all staff members, who come from many disciplines and from all departments (including "nonclinical" areas such as housekeeping, dietary, maintenance, and business office) whose goal is to help the individual child and his or her family. The psychiatrist must become integrated into the organization so to be able to help provide the unique understanding of the complexities of the individual child and to help in the ongoing and often changing need for balance in the many approaches concurrently being used to help that child.

The specific duties and functions of the psychiatrist should be carefully spelled out, understood, and clarified when necessary. Among these, it is recommended that the following should be included:

1. *Direct case consultation with the clinicians:* The psychiatrist consults once a week for 30 to 45 minutes individually with each clinician, to discuss the issues of that person's caseload as indicated. Both client-centered and consultee-centered case consultation should be offered ( [Caplan, 1970](#)). Especially important is a discussion of any psychopharmacologic treatment of a given child for whom the psychiatrist and the nonpsychiatric clinician may be collaborating.
2. *Participation in case conferences with team members:* At least 1 to 2 hours per week of the psychiatrist's time should be available as needed for participation in conferences dealing with any and all issues regarding a given child, ranging from crises to milieu dilemmas to longer-range treatment problems.
3. *Direct psychiatric evaluation of children:* Whenever needed, the psychiatrist will be available to evaluate a given child for various purposes, including intake evaluations, evaluations on admission, evaluations for psychopharmacologic treatment, evaluations for psychiatric emergencies, and direct consultative evaluations to explore clinical issues as requested by staff clinicians.
4. *Continuous case conference with clinicians:* A weekly 1-hour conference is suggested, led by the psychiatrist, at which one of the clinicians presents, for a series of weeks, an informative treatment process. Should there be clinical trainees in the agency, their supervisors may want them to be present at such a conference.
5. *In-service educational conferences:* The psychiatrist provides talks and seminars and participates in panel presentations on topics of interest to all staff of the agency.
6. *Informal visits to classrooms, living units, and other residential activities.* As the consultant becomes more familiar to staff and clients, communication is enhanced, and the consultant's contributions in more formal meetings are enhanced.
7. *Participation in agency committees and working groups:* On an as-needed basis, the psychiatrist should be available to make contributions to agency groups.
8. *Administrative consultation.* In many agencies, the administrators and the psychiatrist have developed a relationship such that the psychiatrist may be asked to provide consultation on the development of new programs, revision of existing programs, or other related problems ( [Caplan, 1970](#)).
9. *Direct monitoring of patients on psychotropic medications:* The psychiatrist not only must take responsibility for prescribing the medications, but also must follow-up the patients directly on a regular basis. A useful way to do this may be to conduct a weekly "medication clinic" wherein the psychiatrist spends approximately half an hour evaluating each child, reviewing the chart, and writing a progress note. In an agency of 40 residents, 20 of whom are taking medication, for example, the "medication clinic" would last for about 2½ hours each week in order that each patient be seen once a month.
10. *Medication rounds with agency nursing staff:* Once a week, a systematic review of charts should be done with appropriate documentation of findings and recommendations. The psychiatrist and nurse conduct the review, but input by other staff members is welcome.

In most RTCs, the psychiatrist does not have complete responsibility for the treatment of a given child or adolescent, but must in some aspects act as a consultant, in some as collaborator, and in the matter of medication, as the responsible physician. It is important that the psychiatrist and agency staff members understand and remain aware of these interrelationships at all times.

### **ADMINISTRATION OF MEDICATION**

Medication is most helpful when there is a clear indication or target symptom for a particular drug; it may help the child to engage in a therapeutic relationship and may facilitate attention and learning. Sometimes a drug is a useful additional measure during the stress of the initial period of residential treatment or later at times of crisis. Many children with thought disorders or hallucinatory experiences can be maintained in the RTC only with appropriate antipsychotic medications. As noted earlier, many children admitted are already receiving medications.

Inasmuch as the total staff, the child, and the family are all involved, it is important to develop a well-understood medication procedure. The initial request for medication may arise from the child, a parent, or any staff member and is then processed by the child's therapist. A full discussion by the entire staff is necessary to clarify the goals for medication use and its anticipated effects on the child. Impulsive proposals for the use of medication are sometimes made by staff members who feel frustrated and desperate in their efforts to manage especially difficult behavior. Countertransference problems are sometimes an unrecognized motivation.

The child should have as thorough an understanding as possible of the medication's effect and the reasons for taking it. Children may have special fears about taking



psychotropic medication. Often, these fears and unrealistic expectations are elaborations of statements or attitudes of peers, staff members, parents, or other family members.

It is important to talk with the child's parents or guardians about the proposal for medication. In addition to obtaining informed consent, the staff members can correct misunderstandings and can encourage the parents to develop a reasonable, supportive attitude toward the child's use of medication. To achieve this end, the following procedure is useful:

1. The therapist, in consultation with the staff, the child, and the parents, defines the target symptoms, the expected goals, and the anticipated effects of the medication. The therapist explains the procedures to the parents.
2. A careful history is taken of significant previous illnesses or allergies in the child or family members and of any history of previous drug experience.
3. The child's physical state is reviewed.
4. A baseline mental status examination is performed, and various behavior ratings may be made.
5. Informed consent is obtained in writing from the parents or legal guardians.
6. All this information is made available to the entire staff for discussion. For this purpose, a brief synopsis of the drugs commonly used should be available to the staff, and the therapist should prepare a summary that follows a standard outline for all to read. A key decision-making conference should then be held.
7. After the decision is made, appropriate laboratory tests are performed, and the medication is prescribed and administered.

Since the early 1990s, the numbers of children admitted to RTCs with severe psychiatric disorders and those taking psychotropic medications have increased steadily. It is not uncommon now that 90% of such children in residential treatment are on such medications, and in some settings two thirds of such children will have received two or more psychotropic medications simultaneously ([Connor et al, 1997](#)).

Appropriate medical records and written procedures and protocols for psychopharmacologic therapy must be in place for these children. The procedures and protocols include the following:

1. *Medical records:* The child's medical records relevant to psychotropic medications should include doctor's orders, prescriptions, laboratory reports, reports from other hospitals or facilities, progress notes, documentation of medication reviews with the nursing staff, copies of informed consent, and authorizations by parents or guardians.
2. *Written protocols for psychopharmacotherapy:* These should include procedures for initiating therapy (including the issues as described earlier) and for maintaining and discontinuing therapy. Staff are sometimes reluctant to discontinue medication because they fear a return of difficult behavior in the child. The reluctance may take the form of rationalizations such as upcoming vacations, schedule changes, room changes, staff changes, parental visits, or other possible stresses. The clinical reasons for discontinuing medication should therefore be carefully documented.
3. *Protocols for laboratory and other studies:* For a given medication, or for a given category of medications, the specific schedules of studies including vital signs, laboratory studies, abnormal involuntary movement scale testing, rating scales, checklists of side effects, electrocardiograms, and other tests should be documented for the premedication phase, for the times of changes of dosage, and for the ongoing maintenance phase. Behavior changes in relation to itemized target symptoms and initial behavior ratings should be regularly documented.
4. *Procedures for discharging clients on medications:* Before discharge, it is important to have established which outside practitioner will take responsibility for continuing the treatment and to document that the parent or guardian accepts the responsibility to implement it.

## COMMUNICATION

To maintain the necessary communication in the residential staff, the staff members must meet as a total group, without an agenda, at least once a week. These meetings allow feelings to come to light that may otherwise remain hidden and silently ferment. Role conflicts, countertransference problems, treatment blind spots, and residential center policies with regard to such issues as limit setting, outside visits, treatment approaches, and work with such agencies as an outside school and a welfare agency are some of the subjects that are discussed. Greater staff cohesion, collaboration, and commitment occur when all staff members are involved in every aspect of the child's treatment. Contributions from social system theory have enabled RTC staff members to view their interaction as part of a complex social system, constituting a further aid to communication.

To monitor the course of treatment, staff members must also attend regular comprehensive case review conferences, at which reports from group living, school, and therapy staff members are presented and discussed, along with additional data derived from neurologic examinations and psychological tests. Diagnoses and treatment goals often need to be revised as the child becomes better known to the staff. The revision may go in either direction; that is, a psychotic process may gradually be revealed, or a child may show strong restitutive capacities once he or she is in a stable environment. [Colligan et al. \(1981\)](#) developed an organizational grid to provide a visual representation of the factors involved in residential treatment. The grid evaluates diagnostic information from four developmental dimensions: physiological, emotional, sociobehavioral, and cognitive—academic. The effects of these dimensions on family, school, and group living can then be monitored.

## COURSE OF TREATMENT

Children who enter a residential center may experience anxiety and feelings of loss, as well as some relief. They may temporarily deal with this anxiety or relief by exhibiting good behavior and a strong apparent wish to please. This so-called honeymoon period, which may last from a few days to a few months, soon gives way to the reemergence of the children's characteristic behavior patterns, albeit in modified form. The children seem to attempt to reestablish many of the characteristics of their relationships with adults and peers. The slow and difficult task of working through unconscious attitudes and fantasies that perpetuate their now maladaptive behavior constitutes a major part of the total therapeutic effort, which may last for 2 or more years. There is no single course of treatment because the variables are many, including the strengths and weaknesses of the particular child, the strengths and weaknesses of the parents, the skills of the staff, and the degree to which a suitable discharge plan can be put into effect.

## TERMINATION

As termination and discharge approach, earlier feelings of loss are often evoked again, and temporary regression may occur, with heightened acting-out behavior, sometimes of an aggressive kind. Intense anxiety about abandonment may also reappear. The child with a diagnosis of borderline personality disorder especially needs a great deal of support at this time. It is necessary to arrange preplacement visits to avoid precipitous changes in the child's life.

## DISCHARGE PLANS AND FOLLOW-UP CARE

Children, parents, and staff members all anticipate with ambivalence the time of leaving. Expectations—some realistic, others unrealistic—tinged with apprehension and anxiety about real or imagined problems abound. In a sense, preparation for departure begins at admission. Actual preparation for leaving should be a planned process, with the child's best interests being of paramount importance. It is important that the children at all times participate in planning their future lives.

Cure, whatever that may mean, is rarely an attainable goal for children in residential treatment. Improved object relations and social adaptation, ability to function in an outside school and later at work, and the working through of certain kinds of trauma are more realistic goals for the child. Similar goals obtain for the parents. The degree to which these goals are achieved is sometimes difficult to determine. The outcomes that may occur range from no change at all to improvement in the whole family.

The range of disposition plans varies according to the outcome. Return to the family with or without further outpatient treatment, day treatment, placement in a group or foster home, boarding school, further residential treatment, and custodial care are some common options. Many RTCs have developed some of these options as part of their own programs, so a child may be admitted, say, into a day treatment program or into a day treatment and on-grounds school program, with full residential treatment reserved as a possible option, or vice versa.

Intensive residential treatment should be seen as a phase of treatment, rather than as the one definitive treatment. Both the children and their families are usually vulnerable. Some may always show a tendency to paranoid behavior when under stress. The residential treatment home may well need to continue to represent the core of stability for these children as they progress through adolescence and young adulthood.

## RESULTS

Many attempts at follow-up studies have been made, but unfortunately, numerous serious methodologic problems plague research in this area. Most studies of outcome are subjective and are difficult to evaluate. Criteria for diagnosis, treatment, and outcome are not sufficiently well defined for research purposes, and the intervening variables make controlled studies virtually impossible. Clinical impressions suggest that good residential treatment programs do provide a beneficial experience for the child during the period of residential treatment and may lead to more satisfactory development and functioning in the child than would otherwise have occurred had the child not been admitted.

[Taylor and Alpert \(1973\)](#) find, however, that the degree of change that children achieved in residential treatment is not significantly related to postdischarge adaptation. [Lewis et al. \(1980\)](#) similarly find that most children who receive about 2 years of residential treatment in one particular setting fare poorly at follow-up according to the objective measures they use to assess outcome. Thus, improvement made in residential treatment does not determine the degree of adjustment experienced on return to the home and community. Instead, Lewis et al. find that ecologic factors and postdischarge factors are important in determining outcome. Successfully adjusted children show a greater increase in family and community support during enrollment and also receive more help from community agencies and special school programs.

Lewis et al. also note that, during the course of residential treatment, crucial attachments are formed between the children and the staff members, and these investigators recommend that these attachments be protected. Thus, the same residential facility staff should be prepared in many instances to continue their commitment and attachment to the children and their families through and beyond adolescence. In effect, the RTC in many instances should serve as a secure base until adulthood. Without that security, the hard-won gains achieved during residential treatment for the vulnerable child and adolescent are essentially undone.

[Wenning \(1988\)](#) reports a high recidivism rate and a high rate of affective conditions in a follow-up study of children diagnosed as borderline while in residential treatment. An outcome study using Child Behavior Checklists of 36 children and adolescents at admission, specific time points, and at discharge found that children who demonstrated oppositional defiant behavior and symptoms of a conduct disorder appeared to do most poorly in the residential treatment setting ( [Joshi and Rosenberg, 1997](#)).

Another study ([Leon et al., 2000](#)) notes that many RTCs are currently struggling to meet the intensive treatment needs of those children and adolescents in residence who have serious psychiatric disorders. One consequence of this struggle to provide the needed treatment is the increased rate at which these RTCs are now inappropriately referring for hospitalization children and adolescents who do not meet usual criteria for hospitalization. Further, RTCs that are struggling in this way are "failing to provide the same levels of supervision as programs whose referrals were largely high risk" ( [Leon et al., 2000](#)). This raises the question of the level of training staff receive in intensive crisis management and the need for state departments of children and families to provide more resources to these struggling RTCs.

## FUTURE TRENDS

The place of residential treatment in the continuum of childrens' services and the vitality of residential treatment programs face serious social, political, and economic challenges in the years ahead. The dearth of follow-up studies demonstrating the efficacy of residential treatment continues to plague its advocates in the presence of increased demands for results and cost effectiveness. Many state and federal policymakers emphasize family preservation or alternative family placement for children currently using residential treatment. Unfortunately, residential treatment is sometimes only viewed as a treatment of last resort, especially when child and family psychopathology has increased to a dangerous level just short of requiring hospitalization of the child. A preferred concept is to view residential treatment within the context of a connected broad range of mental health services.

Staff recruitment and staff retention are serious problems for many residential treatment organizations because budgetary constraints limit resources in many areas for childrens' services. The statistical trend toward an aging society not only increases the demand for resources for the elderly but also potentially pits the elderly against the very young in the increasing competition for mental health, medical, and social services. The resources needed to combat the epidemic of acquired immunodeficiency syndrome and the rapid rise of drug abuse and drug-related violence increase the strain on all social service resources. Emotionally disturbed children are not a voting constituency, and historically their families have not been an effective political force in advocating for their specific needs. Residential treatment organizations often act as advocates for disenfranchised children but are often discredited as being biased or self-serving in their advocacy.

[Davis et al. \(1989\)](#) report that traditional long-term residential treatment facilities are adapting their programs and treatment foci in an attempt to meet the changing needs of children and adolescents. Children are presenting with more intensive treatment needs, whereas family and community resources have diminished for a large population of children. Consumer demands for results and accountability have increased, and agencies must offer a larger range of specialized clinical services to children who are in residential treatment. Residential treatment facilities have responded by developing subacute intensive treatment services within existing facilities. These include 90-day intensive assessment units, 30- to 90-day alcohol and drug treatment units, physical and sexual abuse and trauma response teams, specialized foster care and adoption services, aftercare and independent living programs, outreach family support services, and family preservation services such as Home Builders.

In response to consumer demand for results and accountability, many residential treatment facilities have also modified their programs to be in compliance with the standards of the Joint Commission on Accreditation of Healthcare Organizations (JCAHO). The JCAHO provides comprehensive standards for residential treatment in the areas of administrative and clinical management, patient management, special treatment services, patient services, and environmental management. JCAHO standards emphasize professional staff organization and development, program evaluation, patient safety, patient rights, and all aspects of quality assurance ( [Joint Commission on Accreditation of Healthcare Organizations, 1989](#)).

The Civilian Health and Medical Plan for the Uniformed Services (CHAMPUS) and the American Psychiatric Association have also played a major role in developing comprehensive quality assurance standards and in implementing a peer review system for residential treatment. As of July 1989, 108 JCAHO-approved residential facilities also met CHAMPUS quality assurance requirements and were being monitored through a peer review process.

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# 91 CHILD AND ADOLESCENT PSYCHIATRIC EMERGENCIES

Lynelle E. Thomas, M.D., and Robert A. King, M.D.

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## NATURE AND SCOPE

The emergency nature of a child or adolescent psychiatric problem is defined both by the severity and urgency of the potential threat to the child's and family's safety and well-being and by the community and clinical resources the family is able to access and use to address it. Thus, some situations, such as intense aggressive or homicidal threats or outbursts, acute psychotic or anxiety states, serious suicide attempts, ingestions or intoxications, or acute toxic metabolic states, usually require immediate psychiatric attention in a setting that can muster the full range of acute medical and psychiatric diagnostic and therapeutic interventions. Conversely, the presentation of many other more chronic or less urgent cases to the pediatric psychiatry emergency service reflects the absence of adequate mental health resources in the community or the family's relative inability to access or use clinical and social resources that could have prevented the crisis or permitted its management in a less acute outpatient setting.

Although it is usually clear that these latter cases require prompt intervention, the perception that the case is an emergency and should be seen in a tertiary emergency service is more relative. For example, under what circumstances does an unhappy, neglected child's suicidal ruminations or threats indicate an imminent risk and need for crisis intervention? When does normative adolescent oppositionality or risk taking evolve to illicit substance use, unprotected sexual activity, and other antisocial and high-risk behaviors, and at what point do these become so severe that emergency psychiatric intervention becomes urgently imperative?

The judgment that a given child's thoughts, feelings, or actions constitute a psychiatric emergency reflects some adult's perception that the child's condition is serious, urgent, or unmanageable in the current environment. As a corollary, a multiplicity of adults or agencies may potentially initiate the referral to a child psychiatric emergency service. These include parents, extended family members, teachers, police, mental health clinicians in the community, and child welfare workers (Halamandaris and Anderson, 1999). In addition, many facilities such as youth shelters, residential treatment centers, and juvenile detention use the hospital-based child psychiatric emergency service in the absence of adequate on-site emergency psychiatric capacities.

Psychiatric emergencies were once considered uncommon in childhood. In recent years, however, the number of child and adolescent emergency patients has been on the rise. For example, between October 1, 1963 and July 31, 1964, the number of psychiatric consultations in the Yale–New Haven Hospital emergency department (ED) in New Haven, Connecticut, for children less than 15 years of age, represented only 0.61% of the pediatric ED population ( [Schowalter and Solnit, 1966](#)). In contrast, the annual number of child psychiatry-related visits to the Yale–New Haven Hospital pediatric ED was 2.49% of all pediatric visits in 1995 and 3.94% of those in 1999. Thus, by 1999, as a percentage of all ED cases, the proportion of child psychiatric emergency cases increased almost 60% over 1995 and over 500% compared with 1963. The increase in absolute numbers was even greater, given the increased number of pediatric ED visits for all complaints. The higher number of cases in 1999 versus 1995 resulted, in part, from an increase in referrals for depression and an upward trend in aggression-related complaints ( [Santucci et al., 2000](#)). The magnitude of this clinical burden is apparent if this referral rate is extrapolated to the more than 31 million annual child and adolescent ED visits occurring nationally ( [American Academy of Pediatrics, 2001](#)).

The extent and causes of these dramatic changes are unclear, given the paucity of national data on child psychiatric ED use. However, it may be speculated that increases in the number of children living in poverty and single-parent families may be taking their toll in what many perceive as rising rates and severity of childhood psychopathology ( [Achenbach and Howell, 1993](#)).

Dwindling funds and efforts at cost containment in mental health and community-based social service systems in many states have transformed hospital-based emergency services into a major provider of mental health services. Managed-care–driven strictures have further eroded the availability of mental health resources in the community and have dramatically shortened lengths of hospital stay, thus denying many children and families adequate outpatient treatment in the community and effective inpatient treatment when that becomes necessary. This is apparent in the rising number of repeat visits to child psychiatry emergency services and the number of children receiving repeated “revolving door” short-term hospitalizations. Indeed, in many communities, the psychiatric ED has become the *de facto* mental health system of care.

Availability of community services aside, epidemiologic data help to illuminate the high rates of ED use. For example, although the incidence of completed adolescent suicides has leveled off for the United States as a whole over the past few years after a multidecade rise, the rates of serious suicidal ideation and clinically significant suicide attempts remain strikingly high in the general adolescent population (Centers for Disease Control and Prevention, 2000). In addition, rates of completed, and presumably attempted, suicide continue to rise in certain segments of the youth population, such as children younger than 10 years of age and African–American adolescent boys ( [Centers for Disease Control and Prevention, 1995](#); [Centers for Disease Control and Prevention, 1998](#)).

The increasing rates of child psychiatric ED use and the shifting ecology of these cases raise largely understudied and important practical and research questions regarding how child psychiatric services are actually used, how well they address the emotional and behavioral problems of patients and their families, and how they may be more efficiently organized in the context of comprehensive systems of care.

With this background in mind, this chapter discusses the complexities of child and adolescent psychiatric emergencies and hospital-based assessment and examines the problems confronting ED-based child and adolescent psychiatrists. We also discuss approaches to assessment, treatment, and disposition planning that are unique to this population, with a special focus on the uncooperative patient. For the broader topics of the psychiatric evaluation of the child and the assessment of the suicidal child, see [Chapter 41 \(American Academy of Child and Adolescent Psychiatry, 1995; American Academy of Child and Adolescent Psychiatry, 2001; King et al., 2000\)](#). For emergency psychiatric evaluation in the inpatient pediatric setting see [King and Lewis, 1994](#).



# HOSPITAL-BASED CHILD PSYCHIATRIC EMERGENCY EVALUATION

## Adaptive Context of Child Psychiatric Emergencies

Child psychiatric emergencies presenting in the hospital setting are most often characterized by intense symptoms, perceived danger, and a sense of urgency complicated by the perception of imminent catastrophic outcome and frequent conflict among the parties involved. Despite this acuity, child psychiatric emergencies are usually the outcome of complex ongoing processes rather than sudden, discrete events. Occasionally, a previously well-functioning child with some underlying vulnerabilities may abruptly decompensate and may display psychiatric symptoms in the presence of some critical or traumatic event or organic process. More often, however, the acute emotional or behavioral symptoms that bring the youngster to the attention of the emergency service have been preceded by a longer history of emotional or behavioral difficulties. Thus, a key element of the emergency child psychiatric assessment is to answer the questions: "Who is concerned about the child?" and "Why now?"

A child's functioning and psychological well-being are highly dependent on the family, school, and community setting in which he or she lives and studies. Anything that adversely affects this system has the potential to precipitate a crisis. A child psychiatric emergency usually represents some perturbation or pathology in one or several of the elements in this delicately balanced ecosystem. Either an efflorescence of the child's psychopathology has overwhelmed the caretaking system or the caretaking system has, in some fashion, become less sufficient or less adequate. From this perspective, many child psychiatric emergencies can be conceptualized as a mismatch between needs and resources (Allen, 1999). The corresponding goal of child psychiatric emergency services evaluation is then to clarify the nature and the cause of the imbalance that has arisen and to identify the resources needed (e.g., safe environment, psychoeducation, psychopharmacotherapy, outpatient therapist, family support services) to restore a stable equilibrium. Systematically clarifying the details of the precipitants to the crisis is thus paramount in determining the needed interventions and disposition.

## Goals and Aims of the Hospital-Based Child Psychiatric Emergency Assessment

The primary goals of the child psychiatric emergency evaluation are, as expeditiously as possible

1. To obtain each informant's account of the reason for referral.
2. To develop a working alliance, if possible, with the patient and other involved parties around the assessment and disposition planning.
3. To obtain a focused developmental history of the child's current difficulties and prior functioning against the backdrop of the child's family, current living situation, and any involved clinicians or agencies with particular attention to the possible precipitants of the current crisis.
4. To perform a mental status examination, with particular attention to evidence of suicidal or homicidal ideation, hallucinations, delusions, or thought disorder, evidence of confusion, disorientation, or other signs of delirium, and intense anxiety.
5. To develop a differential diagnosis, including a formulation of what changing factors have precipitated the need for emergency evaluation at the present time.
6. To arrive at a judgment regarding the degree of probable risk to the patient's safety or that of others.
7. To identify interventions that will help to contain and ameliorate the patient's difficulties.
8. To plan and implement a disposition.
9. To collaborate effectively with other clinicians and care providers involved in the case, both within and beyond the hospital setting.

## General Considerations

The hospital ED is designed to contain and resolve urgent or life-threatening situations. The "triage model"—rapid determination of imminent dangerousness, containment, and referral—typifies the process of most hospital-based psychiatric emergency consultation and care. Some beleaguered psychiatric emergency services confine themselves to addressing only two questions: (a) is the child a danger to himself or others? (b) does the child need to be hospitalized or can he or she be discharged back home? Although these dispositional questions must remain at the forefront of the busy emergency clinician's mind, circumscribing the evaluation too narrowly to these areas both precludes an accurate understanding of the clinical situation and renders the ED visit of little ongoing value to the child, family, or treatment effort. Given that most crises referrals arise out of multiple factors in the child's life, it is important that, no matter how expeditiously the evaluation is conducted, it provides the child, family, and clinicians with some useful perspective on how the crisis came about and how it fits into the overall trajectory of the child's life and clinical care.

The assessment and management of child psychiatric emergencies differ from the routine office evaluation in several important ways. The severity, dangerousness, or urgency of the symptoms usually requires rapid clinical decision-making and treatment implementation. Furthermore, the emergency assessment must often proceed under unpropitious circumstances constrained by time pressures, in the relative absence of trained support personnel or optimal physical arrangements, with an unfamiliarity with the patient and family, with the unavailability of key informants, and with the lack of timely, appropriate alternatives for disposition.

Yet another constraint on emergency evaluations is the inconvenient hours at which they often occur—late at night or on weekends, when important informants, such as primary clinicians, teachers, or social welfare agency workers, may be unavailable. The already difficult task of arranging an appropriate disposition at such hours is further complicated by the frequent unavailability of insurance reviewers needed for precertification, psychiatric hospital admissions staff members who can provide prompt information regarding bed availability, or outpatient clinicians who can undertake the responsibility of seeing the patient promptly for follow-up. The availability of sufficient social work, psychiatric nursing, or other professional staff members to assist in these information-gathering and coordinating tasks is essential to prevent burnout of clinicians faced with large volumes of child emergency evaluations.

Time constraints and the urgency of the situation do, of course, require that the clinician be active in eliciting the most relevant data in a time-efficient manner. Right from the onset of the interview process, the experienced emergency clinician begins to prioritize symptoms and to formulate and test tentative etiologic and diagnostic hypotheses that guide further questions. At the same time, the clinician also begins to ponder what interventions and dispositions these diagnostic hypotheses imply. Unlike less urgent settings, the emphasis is on clarifying the child's current symptoms and functioning, the factors in the child's living situation that have served to stabilize or exacerbate difficulties, and the resources and competencies available to the patient and the family.

## Physical Setting

It is important to find a quiet evaluation area, where the clinician, adults, and child will not be frequently distracted by the sights and sounds of physically ill and upset children and their families. The area needs to be free enough of dangerous or delicate medical equipment or furnishings so the clinician need not be preoccupied with keeping the room and the patient safe. The spot needs to be secluded enough that the belligerent or uncooperative psychiatric patient will not disturb other families in the ED, yet near enough to other staff that reinforcements can be called on if needed for safety or calming or to prevent elopement.

## Informants

The clinician's first task is to identify why this particular child has been brought to the ED at this particular time. The impetus for child psychiatric emergency referrals, with few exceptions, comes from adults in the child's life, rather than from the child. Obtaining a full and accurate diagnostic picture for any child psychiatric assessment requires gathering information from diverse sources, including the family, school staff, clinicians, and the child himself. In the emergency situation, however, the initial or primary informant may be an adult other than the primary caretaker, for example, police or corrections officers, school personnel, or representatives of various social service agencies. The wide array of potential referents and perspectives complicates the task of the evaluator, who must efficiently interview multiple informants and, like a detective, rapidly consolidate and reconcile sparse, often conflicting data.

From a practical standpoint, data are collected as they become available. At a minimum, the emergency assessment entails direct interviews with the child and all adults who accompany the child to the hospital setting, as well as any caretakers, clinicians, or caseworkers who are accessible by telephone. On a practical level, if the child is brought in by nonparental parties (e.g., friends, police, correctional officers, or child welfare workers) who may not want to remain in the ED for the evaluation, it is essential to speak with them directly and to obtain immediate contact information for those persons with direct knowledge of the precipitating crises and the child's recent circumstances and responsibility for the child's care and disposition. Even when the child is not accompanied by a custodial parent or legal guardian, most states give the emergency clinicians the latitude to initiate emergency treatment of a child, including contacting of collateral informants, without parental consent. In such cases, it is always clinically desirable to contact and to involve the parents as soon as possible. Many states also permit adolescents to

seek mental health without a parent's involvement. These patients should also be encouraged to involve parents and other adult supports.

The presenting complaint and reasons for the referral are often described very differently, depending on the informant. These discrepancies, referred to in the research literature as *informant variance*, arise for a variety of reasons ([American Academy of Child and Adolescent Psychiatry, 1995](#)). Although these discrepancies complicate the diagnostician's task, when markedly differing accounts or divergences of perspective do occur, they provide potentially important clues to the nature of the child's crisis. At the very least, they point to troublesome lacks of continuity in the child's holding environment and a lack of shared consensus between the child and important adults.

Differences may stem from the different contexts in which the child is observed, the standard of judgment employed, and variations in the demands or stressors impinging on the child in each setting. This is particularly the case when children's symptoms are situation specific (e.g., occurring only at school, only at home, or only at one parent's house, but not the other's).

Informants may differ in their access to information concerning the child's feelings and behavior. Parents may be quick to report a behavior of the child that they find disturbing or annoying, but they may fail to recognize how discord within the family system (e.g., domestic violence, separation, divorce) may directly precipitate crisis symptoms within the child. The child, in contrast, even if aware of and able to describe the problematic behavior verbally, may refrain from doing so out of defensiveness, shame, or fear of reproach. The interviewer must also be aware that vagueness and minimization of problems can sometimes indicate an attempt to maintain some secret within the family system, such as a parental mental illness, illegal activity, child or parental substance abuse, domestic violence, or physical or sexual abuse. In these cases, the evaluation can be extremely difficult, because the events surrounding the crisis may never be completely clarified.

### **Child Interview**

In traditional office-based child psychiatric assessment, several hours and more than one interview with the child are usually desirable to place the child at ease with the interviewer and to obtain a full picture of the child. In contrast, the emergency assessment must be completed within the confines of a single interview. The emergency child interview and mental status examination must also reckon with the characteristic lability of children and their propensity to fall back to more immature or oppositional ways of coping, especially when they are confronted with the anxiety and distress associated with a hospital ED setting. Although it may not accurately reveal the child's optimal or characteristic level of functioning, this "snapshot" of the child in the ED often provides a valid picture of the child's vulnerability to regress under stress and how such regression may have led to the emergency referral.

Although every effort must be made to place the child at ease and to obtain his or her cooperation in understanding what has brought about the crisis, this is often difficult. The high levels of expressed emotion in the events leading up to ED referrals and the coercive processes required to bring the child to the ED often stimulate the child's oppositionality. As a result, the child in the ED is often sullen, mute, withdrawn, or antagonistic.

To the child who is aggrieved or sullenly refusing to talk, the clinician can validly invoke what has been termed the "constructive use of ignorance" by observing that, because they have never met before, the clinician really does not know what has led up to the ED visit and would genuinely like to hear the child's view of what has been happening.

### **Child Mental Status Examination**

The mental status examination is of particular importance in the emergency evaluation ([American Academy of Child and Adolescent Psychiatry, 1995](#)). In trying to understand the nature of the crisis and the interventions needed, the clinician will be especially attentive to evidence of psychosis, delirium, or other organic process, intoxication, dissociation, or extreme anxiety, depression, or elation. The presence of any of these factors is likely to render the patient more labile and vulnerable and points to the need for more intensive interventions and diagnostic studies. Hence, the clinician must be alert to and explicitly note the presence of the following:

- Disorientation, confusion, and fluctuating levels of consciousness
- Incoherence of thought or speech
- Evidence of hallucinations or delusions
- Impaired memory
- Slurred speech, ataxia, or apraxia

Assessment of safety additionally requires explicit attention to the following:

- The presence of suicidal or homicidal ideation
- Aggressive threats or ideation
- Impulsivity
- Proneness to regression or agitation during the interview
- Poor judgment and insight and limited intelligence
- Mood lability

### **Family Mental Status**

The child and parents' attitudes toward the examiner and toward each other during the interview provide valuable clues to whether the child and family can be effectively and safely worked with in an outpatient setting if the child is discharged from the ED. Hence the examiner will note carefully how the child and family interact, how the child relates to the examiner, and to what extent the child can own and reflect on the behaviors that have led to the ED visit.

To what extent can the child and parents, with the examiner's help, at least partially agree about the problems that must be addressed and achieve a working consensus about possible realistic (nonmagical) ways of working on them? In the absence of such a consensus, outpatient follow-up is not likely to be feasible or successful, as for example, when the child persists in denying any problem, the child and parent remain locked in mutual recrimination. If the referral for emergency assessment comes from adults or agencies other than the parents (e.g., neighbors, police responding to a family disturbance, or school personnel concerned about a student's behavior), both child and family may portray the "problem" as the result of the school or neighbors' unreasonable expectations or the derelictions of others. This posture of defensive externalization also bodes ill for enlisting the child and family's cooperation with any treatment recommendations. Conversely, families and children who, with admitted concern, recognize that certain problematic behavior patterns recur in their family may be more likely to have the commitment to make the transition from crisis intervention to ongoing outpatient treatment.

## **UNCOOPERATIVE OR AGGRESSIVE PATIENT**

### **Special Considerations**

We now consider special challenges posed by the uncooperative or aggressive patient. Oppositional and aggressive outbursts at school or at home are frequent precipitants of ED visits. Many such youngsters are brought to the ED by either police or emergency medical services, sometimes in physical restraints. In the ED, such children and adolescents often continue to be agitated, belligerent, and impulsive. Profanity, yelling, and verbal threats are unnerving and upsetting to the clinician, ED staff, and other families and children in the ED.

Aggressive or violently oppositional behavior is perhaps the most difficult management challenge facing the child psychiatry emergency clinician. The clinician must approach the potentially violent patient calmly but cautiously, with the twin goals of performing an assessment and simultaneously bolstering the child or adolescent's capacity to remain in behavioral control.

### **Establishing the Differential Diagnosis**

The assessment of the aggressive, agitated patient begins with an evaluation of the possible causes. Aggressive behavior can occur in the context of a wide spectrum of psychiatric conditions. Thus, establishing a differential diagnosis is important in choosing short-term interventions and developing a disposition and longer-term



treatment plans.

The cause of oppositional, aggressive, and ultimately violent behavior in children and adolescents is usually multidetermined. It evolves from a transaction between the child's temperament and immediate environment, and both social and psychological factors and neurobiological processes influence it.

Poor impulse control is an important risk factor for aggressive outbursts. Impulsivity is a symptom common to several mental disorders of childhood and adolescence, most notably attention deficit disorders, hypomania, and conduct disorder. Children with cognitive deficits and developmental delays, such as mental retardation, autism, or other pervasive developmental disabilities, may have a limited repertoire of social coping skills and hence may react to stress and frustrations with aggressive behaviors.

Children reared in poorly structured, chaotic, or violence-prone families and children who have been chronically or acutely traumatized may exhibit maladaptive, aggressive behaviors in response to stress or confrontation. Especially in adolescents, substance use is a common precipitant of disruptive, aggressive behavior by virtue of impairing judgment, increasing irritability, disinhibiting behavior, and exposing the youngster to potentially threatening situations. Underlying psychotic states, especially those characterized by mania, paranoia, or command auditory hallucinations, are also associated with agitated, aggressive behaviors.

It is also important to be alert for various organic conditions that can result in irritable, aggressive, or disorganized behavior. Toxic metabolic states, whether from ingestions, medication side effects, encephalopathies, or other medical illness, can produce delirium with disorganized and aggressive behavior. Neurologic conditions associated with irritability and aggressive behavioral outbursts include postconcussive states, frontal lobe lesions, and temporal lobe epilepsy. Hence, a careful medical history, including medications and substance use, is important ( [King and Lewis, 1994](#)). Finally, the overstimulating, chaotic, and confining nature of the ED itself can exacerbate anxiety, irritability, and aggressive impulses for many patients and families.

### Specific Aspects of the Assessment

Data crucial for rendering an accurate portrayal of the context include a history of recent and past aggressive behavior, as well as psychiatric, family, and social information. A detailed history of the presenting violent episode is mandatory. This includes preceding and precipitating events, the details of the episode itself, and its aftermath. It is important to elicit the patient's own perspectives regarding the episode and to compare these with the perspectives of the other involved or witnessing parties. The social setting and behavior of the victim and the response of others in the environment should be clarified. The precipitant of such challenges is often a clash between the child and an adult authority figure over limit setting. The role of narcissistic injury, humiliation, intense anxiety, or challenges to the child's autonomy, self-concept, or inability of the youngster to escape from such challenges should be sought in the interview. It is important to assess and document the following in the child or adolescent (Reid, 1988):

1. The degree of premeditation and planning versus impulsiveness.
2. Egosyntonicity or dystonicity.
3. Consistency with the patient's past behaviors or style (including chronic bullying).
4. Extraordinary or uncontrolled rage and use of weapons.
5. The validity of perceived self-defense.
6. Evidence of grossly impaired judgment or consciousness.
7. Bizarre or delusional behavior of thought content.
8. Risk of self-injury during the violent episode.
9. The extent to which the child can remember the details of the episode (including his actions and their consequence), accept responsibility, or express remorse.

Each of these variables has implications for the diagnosis and disposition.

### Management

#### *BASIC TENETS*

In approaching the emergency assessment of the potentially violent patient, the first and overriding consideration should be that of safety: the safety of the patient, the safety of others, and the clinician's own safety. A patient must be under behavioral control before a thorough medical and psychiatric evaluation can be conducted. Safety can be maintained and harm averted in the following ways.

The clinician should always be polite and respectful to the patient and concerned parties. Maintaining a calm atmosphere and avoiding irritable and counteraggressive responses go a long way toward diffusing hostile interactions. The clinician should never be isolated with an aggressive patient without the ability to summon assistance. Placing oneself between the patient and the door permits exiting without obstruction, should the need arise. It is important to be attentive to one's own feelings of discomfort, anger, or threat, because these feelings provide important cues alerting the clinician to the potential for violence and the need to seek help.

#### *SETTING*

The interview and waiting area should also be free of sharps, cords, or other potentially hazardous furnishings or medical equipment that are unfortunately ubiquitous to most emergency examination rooms. Interviews should be conducted in a space that will allow some degree of privacy and decreased stimulation. The interview area should, however, also allow some level of visual surveillance from the outside and ease of physical access should summoning staff assistance or security personnel become necessary. An easily identifiable alarm code should be used to alert other staff members to potential or actual violence. The space must also be adaptable to facilitate seclusion and restraint, if needed; restraints should be available nearby in areas where violence could occur.

#### *BEHAVIORAL INTERVENTIONS*

Protocols for managing disruptive or aggressive pediatric patients should include algorithms for the progression of interventions, from least restrictive and invasive to more restrictive and invasive. Clarity of communication and firm limit setting are essential and may be effective at deescalating a potentially violent situation. Staff should supportively and firmly communicate to the patient and family what will and will not be tolerated in the ED while at the same time offering a small range of acceptable alternatives.

When agitation is the result of organic causes and is accompanied by confusion and disorganization, particular attention is necessary to try to keep the patient carefully monitored and oriented. This can be accomplished by diminishing stimulation while maintaining adequate levels of lighting to avoid sensory ambiguity. It is often helpful to provide a staff companion or family member to remind the patient of where they are and to inform him or her of what is happening. Caution with sedation is needed, so as not to obscure fluctuations in level of consciousness.

#### *"Chemical Restraint"*

If these measures do not succeed, it is at this stage that the need for immediate medication should be assessed. There is no specific acute pharmacologic treatment for violent behavior *per se*. However, "nonspecific sedation" is frequently used in the management of acutely agitated patients, whatever the cause. The choice of agent is usually between a neuroleptic and a benzodiazepine. Haloperidol and lorazepam are perhaps the most commonly used agents and are available in most pediatric EDs.

Lorazepam is a nonspecific, sedative-hypnotic benzodiazepine that is readily absorbed after oral or intramuscular administration. It has a relatively short half-life (10 to 20 hours) and produces no active metabolites. In addition to its sedation and anxiolytic properties, benzodiazepines also have the advantage of reversibility with the benzodiazepine antagonist, flumazenil. Dosing in children ranges from 1 to 2 mg orally or intramuscularly every hour until sedation is achieved.

Haloperidol (Haldol) is a high-potency butyrophenone neuroleptic that has been shown to be more efficacious than lorazepam in controlling violent behavior in adult psychiatric patients ( [Citrome and Volavka, 1999](#)). Haloperidol may be given in doses of 2 to 5 mg intramuscularly or orally. The dose may be repeated in 1

hour if necessary to achieve sedation. When compared with the lower-potency neuroleptics (such as chlorpromazine), haloperidol causes less hypotension and less decrease of the seizure threshold, and it has fewer anticholinergic side effects. Despite these advantages, many clinicians prefer the more sedating low-potency agents, such as chlorpromazine (Thorazine), which is given at 0.25 to 0.50 mg/kg per dose orally or intramuscularly. This dose may be repeated in 1 hour if necessary to achieve sedation.

Droperidol, another neuroleptic in the butyrophenone class, is often used in ED (especially adult) settings to sedate acutely agitated patients. It has an antidopaminergic potency and side effect profile similar to haloperidol, but its sedative properties are similar to those of chlorpromazine. It is usually given in doses of 1 to 2 mg intravenously or intramuscularly and may be repeated in 1 hour if necessary to achieve sedation.

Some pediatric settings use the soporific antihistamine diphenhydramine (Benadryl) for sedating agitated, nonpsychotic pediatric psychiatric patients. It is readily available and is safely administered orally as well as intramuscularly. Caution is required, however, because both diphenhydramine and the benzodiazepines can idiosyncratically cause behavioral disinhibition or agitation in some children (particularly those with brain injury or mental retardation), thereby increasing behavioral dyscontrol.

When choosing a pharmacologic approach to rapid tranquilization of children and adolescents, any concurrent medications and medical conditions must be taken into consideration to identify possible adverse drug interactions or contraindications. If there appears to be an underlying psychotic process, delirium, or agitation caused by substance abuse, a neuroleptic may be indicated. If a neuroleptic is used, the physician should also consider the prophylactic administration of diphenhydramine (Benadryl), 25 to 50 mg per dose, or benztropine (Cogentin) 1 to 2 mg, to prevent dystonia, while taking into account any other anticholinergic drugs the patient is receiving. As with any other medication, ED staff should monitor the patient's vital signs and level of consciousness and should be alert to the possibility of side effects such as acute dystonic reactions (e.g., oculogyric crisis, torticollis).

If the indication for a neuroleptic is less clear (as is often the case in pediatric populations), the choice of sedating agent will depend more on the balance of the risk-to-benefit profiles of neuroleptic versus benzodiazepines, on the patient's history of previous treatment with similar agent, and on physician and institutional experience, preferences, and practices.

The choice of route of administration also depends on available formulations of sedative agents. In general, intramuscular injection of a sedative has a faster onset of action than oral medication. Liquid oral preparations are preferable to tablets, because they cannot be as easily "cheeked" or sequestered. Some children and adolescents may calm down readily after accepting an oral medication. Perhaps this is the result of some anticipatory sedation and the sense of relief that the adults have taken action to control the situation. Offering the child the option of taking the medication orally, rather than by injection, is preferable if the situation permits. Not only is it less invasive, but also it enlists the child in the task of exerting control over his upset. Often, however, the threat of violence quickly escalates beyond the point at which a child or adolescent can ally and cooperate with these less restrictive and invasive measures. Such patients must often be physically restrained before they can be approached to offer or give sedative medication.

### *PHYSICAL RESTRAINT*

The Health Care Financing Administration and the Joint Commission on Accreditation of Healthcare Organizations mandate the standards and guidelines for hospital-based use of behavioral restraint and seclusion and subsequent monitoring. Health Care Financing Administration standards specify that "restraint or seclusion should only be reserved for those situations when a patient's behavior becomes aggressive or violent, presenting an immediate danger to his/her safety or that of others" ([Health Care Financing Administration, 2000](#)). Patients must be released from restraints when the goals of the treatment have been achieved, that is, when the patient, and the patient's behavior are under control and no longer pose a threat to self or others or a further disruption to the therapeutic milieu.

Every accredited facility must have explicitly formulated restraint and seclusion policies and procedures particular to its institution. Staff members of all disciplines should be knowledgeable and trained in protocols for managing dangerous behavior. Elements of such a protocol should include the following:

1. The indications for ordering and application of restraint. Example: Restraints must be used only when less restrictive measure have been found to be ineffective in preventing risk of harm to self or others.
2. Clarification of personnel and roles. Example: The selected leader of the restraint determines the number of staff members required for a restraint and the need for hospital security staff and assigns each staff member to a specific task (e.g., restraining a particular limb, clearing a room).
3. Guidelines for monitoring the restrained child. Example: A staff person must be assigned to monitor the child continuously and to assess for restriction of airway, change in breathing pattern, decreased circulation, or increased body temperature.
4. Guidelines for evaluation, reassessment, and removal of restraints. Example: A physician or other licensed, independent practitioner must see the patient and evaluate the need for restraint within 1 hour after the initiation of this intervention. Reassessment and contemporaneous documentation of the need for continuation of restraint must be done by a designated staff person every 2 hours for a child aged 9 to 17 years and every hour for a child less than 9 years old. Orders for continuation of restraint must be written by a physician or other licensed, independent practitioner. When a child shows behavioral control and can verbally contract for safety, he or she must be removed from restraints.

### **Risk Assessment and Disposition Planning**

The assessment of risk is a difficult and often anxiety-provoking task for the clinician. The history, interview of child and adult informants, and the mental status examination usually provide the emergency clinician with the data for a tentative formulation of diagnostic possibilities and the factors that have led to the current crisis and emergency referral. The crucial dispositive question facing the emergency clinician, however, is whether the child can now return to the current living situation with additional interventions and supports or whether some other more intensive, secure, or restrictive disposition must be found, such as admission to an inpatient psychiatric or pediatric ward or other therapeutic residential setting. This crucial decision hinges, in turn, largely on an estimation of the child's probable risk to self or others.

Some cases are clear-cut. The child with a medically serious ingestion or other suicide attempt, active delirium or acute intoxication, or florid psychosis requires medical or psychiatric hospitalization. Similarly, a child who remains acutely aggressive or agitated despite crisis assessment and interventions in the ED also requires a secure placement.

In other cases, however, the assessment of risk and its implications are more complex. For example, a child's immediate suicidality or assaultiveness may subside during the course of the ED evaluation, but some youngsters remain prone to precipitous decompensations because of psychotic regression, ongoing substance abuse, or extreme reactivity to a chaotic or hostile living situation. When, as is often the case, it appears that these conditions are very likely to recur, hospitalization may be desirable, especially if outpatient treatment or intensive in-home services have already proved unfeasible or insufficient. Among the relative factors suggesting the need for more intensive intervention are a deteriorating course with recurrent crises, poor impulse control, judgment, and insight, escalating risky behaviors (dangerous driving, promiscuous or unsafe sexual activity, physical fights, substance use), or increasing self-mutilative behavior, such as self-cutting, even if not in suicidal intent.

### **Assessing the Context-Specific Aspects of the Crisis**

Assessing the context-specific aspects of the crisis is particularly important in weighing risk in these more relative cases. For example, many children and adolescents can become violent or destructive in one setting, such as home or school, and yet show little or no dangerous propensities in other settings, such as in the hospital or outpatient clinic. Making a prediction based solely on the child's behavior in the ED, then, may have little predictive value as to subsequent behavior after discharge. By the same token, a child who initially presents as aggressive or threatening may not always require inpatient hospitalization if appropriate interventions can be made in the setting in which the crisis occurred (Reid, 1988). For example, most child and adolescent emergency visits for aggressive or suicidal threats or behaviors occur in the context of conflict with immediate caretakers. Effective crisis family intervention, psychoeducation, and short-term problem solving in the ED may result in temporary amelioration and resolution of the family crisis.

Although managed care has disingenuously perpetrated the myth that "medical necessity" is an unambiguously determined criteria and synonymous with imminent risk to self or others, the judgment of risk and clinical indications for hospitalization are not easily decided in many cases. Disposition is often "the art of the possible." Locating an inpatient bed and obtaining insurance authorization when needed are often difficult and time consuming. It is important, however, for the clinician not to



confuse these pragmatic considerations with his or her own clinical judgment of what is optimal for a given child.

## LEGAL CONSIDERATIONS

Mental health clinicians should be familiar with the state laws and institutional regulations that apply to the emergency psychiatric evaluation and treatment of minors, as well as those mandating reporting of physical or sexual abuse. The clinician should also know whom to contact in the hospital administration to obtain legal guidance when necessary.

In terms of the clinician's own legal vulnerability, one of the most important elements in decreasing legal exposure is scrupulous documentation of a thorough clinical assessment and good faith judgment in weighing the risks and benefits of one's actions for the patient. The duty to warn a clearly identified potential victim of a serious threat of imminent harm by a patient (e.g., divulging relevant findings about an adolescent's expressed intent to harm another person) takes precedent over confidentiality ([Simon and Goetz, 1999](#)). Needs for communication should be documented and handled openly, in adherence to state laws and institutional rules.

State statutes vary in their provisions and due process rights governing the certification, psychiatric hospital admission, and discharge of mentally disabled minors. In the state of Connecticut, for example, the statute regarding involuntary psychiatric hospitalization states that "if a physician determines that a child is in need of immediate hospitalization for evaluation or treatment of a mental disorder, the child may be hospitalized under an emergency or diagnostic certificate" ([State of Connecticut Social and Human Services, Department of Children and Families, 1991](#)). In many states, these statutes provide greater latitude in compelling the involuntary psychiatric inpatient treatment of minors than of adults. States vary as to the age under which parents may psychiatrically hospitalize a minor under a "voluntary" admission status, even without the minor's assent, as well as the age at which a minor may contest such a hospitalization or may be entitled to a court hearing.

## SYSTEMS ISSUES

### Optimizing the Emergency Department Setting for Emergency Child Psychiatric Services

Even in specialized pediatric hospitals, ED personnel often have little training or comfort in handling child behavioral health emergencies. Preoccupied with large numbers of seriously ill children, ED staff members may look on psychiatric patients as a nuisance, tangential to the ED's perceived real mission and diverting vital space, time, and personnel resources away from the "truly medically patients." This skeptical atmosphere often pervades the setting and results in implicit and explicit pressures for the mental health clinicians to be quick in their assessment and to discharge psychiatric emergency patients who may appear to the medical and nursing staff as disruptive, uncooperative, or unpleasant.

To counteract these potentially divisive staff tensions and resolve obstacles, ongoing collaborative efforts are needed at both the individual and system levels to communicate with ED staff and for the different services involved to communicate effectively regarding their goals and activities. Periodically scheduled meetings that bring together ED psychiatric and pediatric physicians, nursing, and social work leadership are important, because the large numbers of rotating staff involved (reflecting the nature of emergency work as occurring 24 hours a day, 7 days a week) make collaboration difficult.

The use of child psychiatric nurses to provide in-service training and consultation to ED nursing staff helps to bridge some of the interdisciplinary issues that arise in the ED. In addition, the availability of trained child care workers with inpatient psychiatric experience who can assist with the management of child psychiatric patients in the ED decreases some of the demands on busy ED nursing staff.

Unless designed with mental health service needs in mind, the physical setting of many EDs is also suboptimal for conducting child psychiatric evaluations. One important focus of collaborative planning with ED staff is the identification or development of designated areas suitable for these purposes.

### Coordination of Emergency Department Services with Outside Systems of Care

All too often, child psychiatric emergency visits contribute to a pattern of fragmented care by taking place in isolation from the child's ongoing treatment (if any) and with little or no assurance of adequate follow-up. To some extent, the lack of adequate communication is often the result of the late hour and the unavailability (even by telephone) of key informants and care providers (outpatient therapists, school personnel), as well as the constraints of busy ED clinicians who may have to deal with large numbers of patients with little time, sleep, or support staff.

Both in individual cases and on an institutional basis, however, it is essential to foster better communication. An attempt to contact referring or treating outpatient clinicians should always be made, and, even if not available, they should be notified (with the requisite consents) that their patient has been seen in the ED. Community clinicians and institutions (e.g., residential treatment centers, detention centers, shelters) should be encouraged to call before or at the time of sending a child to the ED, to provide necessary history, information, and disposition collaboration. To as great an extent as possible, it is also important to develop good institutional relationships with referring institutions and receiving acute psychiatric hospitals, including regular channels for communication about children sent from the ED for hospitalization.

When children are discharged home from the ED with recommendations for outpatient follow-up, definite arrangements (specifying time and therapist) should be made if possible before the child leaves the ED. Obviously, this is easiest if the child and family will be returning to the ongoing care of a familiar clinician. Some child outpatient clinics reserve one or two appointment slots each day for urgent visits to which patients seen in the ED the previous evening can be referred. If the child is not already in treatment, it is important for the ED clinician to follow-up regarding whether the family has succeeded in making and keeping the initial outpatient appointment. Empirical studies of young suicide attempters seen in the ED and referred for outpatient therapy find that only about one-third ever keep even a single outpatient follow-up appointment ([Piacentini et al., 1995](#); [Trautman et al., 1993](#)). Although ED staffing patterns conspire against adequate follow-up provisions, with tired on-call mental health clinicians and social workers rotating off service the next morning, explicit institutional policies and staff support to optimize outpatient compliance are desirable.

## FUTURE DIRECTIONS

Given the growing demand for emergency child and adolescent psychiatric services described earlier and the burden of providing around-the-clock staffing, many services find it hard to step back and consider the broader issues and needs these challenges pose. Developing a data base for tracking ED psychiatric services, including chief complaints, discharge diagnoses, length of ED stay, sources of referrals, dispositions, and repeat users, permits identifying changing patterns of use and obstacles to efficient service ([Peterson et al., 1996](#); [Santucci et al., 2000](#)).

In many cases, emergency clinicians struggle with the quandary that it appears unsafe to permit a given child to return home with only traditional clinic-based outpatient services in place. Yet hospitalization, even if available, seems unlikely to provide more than a brief respite, especially if only very short-term and at a great geographic remove from home (hence precluding effective family work). Many cities and states are moving toward the development of regionally consolidated systems of care. These new structures will, we hope, allow for the integration and alliance of existing service providers, as well as facilitate the development of a fuller spectrum of outpatient services. These integrated care models would include intensive in-home, child and family psychiatric services, respite care services, partial hospitalization and day treatment programs, and mobile crisis teams. Moreover, they would ultimately contain behavioral health care costs and provide ED clinicians (and inpatient units) with a greater and much needed choice of viable dispositions. It also seems likely that the wider availability of such services would obviate the need for many ED child psychiatry visits.

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## 92 CONSULTATION PROCESS IN CHILD AND ADOLESCENT PSYCHIATRIC CONSULTATION–LIAISON IN PEDIATRICS<sup>1</sup>

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Studies have demonstrated the clear association between physical disorders and psychiatric disturbances in children and adolescents ( [Shugart, 1991](#); [Steiner et al., 1993](#)). A higher rate of behavioral and emotional disorders occurs in children with chronic disorders (Faird–Rontman, 1992), especially if the child has physical disabilities (see [Chapter 116](#)). Other factors that increase the chances of psychological problems include age (younger children are more vulnerable), male sex, family stresses, single parenthood, and low family income. Psychiatric assessments of randomly selected hospitalized pediatric patients suggest that as many as two-thirds of children admitted to pediatric wards would benefit from a child or adolescent psychiatry consultation ( [Shugart, 1991](#)). Coexistent medical and psychiatric problems in pediatric patients appear to contribute to more complex diagnostic assessments, increased health care costs, and less satisfactory outcomes compared with those in patients without comorbidity ( [Steiner et al., 1993](#)). The child psychiatric consultation, then, is an important intervention to help improve the care of hospitalized pediatric patients and their families. This chapter summarizes common psychological responses in hospitalized children and adolescents and outlines the child and adolescent psychiatric consultation process.

### PSYCHIATRIC CONSULTATION IN PEDIATRICS

The process of child and adolescent psychiatry consultation–liaison in hospital pediatrics rests on a broad foundation that includes a knowledge of normal development ( [Knobloch et al., 1980](#); [Lewis and Volkmar, 1990](#)), psychopathology, diagnosis, and treatment ( [Lewis, 1996](#)), as well as a familiarity with pediatrics and pediatric hospital practices. The purpose of the consultation, the characteristics of the patient and the patient's family and their reactions to the hospitalization, the nature of the patient's illness or injury, and the nature of the relationship between pediatrics and child and adolescent psychiatry all influence the consultation–liaison process. An understanding of the child's concept of his or her body, of how these concepts change with development, and of children's (and family) reactions to illness is particularly useful ( [Lewis and Schonfeld, 1994](#)) as well.

### CHILDREN'S CONCEPT OF THE BODY AND ILLNESS

The child's developing concept of the body is reflected in the child's behavior, verbalizations, and drawings of the body ( [DeLeo, 1977](#)). Initially, the infant seems to experience the body as an external object that either gratifies or causes discomfort. Thus, the infant appears to be either content and oblivious of the body or appears enraged and cries vigorously when the threshold for bodily discomfort is exceeded. There appears to be very little ability in the infant to comprehend or tolerate discomfort or to delay gratification.

By the time children enter preschool, there is clear evidence they possess some knowledge about the body, as well as an increasing capacity to take care of the body. For example, 3-year-old children are curious about the body, can name body parts, including eyes, nose, and mouth, and are concerned about cuts and “booboos.” Very young children seem to view the body as a kind of fluid-filled sac that, if punctured, will ooze precious fluids. Maintaining body integrity is paramount at this developmental stage, and even minor injuries may seem catastrophic to the child. Adhesive bandages (Band-Aids) are very reassuring at this time. At this age also, a simple knowledge of internal organs is beginning, usually with those organs related to direct experience, such as stomach and stomachache.

In kindergarten, children 4 or 5 years of age can draw a simple representation of the body. Frequently, they draw a single circle within which simple features are drawn, such as dots for eyes and nose, a single line for the mouth, and a scribble for hair (the child frequently identifies the sex of the person “by the hair”). Limbs are represented by simple “stick” lines with an indeterminate number of digits emanating from the end of the stick limbs. Simple knowledge of internal organs (e.g., a heart in the shape of a valentine) begins to develop.

As children develop, more details appear in their pictures of the body. By 5 or 6 years, children can identify the jaw, temples, forearms, and shins. From ages 5 or 6, children draw a circle for the head and an ellipse for the torso and add more detail, such as eyebrows and ears. Articles of clothing also become more detailed between 7 and 10 years.

When the child approaches the cognitive stage of formal operations (usually age 12 years and older), the proportions of the body are more realistically represented, and such advances as profile views and representation of movement appear. Knowledge of physiologic functions of certain bodily organs begins to develop; however, adolescents, who may be expected to have a good working knowledge of major anatomic organs and functions, may still be surprisingly ignorant of certain body parts and functions.

Children's concepts of illness follow a similar developmental sequence ( [Lewis, 1994a](#)). In infants, there appears to be no concept of illness other than an external agent (the body) that is attacking the infant and causing discomfort. Very young children who are in the early stages of concrete operations seem to conceive of illness as a result of “immanent justice,” that is, the illness is a punishment for misdeeds committed or imagined and for which the child feels guilty.

The preschool child develops a “contagion” theory of illness, that is, illness is caused by a germ, or “bug.” Usually, the germ or bug is large and imaginary and “infects” in ways imagined by the child. By school age, the child develops a more sophisticated knowledge of the causes of illnesses and the relationship between

symptoms and medical treatments. At this developmental age, children may ask many more questions as they attempt to sort out their understanding of the symptoms, illnesses, and treatments that affect their bodies.

Again, although adolescents are cognitively more capable of understanding illness, they are often psychologically immature and sometimes behave as though they believe they are invincible and immortal. In any event, earlier concepts may persist or may reappear as part of the regression that often occurs with illness. Thus, feelings of guilt may accompany the onset of certain illness, and children and adolescents may have an inaccurate or incomplete understanding of how they became ill.

## TYPES OF REQUESTS FOR CONSULTATION

Requests from pediatricians for child psychiatry consultation in the hospital may fall into the categories described in the following subsections.

### Emergencies

The most common emergency that stimulates a psychiatric consultation is attempted suicide, usually by overdose or physical self-injury, in an adolescent ( [Chapter 91](#)). Other emergencies, also usually seen in the emergency room, include physical abuse (sometimes presenting as Munchausen syndrome by proxy), sexual abuse, drug abuse, acute agitation, acute psychotic reactions, and family crises. In addition, such conditions as anorexia nervosa with critical weight loss may require urgent hospital care.

Emergency consultation in the pediatric ward may be requested for the diagnosis and management of major disruptive behaviors in a child or parent. The consultation may merge into concomitant psychiatric treatment during the hospitalization or may entail making arrangements for psychiatric care after the pediatric hospitalization. Other emergency consultations in pediatric wards may be requested for the diagnosis and management of delirium caused by a wide range of medical conditions (e.g., brain infections and trauma, drug intoxications or interactions, adrenal, hepatic, pancreatic, or renal failure, burns and electrolyte imbalance, and space-occupying lesions) that severely affect the metabolism and function of the brain.

### Differential Diagnosis of Somatoform Symptoms

Anxiety and depression may be the underlying cause of such pediatric symptoms as recurrent abdominal pain, headache, and failure to thrive. Somatoform disorders, including somatization disorder, body dysmorphic disorder, conversion disorder, hypochondriasis, and pain disorder, as well as other related conditions, such as complex regional pain syndrome (formerly reflex sympathetic dystrophy), vocal cord dysfunction, and functional recurrent abdominal pain, characteristically present with symptoms that at first glance suggest a physical disorder but for which psychological factors are of major etiologic importance. The request for consultation and diagnosis may come late in the hospitalization, often after an exhaustive, and often expensive, somatic workup has taken place.

### Collaborative Care of Children with Stress-Sensitive Illnesses

Acute episodes of certain illnesses, including asthma, diabetic acidosis, and ulcerative colitis, are often precipitated by psychological stress in children who are particularly vulnerable and in whom the psychological component of the illness is especially prominent. Psychological assessment and care may be essential for the comprehensive treatment of such a child.

### Diagnosis and Care of Children with Psychiatric Symptoms after a Somatic Illness

Some illnesses seem to linger long after the acute phase in the form of a prolonged depression that may last for a long time. For example, acute infectious mononucleosis and other viral infections are often followed by symptoms of depression that may last for weeks or months.

### Chronic Illness

Chronic pediatric illness of almost any kind, with recurrent hospitalization, is a psychological risk factor for children ( [Lewis, 1994a](#)). The rate of psychiatric illness in children with both chronic medical conditions and disability is three times greater than in noncompromised children for conditions such as attention deficit hyperactivity disorder, overanxious disorder, depression, and conduct disorder ( [Cadman et al., 1987](#)). Many illnesses previously regarded as lethal are now categorized as chronic or curable. For example, children with acute lymphocytic leukemia now have a 90% cure rate.

### Reactions to Major Pediatric Treatment Techniques

Certain kinds of pediatric treatment evoke serious psychological symptoms. Bone marrow transplantation, in particular, gives rise to considerable anxiety and depression ( [Chapter 97](#)). Extensive surgical repair for injury and burns ( [Chapter 99](#)), especially in young children, may give rise to acute behavioral problems. Psychiatric assessment and care may be essential to the child's successful participation in the medical treatment. Some treatments may also give rise to psychological difficulties; for example, cranial irradiation treatment may give rise to cognitive deficits ( [Chapter 96](#)).

### Reactions to Pediatric Illness or Trauma

Finally, every child with a pediatric illness or trauma requiring hospitalization experiences a psychological reaction ( [Lewandowski and Baranoski, 1994](#)). The degree of the reaction varies with the developmental level and premorbid state of the child, the state and reaction of the family, and the seriousness of the illness. The more serious the illness (e.g., leukemia) or injury (especially head injury), the more likely it is that behavioral reactions will arise.

## PSYCHOLOGICAL RISK FACTORS

In general, psychological disturbances are more likely to occur when any of the specific risk factors listed in [Table 92.1](#) are present. Other psychological risk factors include impaired function, immobilization, disfigurement, and loss of autonomy in the child. Additional parental reactions that may increase the risk of psychological disturbance in the child include parental feelings of loss and grief, guilt, depression and anxiety, exhaustion, and isolation, as well as the effects of marital strain, financial drain, and disruption of routine family functioning.

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1. Premorbid psychopathology in the patient
2. Poor parent-child relationships
3. Psychiatric disturbance in either parent
4. Infancy age group
5. More severe or ambiguous medical diagnoses
6. Chronic illness and multiple hospitalizations
7. Inadequate psychological preparation for hospital and invasive procedures
8. Parents' inadequate understanding of the illness, including unrealistic expectations and debilitating parental reactions, including feelings of extreme helplessness and pessimism
9. Involvement of other nonmedical agencies (department of child services, police, law) interested in the patient's welfare

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**Table 92.1. Risk Factors for Psychological Disturbances in Hospitalized Pediatric Patients**

Some characteristics of the treatment team or the treatment process may also increase the risk of psychological disturbances in pediatric inpatients. In general,



psychological distress is more likely to occur in the presence of any of the following risk factors:

1. Use of multiple medical consultants (sometimes with conflicting opinions) without adequate, available, or clearly designated leadership
2. The hospital staff's inadequate response to or understanding of the psychological meaning of the illness, injury, or hospitalization to the child and other family members
3. The hospital staff's inadequate awareness of their transference and countertransference feelings about the child, the family, and the illness or injury

## SIGNS OF PSYCHOLOGICAL DISTRESS

Some of the common signs of psychological distress associated with pediatric illness are shown in [Table 92.2](#).

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1. Biopsychological symptoms: malaise, pain, irritability, disturbances in appetite and sleep
2. Increased attachment behavior: clinging, demanding, heightened separation anxiety
3. Regression: thumbsucking, regression in developmental milestones (i.e., speech, bladder and bowel control, self-care)
4. Passivity and withdrawal: feelings of helplessness and powerlessness
5. Aggressiveness and acting out: tantrums, combativeness, oppositional behavior
6. Frightening fantasies about illness and procedures: ideas of punishment, fear of mutilation and bodily harm
7. Anxiety and mobilization of defenses: denial, projection, phobic symptoms, conversion symptoms
8. Precipitation or aggravation of premonitory psychiatric symptoms

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**Table 92.2. Signs of Psychological Distress in Pediatric Patients**

## MODELS OF CONSULTATION

Several models of consultation–liaison have been developed in response to the kinds of consultations requested by pediatricians and to the kinds of psychological reactions prominent in hospitalized pediatric patients and their families ( [Lewis, 1994b](#)).

### Anticipatory Model

This model derives in part from an earlier concept of *anticipatory pediatrics* ([Senn, 1947](#)), and it is especially useful when serious psychological reactions to an anticipated procedure are to be expected. For example, children undergoing bone marrow transplantation, and their families, can benefit from a pretreatment psychiatric consultation to assess the strengths and vulnerabilities of the family and to prepare the child and family accordingly ( [Atkins and Patenaude, 1987](#)). When the expected psychological reaction does occur, the psychiatric consultation team is then already in place and informed and is in a much better position to offer further help. Sometimes the prior assessment process may be used to help avert serious psychological reactions.

### Case Finding Model

A modified form of the anticipatory model derives from the liaison work that can be offered to the pediatric staff in the ward. Thus, through the use of regular weekly ward meetings with pediatric and nursing staff members ([Lewis, 1962](#)), early detection or anticipation of psychological problems can help to alleviate stress and may possibly prevent severe psychological reactions. Unfortunately, the preventive and therapeutic uses of such liaison meetings are not directly reimbursable by most third-party payers, and the cost has to be absorbed into the general cost of the hospital care ( [Fritz, 1990](#); [Lewis, 1994b](#)). This liaison function is invaluable, but it rarely receives adequate financial support. In some instances, the liaison work may lead to a request for a formal consultation, which is usually then reimbursable.

### Education and Training Model

The consultant can make a useful contribution to the pediatric hospital care of infants, children, and adolescents through regular case conferences and discussions. Often this is most appreciated in the context of acute care units, such as a pediatric intensive care unit. Training for pediatricians can also be enhanced by the use of a study group ([Lewis and Colletti, 1973](#)). Training of child psychiatrists and child psychologists in consultation and liaison in pediatrics can be facilitated by direct, on-the-case supervision as well as by indirect supervision, case conferences, and regular seminars.

### Emergency Response Model

This model, as noted earlier, uses a 24-hour emergency on-call roster and is mostly used for emergency room calls, but it may also apply in the case of urgent consultation requests in the wards. Attempted suicide in an adolescent is a frequent reason for an emergency child psychiatry consultation in an emergency room, whereas the need for containment, restraint, or structure for a behaviorally out-of-control child in the wards ( [King and Lewis, 1994](#)) is a common reason for an urgent response by the consultant ([Chapter 91](#)).

### Continuing and Collaborative Care Model

In many pediatric illnesses, there is a need for consistent, collaborative, and concurrent pediatric and psychiatric treatment. Typical illnesses that require such care include anorexia nervosa, bulimia, and obesity. Children with recurring or chronic pain may also benefit from ongoing collaborative approaches for pain management, including behavioral approaches ([Cardona, 1994](#)) and psychopharmacologic approaches ([Chapter 76](#) and [Chapter 77](#)).

None of these models is mutually exclusive. Indeed, a good child and adolescent psychiatric consultation–liaison service in pediatrics should incorporate all the foregoing models for comprehensive consultative care.

## BASIC CONSULTATION PROCESS

The psychiatric consultation is generally facilitated when the consultant is part of a team consisting of a child psychiatrist, child psychologist, social worker, developmental pediatrician, and child psychiatry clinical nurse specialist. The team approach allows for comprehensive understanding of the medical, psychiatric, and social issues that may be involved in a referral, as well as for greater coordination of services within the hospital and at the time of outpatient referral. Attention to the factors described in the following subsections enhances every form or model of consultation.

### Availability

Prompt, practical, and understandable recommendations ([Leslie, 1992](#)) from an easily available psychiatric consultant are the foundation of effective consultation and liaison work. The presence in the ward of the child psychiatrist at ward rounds, conferences, or other meetings is useful. Liaison work in particular facilitates accessibility, provides opportunities for informal suggestions, and may prepare the way for more formal consultation requests.

### Relationships

Collaboration is much more likely to occur when there are good relationships based on mutual respect and friendship between pediatrician and child psychiatrist. Because pediatricians and child psychiatrists work in different ways ( [Burket and Hodgkin, 1993](#); [Fritz, 1990](#)), misunderstandings may occur regarding the urgency of a referral or the speed of response needed ( [Black et al., 1990](#)). Understanding how the other functions in practice can encourage smoother collaboration between

pediatricians and child psychiatrists ([Lewis, 1994b](#)). Good relationships between pediatric and psychiatric colleagues make communication easier and more straightforward, reduce the tendency for acting out of ambivalent feelings, and ultimately improve the care of patients and families.

### Levels of Consultation

Five levels of consultation should always be kept in mind:

1. The inner life of the child
2. The dynamics of the relationship between the child and his or her family
3. The relationship between the child and family and the various ward staff
4. Interdisciplinary dynamics operating among attending pediatricians, housestaff, nurses, social worker, child psychiatrist, child psychologist, and other consulting specialists
5. In some instances, the relationship of the hospital staff to an outside agency, including state departments of children's services, police, or law

### Preparation for the Consultation

In preparing to respond to a request for psychiatric consultation, the following questions provide a useful checklist to review before going ahead with the clinical interviews:

1. *Who* is requesting the consultation? A consultation request must come ultimately from the physician responsible for the care of the hospitalized child and should include a brief written request by the responsible physician.
2. *What* is (are) the consultation question(s)? The question or questions must be clear and should be of a kind that can reasonably be answered in the often brief period that the child is in the ward and in the context of the particular ward environment.
3. *When* was the request made, and what is the time frame? In some instances, there simply may not be sufficient time before discharge in which to perform a reasonable consultation.
4. *Why* is the request being made at this time? Sometimes the ostensible reason for the request covers a hidden agenda, such as a pending court appearance, a possible custody conflict, or, in some instances, a conflict among hospital staff.
5. *How* much workup has been done or should be done (and by whom) before proceeding with the consultation? Because the available time is often brief, prior information about the child and family may enable the child psychiatrist to focus on specific areas.
6. Have the child and parents been informed and suitably prepared? Occasionally, parents may refuse to have a psychiatrist see their child. The cooperation of the parents is needed for a satisfactory consultation. When a child is a ward of the state, permission for child psychiatry consultation must be obtained in writing from the state agency.
7. Has the child's hospital record been read before one proceeds with the interview?

### Procedure

A flexible interview approach is often required for completing consultations in a pediatric ward. One may have to improvise through the use of a few portable materials (e.g., paper, pencil, crayons, deck of cards, and small rubber doll figures) and by adapting to frequent interruptions and lack of privacy in a pediatric ward.

The psychiatric evaluation is of necessity detailed and complex and should not be rushed, if possible ([Leslie, 1992](#)). If the time available for the consultation is not adequate to complete the necessary evaluation, the focus of the consultation should be adjusted, or the evaluation should be deferred to a time or setting (i.e., the inpatient psychiatric unit or the outpatient psychiatric clinic) when a more appropriate assessment can be made.

Ideally, one would first interview the patient's parents, but this may not be feasible. Suitable preparation of the child and parents by the requesting pediatrician usually reduces the child's and parents' reluctance, if any, to speak with a psychiatric consultant; however, the complete assessment of the child may still require multiple interviews with the child and parents. The basic items of a mental status examination ([Lewis, 1991](#)) ([Chapter 41](#)) need to be performed and additional inquiries or tests ordered as needed. A thorough knowledge of the patient's treatment and medication regimen is particularly necessary when one evaluates a patient for delirium or other possible medication-induced conditions (e.g., depression, anxiety, psychosis, and agitation).

Very useful information about the child and family can often be gathered from the observations of such ward staff as nurses, child life specialists, and social workers. Moreover, conversations with some of the patient's health care providers (inpatient and outpatient) may be necessary to sort out treatment issues that may be in conflict and contributing to the psychological stress of the patient or the patient's family. Clarification of any pending legal or social service issues may also be necessary to comprehend the patient's current stressors and may also determine in part the nature of the consultation and the child psychiatrist's report.

### Report

The findings of the consultation should first be reported to and discussed with the requesting pediatrician. Subsequently, and with the agreement of the pediatrician, one communicates as necessary and as appropriate with the parents (or, in some cases, a state agency), the child, and key ward staff members. The written consultation note should be relatively brief and should be organized in such a manner as to be easily read and understood. Useful headings may include the following: (a) a brief statement of the question and the purpose of the consultation; (b) a concise summary of pertinent historical data; (c) the important findings, both positive and negative, from the mental status examination and comprehensive psychiatric evaluation; (d) diagnosis, using a standard multiaxial classification (e.g., the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* or the 10th edition of the *International Classification of Diseases*); (e) a brief diagnostic formulation and summary; and (f) a recommended treatment or management plan or suggestions for further workup.

### Confidentiality

A hospital chart is not a particularly private or confidential record; it may be seen by the parents or child as well as other people. For this reason, the words used must be carefully chosen to avoid offense, slander, or potential dissemination of possibly damaging information. The written record is at the same time a legal document carrying the consultant's signature and should be written with that in mind.

### Follow-Up

Follow-up after a consultation by inquiring of the requesting pediatrician about the subsequent course of the child is helpful. The consultant obtains general information about the accuracy of the psychiatric assessment and the effectiveness of the intervention, and the pediatrician often appreciates the psychiatrist's continuing interest. Follow-up studies of matched pediatric patients who received psychiatric consultations and those who did not can help assess the efficacy of psychiatric interventions in hospitalized pediatric patients ([Shugart, 1991](#)).

## IMPEDIMENTS TO CONSULTATION—LIAISON IN PEDIATRICS

Difficulties that may interfere with a well-functioning consultation—liaison service ([Anders, 1977](#); [Anders, 1982](#); [Burket and Hodgkin, 1993](#); [Eisenberg, 1967](#); [Fritz, 1985, 1990](#); [Jellinek, 1982](#); [Kanner, 1937](#); [Lawrence and Adler, 1992](#); [Leslie, 1992](#); [Lewis, 1973](#); [Lewis, 1994a,b](#); [Lewis and Vitulano, 1988](#); [Lourie, 1962](#); [Oke and Mayer, 1991](#); [Rothenberg, 1979](#); [Senn, 1946](#); [Shugart, 1991](#); [Wright et al., 1987](#)) include the following:

1. The failure of some child psychiatrists to understand how pediatricians function in practice, and vice versa
2. A perceived, or real, lack of availability of child psychiatrists
3. Professional identity problems in both disciplines
4. Different perceptions of patients (health versus disorder)
5. Different interviewing techniques ("anamnesis" versus "listening")
6. Anxiety among pediatricians in dealing with the emotional problems of children and their families
7. Transference and countertransference issues



8. Time constraints in both pediatric rotation training schedules and inpatient load
9. Financial considerations, including inadequate funding for child psychiatry consultation–liaison services in pediatrics (When payment is determined by the type of insurance plan held by the parents, special rules may apply.)
10. Ambivalent support for the concept of coordinated multidisciplinary care for the whole child and his or her family
11. Limited opportunities for continuity of care in pediatric training
12. Compartmentalized, disease-oriented research, rather than collaborative biopsychosocial research
13. Inadequate outcome studies

None of these impediments is irremediable. With positive motivation and cooperation among all the disciplines involved, one should expect to have a thriving child and adolescent psychiatric consultation–liaison service in pediatrics ( [Lewis and Vitulano, 1988](#)).

## RESEARCH DIRECTIONS

Current national health policies and health plans have affected the role for child psychiatry consultation in pediatrics. At the same time, advances in pediatric medical knowledge and technology sometimes give rise to an increase in requests for child psychiatric consultation and liaison. For example, advances in bone marrow and solid organ transplantation, neonatal and pediatric intensive care, and prolonged survival from cancer, cystic fibrosis, chronic renal disease, and other once fatal pediatric conditions not only illustrate the effects of technology on pediatrics but also identify new regions of pediatrics in which the expertise of the consultation–liaison child psychiatrist is relevant.

Increased awareness about the coexistence of psychiatric and medical problems in pediatric patients has contributed to the development of pediatric medical–psychiatric units ( [Fritz, 1990](#); [Sexson and Kahan, 1991](#); [Steiner et al., 1993](#)) and triple-board programs, which prepare physicians to be board eligible in pediatrics, general psychiatry, and child and adolescent psychiatry. The role of the consultation–liaison child psychiatrist in such settings, as well as in the general pediatric setting, continues to undergo changes ( [Fritz, 1990](#)).

Problems of training, funding, and organization continuously need to be addressed, particularly in the changing health care environment. New ethical and legal issues continue to arise and need to be considered as well. Perhaps most important, research projects designed with clear short-term and long-term outcome measures (e.g., hospital length of stay, improved compliance with medical care, decreased use of medical services, improved functioning, or improved quality of life) are needed to assess the efficacy and cost-effectiveness of particular psychiatric consultation–liaison interventions ( [Shugart, 1991](#)).

<sup>1</sup>An earlier version of this chapter by Melvin Lewis was published in Lewis M, King RA (eds.): Consultation–liaison in pediatrics. *Child Adolesc Psychiatr Clin North Am* 3:513–529, 1994.

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# 93 COLLABORATION BETWEEN CHILD PSYCHIATRISTS AND PEDIATRICIANS IN PRACTICE

Richard H. Granger, M.D., and Elsa L. Stone, M.D.

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Since the 1950s, the introduction of and increased use of preventive vaccines and more effective antibiotics (as well as improvements in nutrition and sanitation in general) have greatly decreased the number of children presenting with acute or chronic contagious and infectious diseases in pediatric practice. Similarly, new technology and better science have expanded the number of children with chronic diseases and infirmities of many kinds and have prolonged their survival enormously. At the same time, the swelling pressure on families and environments has helped to bring to attention a growing number of children with developmental and psychological problems. Whether this is real or seems so only because of more attention to these issues and the availability of better diagnostic procedures and criteria, it is now widely acknowledged that 12% to 15% of children in the general population are in need of direct, intensive mental health services, and 20% to 40% could benefit from less intensive mental health services ([Garralda and Bailey, 1989](#); [Starfield et al., 1980](#); [Starfield et al., 1984](#); [Sturner et al., 1980](#)). It is probable that in populations where poverty, violence, drug use, broken families, inadequate housing, and run-down schools are present, the morbidity is closer to 100%.

These facts present a clear case for the need for close collaboration between pediatricians and child psychiatrists in the health care of children. Children with serious psychopathology need to be referred to and managed by mental health professionals, preferably child psychiatrists. Failure to receive, or even delay in receiving, such services leads to greater psychological dysfunction, school failure, substance abuse, and other sociopathies. Children with more ambiguous mental health needs and those with chronic organic disease need to be managed in consultation between practitioners in the two fields. It has been well demonstrated that intervention informed by mental health principles can reduce pain, suffering, and time of recovery from injury and illness in patients with chronic organic problems ([Granger, 1990](#)).

Despite all this evidence, the relationship between the two fields has been less collegial than it needs to be to serve the best interests of children and their families. Both sides have much to answer for in this regard. Myriad reasons have been suggested for the mutual discomfort ranging from time constraints and financial considerations to differences in personality and temperament, in training, in theoretical framework, and in therapeutic approach between those who choose the two fields ([Greene, 1984](#); [Lewis and Vitulano, 1988](#); [Work, 1989](#)). As usual, these reasons hold up better when group averages are discussed than when individuals are the subject of comparison. Be that as it may, strong forces are now at work that make a rapprochement between the fields more imperative than ever.

## FACTORS IN COLLABORATION

The nature and needs of children have not changed, but four factors introduced into the ecology of medicine in general and into child psychiatry and pediatrics in particular are helping to blur the borders between them. These factors are (a) managed care, (b) the increasing use of psychotropic medications in treatment, (c) the creation of a subspecialty board in developmental/behavioral pediatrics, and (d) the decrease in number of child psychiatry training programs and applicants for them.

### Managed Care

The mushrooming growth of managed-care programs has had more effect on mental health care than on any other area of medicine. It has become the policy of almost all these programs to minimize both the availability and length of treatment—other than drug treatment—for even the most serious psychopathology. Psychiatrists are discouraged or prevented from participating with many managed-care organizations because of closed panels and inadequate reimbursement levels. Patients and referring physicians find it difficult to identify participating psychiatrists because many plans do not make these lists readily available. Instead, clerks in distant cities maintain lists of participating mental health professionals of all types in various regions and often provide only a few names from that list. These lists do not include information on the training of these professionals. The source seeking referral (often the family itself) has no way to distinguish child psychiatrists from general psychiatrists, and many of the latter have taken to listing themselves as capable of dealing with the mental health problems of children. This makes it more important than ever that the child psychiatrists in any community help the pediatricians in that community to know who they are and how they may be reached.

### Psychotropic Medications

In recent years, child psychiatry as a field has learned much about basic brain science—physiology, biochemistry, pharmacology—and this has led to the development of a rapidly increasing pharmacopeia aimed at the treatment of various psychopathologic conditions. At the same time, the use of drug treatment has been encouraged by managed care, which, in the interest of the bottom line, looks for the least time-consuming and labor-intensive treatment available. One of the byproducts of this trend has been that child psychiatrists have prescribed medications for patients and then left it to the pediatricians to write continuing prescriptions and do follow-up care. This can be a good source of collaborative effort when the psychiatrist communicates with the pediatrician at the time of prescription, informs him or her of the rationale for the treatment, and maintains periodic contact with the pediatrician to monitor the course. It is *not* a collaboration at all when the child psychiatrist merely tells the family to get in touch with the pediatrician and leaves the latter with an unsought and poorly understood responsibility. Pediatricians are under increasing pressure from families and nonphysician mental health clinicians to prescribe psychotropic medication without psychiatric evaluation. Most pediatricians are not as knowledgeable as psychiatrists about these medications and would welcome a child psychiatrist's collaboration in the care of their patients.

### Pediatric Subspecialty Board

For more than 50 years, some pediatric centers have provided some of their trainees with knowledge and skills derived from some of the mental health fields. The number of such trainees has slowly increased over the years aided by support, both financial and organizational, from the National Institute of Child Health and Development, and most centers currently have some representation in the field. After long discussions with other specialty groups, the American Board of Pediatrics established a subspecialty board in developmental/behavioral pediatrics that is currently generating a curriculum for a fellowship program in this area and will soon begin certifying graduates of such programs as well as some others who will be allowed under a "grandfather clause." The institutionalization of this program means that every pediatric training center in the country will now have to have such a program and faculty with expertise in the subject. The new subspecialists will not be trained to be junior child psychiatrists. Much of their training will be in early child development, in the development and management of community outreach programs, in the management of common psychosomatic problems such as enuresis and encopresis, in some family counseling and in the detection and appropriate referral of serious psychopathologic entities. They will have been trained, in many cases, in conjunction with child psychiatrists or psychologists and will be natural collaborators with mental health professionals in a plethora of activities.

### Decreasing Number of Child Psychiatrists

This has been an alarming trend in recent years. Driven by economics as well as other issues, there has been a considerable decline in the number of child psychiatry training programs in the United States and an overall lessening of the numbers of candidates applying for and completing such training. Many of those completing training are entering academic research programs rather than going on with clinical work. This trend will certainly help extend the breadth of the field, but it does indicate a continuing diminution of the number of clinical resources available to deal with the many children and their families who need psychiatric treatment. This will put more and more pressure on those child psychiatrists doing clinical work in the community, and most of them are already overloaded. It may be

that some of their time spent in consultation or collaboration with pediatricians, as well as others, may serve as an extender of services.

Nothing in this chapter is meant to suggest that collaboration is automatic or even easy—or even desired by either participant in a case. At the present time, many pediatricians in practice are still relatively uninformed about most aspects of mental health work and may not be particularly interested in ongoing contact with the psychiatrist. However, there are more pediatricians who are interested in such a collaboration today than there have ever been, and the increased emphasis this work will now assume in pediatric training programs will ensure more in the future. Those pediatricians who are interested can be a valuable source of information for the psychiatrist. Most often, they have known the family for a long time and may have watched the problems develop while working with the family to find and accept help.

## NATURE OF COLLABORATION

In community practice, collaboration will consist primarily of the interactions between a pediatrician and a child psychiatrist around the clinical care of a child and family. The source and the nature of this interaction may vary considerably. The child may be referred directly to the psychiatrist by the pediatrician for evaluation or treatment. The child may be referred to the psychiatrist by a school or other agency with or without the knowledge of the pediatrician. The pediatrician and the psychiatrist may have an ongoing relationship that allows them to consult with one another—in either direction—around a case they have already worked on together or on one unknown to one or the other but about which advice is sought. There may also be times when information is sought about a subject (an article, a community activity, a disease) without a specific case at reference. Each of these modalities requires give and take, but of a different nature.

## REFERRAL BY PEDIATRICIANS

In the first mode, the pediatrician essentially indicates confidence in the child psychiatrist by asking him or her to take the case on for evaluation and treatment. Sometimes the family is well prepared for the referral, and at other times they are not. Sometimes the referral is both thoughtful and appropriate, and at other times it seems more like an impulsive rejection of the child and family by the pediatrician. This depends both on the sophistication of the pediatrician and on the anxiety level of the parents. The pediatrician may have made more effort to prepare the family than is evident on the first visit, and the psychiatrist should withhold judgment of this issue. The child psychiatrist should remember that the pediatrician is usually asking him or her to share the burden of managing what has become, for the pediatrician, a difficult and problematic family or child. At the very least, collegiality suggests that the psychiatrist try to sort these factors out on behalf of both the patients and the pediatrician, rather than abandoning them in turn.

For many pediatricians, the process of assessing the mental health needs of a child and family is difficult, and the act of suggesting and making a referral often puts a strain on the pediatrician–family relationship. Although some parents initiate this process themselves, most parents are still strongly ensconced in some level of denial and are resentful of the pediatrician's intervention. The child psychiatrist can help to strengthen the pediatrician–family relationship by the way in which he or she supports the actions of the pediatrician and encourages and facilitates the family's maintenance of close ties with the pediatrician. Child guidance agencies often do this quite poorly. The agency's refusal to communicate with the pediatrician gives the family a message about the pediatrician that is, at the very least, ambiguous and ambivalent. Sometimes this results in the family's leaving the pediatrician, but at other times the ongoing tie to the pediatrician is stronger than the developing tie to the mental health agency, and the result is the shortening or interruption of treatment. The private child psychiatrist should be aware of all the possibilities and dangers in these dynamics and should act accordingly. The child will certainly continue to need access to ongoing health supervision and illness treatment, and it is not likely to be desirable that such access be blocked or changed in the middle of psychiatric treatment. One way the psychiatrist can help in this process is by providing regular and periodic feedback to the pediatrician about the status and progress of treatment. The child psychiatrist is likely to obtain useful feedback from the pediatrician, in turn, about new or ongoing illnesses or complaints. Especially if the family has more than one child, the pediatrician is likely to have continuing contact with the family in a way that can provide important information for the therapist. Successful collaboration also enables the pediatrician to encourage the family to remain in treatment with the child psychiatrist when resistance emerges, rather than inadvertently colluding with the resistance.

There is no suggestion in this that confidentiality should be breached. However, over the years, a dangerous mystique has arisen about this issue. It certainly is no longer true that most patients wish to hide all evidence of their being in treatment or that they want no communication to occur between the therapist and other important caretakers in the child's life. In our experience, most families, if asked, encourage ongoing contact between the child psychiatrist and the pediatrician. There is no need for the therapist to provide the pediatrician with details of dynamics or treatment processes. He or she should merely make a brief phone call or provide a short note about the continuing nature of the treatment.

In regard to this mode of referral, many pediatricians choose to refer many kinds of cases to mental health professionals other than psychiatrists—social workers, psychologists, even other psychologically oriented pediatricians. A major reason for this is the pediatricians' belief that child psychiatrists are less available, less responsive, and less communicative than these other professionals. However, there is also evidence that pediatricians often do not understand what types of problems child psychiatrists are trained to treat ([Fritz and Bergman, 1985](#)). Finally, the problems and issues superimposed by mental health managed-care organizations have resulted in decreased access for patients to child psychiatrists in many communities, a further challenge for child psychiatrists.

## REFERRAL BY OTHER SOURCES

When the referral comes to the child psychiatrist from a source other than the pediatrician, the child psychiatrist may assume that the pediatrician does not know about the problem or is not interested in it. This may sometimes be true, but it is not a safe or useful conclusion. In these cases, just as in those mentioned earlier, the therapist should ask for permission to communicate directly with the pediatrician. If the family really does not want such communication, they will not hesitate to say so. However, even in these cases, it is likely that the pediatrician may have information that will be both useful and germane to the evaluation and treatment process. Occasionally, the family may want to exclude the pediatrician specifically because he or she does know a great deal about the family, and the family wants an unbiased, "blind" opinion about the problem from the psychiatrist. This dynamic is seldom useful to the child, and the psychiatrist may want to examine his or her options in these cases. Finally, inasmuch as the pediatrician has ongoing supervision of the health of the child, he or she may be able to provide some insights into other areas in the child's life about which neither the child nor the parents may be forthcoming.

## ONGOING CONSULTATION WITH PEDIATRICIANS

In the course of busy, ambulatory practice, pediatricians assume the management of many children with difficult and complicated illnesses and difficult and complicated families and psychosocial situations. Studies of pediatric primary care practices ([Goldberg et al., 1984](#); [Starfield et al., 1980](#); [Starfield et al., 1984](#); [Sturner et al., 1980](#)) reveal that between one-fourth and one-half of all well-child visits to pediatricians revolve primarily around psychosocial issues. The pediatrician may choose to ignore all the signs and symptoms involved in this, but he or she will almost certainly need and want to manage some of them more directly. In doing this, pediatricians often want consultation from a mental health colleague, and many of them have developed some ongoing pattern of presenting such issues to a child psychiatrist in their area—if they are lucky enough to have one. This consultation may take the form of telephone calls or of more formalized meetings. It may be seen as an intrusion by the psychiatrist, but this type of contact is useful in helping the pediatrician to assess the kind of help available from the mental health field and from individual colleagues in particular. It is an important and valuable service that the child psychiatrist would do well to foster in the community. Such initial calls often lead to referrals for more extensive evaluation and treatment, but even if they do not, they create a goodwill that cannot be overestimated. The need for such consultation is especially clear and acute around emergency situations such as suicide attempts, but it is equally important for less dangerous and acute problems as well.

In some communities, a specialized form of this type of collaboration has been in place for varying periods ([Granger, 1985](#); [Solnit, 1968a](#); [Solnit, 1968b](#)). In this model, a group of pediatricians meets regularly with one or two child psychiatrists to discuss difficult cases from the pediatricians' practices. The pediatricians present the cases to the group, and the group discusses them under the leadership of the psychiatrist and perhaps a senior pediatric colleague as well. The ongoing nature of the group provides more benefit for its members than just the case discussions themselves. The members, pediatricians and psychiatrists alike, come to see the group as a support in the continuing struggle against the vicissitudes of practice. This has become even more important as access to direct mental health services has been limited by managed care. Pediatric members of the group unquestionably develop both knowledge and skills in the management of psychological and developmental problems in their practices. They become more sophisticated in their understanding of psychotropic medications. Psychiatrists often use the process to keep up with the changing biological factors in pediatric practice, many of which impinge on the etiology and treatment of psychological disease. In fact, studies in the educational field suggest that the only mode of continuing education that actually results in changed behavior among practitioners is the one in which they interact around cases from their own practices in a continuing manner.

One common ground for collaborative work between pediatricians and child psychiatrists is the use of the concept of temperament in pediatric practice ([Carey, 1986](#); [Carey, 1994](#); [Chess and Thomas, 1986](#)). Temperament measurements can be useful, for example, in educational discussions with parents as well as in formulating



first-time intervention measures when an interference with the “goodness of fit” has given rise to behavioral problems in the child ( [Cameron et al., 1994](#)) (Chapter 15). The concept of temperament has also proven useful in the collaborative care of certain children with chronic illnesses ( [Lewis, 1994](#)) and with uninfected children in families affected by human immunodeficiency virus ( [Lewis, 1985](#)).

The collaborative relationship also opens the door for the possibilities of joint research between child psychiatrists and pediatricians. Some of the most interesting aspects of clinical case management come out of such collaborations. There is ample room in the process, especially in the group process outlined earlier, for more definitive intervention research in pediatric practice settings. Such research can be set up to conform with good psychological practice and can be carried out in multiple practice settings to offer truly useful information on the possibilities of what may be called preventive mental health.

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#### CASE ILLUSTRATION

Jim Smith is an 18-year-old adolescent, the only child of a Midwestern couple in their midforties. Jim was born in Milwaukee and appeared to be developing normally until around 18 months, when he suffered from a severe, persistent bout of otitis. During this time, his language development was believed to regress, and he was referred to numerous specialists. He eventually was seen by a child psychiatrist, who reported that although Jim had some developmental delays, he was not autistic. Mr. Smith had just received an attractive job offer on the east coast, and the psychiatrist recommended that the family seek the services of a child development resource when they relocated. After the family's move, Jim was evaluated at 22 months of age by a child psychiatrist at a child development center and was referred by the center to a developmentally knowledgeable general pediatrician. This child psychiatrist's assessment contrasted markedly with the first evaluation, and she informed the family that Jim was autistic. The family was ill-prepared for this diagnosis; the psychiatrist had not involved the pediatrician in helping to prepare the family for it, and the family rejected the diagnosis and fled from the child development resource.

Dr. Marks, the pediatrician, consulted with another child psychiatrist, Dr. Cooper, with whom he had an ongoing collaborative relationship, about the management of this child and family. They agreed that the child and family would need mental health help as the child grew, but that Dr. Marks should proceed slowly to pave the way for them to accept this. In the meantime, Dr. Marks was able to arrange speech and language resources for this child in his hometown. He arranged for an educational specialist to consult with the preschool to modify the regular program to meet Jim's needs. Jim responded well to the speech and language intervention, and gradually language ceased to be a problem, whereas Jim's social awkwardness and lack of relatedness became more central.

By the time Jim was 4 years old, his parents were ready to talk with a child psychiatrist, and they were referred to Dr. Cooper. He worked with the parents and evaluated the child. In the first few years, most of Dr. Cooper's work was with the parents. It became apparent that Mr. Smith shared some of Jim's relatedness issues. Mrs. Smith, in contrast, was extremely nurturing and supportive and was the crucial family resource facilitating the work of both the psychiatrist and the pediatrician. When Jim was 6 years old, Dr. Cooper began to see him on a more regular basis. During Jim's "latency" period, some of the content of the sessions suggested a thought disorder, although Jim appeared to have no trouble distinguishing between fantasy and reality. In adolescence, Jim's verbalizations and imagery appeared more psychotic. The guidance counselor at school was frightened by some of his statements and needed help from the physicians to understand him. Drs. Cooper and Marks both believed that Jim would benefit from medication. Risperidone was helpful with his thoughts, but it exacerbated his exercise-induced asthma, thereby interfering with his swimming, which was very important for him. Again, the pediatrician and the psychiatrist were able to work together to balance Jim's myriad needs.

Jim currently carries the diagnosis of Asperger's disorder. He is taking medication and is functioning well, with reasonable self-esteem and self-awareness, in a vocational training program.

#### Comment

The favorable outcome that was achieved in this case was far from certain, given its beginnings. The complexity of developmental problems in children with spectrum disorders requires the utmost collaboration between child psychiatrist and pediatrician. Pediatricians have ready access to these families and are generally perceived as nonthreatening. They can help families to cope with and confront the problems of their children, and they are often first to become aware of new issues. Child psychiatrists can help families and children to understand and work through their difficulties for an optimal outcome. Close communication between the two professionals significantly improves the result.

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## SUMMARY

Given the differences between their disciplines, pediatricians and child psychiatrists have not found it easy to collaborate. However, more and more of them are managing to do it and, as they become more experienced with the process, are finding it a rewarding way to manage their professional lives. The process results in an improvement in the management of health care for children that neither discipline can achieve alone. It can only be hoped that more and more pediatricians and child psychiatrists will discover the satisfactions and rewards of true collaboration.

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## 94 CHILD'S COGNITIVE UNDERSTANDING OF ILLNESS

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Physicians realize how difficult it is to provide adequate and effective explanations of illness and proposed treatment to their adult patients. Medical education has left the physician with a qualitatively different way of understanding illness, both physical and mental, that is beyond the grasp of the lay public. Explanations must be translated by the physician so the patient can understand. This is not a simple process of merely replacing unfamiliar medical terminology with words from the patient's own vocabulary, but it often involves the much more difficult task of restructuring the explanation to comply with a conceptual framework that is comprehensible to the patient. When the patient is a child, the clinician is confronted with a broad range of conceptual frameworks, some of which may initially seem idiosyncratic. The challenge of ensuring effective communication therefore becomes far greater, but it is of no less importance.

The prospect of determining each child's unique conceptual framework for understanding illness may at first seem difficult. Fortunately, systematic research has been conducted that provides important insights into the developmental process by which children obtain an understanding of the fundamental concepts of physical illness, such as causality, prevention, and treatment. These cognitive–developmental studies ( [Burbach and Peterson, 1986](#)) demonstrate a systematic and predictable sequence by which children acquire these concepts to explain both general physical illness, as well as specific conditions such as acquired immunodeficiency syndrome ( [Schonfeld et al., 1993](#)). This developmental process is comparable to that of the acquisition of causal understanding as described by Piaget ( [Piaget, 1929](#)). According to Piaget's model ( [Brewster, 1982](#); [Ginsburg and Opper, 1969](#); [Perrin and Gerrity, 1981](#); [Piaget and Inhelder, 1969](#)), as the result of biological maturation and the accumulation of experience, the child progresses through four sequential stages of cognitive development: the sensorimotor period (infancy), the preoperational period (early childhood, roughly 2 to 7 years), the concrete operational period (middle to late childhood, roughly 7 to 11 years), and the formal operational period (adolescence to adulthood). Although experience has shown that children often progress through these stages at a faster rate than was initially predicted by Piaget, the nature and sequence of these stages, described by Piaget as invariable, have been confirmed by extensive research. Children in the preoperational period rely on direct personal experiences and have little ability to generalize to related situations or to appreciate multiple aspects of one situation; their thought processes tend to be empirical rather than logical. Later stages involve an increasing ability to use logical thought processes, with abstract thought attained only during the formal operational period. Later stages also are characterized by the child's increasing ability to differentiate self from others and to distinguish internal wishes, needs, and thoughts from the realities of the external world.

Efforts to promote the child's adjustment to physical illness and its treatment require an appreciation of this developmental process. Effective support and assistance are predicated on an understanding of not only the child's feelings but also the child's beliefs about being ill ( [Bibace and Walsh, 1980](#)). For example, it is often assumed that children fear blood drawing because of the pain associated with the procedure, and reassurances thereby take the form of "It will only hurt a little." In reality, the child's feeling of fear may be related to a belief that the phlebotomist will remove too much blood and the child will die ( [Licamele and Goldberg, 1987](#)). In this situation, the reassurances offered are ineffective, and the child is left to deal, alone, with a terrifying misconception about the treatment process. Efforts to facilitate the child's adjustment to physical illness and its treatment must therefore be based on an appreciation of the child's knowledge of the relevant concepts of illness. Otherwise, the ill child will be left, as depicted by Anna Freud, "to submit uncomprehendingly, helplessly and passively" to both the illness and the treatment process ( [Freud, 1977](#)).

The child's knowledge and understanding of the concepts of illness can thereby be viewed as vital determinants of the child's adjustment to illness. The focus of this chapter is to review what is known of the developmental process by which children acquire an understanding of the concepts related to illness and the implications of this knowledge for clinicians.

### PRIOR RESEARCH

On the basis of a review of the cognitive–developmental literature on children's concepts of physical illness, Burbach and Peterson (1986) conclude that a positive relationship exists between the level of understanding of the concepts of illness and the child's chronologic age and cognitive maturity that is consistent with Piaget's theories of cognitive development. Wide variability in children's understanding as a function of age is noted across studies; a similar but somewhat smaller variation persists when analysis is conducted as a function of cognitive–developmental level (frequently measured in terms of piagetian tasks). Global estimates of a child's cognitive abilities are thereby often inaccurate predictors of the level of understanding of concepts about illness; on average, the understanding of the causality of illness typically lags behind that of general causality ( [Perrin and Gerrity, 1981](#)). Children bring their own personal experiences to bear on their understanding of illness; some of these experiences may promote understanding of the concepts for a particular child, whereas others may have been negative experiences that only serve to heighten anxiety and interfere with the acquisition of knowledge. It is not surprising, then, that it is still unclear what role personal experience with illness plays in the process by which children develop an understanding of the concepts of illness ( [Burbach and Peterson, 1986](#); [Perrin et al., 1991](#)). For this reason, in the sections to follow, the focus is not on what "a typical child" knows or thinks at a particular age. When possible, generalizations about representative age ranges are provided, but the emphasis is on an overview of the process and not a time line. Clinicians are advised not to rely on age-based normative data but to develop instead an appreciation of the process of cognitive development in this area. Simple inquiry into the child's views and understanding will then identify the child's level of comprehension and can lead to an appreciation of the child's unique misconceptions and concerns.

### Immanent Justice, Guilt, and Shame

With increasing cognitive maturity (and age), children's understanding of the causality of illness increases in a predictable manner. Younger children, lacking an adequate explanation for the cause of illness, are apt to resort to explanations that attribute the cause of illness to immanent justice, "the belief that a form of natural justice can emanate from inanimate objects," wherein misdeeds will be automatically punished. Such a belief leads to the acceptance of personal guilt and shame for the origin of illness. It is hypothesized that magical thinking and immanent justice concepts are employed by the young child in preference to the concept of chance, thereby allowing the child to retain the illusion of order and personal control in what would otherwise appear to be a random and often unfair world ( [Wilkinson, 1988](#)). Freud also appreciates the child's tendency to assume guilt for personal illness: "[Illness] appears to the child as a confirmation of the belief that wrongdoing, however secretly performed, is open to punishment, and that other, still undetected misdeeds, whether actually carried out or merely contemplated in fantasy, will likewise be followed by retribution of some kind" ( [Bergmann and Freud, 1965](#)). As one 11-year-old boy related about his diagnosis of polio 2 years earlier: "I was charged with having polio. I had to plead guilty, and my sentence was life imprisonment in a wheel chair" ( [Bergmann and Freud, 1965](#)).

Research has shown that explanations based on immanent justice are more persistently used for ailments for which the child has limited personal experience and for which another explanation is not readily available ( [Siegal, 1988](#)). With increasing cognitive maturity and with the accumulation of experience with illness, the child is more likely to reject the notion that illness and misbehavior are linked and instead acquires more accurate perceptions of personal control over illness and recovery ( [Burbach and Peterson, 1986](#)). It is hoped that, by providing the child with more appropriate explanations for the cause of illness, the child will be able to abandon immanent justice explanations at an earlier age ( [Kister and Patterson, 1980](#)). Such immanent justice explanations can be expected to result in guilt or shame, to impede competent understanding of the cause of disease and thereby hinder attempts to promote compliance with preventive health measures and treatment regimens, and to impair adjustment and coping to illness of self and significant others. It has been demonstrated that even preschoolers dismiss immanent justice as an explanation for familiar ailments with which they have had personal experience (e.g., colds) while they retain this concept in their explanations of causality for ailments with which they are less familiar (e.g., toothaches) ( [Siegal, 1988](#)). Such findings lend support for the role of personal experience, and indirectly education, in



advancing the child's understanding of the causality of illness.

### Further Understanding of Illness Causality

As children begin to develop an understanding of the true causes of physical illness, the concept of contagion appears in their explanations for the causality of disease. Younger children (preoperational stage) initially tend to overextend the concept of contagion to include noncontagious illness; the concept is applied most appropriately by children reaching the later stages of cognitive development (concrete and formal operational stages) ( [Kister and Patterson, 1980](#); [Potter and Roberts, 1984](#)). Yet in appropriate circumstances, proper discriminant application of the concept of contagion is within the grasp of children as young as the preschool level. Siegal (1988) demonstrates that most preschool children in his study (the mean age for study participants was 4 <sup>11</sup>/<sub>12</sub> years) could accurately attribute contagion to the common cold and differentiated this from certain noncontagious ailments (e.g., a scraped knee). As such, it is concluded that knowledge of the causes of illness is within the cognitive grasp of very young children, a finding offering support for the incorporation of causal knowledge into health education efforts for children even at the preschool level.

Subsequent to the acquisition of the knowledge that illness can be contagious, an understanding of the physical process by which this can occur must be developed by the child. Such an understanding typically takes the form of a germ theory. In a study of Scottish nursery children (3 to 5 years old), Wilkinson (1987) describes the early stages of children's understanding of the role of germs in the spread of disease. Initially, the child is unfamiliar with the word and often confuses *germ* with *bug*, and thereby *insect*, in addition to other terms ("I don't know what a germ is but I have heard of a German person."). Children view germs within their own magical and egocentric context ("Germs are not there all the time. They come when mummies go away."). They also attempt to draw on concrete experiences to explain what germs look like. Many children in Wilkinson's study stated that germs are blue and 1 to 9 inches in diameter, and although they had not seen one, they claimed "I think I will see one one day." Discussion with parents disclosed that the children were drawing these images from a television commercial for a cleaner wherein hidden imaginary blue germs were destroyed by the application of the product. Little understanding is seen at this age regarding how germs are transmitted or cause illness. One mother, attempting to have her son describe the nature of germs, inquired, "Why are you made to wash your hands before eating?" His reply illustrated his lack of comprehension: "So my plate doesn't get dirty."

Perrin and Gerrity (1981) found that by about 9 or 10 years of age, children generally believe that illness is caused by germs but still have little understanding of how this is accomplished once the germ is internalized. By about 12 or 13 years of age, illness is seen as the result of multiple causes; with this comes an appreciation of the role of host factors and the beginning of an understanding of the subtle interactions between host and agent in the causality of disease and the recovery from illness. It is therefore not until at least adolescence that the child can associate apparently unrelated symptoms (e.g., headache and rash) and view them as belonging to one illness and can identify and relate the various phases of an illness into a coherent progression of one disease process. During the formal operational period, children also develop an increased understanding of their bodies, which allows an appreciation of internal physiologic structures and functions that is demonstrated in the emergence of physiologic explanations for the causality of illness in early adolescence ( [Bibace and Walsh, 1980](#)). Only at this stage, with this improved understanding of internal physiology and the causality of illness, can the child be expected to comprehend many of the simplest treatment regimens that adults take for granted, such as the use of oral antibiotics to treat an ear infection or the use of injections of insulin in diabetes.

### IMPLICATIONS

Knowledge of what children understand about illness and its treatment at various stages of cognitive development has practical implications for child psychiatry consultations to clinicians. It provides a framework for offering guidance on how to approach discussions about illness and its treatment with children, both to advance their understanding and coping skills and to promote their attainment of an active role in the decision-making process for their health care. As children acquire increasing knowledge about the concepts related to illness and the cognitive skills to process this information, they become more able to report symptoms of illness accurately, to comply with the treatment regimens prescribed, to adjust to the illness and its treatment, and to make informed decisions regarding preventive health measures.

#### Child's Ability to Report Symptoms

Young children, with their immature understanding of disease processes, may fail to report important symptoms of serious illness. The reason may be, in part, a lack of sensitivity to the relevant internal cues and objective signs of even serious illness ( [Pidgeon, 1985](#)). However, even when children are aware of their symptoms, egocentrism and magical thinking may lead them to withhold this information from caregivers, and they may prefer instead to rely on internal mechanisms of dealing with the illness. They may assume that if they do not wish to be ill, then they will not be ill; they may worry that the mere vocalization of their concerns may be sufficient to bring their fears to reality. Children who persist with immanent justice explanations for the causality of illness may also be disinclined to voice their symptoms out of shame or fear of retribution. These issues are particularly relevant for children with chronic illnesses that require prompt recognition and management of often subtle internal symptoms, such as hypoglycemia in the child with diabetes.

#### Child's Compliance with Treatment

Attempts to communicate effectively with children about illness and its treatment also maximize efforts to enlist the child's cooperation in the treatment process. Such efforts should maintain as a long-term goal the child's attainment of an active role in the decision-making process in the child's own health care. In times of frustration, it is easy to lose sight of this goal, such as when a child with asthma is noncompliant with treatment and is warned, "If you don't take your medicine, you'll have to go to the emergency room and get a needle," but is not provided with a developmentally appropriate explanation of the treatment process. Parents need help in appreciating that such threats may yield temporary compliance, but at the expense of reinforcing maladaptive concepts of illness, that may, in the long run, lead to decreased compliance. This approach could be justifiable if it were shown that the concepts of illness were beyond the grasp of children and that illness in the child could be effectively prevented through compliance with recommendations. Both assumptions are incorrect. First, the preceding discussions illustrate that an understanding of the concepts of illness is possible even in very young children, if they are given appropriate explanations and are provided with constructive learning experiences. Second, even if children are able to comply fully with all the numerous restrictions and requirements placed on them by their parents in the hopes of preventing illness (e.g., to get enough sleep, to eat the right food, to dress correctly), many will still become ill. If the only explanations provided about the causality of illness reinforce immanent justice, then the child is left with no other recourse but to accept self-blame for personal illness. Hospitalization and increasingly invasive medical management, when required, are then not perceived as a result of the natural process of the disease but instead as a personal failure on the part of the child. The child believes that he or she has failed and is being punished; in addition to feeling ill and frightened, the child is now apt to feel angry and rejected. As one child remarked about his failure to be discharged from the hospital: "I did everything the doctors told me to do, and now they won't let me go home." He had been told in simple terms what he needed to do to get better, but he had not been given explanations that he could understand of how these procedures would influence his illness. After complying fully with the requirements placed on him and failing to become well and be discharged, he was understandably hurt and angry.

#### Child's Adjustment to Illness and the Treatment Process

Explanations for children about their illness and treatment should seek not only to achieve compliance but also to promote the child's understanding. The explanations should be presented in a manner that is consistent with the current level of understanding of the child or perhaps is slightly advanced ( [Bibace and Walsh, 1980](#); [Brewster, 1982](#)). These explanations should aim to replace frightening misconceptions and to supply constructive information in a manner that will promote coping and adjustment. This is, of course, an ongoing process. Children need to be questioned about their understanding of the explanations provided, thereby allowing the discussion to become a true dialogue. Vocabulary used should be simple and direct. Indeed, even adult patients misinterpret terminology at a rate that exceeds the expectations of most physicians. In one study of women attending a public health clinic, 80% of patients confused "anemia" with "enema," and more than 50% defined "well-nourished" as "nervous body" ( [Collins, 1955](#)).

When under stress, such as during an acute illness, children often regress in their developmental abilities and are least amenable to techniques to advance their developmental progress. Even in this setting, recognition of children's current level of functioning still has practical implications for efforts to restructure their thinking and provide support during the illness. For example, when one is constructing a plan to help control pain for a child in the preoperational period, the connection between the medication taken orally and the relief of the pain can be emphasized, but more concrete measures that will be reassuring to the child (e.g., application of a heating pad for abdominal pain) should also be employed. As much as possible, the use of intramuscular pain medication should be avoided, in view of the child's tendency to associate treatment with punishment and to fail to comprehend the logic of giving a painful intramuscular injection to treat abdominal pain. Moreover, when one is selecting the oral medication to be given, knowledge of the child's cognitive abilities regarding comprehension of quantity and number may have further practical implications; it may be preferable when the dose of pain medication is to be adjusted to give two 15-mg pills instead of one 30-mg pill, or to use an elixir instead, because of the even clearer concrete representation of increasing dose with increasing amount ( [Clark, 1985](#)). Appreciation of children's understanding of

their illness and its treatment and knowledge of their cognitive abilities can thereby provide guidance not only in the selection of explanations but also in the selection of the most appropriate techniques for the delivery of health care.

Research also has shown that, parallel to the process of conceptual development wherein children acquire an understanding of the causes of illness, children also develop increasing appreciation for the intent of medical procedures. Brewster (1982) describes three stages in this process: The child initially views the procedures as punishment, is subsequently able to understand the intent of the procedures but feels that the staff will be empathetic only if the child outwardly expresses pain, and, finally, is able to infer both empathy and intention. To enlist cooperation and to decrease anxiety and fear surrounding the procedures, children should be prepared for procedures with information and explanations that are consistent with their level of understanding of the relevant concepts. Preoperative teaching for the child in the preoperational period, for example, could focus on acquainting the child with what the concrete experiences will be (e.g., what the room will look like, who will be there), whereas preparation for the child in the formal operational period could also include a more detailed discussion of the procedure, including mention of anatomic and physiologic principles within the cognitive grasp of the more mature child ( [Perrin and Gerrity, 1981](#)).

Programs to prepare children psychologically for procedures have been shown to be effective in helping them to cope with the associated anxiety. Edwinston (1988) demonstrates that such programs are effective in reducing anxiety, even in the setting of emergency surgical procedures (e.g., acute appendectomy), when compared with standard, unstructured preoperative preparation. Research is also just beginning to demonstrate empirically that preparation programs for hospitalization and medical procedures that are consonant with the child's cognitive–developmental level are more effective in decreasing distress and anxiety in response to procedures when compared with preparations that are not designed with these concepts in mind ( [Rasnake and Linscheid, 1989](#)).

### Preventive Health Education for Children

Health education only at the time of illness or hospitalization may not be the most effective means of advancing the child's understanding of illness. During times of illness, as in other stressful periods, children often regress in their developmental abilities. At these times, they are least prepared to understand the situation confronting them and least able to benefit from educational efforts to promote this understanding, but most in need of this information to facilitate adjustment and successful coping. This observation provides a strong argument for the need to promote the acquisition of these concepts before they are needed to deal with acute crisis in the form of illness. Health education efforts, then, should begin when the child is well, with the aim of promoting increased understanding, knowledge, and skills in this area. Research has demonstrated that developmentally appropriate health education in elementary school can advance young children's conceptual understanding and factual knowledge of illness ( [Schonfeld et al., 1995](#)). Children should be taught to do more than “just say no” to bad health decisions; they should be helped to understand the rationale behind these choices and to acquire the skills to make informed and responsible health decisions that affect them in the future. For health education to be effective, it must be developmentally based, so discussions coincide with the child's level of understanding ( [Schonfeld, 2000](#)).

Physicians should use preventive health visits during the pediatric years to educate not only the parents but also the child. Unfortunately, research has shown that, during pediatric visits, the pediatrician spends little time talking directly to the child. In one study of children 4 to 7 years of age who were attending private pediatric practices for routine health maintenance visits or visits for minor illness, only about 5% of the discussion during these visits was directed by the physician toward the child. Interviews of the children after the visits illustrated that most of them desired more information about their medical problems; their concerns were well formed and relevant to the reason for the current visit, yet these concerns were not addressed by either the parent or physician. In fact, explanations about treatment and symptoms were not provided to the children in this setting, and the children were treated instead as passive recipients of health care ( [Perlman and Abramovitch, 1987](#)).

Other studies have shown similarly low rates of communication between physicians and pediatric patients. This is unfortunate, because children are interested in clinical information even at a young age and are capable of comprehending the information if it is presented in an appropriate manner. Pediatricians should take advantage of this opportunity to advance the child's understanding of the concepts of physical illness in a salient and relevant context—that of the child's own medical visit. One reason for this oversight may relate to the observation that pediatricians, and other health care professionals who work with children, are unfamiliar with the developmental stages of children's understanding of illness: “Professionals do not operate with an intuitive sense about cognitive development. . . . They lack an understanding of the important qualitative differences in the very basic ways in which children at different stages of development see, interpret, and understand the world around them” ( [Perrin and Perrin, 1983](#)). One study ( [Lewis et al., 1991](#)) demonstrated that a brief educational intervention targeted to 5- to 15-year-old children, parents, and physicians enhanced physician–child communication and rapport during pediatric office visits and allowed children to take a more active and effective role in their health care.

### CONCLUDING REMARKS

The focus of this chapter has been on the cognitive process by which children develop an understanding of their illness and treatment and on the implications of this for health care professionals. Such a cognitive–developmental approach to viewing the child's understanding of illness is not in conflict with other theories that aim to explain the child's psychological reaction and behavioral response to the illness experience. Conscious understanding (cognitive factors) is only one component of the child's response to illness. Unconscious developmental fantasies, too, may alter the child's perceptions of the illness and treatment process, despite the presence of mature cognitive abilities. For example, a fear of surgery may be heightened by underlying castration anxiety ( [Lewis, 1982](#)). Even adolescents who have reached the formal operational stage of cognitive development may still be overwhelmed by anxiety from unconscious developmental conflicts. These conflicts may distort their perceptions and may result in misconceptions and fears out of proportion to what would be predicted by a purely cognitive–developmental approach. An integrated approach to viewing the ill child that incorporates both the child's cognitive abilities and an appreciation of the psychological factors pertinent to his or her personality development is therefore likely to yield the most insight into the complex process by which the child comes to understand, accept, and respond to both the illness and treatment process.

Subsequent chapters highlight the child's unique response to specific illnesses and components of the treatment process, such as hospitalization and surgery. An increased appreciation of the stages of children's understanding of illness will allow the physician to communicate more effectively with children, to improve their medical care, to minimize their anxiety and fear, and to promote their adjustment to illness and its treatment. Health care professionals will thus be able to assist the children to acquire the requisite skills to become increasingly active and effective partners in their health care management.

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# 95 PREMATURITY, BIRTH DEFECTS, AND EARLY DEATH: IMPACT ON THE FAMILY

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As child psychiatrists have become more involved with infants, toddlers, and their families, they are often asked to consult when a child is medically compromised. These consultations involve assessment of the child, the family, and often the medical care system responding to the child's illness. Conditions such as prematurity, birth defects, and/or serious genetic malformations are accompanied by profound psychological disturbances in the child, family, or both; these psychological disruptions contribute to the family's and child's recovery or continued psychological debilitation. In the instance of early childhood death, the impact on the family and subsequent children may be pervasive and long lasting. In this chapter, we review the role of child psychiatrists in three situations—preterm birth, birth defects, and sudden infant death. We discuss the instance of preterm birth in greatest detail, with the understanding that many of the clinical issues a child psychiatrist encounters in that situation are also applicable to birth defects and infant death.

## HISTORICAL NOTE

Although the topics discussed in this chapter represent forms of family crises experienced since the dawn of humanity, the child and adolescent psychiatric literature was relatively sparse in these areas until approximately 30 years ago. Prior relevant literature appeared in the early 20th century, when the topic of loss and its psychological implications was discussed by [Freud \(1957a\)](#) in the classic paper, "Mourning and Melancholia." Although Freud used normative mourning in order to understand the more pathologic state of melancholia, his interest appears to have legitimized the normative experience as an area for further study. It was not until the 1940s, however, that a formal descriptive study appeared in the literature. In 1944 Erich Lindemann and colleagues reported on studies of acute traumatic loss in families who had lost a close relative in the catastrophic "Coconut Grove" fire in Boston. In one case vignette unrelated to the fire, they alluded to mothers of infants who had died being especially vulnerable to severe grief reactions. Many of the victims in this study were very young servicemen and their escorts and might therefore be considered to be late adolescents.

In the 1950s child and adolescent psychiatrists began more specific inquiries into the stresses of prematurity as a family crisis. Dane Prugh, one of the first consultation-liaison child psychiatrists, published in 1953 an anecdotal and theoretical article on parents and premature infants. Slightly later, Gerald Caplan, the founder of community psychiatry, and associates studied families with premature infants as a paradigm for acute stress in normative families ([Caplan et al., 1965](#)). Prematurity was one reason for early infant loss; crib death was another. The term sudden infant death syndrome (SIDS) was not present in the medical nomenclature until the 1970s. Furthermore, although the term crib death had been used in the pediatric literature, no family studies were conducted, most likely because these families were inaccessible at the time. Instead, efforts were directed toward parents with children dying in the hospital ([Solnit and Green, 1959](#)). In 1961, Solnit and Stark directed their attention to parents of children with birth defects, particularly retarded children. All of these early papers were primarily descriptive and anecdotal in nature, with theoretical postulates and management approaches being suggested. It was not until the mid-1960s that formal studies of significant numbers of parents began to appear in the literature.

In 1963, Friedman and associates studied prospectively the parents of 27 children admitted to the National Cancer Institute and followed the parents' adaptation to their child's illness irrespective of the course of illness ([Friedman et al., 1963a](#)). Special emphasis was placed on families whose children died; measurement of corticosteroids in the parents' urine was used during the study for the first time to document the biological effects of chronic stress ([Friedman et al., 1963b](#)). These studies can be said to have ushered in the modern era of family studies of a more specific nature, which have produced management techniques in dealing with parents in the premature intensive care unit, general hospital unit, and at home. Also, as the care of preterm and/or very sick infants and young children has advanced technologically, child psychiatrists are called on to act therapeutically at the interface between complex medical systems and parents' psychological reactions and adjustments to such systems.

## THE FAMILY AND PREMATURE BIRTH

### Early Studies and Crisis Theory

The premature birth of a baby has been viewed historically in psychiatry as a crisis for the family as a whole. The crisis has been described as a period of normative emotional disequilibrium, during which individuals demonstrate symptoms resembling psychopathology ([Caplan, 1960](#); [Caplan et al., 1965](#); [Friedman et al., 1963b](#); [Kaplan and Mason, 1960](#)). Early authors, such as [Prugh \(1953\)](#), highlighted the mother's emotional responses to the baby, staff caregivers, and family members. He pointed out that the complex psychological reactions on the part of the mother included anxiety (particularly related to lack of confidence as a caretaker), guilt about the prematurity, resentment and disgust toward the baby, and competition and jealousy toward the nursing staff. Furthermore, Prugh described an "emotional lag" or period of alienation on the part of the mother during the first few days, as well as an occasional tendency to overprotect. Suggested interventions included early contact with the baby, provision of information, encouragement of emotional expression, involvement of the father and other family members, and discussion groups of mothers on the ward.

Caplan, in another early description in the literature, studied 30 cases of prematurity ([Caplan, 1960](#)). He depicted "healthy" and "unhealthy" outcomes based on the mother's sensitivity to the infant's needs, her respect for and satisfaction of these needs, and the maintenance of other family relationships. Caplan related the patterns of parental response to their cognitive grasp of the situation, handling of associated feelings, and seeking of help.

[Kaplan and Mason \(1960\)](#) presented the hypothesis that maternal reactions to the stress of the premature birth, although expected and normative, are usefully described as acute emotional disorders. Unique psychological tasks related to the prematurity were enumerated sequentially. The first task included preparation for the possible loss of the child ("anticipatory grief"). The second task related to the mother's failure to deliver a normal full-term baby. (They noted that the first two tasks were often associated with grief and depression.) The third task was the resumption of the process of relating to the baby, which had been interrupted by the



premature birth. The fourth task related to the mother's understanding and attention to the baby's special needs and growth patterns. These authors further described pathologic deviations from the normative accomplishment of the tasks. These included maladaptive denial and failure to respond positively to the progress of the baby.

In a pivotal integrative paper, [Caplan and colleagues \(1965\)](#) described patterns of parental response to the crisis of prematurity. They defined the crisis state as analogous to [Lindemann's \(1944\)](#) classical description of bereavement. Caplan and coworkers more fully elaborated "healthy" and "unhealthy" adaptations to the premature birth. For instance, they questioned whether the parents continuously attempted to gather information about the baby. Did they acknowledge ambivalent feelings about the premature baby, with accompanying verbal and nonverbal expression? Was there an active seeking of help for the family, related to the child's special needs and the need of the parents for emotional expression?

### Development and Bonding

Somewhat later, there appeared many articles in the literature relating to the associations among premature birth, neonatal complications, developmental difficulties, and subsequent problems with bonding between the mother and baby. Several authors postulated a link between dysfunction in bonding and later potential for abuse and/or neglect ([Elmer and Gregg, 1967](#); [Hunter et al., 1978](#); [Klein and Stern, 1971](#)). This association has remained controversial and was not confirmed by other investigators ([Minde, 1980](#); [Steele and Pollock, 1974](#)). Several researchers describe the behavioral and interactional difficulties that preterm babies have during the first years of life ([Als, 1981](#); [Als and Brazelton, 1981](#)). They and others also presented the difficulties parents have in engaging the attention of their babies ([Als, 1981](#); [Als and Brazelton, 1981](#); [Brown and Bakeman, 1980](#); [Field, 1979](#)).

[Minde and colleagues \(1983\)](#), in a well-controlled study of 184 low birth weight infants in a neonatal intensive care unit, demonstrated that parents of "sick infants" interacted far less with them than parents of well babies. Furthermore, they found that this pattern continued at home after discharge from the hospital. [Bidder and associates \(1974\)](#), comparing mothers' attitudes to their preterm and term babies, found that the mothers perceived the preterm baby to be "weaker" than the term babies. They highlighted two specific periods of particular anxiety for the mother: immediately after the birth and when the baby goes home.

[Jeffcoate and colleagues \(1979\)](#) compared attitudes of parents of preterm infants to those of parents of full-term infants. They found that there was evidence of some disturbance in parent-child relationships in the preterm group. This consisted of delays in maternal attachment, negative maternal perception, and continuing parental anxiety about caretakers. Two of their sample of preterm infants had been abused or neglected. Jeffcoate and colleagues emphasized the need for ongoing psychological support for these parents.

[Silcock \(1984\)](#) utilized the previously mentioned four-task framework of Caplan and coworkers to investigate the intensity of maternal experience of these tasks, mothers' success in dealing with them, and the relationship between successful task completion and later mother-infant interaction. The author studied 24 mothers of preterm infants and found that the ability to cope with the crisis was correlated with better mother-infant relationship at 1 month and 4 months of age. She also ascertained that the degree of intensity of emotion accompanying each task varied widely among the mothers and related to individual prior life experience. [Macey and colleagues \(1987\)](#) reported that mothers of preterm infants felt overprotection toward them, were apprehensive about surrogate caretakers, and believed the baby's birth had had an initially negative effect on the family. These authors also noted that preterm infants demonstrated less exploratory play and stayed close to their mothers while at play.

### Demographic Characteristics of Prematurity and Acute Hospital Care

Approximately 10% of all newborn infants are born at a gestational age less than 37 weeks and between 1% and 2% of infants are born at a birthweight of less than 1,500 grams ([Goldberg and DiVitto, 1995](#)). Between 80% and 85% of infants weighing 1,000 to 1,500 grams at birth, but only 50% to 60% of infants less than 1,000 grams, survive the perinatal period ([Pharoah and Alberman, 1990](#)). Preterm births are also now more common among families with limited resources and much increased among single, young mothers with little to no prenatal care. These factors define not only a postnatal (or postnursery) environment of risk but also describe families who have few psychological as well as physical resources to deal with the crisis of a preterm infant's birth and subsequent care needs.

The severely preterm infant may be hospitalized for several weeks to months. Longer hospitalizations exact greater psychological tolls from parents and siblings as their daily lives are rearranged for several months to encompass the infant's hospital stay. Also, although it is rarely written about ([Mayes, 1995](#)), longer lengths of stay for preterm infants exact a cost from nursery staff as well and the impact on nursery staff affects how much they are available to parents. Both nurses and physicians become more deeply involved with an individual infant and family and must repeatedly work through the infant's multiple medical crises, debilitating complications that are sometimes fatal, and the infant's eventual discharge to someone else's care. As the infant improves, nurses begin to play, talk, and hold the baby beyond the requirements of routine medical care ([Corter et al., 1978](#)). These greater demands on forming, remodeling, and mourning personal attachments also occur in the context of multiple acute, life and death crises that are the daily substance of an intensive care nursery. Thus, psychological processes that require time and reflection are evolving in an atmosphere of urgency and a compressed sense of time. These disjunctions between psychological time and the urgent, crisis-colored time frame of the infant's acute illnesses are equally powerful for parents and shape certain aspects of their later relationship with their infant ([Mayes, 1995](#)). At the very least, for many parents, they find themselves always feeling vigilant and crisis-ready, even years later as their infant boy becomes an active, athletic, bright teenager. In addition to long hospitalizations with the attendant stresses on family functioning, very low birthweight preterm infants are at greater risk for acute medical complications that often carry severe risks for neurologic and physical sequelae. These include intraventricular hemorrhage, necrotizing enterocolitis, and cardiopulmonary complications. Not only do these prolong an infant's hospitalization and increase the risk for later neurodevelopmental compromise, but they also define acutely life-threatening conditions that further lead to the sense of fragility and uncertainty surrounding the preterm infant. Recurrent threats to survival pose recurrent threats to the ability of parents to make a place for their very sick infant in their lives and to construct within their inner object world even a nascent sense of the child as a part of them and their family. Mourning and "letting go" conflict with forming attachments and "taking in," as occurs in the birth of a full-term, healthy infant.

The neonatal intensive care unit is also like no other place parents or their other children might encounter. The primary goal of the unit is to ensure the physical well being of the infant by whatever means necessary, and this often involves highly sophisticated mechanical technology. Many studies document the relatively high noise and lighting levels, the large number of adults, frequent painful, stressful procedures for the infant, and the lack of relation between the infant's behavior and environmental structure or events ([Goldberg and DiVitto, 1995](#)). For example, the need to monitor the infant's physiologic status continuously precludes altering levels of light or sound to approximate a day-night cycle, as would occur for a term infant. Additionally, preterm infants may be more sensitive to levels of environmental stimulation and thus more easily overwhelmed by standard nursery environments than term infants ([Thoman, 1993](#)). Indeed, the extent to which nursery conditions affect long-term development is not at all clear, and it is very difficult to separate potential effects of the environment or interventions from the medical condition that necessitates them. In any case, the neonatal intensive care environment is surely stressful and often overwhelming for parents and is continuously functioning at a level of intensity and vigilance found in few other environments caring for infants and young children.

### Parenting and the Preterm Nursery Experience

The nursery experience and interventions required to medically support a preterm infant often stress and in some instances severely compromise the development of expected parent-infant relations. Traditionally, as medical expertise in intervening with and caring for the very preterm infant improves and technology becomes more advanced, families are more often separated from their infants during the most acute phases of their hospitalization. In the early 1960s, neonatal intensive care units stressed maintenance of a sterile environment to prevent infection and parents were often excluded. Furthermore, parents often felt overwhelmed and frightened by the sophisticated technology designed to support the infant and were sometimes more likely to stay at a distance. Their reluctance to approach the baby was given functional credence also by the prevailing wisdom of that era that touching and handling the preterm infant was stressful and only medical procedures should be allowed, with touching or caressing kept to a minimum.

Policies such as these began to change in the 1970s when careful studies revealed no increase in infection rates after parents were allowed in the nursery ([Barnett et al., 1970](#)). Parents were encouraged to visit their infants, and these shifts in nursery policies have generated a number of studies on the effects of holding, massage, and skin-to-skin contact on the immediate status of the infant ([Field, 1990](#); [Goldberg and DiVitto, 1983](#); [Goldson, 1992](#)). As these physical barriers were removed, more psychological interventions for parents also were instituted, including direct involvement in the infant's care, areas within the nursery for parents to be alone with their infant, and therapeutic groups for families with infants in the intensive care unit. Liberalized contact between family members and the baby has become routine in the vast majority of special care nurseries. As a result of such increased access, several authors have maintained that the emphasis of concern on the part of the mother has shifted from her own feeling of psychological loss to the outcome for the baby ([Pederson et al., 1987](#)). How these types of liberalized interventions impact on later parenting after the infant is discharged from the nursery has not been systematically studied, but at least acutely in the nursery, more direct parent involvement potentially increases parental self-confidence and self-esteem and likely impacts on the early formation of internal working models and attachment

patterns between parents and infants.

Many writers have alluded to the significance of networks of social and emotional support to aid parents who are dealing with a preterm birth crisis ( [Bidder et al., 1974](#); [Blackburn and Lowen, 1986](#); [Fox and Feiring, 1985](#); [Pasco and Earp, 1984](#); [Prugh, 1953](#)). [Pederson and associates \(1987\)](#), utilizing an interview format, confirmed the continuing need for such a supportive network to aid the mother in particular with the ongoing stress of caring for the infant. They reported that husbands, parents, and members of church were viewed as the major sources of ongoing support. Several authors ( [Leifer et al., 1972](#); [Trause and Kramer, 1983](#)) have also investigated the stresses a premature infant places on the marital unit. [Leifer and associates \(1972\)](#) showed that parents of such an infant are at increased risk for divorce; however, this finding was not confirmed by another clinical investigation ( [Boyle et al., 1977](#)). [Trause and Kramer \(1983\)](#) evaluated a middle-income group of parents of premature infants in comparison with a group of parents of healthy full-term infants. They found that the premature birth caused a crisis in the immediate postpartum period, but the stress decreased significantly after discharge home. With time, parents of preterm infants reported themselves to be "more attuned to each other" than parents of full-term infants. They also found that mothers in both groups consistently rated themselves as more distressed than fathers.

Blackburn and Lowen examined the impact of premature birth on grandparents (1986). Referring to the grandparents as the "forgotten grievers." In a retrospective exploratory study, they ascertained that grandparents experienced intense stress as well as parents. The stress was exacerbated by the fact that the grandparents, lacking direct access to physicians, were forced to rely on the distressed parents as their sources of information about the infant. In a study of mothers of preterm versus full-term infants, [Crnic and associates \(1983\)](#) confirmed the positive effects of social network support on lowering overall stress and increasing positive parenting interactive behaviors. [Beckman and Pokorni \(1988\)](#) recently reported that stresses related to preterm birth are specific but also change over time. They found that lowered levels of stress were associated with the continuity of informal, intimate environmental support. Although of significant clinical interest, the majority of these studies of network support suffer from small sample size and lack of adequate control groups.

In addition to the stressors of the nursery experience itself, there are the psychological effects of delivering an infant before term. During pregnancy, parents undergo a complex psychological reorganization as they form expectations and fantasies about their growing infant ( [Leckman et al., 1999](#)). They prepare themselves, and late in the pregnancy their physical surroundings, to meet their new infant. For mothers, these processes take vivid shape as fetal movement becomes more regular and active. Preterm births interrupt these normal developmental processes and for the very preterm infant, the birth may occur just a few weeks after the mother begins to appreciate movement. Parents' expectations and fantasies about when their baby will arrive and about a normal delivery with a healthy, full-term infant are shattered by a preterm delivery. As they adjust to the disappointment, they must also face their fears for the infant's life and reorient their views of their future and the infant's future ( [Pedersson et al., 1987](#); [Zeanah et al., 1984](#)). There is also the added stress and disappointment that they cannot care for their new baby on their own, and they are often, at least acutely, separated from their infant as he or she is moved to another hospital with the appropriate level of technical support needed to care for the infant. Instead of imagining joyfully bringing their infant home to friends and families, they are confronted with the uncertainty of a prolonged hospitalization, potentially fatal complications, or at least compromising sequelae and are often left isolated from their usual social supports ( [Jeffcoate et al., 1979](#)).

When the time comes for the preterm baby to go home, parents who for weeks or months have watched professionals care for their infant may be frightened and intimidated by the responsibility of caring for an infant who still seems fragile and vulnerable ( [Mayes, 1995](#)). Families are abruptly isolated from the psychological support of hospital staff and other families. Although many nurseries now make ongoing parent groups available after discharge, more systematic study of the effectiveness of these types of interventions and involvement of child psychiatrists are needed. [Minde and colleagues \(1983\)](#) demonstrated that mothers of preterm infants who participated in a self-help group were more comfortable encouraging autonomy and sociability in their infants and overall more confident in their caregiving roles.

It has often been observed that mothers of preterm infants express continued anxiety and low confidence in their caregiving competence during at least the first year of their infant's life ( [Corter and Minde, 1987](#); [Crnic et al., 1983](#)). Preterm infants are often difficult to care for and many of the apparent alterations in parenting behaviors seen among parents of preterm infants may reflect the preterm infant's altered state regulatory patterns and potentially slower information processing times ( [Mayes, 1995](#)). Limits in the perceptual and cognitive skills of preterm infants may also modify the impact of parent behaviors on the preterm infant. [Field \(1982\)](#) conceptualized the preterm infant as having a relatively high threshold to adult social stimulation coupled with a low tolerance for stimulation. In response to the differences in stimulation tolerance and perceptual and cognitive skills, mothers of preterm infants appear to take a more active role than mothers of term infants, indeed almost to appear overstimulating ( [Minde, 1993](#)). They direct attention more, hold and touch more, and provide more tactile and kinesthetic stimulation ( [Barnard et al., 1984](#); [Crnic et al., 1983](#); [Field, 1979](#)). These strategies in parental activity increase the amount of mutual gaze between mother and infant and, in effect, structure the infant's attention ( [Goldberg and DiVitto, 1995](#)). In turn, differences in parental activity also influence preterm infants' performances on novelty preference and exploration tasks ( [Landry, 1986](#); [Landry et al., 1986](#); [Rose, 1980](#); [Ruff, 1986](#)).

### **Child Psychiatric Consultation in the Intensive Care Unit**

In the literature there is overwhelming agreement among authors about the type and frequency of psychosocial intervention that are most constructive for the families of premature infants. Liberal access to the babies by family members is desirable, particularly in light of technological advances in neonatal intensive care units that otherwise tend to isolate babies for extended periods of time. [Harper and colleagues \(1976\)](#), in a retrospective questionnaire format study of 58 families, determined that perceived parental anxiety correlated with the degree of the infant's illness. However, despite the associated anxiety the parents felt at the time, parents did maintain much contact with the infants and would have been opposed to more restricted contact. They noted that even parents of babies who died expressed no regrets about the time spent with the infant in the intensive care unit.

Child psychiatric involvement in the intensive care nursery (or, for that matter, on a pediatric inpatient service for infants and toddlers) may occur on at least three levels ( [Mayes, 1995](#)). The most traditional is direct involvement with the infant and his or her parents. The second involves consultation to the staff of a nursery or medical unit who are involved in the care of that infant and family. The third level consists of more general and long-term consultation to the nursery to effect changes in nursery policy and standards of care that directly and indirectly impact on parents and families. These changes may consist of interventions for families (e.g., assigning a mental health professional to each family on admission to the nursery), alterations in nursery routines in ways potentially more appropriate for the infant's developmental needs (e.g., alterations in the amount of sensory stimulation), and organizing parent groups for families during their nursery stay. The work in each of these areas may be done by a child psychiatrist who is a regular part of the nursery team (a model available in only a few nurseries) or a clinician who has special training in issues of infancy and preschool-aged children and regularly does consultation-liaison work with pediatric wards and nurseries. The more the clinician is familiar with nursery routines, knows the staff, and is a familiar presence in the nursery environment, the more likely it is not only that he or she will be consulted but also that the consultative work will be effective. Each intensive care nursery has its own individual ecology and structure that markedly influences how families and staff interact. Understanding these individual system differences is critical for understanding how best to help families and medical personnel. Participating in nursery activities may include joining in regular medical rounds, staff rounds, or psychosocial rounds, or offering regular in-service conferences. Also, the more mental health clinicians are present on a day-to-day basis in the nursery, the more likely they will be to see families coming at unusual or irregular hours and the more comfortable families will feel in approaching the clinician.

Direct work with infants and their families in the intensive care nursery is usually requested because of concerns about the parent's reaction or adjustment to the infant's condition. Rarely do parents themselves request such consultation. More often, staff or concerned family physicians request additional support for the family. Child psychiatrists are able to evaluate the severity of parental depression and limitation owing to anxiety that may be both acute and chronic and develop a therapeutic plan before the infant is discharged to the parent's care. For parents with preexisting affective disorders, this is particularly critical given the increased stress created when preterm infants are discharged from the hospital.

Child psychiatric consultation in the nursery may involve several different approaches. Most often in the acute phases of the preterm infant's nursery course, neurodevelopmental assessments are neither indicated nor warranted. But the child psychiatrist's appreciation of the level of neurodevelopmental competency of a preterm infant at a given gestational age may be used as an intervention with the parents. For example, knowing that infants as young as 32 weeks respond differentially to voices or touch may be used to help parents feel like they can contribute something to their infant's care, and that their presence is important. Interventions such as these grounded in understanding of early neurologic integrity may also be the beginning of helping parents start to build an internal view of their infant as a person with awareness and an individual identity. Additionally, as already outlined, parents with a preterm infant have had an essential developmental process for their role as a parent abruptly interrupted. Child psychiatrists working with families bring an appreciation of the psychobiologic importance for parents of the third trimester of pregnancy, which they will not experience with a preterm birth. Because many parents who give birth to premature babies have had previous miscarriages or other serious obstetric complications, they may be especially sensitized to the threat of loss of their infant. They may experience acute depression and withdrawal that requires intensive individual intervention for one parent or both. The child psychiatrist also is able to help parents understand their feelings and actions and help them feel less out of control.

Child psychiatrists also may be asked to consult about the older siblings in the family whose lives are acutely affected by the loss of their parents, when one or both



are in the nursery, and by the disruption in daily routines. Siblings have recently been permitted as visitors in a number of nurseries ( [Ballard et al., 1984](#)), but their reactions to the disruption in the family have been less systematically studied. Younger siblings may experience acute changes in eating or sleeping patterns, withdraw, or become more irritable and difficult. Older children may have more trouble in school, resist leaving their parents, and also withdraw from peer groups or usual activities.

Child psychiatric consultations to the nursery staff may also be about and for the parents. Nursery staff may be helped to understand an individual parent's response that they have found anxiety provoking or excessively frustrating. For example, the parent who requests to know the exact respirator settings, values of the last blood oxygen level, and the amount of fluid intake is likely attempting to maintain a form of control over a debilitating sense of uncertainty and the unknown. But such strategies often run counter to the routines of any medical unit and an implicit philosophy of protecting parents from the weight of technical details. Similarly, the parent who anxiously refuses to touch or hold the baby may also make nursery personnel uncomfortable and worried about the parent's capacity to care for the baby. The staff's insistent efforts to bring the parent and baby closer may be met with increasing resistance and perhaps avoidance of the nursery altogether. In these instances, the child psychiatrist's initial task is not to help the parent be in contact with the baby but rather to understand what the baby means to the parent and what he or she imagines it will be like to care for the infant. The essential challenge of this type of individual work with parents of infants in an intensive care nursery is that it has to be done in an atmosphere that conveys time for reflection against the backdrop of the urgency and life-and-death nature of the nursery. The child psychiatrist may begin his or her interview with a parent of an infant while standing by the infant's bed. New admissions and emergencies may be going on around the family and clinician and become also the context of the interview. It is important that the consulting clinicians be familiar enough with the nursery environment that they are not initially overwhelmed, but also so that they can stay in touch with the experience from the perspective of the parent struggling to accept his or her infant.

Consulting to the staff of the nursery involves not only case management around the work with a particular family but also consultation to the staff around their own reaction to a particular infant. Nursery staffs often develop deep attachments to an infant and family and need support when it is time for the infant to go home. These issues become even more critical when the infant is going home to a high-risk environment such as a single parent or a family affected by substance abuse. In these instances, nursery personnel may feel extraordinarily protective and unable to let the infant go. Careful group work and consultation in the days before discharge may be effective in both allowing the staff to move on and also helping them to facilitate the family's taking over the care of the infant. Also, in high-risk situations, the child psychiatrist can be influential in helping with discharge planning that includes family support services, immediate referrals to early intervention services for the infant, and detailed plans for follow-up developmental assessments.

## **BIRTH DEFECTS AND THE FAMILY**

### **History and Special Considerations**

The birth of a child with congenital abnormalities presents the family with a severe crisis that shares some features of a premature birth but has specific characteristics of its own. Although there is usually less uncertainty about the survival of the child compared to the situation of premature birth, the defective baby's birth is generally associated with mourning over the loss of the expected child ( [Solnit and Stark, 1961](#)). In addition, continuing conflicts surrounding the appearance of the baby, and the difficulties of physical care of the deformed child complicate the family crisis as well. The societal stigma often associated with physically defective children is a continuing issue for the parents and siblings and must be considered in planning psychosocial intervention strategies.

Descriptions of family adaptation to the birth of an infant with congenital abnormalities began appearing in the literature in the 1950s and 1960s. [Schonell and Watts \(1957\)](#) wrote about the effects of a "subnormal child" on the family unit. This early report from Australia, focusing on a heterogeneous group of retarded children, documented the frustration experienced by, and lack of information and psychosocial supports provided to, parents who were attempting to rear their children at home.

In their paper on the crisis of the birth of a child with congenital abnormalities, [Solnit and Stark \(1961\)](#) referred to the acute grief reaction originally described by [Lindemann \(1944\)](#). They referred to the initial shock, denial, and sense of disequilibrium observed in these families. In addition, they utilized a "theoretical approach founded on the psychoanalytic explanation of the process of mourning as applied to the mother's reactions to the birth of a defective child" ( [Solnit and Stark, 1961](#)). Referring to the original descriptions by Freud ( [1957a,b, 1961](#)), and later by [Engel \(1961\)](#), Solnit and Stark explained the mother's psychological reaction as relating to the loss of the image of the expected baby. During the pregnancy, the mother's normative anticipatory process of expecting a healthy baby is acutely interrupted by the birth of a child with congenital abnormalities and possibly disabilities. Furthermore, her reaction is influenced by her own past experiences with family members and significant life events. Solnit and Stark suggested that because of the acute crisis, there is no time for the mother to work through the loss of the expected child before there is a demand to bond to the ambivalently viewed new baby. Although the emphasis of these authors was focused on the mother's emotional reactions, their advice was to clarify continuously to both parents the situation and needs of the new infant. They also strongly suggested that the mother view the new child as soon as possible, a step that had often been omitted based on a well-intentioned attempt to spare the mother pain.

### **Family Stress in Relation to Specific Birth Defects**

An early description of the psychosocial sequelae of the birth of a child with birth defects was by [Tisza and Gumpertz \(1962\)](#), writing about infants with cleft palate. They described parents who, after experiencing the initial shock and disbelief components of acute grief, became obsessed with etiology (e.g., genetic linkages, which are associated with guilt). They emphasized the need for early imparting of information about the baby to the parents, preferably by the physicians, who should be available to answer the variety of questions posed. They described occasional pathologic attachment between mother and child, based on the mother's viewing the child as an extension of herself.

Recent studies have studied the adaptation of parents to a disabled infant in relation to the type of disability presented. [Pelchat and associates \(1999a\)](#), utilizing a self-report questionnaire, indicated that parents of infants with Down's syndrome and congenital heart disease reported greater levels of parenting stress and psychological distress than parents of children with cleft lip and palate. They also found mothers to report greater levels of distress overall, but differences across diagnostic groups were similar for mothers and fathers.

In Wales, [Hare and associates \(1966\)](#) studied family stress in relation to an infant born with spina bifida via the use of a prospective interview format. Many of the parents reported that the medical staff did not seem to appreciate their initial worries and concerns. The study pointed to the fact that many mothers were unable to comprehend much of what was explained to them soon after the delivery. The parents also expressed a keen interest in meeting with other parents of children with spina bifida.

[Walker and associates \(1971\)](#), in a prospective questionnaire study of 107 British families of infants with spina bifida, reported the parents' dissatisfaction with the manner in which they were informed about the abnormality. The parents complained of chronic tension in the home, difficulties with siblings, and a sense of isolation because of the arrival of the baby. Walker and associates suggested to physicians that a psychosocial team closely monitor these at-risk families.

Very few investigators have applied a psychoanalytic point of view to the issue of birth defects and parental reaction. [Lax \(1972\)](#), in an anecdotal report of only a few patients, described the mother's "narcissistic trauma" in relation to the arrival of the impaired child. Interestingly, she noted that the severity of the mother's depressive reaction often was unrelated to the magnitude of the birth defect ( [Lax, 1972](#)). Lax emphasized preexisting personality features in the mother as clinical clues to her adaptation and acceptance of an impaired child. She presented a dramatic illustration of the rejection by the mother of a newborn with a large nevus of the cheek: "On seeing the child for the first time, the mother turned away and said: 'She's ugly—it can't be a two-faced monster' (referring to the normal side of the child's face and the deformed one)" ( [Lax, 1972](#), p. 341).

### **Studies of Heterogeneous Defects**

[Johns \(1971\)](#), in an Australian study, performed a prospective 6-month investigation of 12 consecutive infants born with a congenital abnormality. Initially and subsequent reactions of the mothers and family were observed. Johns found that parents of children with more visible or more severe defects were the most anxious and accessible to offers of psychological help. Facially disfiguring lesions were of particular concern to the parents. The author suggested an early but brief announcement to the parents of the abnormal finding, an early statement of the plan to correct the deformity, ready access to genetic counseling, and continuing emotional monitoring and support.

[Drotar and associates \(1975\)](#) reported a large sample of parents of 20 children with a wide range of malformations. The malformations included Down's syndrome, cleft palate, and congenital heart disease. They arranged structured interviews after the birth of the child (elapsed time ranged from 7 days to 5 years). They

emphasized the more adaptive elements in the coping of these parents, which had previously been largely overlooked. Successive stages of parental reactions were outlined by this group as components of a normative pattern; shock; denial (feelings of unreality); a mixture of sadness, anger, and anxiety; eventual adaptation; and reorganization. According to Drotar and colleagues, mixtures of sadness, rejection, hatred, and fears of becoming attached to babies who might die were often seen in the middle phases of coping. The last stage, reorganization, included positive long-term acceptance of the child with mutual support between parents. These investigators emphasized the importance of early physical contact (bonding) with the child, referring to earlier work by [Klaus and Kennell \(1970\)](#). They maintained that the early initiation of a relationship, specifically through physical care, served as a major means of anxiety reduction in the parents.

Recently, in a study of predictors of parental distress after the birth of a child with congenital disabilities (including cerebral palsy, spina bifida, and limb deformities), it was found that families with adaptive distribution of responsibilities, including domestic tasks and unrelated life stressors, were at less risk for dysfunctional distress ([Wiegner and Donders, 2000](#)).

Many of the descriptive studies noted in the preceding, although clinically rich in detail, generally lack relevant control groups and large sample size of subjects.

### Effect on the Marital Relationship and Siblings

Several clinical investigators have studied the significant effects of the arrival of a child with a birth defect on the marital relationship. [Tew and associates \(1974\)](#), utilizing a prospective interview format, were the first researchers to note significant deterioration in the couple bond after the birth of an atypical child. The divorce rate of the index parents (of offspring with spina bifida) was twice that of the control group and the national average (in Wales). Only one-fourth of the index families appeared to be free of marital difficulties; mothers pregnant at the time of the marriage appeared to be at the greatest risk for divorce or separation subsequent to the birth. [Martin \(1975\)](#), in a slightly later study, also documented a high rate of marital breakdown in families of babies with spina bifida.

[Drotar and associates \(1975\)](#) underlined the importance of synchrony between the parents and their reactions. Parents who were viewed to be “out of synch” with each other were more prone to emotional separation coinciding with the stresses on the family of such a child.

[Gath \(1977\)](#) reported on a prospective controlled study of 30 families of infants with Down's syndrome. This author found few differences in the mental or physical health of the two groups of parents, but marital rupture or severe disharmony was found in nine of the families with a child with Down's syndrome and in none of the controls.

Little work has been presented in the literature regarding the specific reactions of siblings to the arrival and presence of a child with birth defects and/or physical handicaps. In a clinically descriptive case study, [Poznanski \(1969\)](#) pointed to the resolution of stress and conflict in the family through displacement onto a “well” sibling. The author pointed to the frequent overinvolvement on the part of the mother with the handicapped child, and her lack of sensitivity to the more subtle needs of the other siblings. According to Poznanski, this constellation of dynamics often led to “behavioral reactions as a consequence of emotional neglect.”

In a more recent longitudinal study, [Fisman and associates \(2000\)](#) reported that of 137 normal siblings of children with Down syndrome, Pervasive Developmental Disorder, and a normal control group, significantly more adjustment problems were found in the sibs of children with Pervasive Developmental Disorder when compared with the others.

Several investigators have examined the respective responses of mothers and fathers in relation to the birth of a disabled child. [Hobdell and Deatrick \(1996\)](#) surveyed mothers and fathers of 69 children with a neural tube defect. Mothers reported a mood state change in the very intense category, from more pessimistic at the time of diagnosis to more optimistic later on. Fathers, although also indicating severe mood change, had somewhat more pessimistic ultimate responses.

[Hunfeld and colleagues \(1999\)](#) assessed the burden on the parents and grief 1 year after having a child with a congenital anomaly. The parents of 27 children with defects who had been admitted for surgical treatment as newborns participated in the study. Results showed that the mothers and fathers showed no significant differences in overall burden and grief; however, mothers reported more personal strain. The authors suggested that foreknowledge from prenatal diagnosis, a low perceived functional health status of the child, and multiple anomalies increased the sense of burden and grief. A perinatal counseling team providing clear and consistent information about the defects, treatment, and prognosis would help to reduce stress and uncertainty, particularly for parents who received prenatal information.

### Intervention and Counseling Techniques

Intervention strategies for counseling families of infants with defects have been discussed in the literature since the 1960s. Focusing on 140 families of infants with Down's syndrome, [Giannini and Goodman \(1963\)](#) emphasized the point that, through immediate crisis intervention counseling, families were better able to participate in decision-making relating to institutional placement outside the home. [Goodman \(1964\)](#), in reference to the same population with Down's syndrome, recommended group counseling for the parents to provide peer support, as well as counseling in the home if possible.

The [British Working Party \(1971\)](#) suggested to clinicians guidelines of communication with the parents of an abnormal child. They recommended that a familiar staff person tell the parents as soon as possible to prevent unfortunate discovery of the problem by the parents. They warned of psychological reactions to be expected, such as anger, projection of blame and guilt onto staff, and, at times, temporary rejection of the infant. A multidisciplinary team approach, including the pediatrician and medical social worker, was suggested for communications with the family during the crisis.

In a description of work with families of children with Down's syndrome, [Golden and Davis \(1974\)](#) emphasized that clinicians should carefully evaluate their own feelings about the child in order to better counsel the family. They found that a surprising number of parents are able to accept raising these children at home if their questions are answered and psychosocial needs met.

[Drotar and associates \(1975\)](#) suggested pediatric management guidelines as a result of their study of parents of children with congenital malformations. Familiarity on the part of the physician with the predictable psychological stages of adaptation to the birth crisis is critical. Furthermore, they stated:

Since the parents' initial shock and disbelief limit the amount of information that they can absorb in regarding their child's condition, information regarding the child's handicap may have to be communicated very clearly and repeated many times ( [Drotar et al., 1975](#), p. 716).

Again, early physical contact with the baby was highly urged in order to minimize the parents' feelings of estrangement.

Drotar and associates recommended that more senior staff physicians assist young house staff officers in intervening with these parents. Family counseling by the physician during the infant's first year of life was strongly urged to maximize the development of the child within the family. Van Riper and associates, in a study of 90 parents of children with Down's syndrome, found that pediatric house officers often avoided interacting with parents of the affected child. They suggested that all involved clinicians need to adopt a more interactional and positive approach to the family ( [Van Riper et al., 1992](#)).

Various clinicians recently have described the positive and preventive effects of early counseling intervention. A British group lately described a questionnaire study to determine anxiety among mothers who had undergone a neonatal screening for occult spinal defects. They concluded that a program of investigation for neonatal abnormalities need not cause excessive parental anxiety; they suggested ongoing psychological monitoring and intervention for the parents of affected infants. An interesting finding was that some of the mothers remained anxious even after receiving normal results ( [Gibson et al., 1997](#)). [Pelchat and associates \(1999b\)](#) described better adaptation among parents of children with congenital disabilities (Down's syndrome, cleft lip and palate) who participated in a psychosocial intervention program compared with those without such intervention ( [Pelchat et al., 1999b](#)).

The child psychiatrist's role in management is critical and requires both an understanding of the psychological reactions of the parents and a willingness to deal with personal responses to these infants.

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#### CASE ILLUSTRATION

Mrs. C., a 32-year-old mother, gave birth to a baby boy with Down's syndrome. This was the first child for the mother, a college professor, and her husband, a 36-year-old attorney. The mother had carefully arranged her work schedule around the planned birth



and expected to resume teaching soon afterward. The father was present for the delivery, which was by natural childbirth. Amniocentesis had not been performed during the pregnancy.

When the baby was born, Mrs. C. noticed the concerned expressions of doctors, nurses, and her husband. Mr. C.'s first instinct was to protect his wife, but he was encouraged by the medical staff to show her the infant. There was medical evidence of an endocardial cushion defect for which surgery would most likely be indicated by age 5.

Mrs. C.'s response was to reject the baby because of his physical appearance, future retardation, and cardiac status. She refused to breast-feed the infant and directed much of her anger toward the nursing staff, whom she felt were forcing her to deal with the child. Although the father expressed concern about the future, he was certain that he preferred to keep the baby at home. Mrs. C. continued to be obsessed with the stigma of having such a child and expressed the desire to place him in an institution because he'd "never be perfect."

A pediatric social worker was enlisted to discuss disposition options with the parents. The child psychiatrist was regularly consulted by the social worker regarding the counseling.

This case underlines several important features and implications of psychosocial support in relation to modern pediatric practice. Because there are too many children born with congenital defects for all to be seen by child psychiatrists, the management of these families is best undertaken by social workers or child life specialists, with psychiatric consultation and intervention as needed.

The families generally experience such psychological reactions as shock, a sense of unreality, denial, sadness, anger, rejection of the baby, and eventually adaptation. It is crucial that the family members work through the mourning process of the loss of the "expected" intact child and be encouraged to involve themselves rapidly in the physical care of the infant. In our society, the family more readily accepts nonvisible remediable physical defects and nonmentally handicapping conditions. On the other hand, facial deformities and retardation syndrome elicit more severe emotional reactions in family members.

Although some families eventually do place defective, particularly retarded, infants in institutional settings, it is generally agreed that the outcome for children with special handicapping conditions is better if they are raised in the home environment.

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## THE FAMILY AND EARLY INFANT DEATH

### History and Medical Considerations

The sudden death of infants has been familiar to clinicians for centuries, indeed since biblical times. Sudden infant death syndrome (SIDS), previously referred to as cot death or crib death, is defined as the sudden death of an infant that is unexpected by history and unexplained by a thorough post mortem examination that includes a complete autopsy, investigation of the scene of death, and review of the medical history ( [Hunt, 2000](#)). An autopsy is essential in all unexpected infant deaths because all known causes need to be investigated, even in the context of a detailed history and scene investigation. Sudden infant death syndrome is the most common cause of postneonatal infant mortality in the United States after congenital anomalies and disorders relating to short gestation and low birth weight. In developed countries, it is the most common cause of postneonatal infant mortality, and accounts for 35% to 55% of infant deaths between 1 month and 1 year of age and about 20% of all deaths in infants discharged from a neonatal intensive care unit. In 1996, about 3,000 infants in the United States died of SIDS, a rate of 0.74/1,000 live births. Among full-term infants, SIDS is rare before 1 month of age, with the peak incidence at 2 to 4 months, and 95% of all cases occurring by 6 months of age ([Hunt, 2000](#)).

Over the past three decades, SIDS has been described more specifically, and its etiology has been aggressively studied. The most common hypothesis to explain SIDS seems to be a brain stem abnormality in cardiorespiratory control, including arousal responsiveness, and perhaps other autonomic controls such as blood pressure and sleep-wake regulation. The post mortem data are consistent with this hypothesis ( [Hunt, 2000](#)). An increased incidence of SIDS has been associated with premature and low birth weight infants, lower socioeconomic status, nonwhite ethnicity, young age of mother, smoking during pregnancy, and male sex of baby ([Hoffman, 1987](#); [Valdes-Dapena, 1980](#)).

Over the years, the hypothesized causes of sudden infant death have been numerous. In the remote past, it was thought to be associated with "overlying" or smothering by the parents (Templeman, 1892). In the 1930s, spasm of the glottis and cardiac failure were suggested ( [Goldbloom and Wigglesworth, 1938](#)). Other theories of causation have included enlarged thymus gland, allergy to cow's milk, suffocation from bedding, infection, and cardiac immaturities ( [Weinstein, 1978](#)). Other research has focused on sleep apnea, enzyme irregularities, and chronic oxygen deficiency ( [Naeye, 1974](#); [Steinschneider, 1975](#)). Newer theories have pertained to brain stem function and metabolism, cardiac predisposition, and possible genetic linkages ( [Giulian et al., 1987](#); [Kinney, 1987](#); [Sadeh et al., 1987](#)). [Beal \(1992\)](#) found a slightly increased incidence of SIDS in siblings, which may be attributable to biological or environmental factors. The definitive etiology remains obscure at present, and researchers ultimately may conclude that the syndrome is related to multiple factors.

The sleeping position of the infant has been implicated most recently in the etiology of SIDS, and in 1994, the American Academy of Pediatrics initiated a program that warned of the dangers of SIDS when infants were placed in prone position (on their stomachs) to sleep as opposed to their backs (supine) ( [Willinger et al., 1994](#)). In the past several years, the decrease in the incidence of SIDS in the United States has been attributed to the change in infant sleep positions and a reduction in bed sharing with parents ( [Thogmartin et al., 2001](#)).

### Psychological Reactions

It has often been noted in the literature that the acute psychological reaction of the family to SIDS death is extremely intense and severe in quality. Most authors refer to Lindemann's (1944) description of normative acute grief reactions as a reference point. These descriptions included various somatic symptoms, guilt secondary to perceived negligence, and anger and hostility directed toward health professionals. Pathologic or "morbid" grief reactions presented by Lindemann and observed among SIDS parents include delayed mourning, overactivity without a sense of loss, alteration in relationship to friends and relatives, furious hostility directed against specific persons, schizophrenia-like symptoms, and agitated depression.

According to [Friedman \(1974\)](#), the intensity of reaction to the loss of the infant is related to the absence of "anticipatory mourning." The parents lack the time to prepare themselves psychologically for the overwhelming event. In addition, the death occurs in otherwise healthy infants, the etiology is unclear, and parents often feel blamed or responsible for the child's death ( [Friedman, 1974](#)).

### Descriptive Studies

[Bluglass \(1981\)](#), in a review of psychosocial aspects of SIDS, stated that few well-controlled prospective studies have been done relating to bereavement, and none at all in relation to SIDS. However, some descriptive studies of family reaction to SIDS have been reported. [Bergman and associates \(1969\)](#) delineated the acute phase of family reactions to the sudden death of the baby. They mentioned feelings of disbelief, anger, helplessness, severe guilt, and loss of meaning of life. Parents felt they were "losing their minds," and there was associated disruption of usual routines. Following the death, they described some of the parents demonstrating denial of the event and experiencing persistent dreams of the child. Hostility to friends and relatives was common as well.

[DeFraun and Ernst \(1978\)](#), in a clinical study of 32 parents of infants who died of SIDS, found through mailed questionnaire responses that SIDS was the most severe crisis the parents had ever encountered. In addition, they found that the majority of parents suffered feelings of personal guilt and other psychological difficulties and that relatives were also affected. An interesting finding of their study was that 60% of the parents who had experienced SIDS had moved from their hometowns within 2½ years of the death. They also reported that behavioral difficulties were found in siblings and that better psychological recovery of the family was associated with higher income.

[Cornwell and associates \(1977\)](#) reported on a small Australian prospective questionnaire study. They stated that three families sought formal psychiatric attention for severe difficulties (e.g., delusional hypochondriasis, severe depression, and extreme anxiety). In addition, marital difficulties were common (one-third of the group), siblings were noted to be overanxious, and "anniversary reactions" were usual. Physical health was reported to have deteriorated, with the emergence of psychosomatic symptoms. They also noted parents' overprotective tendencies toward surviving siblings. There was a preoccupation with subsequent fertility, and thoughts of a "replacement child" were common. The questionnaire studies may be critiqued on the basis of sample bias; specifically, it is possible that the families who replied to the questionnaire were more disturbed and dysfunctional than were the nonrespondents. [Salk \(1971\)](#) discussed the prominent role of guilt in the reaction of SIDS parents, which is exacerbated by the mysterious etiology. In an anecdotal summary, he argued that parents who are prone to self-blame are more likely to do so after SIDS death because of the ambiguity of cause. He underemphasized the role of the physician in helping families to cope with the crisis of SIDS.

[Smialek \(1978\)](#) conducted a 2-year follow-up study involving 351 families, most of which (75%) had lost a baby to SIDS. (The remaining 25% lost infants suddenly owing to other causes.) In addition to the common psychological reactions noted by other clinicians, the author found that certain parents expressed (relief) at the death because of extreme ambivalence about the child. She noted that the stages originally described by [Kübler-Ross \(1969\)](#) in relation to dying patients were useful in understanding the adaptation of parents to sudden infant death.

[Zebal and Woolsey \(1984\)](#) reviewed parent's reactions to sudden infant death and emphasized the relative youth of the parents, for whom this may have been the first death experience. Consequently, the parent may misinterpret normative grief reactions and fear he or she is becoming mentally ill. The authors noted that individuals in the surrounding social network tend to underestimate the intensity and duration of the grief reaction and therefore are less supportive than they might be. Furthermore, police investigations and legal proceedings are usually involved, which heightens the initial stress. Parents are not infrequently accused of child abuse and/or neglect pertaining to the death of the infant. A recent study showed no correlation between death from SIDS and prior reports of the family to child protection agencies ([O'Halloran et al., 1998](#)).

### Family Adaptation

A "long-term adaptation" model with key transitional points was presented by [Zebal and Woolsey \(1984\)](#) in their review. After several days or weeks, the initial shock or denial reactions give way to the acute grief reactions described previously. Six or 8 weeks later, the reality of death is more clear, coinciding with the gradual withdrawal of external supports. Four to 6 months after the death, there is a shift to future-oriented concerns. Significant dates (birthdays, holidays) are associated with anniversary reactions. After 1 to 2 years, consideration of a new pregnancy usually occurs; if a pregnancy occurs, there is heightened anxiety about the vulnerability of the baby to SIDS and other dangers.

A recent Scandinavian study surveying 251 parents after infant death demonstrated that the women received more and continuing emotional support for their loss than the men in the group. The study also found that social support in connection with the death was related to long-term psychological adaptation to some extent ([Thuen, 1997](#)).

Several authors have noted that there are sex differences among the parents regarding their psychological adaptation to SIDS ([Benfield et al., 1978](#); [Cornwell et al., 1977](#)). [Benfield and coworkers \(1978\)](#) reported on 50 parent pairs whose children had died suddenly. They noted that fathers appeared to experience less intense and more accelerated "grief work" because of involvement in practical matters (funeral arrangements, etc.). They postulated differences in societal expectations for men and women regarding expression of grief and mourning. Mandell, in a clinical study of paternal response, described fathers of SIDS infants as driven to "keep busy" through increasing involvement with work; they also demonstrated stoicism and a limited ability to verbalize feelings or ask for support ([Mandell et al., 1980](#)). A recent European study demonstrated that a high proportion of the mothers who had lost infants to SIDS 5 years previously still had higher levels of symptoms compared to a normal control group. In general, the male respondents displayed fewer symptoms and a higher level of subjective adaptation than the females ([Thuen and Schlytter, 1996](#)). A contrasting study of sex differences in mourning in 13 couples rated 6 months after the infant's death showed no significant difference between the mothers and fathers. The study found, moreover, that the correlations between mothers' and fathers' subscales and total scores on a grief scale were low, indicating that parents do not grieve in the same ways or over the same time ([Hunfeld et al., 1996](#)).

### Marital Issues

Regarding the effect on the marital bond, Benfield and associates noted that, in couples where communication was poor, it appeared that critical emotional issues were avoided for extended periods of time ([Benfield et al., 1978](#)). [Zebal and Woolsey \(1984\)](#) noted that the differing styles of adaptation of men and women could lead to difficult interactional cycles in the marriage. There is often misunderstanding of the respective styles of grieving, which often leads to conflict. [Mandell \(1980\)](#) observed that, of his study sample of 28 parent pairs who had lost babies, six marriages had dissolved after the death.

Several authors have referred to the desire of some parents to replace the dead infant with another child. [Cornwell and associates \(1977\)](#) found that most parents they studied seemed to be aware of the psychological risks of replacing a lost child. [Rowe and coworkers \(1978\)](#), in a follow-up study of 26 families who had experienced perinatal death, found that a prolonged grief reaction in the parents was associated with early subsequent pregnancy. The implication of this work is that the necessary grief work had been interrupted by the new pregnancy and birth.

### Effect on Siblings

The effects of SIDS on surviving siblings have been presented often in the literature, although there have been no published long-term longitudinal studies. Most authors highlight the particular difficulties older siblings have in dealing with the infant's death, owing to preexisting rivalrous feelings. [Cain and associates \(1964\)](#) described the sense of responsibility and guilt reactions the older children suffer. They noted distorted views about the concept of death, death phobias, and a subsequent feeling of deprivation experienced by the surviving sibling. [Halpern \(1972\)](#) mentioned the tendency of some mothers to project blame and guilt onto the older sibling, which may lead to behavioral problems. He also described painful anniversary reactions experienced by the family, which affect surviving siblings.

Several authors have presented the frequent occurrence of behavioral difficulties in siblings of SIDS infants, as well as the tendency on the part of the parents to overprotection of the remaining siblings ([Cornwell et al., 1977](#); [Mandell et al., 1988](#)). [Williams \(1981\)](#) studied a group of surviving siblings and noted developmental age-specific differences in response. He found that the children between 6 and 9 years of age showed the least direct expression of their distress. The younger children (preschool age) were confused and angry, and the older children (over 10 years) demonstrated an adult-like pattern of grief. [Gaffney \(1992\)](#) has recently described that well-intentioned attempts to spare the feelings of the siblings often result in increasing isolation and tendencies to fantasize in a maladaptive manner.

[Swoiskin \(1986\)](#) also noted age-specific developmental reactions and cautioned that misleading euphemisms about the baby's death often exacerbated the emotional reactions of the surviving siblings. She also described occasional parental distancing from a sibling, related to idealization of the lost child.

[Burns and colleagues \(1986\)](#), in a questionnaire study of 50 siblings of 43 families, noted that 54% of them experienced extended grief reactions lasting over 1 year. In a recent prospective interview study of 45 surviving sibs of 36 families, [Mandell and colleagues \(1988\)](#) described changes in sleep patterns, social interaction, and parent-child interaction. They noted that these behavioral changes reflected both patterns of adjustment by the child and persistence of parental concerns.

### Effect on Grandparents

In a questionnaire study, [DeFrain and Jakub \(1992\)](#) examined the psychological and social impact of SIDS on surviving grandparents. The loss of the baby was found to be devastating for them as well, with mixed emotional reactions of disbelief, anger, guilt, anxiety, and depression identified. They described difficulties inherent in grieving for the baby, at the same time attempting to console and comfort their own grown children. Bereavement support groups were found to be quite helpful for the grandparents involved.

### Interventions and Counseling Techniques

In the two decades of active recognition of SIDS, much has been written about intervention and management techniques. Most authors have pointed to the tendency of medical staff to avoid dealing actively with the psychosocial issues confronting these families. Referring to the work by [Kennell and colleagues \(1970\)](#), correlating more adaptive mourning with prior physical contact with the infant, the majority of clinical investigators have urged increased visual and physical contact between the parents and the dead infant. Many authors suggest that autopsies be routinely performed, in order to decrease the atmosphere of confusion, ambiguity, and mystery surrounding the death of the infant (Bergman, [1974, 1979](#); [Weinstein, 1978](#)). Any available information should be rapidly transmitted to the family by the medical and nursing staff to minimize tendencies of self-blame or blame of others. [Powell \(1991\)](#) has suggested that providing medical information to the family within the first 3 months after the death leads to more normative grief reactions and acceptance. The vast majority of investigators suggest that the medical team, including active involvement by the physician, participate in ongoing psychosocial follow-up of the affected families.

More specific counseling interventions have been suggested in the literature. To foster mutual support, [Salk \(1971\)](#) recommended regular group meetings of parents of infants who have died of SIDS. [Goldberg \(1992\)](#) has suggested that group parental sessions comprised of recently affected parents and "veterans" are quite effective and should contain a significant educational component regarding recent medical research findings in SIDS. [Smialek \(1978\)](#) outlined a multiple-step counseling model based on a large prospective study of 351 families of infants who died of SIDS. These steps include accepting parents' grief reactions, encouraging ventilation of feelings, clarifying misconceptions about the death, allowing private access to the dead infant, and assisting with autopsy and funeral arrangements. Smialek advised that siblings be permitted to attend the funeral of the infant to encourage their grieving process.

[Zebal and Woolsey \(1984\)](#) and later [Woolsey \(1988\)](#), writing independently, made the point that many families experience a withdrawal of social support several months after the death of the infant. [Buschbacher and Delcampo \(1987\)](#) recently reported that relatives in the extended family network often are unable to provide



necessary support because of their own grieving. Most authors agree that a network of aid should be arranged to be available to the family over an extended period of time, considering that the grieving process for the lost child often takes place over years. The network might include other parents whose infants have died of SIDS, family members, friends, and members of the psychosocial or medical team. Many states provide services through SIDS Information and Counseling Projects and the National SIDS Foundation.

In a critical review of adaptation following perinatal loss, [Zeanah \(1989\)](#) emphasized several important points. He maintained that hospital management of these families, including encouragement to view the deceased baby, has not been based on adequately tested assumptions. He also stated that other specific areas need to be more systematically tested as well, including differing mourning patterns of mothers and fathers, differentiation of grief and mourning during the course of bereavement, and advice-giving about subsequent pregnancies.

[Maclean \(1999\)](#) has described the experiences of a group of parents who lost infants to SIDS and who monitored their subsequent infants at home for signs of apnea. The caregiving experiences were explored in comparison to children with other life-threatening chronic illnesses; in both groups, the condition of "sustained uncertainty" was the continuing psychological challenge to the family.

[Davis and associates \(1989\)](#), in an investigation about subsequent pregnancy, interviewed mothers about the advice they had received from physicians about postponing pregnancy. Significantly, they found that, regardless of the specific advice given, most mothers felt that their individual psychosocial situations had not been considered enough at the time. They noted that physicians and other caregivers may be most effective and helpful by educating parents about the advantages and disadvantages of postponing pregnancy, in order to help them make a more informed decision.

In a recent study, [Leitch and associates \(2000\)](#) assessed demographic data, obstetric history, experience of child loss, pregnancy and delivery complications, infant morbidity, family-child interaction, family support, and the relation of each of these factors to SIDS-related anxiety. They found a strong and significant relationship between previous experience of child loss and SIDS-related anxiety, and concluded that psychological help should be offered preventively to parents who have already experienced the loss of a child and are considering another pregnancy.

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#### CASE ILLUSTRATION

A 19-year-old mother gave birth to a 4½-pound baby girl after a 36-week gestation. This was the mother's second pregnancy; there was a 2½-year-old brother at home at the time of the birth. The baby had been delivered by cesarean section because of premature onset of labor and fetal distress. There had been a possible history of alcohol or drug abuse; the mother tested negative for HIV infection. She had also been a cigarette smoker throughout the pregnancy. Apgar scores were 7 and 6, and the baby was observed for 3 days in the neonatal intensive care unit for labored breathing.

The infant was doing well until age 2 months, when she had several episodes of apnea at home, none requiring resuscitation. After a careful evaluation, it was decided not to place the child on a cardiorespiratory monitor.

At age 5 months, the older brother, now almost 3 years of age, discovered the baby not moving in her crib. He reported this to the mother, who impulsively yelled out "What did you do to her?" The boy had previously demonstrated some behavioral difficulties indicative of sibling rivalry. The baby was rushed to the pediatric emergency room, where she was pronounced dead. Counseling was recommended to the family at the time, but they did not seek help, and little outreach by hospital staff took place.

Several weeks later, the parents brought their son to the pediatric emergency room one evening, complaining that he had a cough and chest congestion that worried them. In the course of the evaluation, mother herself presented vague somatic complaints, stated she felt she was "going crazy" because she saw visions of her daughter choking, and feared she was losing control of her temper with the son. The child psychiatrist was consulted.

It was ascertained that the father was attempting to support his wife, but he indicated that he was in danger of losing his job if he kept attending to the problems at home. Extended family members were unavailable, and the parents felt rather isolated and helpless, particularly in relation to their son's behavioral problems. The child was also demonstrating nightmares and new animal phobias. Counseling was arranged for the child and family by the pediatric social worker, and the primary pediatrician made himself available for continuing support. One year after the death of the infant, all family members were functioning more adaptively, the brother was enrolled in nursery school, and the parents were considering another pregnancy.

#### Comment

This case presents a fairly classic paradigm of the problems SIDS families encounter. The families demonstrate the following general characteristics: The mothers tend to be young, multiparous, of low socioeconomic level, and cigarette smokers. The pregnancies are relatively complicated and are more prone to premature birth. The infants often present with difficulties at birth, which may resolve acutely, are discharged home, and later experience episodes of apnea that presage the sudden death.

The acute psychological reaction to such an event often involves displacement of blame onto a family member, such as an older sibling, or medical staff. Fathers frequently present as somewhat distant, stoic, and relatively unsupportive of the mother's emotional state.

Ideally speaking, follow-up visits should be planned at the time the infant arrives at the emergency room. However, these families are too often inadequately followed, and those without network support often experience delayed emotional sequelae. Typically, well-intentioned friends, family members, and physicians suggest premature replacement of the infant, which may lead to exacerbation of symptoms on the part of the parents. A sibling who feels guilt about the death may present with symptoms. The mothers frequently develop psychosomatic or depressive features.

Immediate psychological intervention is usually necessary; involvement of the father and surviving siblings is critically important. Continuous monitoring by the pediatrician and pediatric social worker is indicated, with psychiatric consultation as needed. Outcome and prognosis depend on premorbid adjustment as well as management by the interdisciplinary team.

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## CONCLUSION

The immediate effect of potential and acute loss of an infant on the nuclear family is the major theme of this chapter. Each condition presents young and relatively inexperienced parents with a crisis for which they have usually had little preparation and no prior education. Furthermore, in our technologically and medically advanced culture, with its overinvestment in physical beauty, high intelligence, and medical expertise, the negative impact of prematurity, disability, and death tends to be exaggerated. It is almost impossible for the average American to comprehend that there are parts of the world where infant morbidity and mortality are daily experiences. Actually, this was, and in a few places still is, true of the United States in the 21st century. Universally, the potential or actual loss of an infant is a severe stressor for the family.

Prematurity represents a state of suspended animation for most parents. Even large premature infants with good prognoses induce anxiety and symbolize potential death and disability. Furthermore, the specter of mental retardation, an anathema in our society, presents parents with a long period of ambiguity and chronic anxiety. During this period, they must be helpless observers rather than active participants. Recent research has indicated that the active involvement of parents in the care of their premature infants can be helpful in alleviating both the guilt and anxiety related to loss and impairment. Even if the ultimate complexities of bonding have yet to be fully delineated, this is a practice and useful approach in the prematurity nursery. Clearly, child psychiatrists have important roles to fulfill both in helping staff members to deal with increased parental participation and in the direct management of family members with intense distress related to their infants' fragility.

Parents whose infants are born with congenital anomalies may face a lifetime of adjustment to a child who places special demands on them. Although some congenital anomalies are not obvious to the culture at large or are surgically remediable, those presenting visible deformities to the world or resulting in mental retardation place special emotional burdens on the parents. This situation usually requires the health care professional to deal with both chronic guilt and significant ambivalence on the part of one or both parents. In addition, considering the social and economic burdens that must be endured, major realistic stressors must be recognized and attended to if the family is to remain a viable unit.

Last, the literature on sudden infant death has been reviewed. Although infant mortality has been a reality throughout human history, the psychological study of families experiencing the death of an infant or child is a very recent endeavor. Mourning, in a secular society, has evolved from being a primary purview of the religious counselor to the legitimate scientific pursuit of the psychological investigator. Counseling efforts are now being introduced and implemented in hospitals and clinics to provide support for families with children who die suddenly at home or in the hospital. Child psychiatrists have important roles to fill at all levels of liaison activity regarding SIDS, be it in direct consultation and service or in training and supervision of other health care professionals.

It is clear that there will never be enough child and adolescent psychiatrists to treat all families of premature, disabled, and deceased infants. Knowledge of normative responses has advanced to the point where basic skills can be utilized by and transmitted to others who can provide basic services. However, there is much to be learned about the short- and long-term sequelae of such stressful situations on individuals and family systems with preexisting psychopathology. For such families, child and adolescent psychiatrists are uniquely suited to play a further role in research and treatment.

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## 96 PSYCHIATRIC ASPECTS OF CANCER IN CHILDHOOD AND ADOLESCENCE

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In the last 15 years, there has been significant progress in understanding the psychiatric and psychosocial aspects of cancer in children and adolescence, an important development given the persistence of childhood cancer as the leading cause of death from disease other than congenital illness ( [Haase et al., 1998](#)). The most common types of childhood cancers include leukemias, lymphomas, neural tumors, and sarcomas; and each has a specific age, sex, and racial distribution ( [Steinherz and Simone, 1998](#)). Pediatric cancers are distinguished from adult cancers in their tissue type, embryonic origin, and presumed causative factors ( [Steinherz and Simone, 1998](#)). Pediatric cancers also have higher survival rates, perhaps in part because of children's ability to tolerate more robust therapy ( [Steinherz and Simone, 1998](#)).

Survival of children is maximized when the children are part of treatment protocols sponsored by national pediatric cooperative groups ( [Haase et al., 1998](#)). The importance of a coordinated multidisciplinary approach that encompasses psychosocial aspects of treatment and survival cannot be overstated. The mortality from pediatric cancer underwent a significant decline between 1975 and 1995 ( [Gloeckler Ries, 1999a,b](#)), with mortality declining overall by 40%. When looking at cancer mortality statistics, it is important to look not only at pooled data, but also at data pertaining to individual cancers and age groups; for example, mortality rates in children 5 to 9 years of age declined most, and the mortality rate from acute lymphocytic leukemia (ALL), which represents about one-third of pediatric malignancies, fell by 50% between 1975 and 1995 ( [Gloeckler Ries, 1999b](#)). The survival rate for children under the age of 20 with ALL is now approximately 80%, Hodgkin's lymphoma is 91%, and non-Hodgkin's lymphoma is 72% ( [Ries et al., 1999](#)). Survival rates for central nervous system (CNS) tumors are considerably less.

Physical sequelae of illness and chemotherapy can include affects on cognition, growth/endocrine systems, liver, cardiac, and kidney function, sexual maturation and function; all can have psychological and cognitive correlates, including effects on academic achievement and social competence ( [Eiser, 1998](#); [Parsons and Brown, 1998](#)). Parsons reviewed studies showing a fourfold increased risk of school-related difficulties in this population ( [Mulhern et al., 1989](#); [Parsons and Brown, 1998](#); [Rubenstein et al., 1990](#); [Williams et al., 1991](#)). She cites one study that found that 50% of children surviving leukemia had academic problems at 5-year follow-up despite normal IQ ( [Deasy-Spinetta and Spinetta, 1980](#); [Parsons and Brown, 1998](#)), with 61% having attentional problems ( [Jannoun and Chessells, 1987](#)). Another study looking at pediatric ALL survivors found that 33% showed impaired cognitive functioning and 28% demonstrated impaired emotional functioning ( [Barr et al., 1993](#)).

Depression seems to be correlated with hospitalization and greater time since diagnosis ( [Mulhern et al., 1992](#)). Importantly, psychosocial functioning *may not* correlate with physical health and vice versa ( [Parsons and Brown, 1998](#)). The earlier the diagnosis and treatment, the greater is the likelihood of subsequent cognitive and behavioral sequelae ( [Eiser, 1998](#)).

Multimodal approaches, according to the American Cancer Society Task Force on Cancer in Children, hopefully will bring the overall cure rate of childhood cancer near 80%. The incidence rate is rising at 1% per year, and the cure rate is increasing at 1.4% yearly ( [Haase et al., 1998](#)), figures that are felt to be predicated on assurances of attention paid to "the physical, emotional, and social quality of life" ( [Haase et al., 1998](#), p. 822). The American Cancer Society organized a quality of life workshop in 1995 to address these issues, which noted that quality of life includes issues such as the family (siblings and parents) and community as well as the individual, with serial assessments over time. Specific aspects of the patient's life that must be included are education, insurability, development, and employment. Potential side effects of antineoplastic medication, including secondary malignancies and effects on growth and cognition, are important aspects to be considered. It was recommended also that the effects of managed care systems on reimbursement for medical costs be examined closely ( [Haase et al., 1998](#), pp. 822–823).

It was estimated that by the year 2000 there would be over 200,000 survivors of childhood cancer, which would represent 1% of the young work force in America ( [Haase et al., 1998](#)), with these children comprising "1% of the cancer incidence but . . . 100% of the future" ( [Haase et al., 1998](#), p. 823). Statistically, the loss of a child's life from cancer represents over 100,000 person-years of life lost annually, whereas survival of each child represents 400,000 person-years ( [Haase et al., 1998](#)). Critical ongoing investigations are necessary to evaluate the following:

1. Pediatric cancer control
2. Issues of cancer survivorship
3. Late effects of treatment
4. Psychosocial outcomes
5. Supportive and transitional care

Research findings have influenced the manner in which children are treated for cancer. As [Redd \(1994\)](#) notes in his summary of advances in psychosocial oncology, for example, an understanding of how central nervous system irradiation for leukemia affects neuropsychological function has informed how radiation treatment and intrathecal chemotherapy are delivered ( [Copeland, 1992](#)). Other major areas of research have been behavioral techniques to reduce distress associated with painful procedures, and the psychosocial effects of cancer treatment on children and their families. As [Redd \(1994\)](#) observed, recent research has demonstrated that cancer treatment does not seem to necessarily increase the risk of psychopathology. He noted specific factors that have promoted recent research in this area, and underscored that this area is amenable to the study of the interactive effect of psychological, social, and medical factors, such as the following:

1. Advances in research methodology (e.g., to assess distress reliably)
2. Increased interest in the study of children with cancer among professionals not otherwise associated with cancer
3. Wide availability of professional outlets for research findings
4. Support for psychosocial research by departments of pediatric oncology at major medical centers
5. Patient and parental cooperation
6. Efforts made by the American Cancer Society (ACS) and the National Institutes of Health (NIH) to support research and research training in psychosocial oncology

He also pointed to factors that interfere with continued movement in this direction, including the following:

1. Methods of psychosocial assessment
2. Clinical significance of research findings
3. Availability of appropriate comparison groups

4. Communication of research findings
5. Balance in conceptual focus

For example, although there have been advances in the ability to measure parameters of psychosocial function, less obvious effects may not be discerned. Also, certain research findings may not be clinically relevant. Identification and selection of appropriate comparison groups is a challenge because of the relatively small number of children being treated. There are relatively few researchers in this area, and there is a need for greater liaison with researchers in other fields as well as adequate communication of clinically relevant research results with health care workers primarily responsible for the care of these children. Last, Redd wishes that the focus of clinical work might include identification of strengths and protective factors in these patients as well as psychopathology.

In this chapter, a review of recent clinical and research findings is summarized, including the following:

1. Psychosocial impact of cancer
2. Assessment instruments
3. Individual psychological factors affecting adjustment and outcome
4. Family factors influencing adjustment and outcome
5. Pain and distress in the pediatric cancer patient
6. Cognitive sequelae of cancer treatment
7. Posttraumatic symptoms related to cancer treatment
8. Behavioral and psychosocial interventions
9. Psychopharmacology in the pediatric cancer patient
10. Hospice care and the terminal patient

## PSYCHOSOCIAL IMPACT OF CANCER

There has been a greater recent appreciation of the psychological sequelae of surviving cancer in childhood because of increasing survival rates ( [Die-Trill and Stuber, 1998](#)). The long-term effects of cancer survival have potential consequences on multiple parameters of the survivor's life, including growth and development, social relationships, body image, employment, marriage, and vulnerability to anxiety and depression ( [Hill and Stuber, 1998](#)). Conflicting reports of the prevalence of psychological problems in this population can be explained to a large degree by methodologic issues involving the definition of terms, samples, and measures used. There is ample evidence of a lasting psychological impact from cancer and its treatment ( [Hill and Stuber, 1998](#)). Locus of control may affect different patients in unique ways, either by combating a sense of helplessness in patients who are aided by feeling more in control of their illness, or absolving patients from the responsibility for the illness if they prefer to believe that doctors or fate determine the outcome ( [Hill and Stuber, 1998](#)). The psychological defenses of denial and repression may be adaptive in this population ( [Hill and Stuber, 1998](#); [Phipps et al., 1995](#); [Worchel et al., 1992](#)).

[Redd \(1994\)](#) and other authors ( [Katz et al., 1988](#)) have pointed out that issues facing children and adolescents who have been treated for cancer include those related to isolation from peers and reentry into school. It is important to understand the differential developmental impact of cancer on the preschooler struggling with issues of mastery of the environment, on the school-age child undergoing physical development, and the adolescent searching for a sense of identity ( [Die-Trill and Stuber, 1998](#)). Children under 6 years of age perhaps are less susceptible to the subsequent development of posttraumatic stress disorder (PTSD) but more vulnerable to the neuropsychological sequelae of CNS irradiation; patients diagnosed in adolescence seem to be more susceptible to adjustment problems ( [Hill and Stuber, 1998](#)).

Psychosexual sequelae of pediatric cancer also have been reviewed recently; they traverse physical, psychological, and interpersonal domains ( [Woolverton and Ostroff, 1998](#)). Physical sequelae include the impact on the timing (delays) or status of puberty (including the loss of previously acquired pubertal milestones such as fertility, menstruation, libido, erections, and pubic hair) ( [Grant and Demetriou, 1988](#); [Woolverton and Ostroff, 1998](#)). Sperm-banking in males, although quite ethically compelling, can pose unique stresses for the adolescent. Psychological effects include those on autonomy, one's concept of the future, and sexual self-concept (one's sense of identity related to physical attractiveness and sexual function). Effects on the latter are easily neglected by those involved in the care of the pediatric cancer patient. Effects on interpersonal aspects of psychosexual development proceed from the impact on peer group affiliation and dating caused by the isolating effects of cancer and its treatment. The social impact of cancer has both vertical (adult) and horizontal (peer group) impact ( [Woolverton and Ostroff, 1998](#)). Survivors experience the unique stress of cancer's differential effects on maturity, because they often have a precocious sense of empathy, responsibility, and appreciation for life, but delayed social and sexual development.

Effects on oral intake are common, both secondary to illness and chemotherapy, and as well can be ways in which the child or adolescent attempts to maintain some sense of control. The response to the physical limitations imposed by procedures, intravenous lines, feeding lines, and so on, exacerbate those resulting from pain, surgery, nausea, and vomiting, and can both interfere with development in the younger child or infant, as well as cause psychological regression and social isolation ( [Die-Trill and Stuber, 1998](#)). There seem to be developmental differences; younger children experience more social anxiety and shyness, and teenagers experience more social isolation, although not necessarily depression. Research indicates that social support is a crucial element in the adjustment of this latter group ( [Katz and Varni, 1993](#); [Katz et al., 1992](#); [Noll et al., 1993](#); [Varni et al., 1993, 1994, 1997](#)).

Although children with cancer often may return to school early in the outpatient treatment phase, they frequently experience fears about not being able to catch up with work, and how peers or school staff will react to their illness and its physical manifestations ( [Die-Trill and Stuber, 1998](#)). High rates of school absence are reported. Four groupings of school difficulties are noted in these patients:

1. Patients exhibit school anxiety secondary to the illness or its treatment-induced side effects (hair loss, weight changes, nausea and vomiting, surgical disfigurement).
2. Patients, parents, and schools have difficulty reintegrating the patients into school after prolonged absences.
3. Patients have illness-related learning disabilities requiring psychological evaluation and possible school arrangements.
4. Newly diagnosed patients require preventive intervention and guidance to be reintegrated.

Social stressors for pediatric cancer patients also include separation from peers, functional changes in physical ability, and responses from peers and adults, which can lead to diminished self-esteem ( [Die-Trill and Stuber, 1998](#)). One study found that children receiving social skills training noted higher levels of support from teachers and peers at 9 months following diagnosis compared with before treatment, and their parents noted a decrease in both internalizing and externalizing problems, along with improvements in school competence, during this interval ( [Varni et al., 1993](#)). [Varni and associates \(1994\)](#) noted that support from teachers, classmates, and friends correlated with lower psychological distress and higher self-esteem. Peer support has the greatest impact, which emphasizes the importance of the social network at school. In a later study, [Varni and Katz \(1997\)](#) found that "perceived stress" and social supports have significant impact, particularly at 9 months following diagnosis.

The importance of an orientation that looks at "transitions" in the life of the pediatric cancer patient has been underscored by an American Cancer Society Workshop on Quality of Life in Children's Cancer ( [MacLean et al., 1996](#)). These transitions include moving from active treatment into completion of a treatment protocol and from there to long-term survival, changes in health care providers associated with moving from pediatrics into adult medicine, and changes in the place of treatment delivery ( [MacLean et al., 1996](#)). Transitions often involve significant stress and anxiety because of accompanying changes in procedures. The psychological importance of acknowledging what such transitions mean to pediatric patients and their families is an important part of medical management. Obstacles to successfully negotiating these transitions can include the failure of such acknowledgment along with other factors such as lack of financial resources, inadequate education for health care providers, insufficient involvement of parents, or "unevenness" of care, such that psychosocial interventions are underrepresented ( [MacLean et al., 1996](#)).

Redd (1974) reviewed research suggesting that parents often have greater adjustment problems than their children, with 30% of parents experiencing depressive symptoms ( [Manne et al., 1993](#)) in the phase following diagnosis, and children expressing more context-specific distress around procedures ( [Manne et al., 1992, 1993](#)). One-fourth of husbands and wives report increased marital discord, related to individual distress, and differences between spouses with regard to anxiety or coping style ( [Dahlquist et al., 1993](#); [Katz et al., 1992](#)).

Elementary school-age children have a limited understanding of what causes cancer, less so than their comprehension of what causes colds, although the understanding of what causes cancer seems to increase with grade level. Misconceptions that casual contact and contagion are important etiologic factors persist into



preadolescence, underscoring the importance of education ([Chin et al., 1998](#)). This has obvious implications for both the child diagnosed with cancer, as well as the education of his or her peers.

Interventions designed to address these issues (see the following) must target the psychosocial, psychological, and neuropsychological sequelae of cancer and its treatment ([Hill and Stuber, 1998](#)).

## ASSESSMENT INSTRUMENTS

Quality of life in pediatric cancer patients has received increased scrutiny, and is being studied as part of Phase III Clinical Trials in order to better evaluate outcome ([Bradlyn et al., 1995](#); [Seid et al., 1999](#); [Varni et al., 1998a,b, 1999a,b](#)), although overall it remains understudied, perhaps largely because of methodologic challenges ([Parsons and Brown, 1998](#)). Important parameters to take into account in doing such research include the manner and validity in which such information is obtained (ideally from the child, teachers, parents, and health care providers), developmental factors, timing of the assessment, sensitivity and specificity of the instrument, importance of measuring function against reasonable norms for a given patient, and need to study different domains of functioning (academic, social, and family).

Unfortunately, earlier research has tended to focus on studying individual deficits or parameters of function ([Parsons and Brown, 1998](#)). Domains that are assessed must be altered from those used to evaluate adult quality of life, to account for the relative dependence of children on their parents, the emotional life of children, and the ability to modify assessment to account for longitudinal changes associated with development ([Bradlyn et al., 1995](#); [Parsons and Brown, 1998](#)). The lack of clarity regarding proxy assessment, especially in young children, has been identified as a major challenge in doing quality of life research, as is the importance of having adolescent patients treated by pediatric rather than adult oncologists, because of their close familiarity with developmental issues particular to adolescents ([Bradlyn et al., 1995](#)). A more recent instrument, the Pediatric Cancer Quality of Life Inventory (PCQL) ([Seid et al., 1999](#); [Varni et al., 1998a,b, 1999a,b](#)), has included disease-specific symptom modules (e.g., pain and nausea) and treatment-specific modules; patient and parent concordance on these measures is high. Other measures of quality of life also have been developed ([Feeny et al., 1998](#)).

Parsons notes that objective measures involving clinical status or academic performance should be used whenever possible. Significant variability in assessment of children among respondents is an important factor, a phenomenon mitigated especially by family dynamics, necessitating multiple informants and self-report ([Parsons and Brown, 1998](#)). Chronic illness necessitates longitudinal as opposed to cross-sectional assessments in order to be sensitive to long-term consequences of cancer and its treatment. The knowledge gained from studying long-term outcome can be used to help direct health care policy, given the fact that childhood cancer survivors live an average of seven decades following "cure," and continue to be affected by the psychological, physical, and cognitive effects of cancer and its treatment ([Parsons and Brown, 1998](#)), although they are outside the domain of the medical system. Therefore, it is vital to extend the scope of "cure" and assessment of patient needs beyond that of medical treatment and include the rest of life.

Body image instruments (Kopel et al., 1998; Mulhern, 1999) have been developed to measure this parameter in adolescents and young adults with cancer, and may be useful in screening adjustment difficulties that emerge from body image disturbance. Die-Trill and Stuber assess that

weight changes, hair loss, amputations, placement of catheters to facilitate treatment administration, surgical scars, and alterations in skin coloration and texture not only make the child feel different from peers, but may represent frightening changes in the body to the child and may impact self-esteem adversely. Fear that the body will never return to its original appearance, fears of not being recognized by others, or of being mistaken for an individual of the opposite sex (frequently experienced by adolescent girls who lose their hair) often lead to shame, social isolation, and regressive behavior ([Die-Trill and Stuber, 1998](#), p. 900).

Psychosocial assessment at the time of initial diagnosis should include an assessment of various domains, including development, socioeconomic and cultural issues, coping capacity, family cohesion and communication, and personal and family history ([Adams-Greenly, 1991](#)). [Feeny and associates \(1998\)](#) developed a tool designed to systematically assess health status in survivors of childhood cancer 7 years old and older that used seven attributes—including senses, mobility, emotion, cognition, self-care, pain, and fertility. This tool was used later in a study by [Billson and Walker \(1994\)](#). In their study of children who had completed treatment for cancer, 30% of the parents' and 40% of the doctors' assessments identified no deficits in any attributes. The attributes most often disagreed on between doctor and patient/parents were those of pain and emotion. Doctors generally noted fewer deficits in all categories compared with patient/parents, suggesting that doctors unfamiliar with the patient might underreport problems, and/or that parents might overreport them. The survivors of neuroaxial tumors tended to have the worst health status outcome of the 48 assessment pairs, although 66% of patient/parents reported being satisfied with their or their child's life overall.

## INDIVIDUAL FACTORS AFFECTING PSYCHOLOGICAL OUTCOME

Factors such as the effect of differing cognitive orientations toward achievement on response to cancer treatment ([Elkin et al., 1998](#)), as well as the effects of body image on social adjustment have been studied ([Pendley et al., 1997](#)). The latter study found that the effects of negative feelings about one's body or low self-worth as social anxiety may not manifest until several years after treatment is completed. In another study, avoidance coping, depressive attributional style, and social competence predicted almost half the variance in child depression. Anxiety in children was predicted by a depressive attributional style, parental anxiety, and socioeconomic status ([Frank et al., 1997](#)); based on these findings, interventions should foster more positive attributional and assertive styles, as well as support from friends and training in social skills.

Children who *both* perform more self-care *and* receive more dependent care from their mothers were found to have higher self-concept scores in one study ([Mosher and Moore, 1998](#)). One review examined the construct of the "sense of self" in children who survive cancer ([Woodgate and McClement, 1997](#)), encouraging an analysis of the child from multiple points of view of, including the following (borrowing from William James):

1. How the child defines himself or herself (the "Me-self")
2. The child as the subject, who experiences and organizes experience (the "I-self" or object)
3. The child within a social context (how the self mirrors the reactions of others)
4. The child in relation to feelings about himself or herself (self-reflection)
5. The child within a cultural context
6. The child in a self-evaluation context (self-concept, or how the child distinguishes himself or herself from others)
7. The child's self-esteem; that is, how the child feels about himself or herself, such as self-worth or feelings about competence

This construct is relevant to an understanding of how the child with cancer experiences himself or herself: How does the child conceptualize his or her illness? For example, does the child anticipate recovery?

Studies have suggested that adolescents with cancer attempt to maintain a sense of normalcy in their appearance, school attendance, and social connections ([Woodgate and McClement, 1997](#)). Adolescents may try to maintain a separation between the world of illness (cancer) and normalcy as a way of sustaining the integrity of their sense of self, although at the same time they can feel "different" because of the uniqueness of their experience and fortitude ([Rechner, 1990](#); [Woodgate and McClement, 1997](#)). Self-worth can positively influence affect, which in turn is related to the extent to which emotional reserves can be mobilized to allow the child to participate in life's activities. Self-esteem and self-confidence are believed to correlate with persistence and flexibility when faced with adversity ([Rutter, 1985](#)). Little research is available regarding how surviving cancer affects children's self-concepts ([Woodgate and McClement, 1997](#)). [Koocher and colleagues \(1980\)](#) found that developing an understanding of their experience and themselves related to successful coping strategies in three patients. Incorporating some assessment of the patient's sense of self into the evaluation of a child or adolescent newly diagnosed with cancer thus may be quite useful in developing an understanding of the patient's experience ([Damon and Hart, 1982](#); [Woodgate and McClement, 1997](#)). [Woodgate and McClement \(1997\)](#) reviewed [Damon and Hart's \(1982\)](#) interview techniques, which consist of: self-as-object items involve examining self-definition, self-evaluation, self in past and future, and self-interest and include questions such as the following:

1. What are you like?
2. What are you not like?
3. What do you like most about yourself?
4. What do you like least about yourself?
5. Do you think you'll be the same or different 5 years from now?
6. How about when you are an adult?

## 7. What do you want to be like?

Self-as-subject items involve examining aspects of continuity, agency, and distinctness, and include questions such as the following:

1. Do you change at all from year to year?
2. How did you get to be the way you are?
3. How did that make you the kind of person you are?
4. What makes you different from anyone you know? ([Damon and Hart, 1982](#); [Woodgate and McClement, 1997](#))

Cognitive and behavioral strategies designed to enhance self-worth and self-esteem can be targeted more accurately from such an understanding of the factors impacting on the child or adolescent's sense of self ([Woodgate and McClement, 1997](#)). Play and drawings must be used as tools for eliciting the same information in younger children. [Varni and coworkers \(1995\)](#) systematically studied this phenomenon. They found that perceived physical appearance in children with cancer had direct and indirect effects on depression and social anxiety, with the indirect effects proceeding from self-esteem. Perceptions of athletic, social, and academic competence were found to attenuate the effects of perceived body image on self-esteem to some degree. These findings highlight the importance of interventions such as those described by Varni in other papers that address social functioning and support ([Varni et al., 1994](#)), and cognitive interventions that might successfully address negative feelings about physical appearance.

## FAMILY FACTORS INFLUENCING ADJUSTMENT AND OUTCOME

Research has explored the effect of family factors on the psychological morbidity of cancer in children. This has been examined from a social-ecological perspective ([Shapiro et al., 1998](#)) in mothers of children diagnosed with leukemia, where both intrapsychic factors (caregiver adaptation as reflected by maternal depression and sense of burden) and instrumental adaptation (reflected by maternal ability to function successfully within the health care system) were affected by positive relationships in the family. Mothers who noted less depression and burden were more likely to report improved relationships and communication with their spouses; mothers noting more positive doctor–parent relationships also reported better relationships with both spouse and child. Others also have looked at adjustment and coping by parents of children with cancer ([Grootenhuis and Last, 1997](#)), examining psychological distress, marital distress, and family functioning, as well as the use of coping strategies such as social support, communication, search for meaning, and factors that affect adjustment.

Parents commonly experience feelings of isolation, incompetence, and self-blame. [Die-Trill and Stuber \(1998\)](#) reviewed two models of parental response to a diagnosis of cancer, one of which describes stages including the anxiety and disorganization of the impact of the diagnosis, denial, grief, focusing attention on the situation, and closure, which is marked by acceptance and adjustment to the ramifications of the illness on the family ([Fortier and Wanlass, 1984](#)). A second model frames potential response patterns of chronic sorrow ([Buschmann, 1988](#)), and is characterized by profound feelings of losing the “complete” child and his or her hopes over the long-term.

There have been conflicting reports on the specific effects of pediatric cancer on family functioning because of the lack of methodologic consistency in studies ([Die-Trill and Stuber, 1998](#)). Studies vary as to whether the child was on or off treatment, prospectively or retrospectively, or was involved in a specific phase of treatment (initial, inpatient, outpatient, or during remission), rather than longitudinally. It appears that preexisting family stressors—parental psychiatric illness, child neglect, divorce or marital conflict, conflict between parent and child, cultural and language barriers, and multiple losses within the family—put families at risk for adjustment difficulties ([Brown et al., 1992, 1993](#)). Acute emotional disturbance during the initial phase of diagnosis, including intense separation anxiety, sleep disturbance, and obsessive-compulsive symptoms, have been described in over 50% of parents, with the persistence of anxiety and depression 8 months later during remission, and sleep problems and depression 20 months later ([Magni et al., 1986](#)).

Positive parental adjustment has been found to correlate with marital satisfaction, family support, open communication, less stressors, family expressiveness, and a greater moral or religious emphasis ([Barbarin and Chesler, 1986](#); [Cassileth et al., 1985](#); [Die-Trill and Stuber, 1998](#); [Kupst et al., 1984](#); Spiegel et al., 1983). Siblings also can be specifically affected and experience ambivalent feelings, including intense fears about their ill sister or brother. Siblings also may develop feelings of abandonment and resentment because parental attention is focused on the ill sibling, with consequent feelings of guilt because of their anger or being the well child. Siblings may harbor shameful feelings about the appearance of their ill brother or sister, or develop somatic preoccupations ([Die-Trill and Stuber, 1998](#)).

[Manne and Miller \(1998\)](#) found that perceived conflict between adolescents and their mothers was associated with psychological distress; the magnitude of physical impairment accounted for most of the variance, supporting interventions that address this relationship. [Sahler and associates \(1997\)](#) found that mothers with dysfunctional siblings of a child with cancer displayed the lowest levels of well-being and were more likely to look for professional services, and least likely to benefit from social support, indicating that specific problem-solving training for mothers was likely to be more effective. [Hoekstra-Weebers and colleagues \(1999\)](#) found that trait anxiety, considered to be a personality factor, predicted risk most robustly for maladjustment to having a child with cancer, and social support seeking by fathers tended to mitigate risk. With regard to mothers, the experience of positive events that preceded the child's diagnosis and assertiveness seemed to reduce the risk of maladjustment. [Sawyer and coworkers \(1997\)](#) prospectively studied children aged 2 to 5 who were diagnosed with cancer and their families, and reported that considerable distress was reported in the period immediately following diagnosis, although this effect became attenuated during the year following diagnosis, approaching levels of problems seen in the general community. In a later paper, [Sawyer and coworkers \(1997\)](#) reported that maternal adjustment following the diagnosis of childhood cancer predicted the child's adjustment 2 years later; it was hypothesized that impaired maternal functioning from maladjustment might impact on the delivery of care for their children.

[Enskar and colleagues \(1997\)](#) described eight categories of problems faced by the families in their sample of 15 children and adolescents with cancer, including: (a) feelings of powerlessness associated with the child's suffering or reaction to the disease or treatment; (b) having their lives governed by the child's disease (e.g., with respect to work and finances); (c) shifting family dynamics, including the lack of privacy or integrity in the marital relationship, not devoting enough time to well siblings, and tendency to spoil or overprotect the sick child; (d) significant change in parental self-image governed by a shift in priorities, feelings of despair, sadness, and uncertainty; (e) attempts to cope; (f) dealing with the reactions of others; (g) finding support from others (including immediate family, friends, parents of other ill children, and health care staff); and (h) evaluating the quality of care, with an emphasis on professionalism of staff, organization of the delivery of care, information supplies, and types of equipment available for activities (e.g., VCR).

[Brown and associates \(1993\)](#) found that 34% of mothers with children diagnosed with ALL met the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition, revised (DSM-III-R) ([American Psychiatric Association, 1987](#)), criteria for at least one psychiatric disorder, and that psychopathology in either parent accounted for a major portion of the variance seen in the children's' psychological function. Children with mothers who had a psychiatric diagnosis noted more anxiety symptoms and were more likely to have a maladaptive attributional style; mothers with psychiatric diagnoses reported more internalizing symptoms in their children, although this could have represented biased reporting. The higher rates of psychiatric disorders in mothers in this sample as compared with the community at large seemed not to be explained simply on the basis of a stress response to the child's illness, and may have resulted from higher rates of premorbid disorders. The majority of the children in this sample did not evidence elevated rates of psychopathology, although the children who had parents with psychopathology did have greater adjustment difficulties; the direction of this relationship remained unclear, and the authors supported a multifactorial model including the interactive effects of chronic illness in the child and vulnerability to parental psychopathology. Other research in this area has found that the constructs of family cohesion and expressiveness most positively predicted favorable adjustment in pediatric cancer patients at 6 and 9 months following diagnosis ([Varni et al., 1996c](#)).

Treatment adherence in this population also has been reviewed ([Die-Trill and Stuber, 1998](#)), with 33% of children under 13 years of age and 59% of adolescents found to be noncompliant with oral medication; nonadherence is causally associated with poorer prognosis. Factors predictive of adherence differ by sex, anxiety (in female patients), and anxiety, anger, and obsessive-compulsive behavior (in parents of male patients) associated with greater adherence. Medication intake, number of siblings, and poor-self image also correlate negatively with adherence. Cultural differences also play an important role in adherence, and more intensive treatment, parental depression, and child behavioral problems are predictive for less adherence ([Die-Trill and Stuber, 1998](#)). Paradoxically evidence does not support increased adherence in more serious illnesses; those adolescents at risk seem to have less insight into causality and prognosis, and use denial more defensively ([Die-Trill and Stuber, 1998](#)).

Adherence has been found to relate to family factors ([Manne et al., 1993](#)). Problems with adherence were found to be more common in younger children (especially around procedures such as venipuncture, lumbar puncture, or bone marrow aspiration) and families with lower socioeconomic status (decreased adherence to appointments and the reporting of child's reactions to treatment). A more “supportive” parenting style seemed to be associated with greater adherence, which is perhaps mediated by the child's perception of the parent's receptivity to hearing complaints or concerns voiced by the child, or the parent responding more sensitively to child distress, such as a reluctance to get out of bed on the day of a clinic appointment. Treatment implications include interventions designed to behaviorally



assess and improve parental supportive functions. The success of specific types of behavioral interventions designed to treat noncompliance probably can be optimized by determining the acceptability of these measures by parents and nurses before instituting them ( [Miller et al., 1998](#)); in this study, positive reinforcement and reprimand were tolerable to parents, whereas response cost and time out were not.

## PAIN AND DISTRESS IN THE PEDIATRIC POPULATION

An understanding of the psychological meaning of pain within a developmental and individualized context is crucial to working with pediatric cancer patients, and can be associated with disease, procedures, or treatment ( [Frager and Shapiro, 1998](#)). Pain from disease can be somatic, visceral, or neuropathic in etiology; treatment-related pain includes mucositis, peripheral neuropathy, and obstipation; pain syndromes also can be specific for certain stages of treatment, including pain in the initially diagnosed patient, during relapses in the terminal patient, or chronic pain syndromes following treatment ( [Frager and Shapiro, 1998](#)). Children may minimize or deny pain symptoms owing to associations of pain complaints with procedures or doctor visits, out of fear of worrying their parents, or out of a sense of guilt for somehow being responsible for the pain. Procedural pain in and intense anxiety in anticipation of such procedures in particular is often highlighted as most distressing by these patients, yet this area is often one not given enough attention by health care professionals; this exacerbates the feelings of loss of control that accompanies cancer treatment. Parents also may minimize or deny the extent of their child's pain because it symbolically heralds a more ominous prognosis for the illness, or to protect them from experiencing the impact of their child's pain. Pain can worsen preexisting marital tension and lead to parents blaming each other for the suffering of their child; siblings may experience serious distress too, especially guilt about witnessing their brother or sister suffer ( [Frager and Shapiro, 1998](#)).

Guidelines recommended by [Frager and Shapiro \(1998\)](#) are: (a) including the entire family in intervention for pain; (b) viewing pain as an "emergency;" (c) addressing the physiologic, emotional, and social aspects of pain; (d) facilitating parental involvement in pain management; (e) frequently reassessing pain and the success of interventions; (f) using both pharmacologic and nonpharmacologic strategies; and (g) being vigilant to treatment-associated pain, both in advance and concurrent with treatment. They provide an excellent manual on the assessment and treatment of pain in this population, noting the need for a comprehensive initial pain assessment, with a detailed history and characterization of the pain complaints and coping strategies, and the use of self-report aided by visual analog scales as the "gold standard" ( [Frager and Shapiro, 1998](#)). They stress the importance of issues such as privacy during procedures, trust, and (safe time" when the child knows that procedures will not take place. The presence of an involved parent who can use transitional objects, story-telling, books, or other developmentally appropriate interventions specific to a child's interest can help these children tremendously. Pharmacologic interventions ( [Frager and Shapiro, 1998](#)) include nonsteroidal antiinflammatory drugs, acetaminophen, opioids, or adjuvants such as tricyclic antidepressants or anticonvulsants such as gabapentin, depending on the type of pain (acute versus chronic, neuropathic versus somatic or visceral, localized versus diffuse); topical analgesics include eutectic mixture lidocaine-prilocaine topical anaesthetic (EMLA) cream. An essential principle is that adequate pain management is given as "standing orders," not pro re nata (PRN) as needed. Patient-controlled analgesia is an extremely effective way to achieve around-the-clock pain management. Identification of comorbid conditions (e.g., anxiety, insomnia) can help guide the use of other adjuvant medication such as benzodiazepines.

The American Academy of Pediatrics published guidelines regarding the management of pain associated with procedures in children with cancer ( [Zeltzer et al., 1990](#)). Despite these guidelines, The American Pain Society noted in its 1995 Consensus Statement that pediatric pain continued to be an under treated phenomenon, owing to a lack of education in health care providers, misled concerns about addiction to opiates, state and federal regulations regarding the prescribing of controlled substances, and insurance reimbursement for the same ( [American Pain Society Quality of Care Committee, 1995](#)). In this statement, they reviewed the American Pain Society Guidelines for pain management, which had been established in 1991, and included: recognizing and treating pain promptly (including charting/displaying of pain self-report, identifying outcome variables, and documenting outcome); making information about analgesics readily available; promising patients attentive analgesic care; defining explicit policies for use of advanced analgesic technologies; and examining the process and outcomes of pain management with the goal of continuous improvement ( [American Pain Society, 1991](#)). These guidelines were revised in 1995 ( [American Pain Society, 1995](#)), noting five key elements: (a) assuring that a report of unrelieved pain raises a "red flag" that attracts clinicians' attention; (b) making information about analgesics convenient where orders are written; (c) promising patients responsive analgesic care and urging them to communicate pain; (d) implementing policies and safeguards for the use of modern analgesic technologies; and (e) coordinating and assessing implementation of these measures. The Agency for Health Care Policy and Research also published guidelines for the management of cancer pain (1994), and these guidelines have been applied to children ( [Buchanan et al., 1997](#); [Schmidt et al., 1994](#)).

Other guidelines relating specifically to children also have been published ( [McGrath et al., 1996](#)) that stress the combined modalities of behavioral, physical, and supportive therapies along with appropriate pharmacologic treatment, which should be given as a standing order (not a "PRN") to help children sleep through the night, and orally to avoid painful routes of administration.

Specific articles have been written about the use of local anesthetic agents such as EMLA cream ( [Kapelushnik et al., 1990](#)), such as in reducing pain associated with lumbar puncture. [Varni and associates \(1996a\)](#) described a "Multidimensional Biobehavioral Model of Pediatric Pain," and used this model to look at psychosocial mediators of pain, such as "perceived stress." Perceived stress is a construct that reflects the meaning of day-to-day events that are distressing to children, also referred to as "daily hassles" ( [Varni et al., 1996a](#)). Varni concluded that targeted cognitive-behavioral interventions could be effective in reducing pain, such as "cognitive refocusing" or "stress management techniques (progressive muscle relaxation, meditative breathing, and guided imagery)," would be effective in ameliorating pain, based on his model of "precipitating events" and "intervening variables," which mediate pain perception in children ( [Varni et al., 1996a](#)).

Standardized assessment of pediatric pain also has been an area of scrutiny ( [Varni et al., 1996b](#)). There is some evidence that different phenomenon might be measured depending on the informant (child, parent, or nurse). [Manne's \(1992\)](#) study found that during venipuncture in pediatric cancer patients, when compared with an independent rater, the child's report correlated most with chronologic age, the parents' report was based on their subjective perception of the child's pain, and the nurse's report reflected overt distress. Distress is five to ten times greater in children under 7 years of age ( [Jay et al., 1985](#); [Redd, 1994](#)). Some of this can be attributed to the cognitive level of the child, as the understanding of illness and procedures follows along piagetian lines ( [Perrin and Gerrity, 1981](#)). Young children often feel illness and/or procedures represent punishments for misdeeds, and may attribute malevolent intentions to health care providers, especially in the context of a procedure. A recent review of 13 treatment-outcome studies ( [Powers, 1999](#)) indicated that cognitive-behavioral intervention is a "well-established treatment" for pain accompanying procedures in children.

As [Redd \(1994\)](#) noted, parents may influence their child's emotional state during procedures via a "contagion" model; the child may read nonverbal cues indicating the presence of a threat and become as anxious as his or her parents. This phenomenon could result from a parent's anxiety or depression interfering with his or her support during a procedure, or from the lack of distress reducing skills (i.e., simply trying to reassure the child instead of using distraction or other behavioral techniques). Depending on the developmental level of the child, the parents can play a key role in using cognitive-behavioral interventions, which might include the child blowing on a party blower, the use of video games, guided imagery, or hypnosis, accompanied by positive reinforcement (stars, stickers, etc.) ( [Redd, 1994](#)). Behavioral interventions, including parent coaching, attentional distraction, and positive reinforcement can reduce distress during venipuncture, with less physical restraint needed ( [Manne, 1990](#)). Contingency management, hypnosis, systematic desensitization, modeling, and behavioral rehearsal allow parents to play an important part in these interventions ( [DuHamel et al., 1999](#)). Taken together, the data suggest that the emotional state of parents exerts a powerful effect on the way children's feelings are interpreted, and directly on psychological morbidity in children in and of itself.

Pediatric cancer patients undergo repetitive invasive, frightening, and painful procedures that are often sensitizing (i.e., the child does not necessarily "get used" to the procedure); and there may be resistance, refusal, and anticipatory fear because of cognitive limitations, especially in younger patients. Die-Trill and Stuber have described childhood manifestations of distress:

distress is usually manifested by crying, screaming, requesting emotional support or physical contact, verbal resistance, verbal expression of fear, information seeking, and requesting delays in the administration of the procedure. Common fears include disfigurement, losing all their blood or being overloaded with it during transfusions, and death. Shame for not being able to control oneself is frequently experienced by older children ( [Die-Trill and Stuber, 1998](#), p. 899).

Parents and staff alike may feel overwhelmed and guilty when observing such distress in a child.

## COGNITIVE SEQUELAE OF CANCER TREATMENT

Cancer also can impact on neuropsychological function directly, in the case of brain tumors and metastases, and indirectly, through the effects of infections, fever, medication, and complications ( [Silberfarb and Oxman, 1988](#); [Walch et al., 1998](#)). A significant mediator of the effect caused by brain tumors is younger age at diagnosis and treatment.

Work done in the last decade found that CNS irradiation and intrathecal administration of chemotherapeutic agents (e.g., methotrexate, which is used as prevention against disease spreading to the CNS in leukemia) could have adverse effects on cognitive function, although they improve survival rates dramatically. These effects are most commonly seen in performance skills, arithmetic, perceptual motor skills, visual processing, visual motor integration, sequencing ability, and short-term memory (Redd, 1994), although some researchers have argued that more specific individual neuropsychological batteries might measure effects more specifically and validly than overall global tests such as the Wechsler Intelligence Test for Children (WISC). The size of the effect has many determinants, including the child's age and sex, dose of radiation, size of the area irradiated, and dose schedule (Redd, 1994; Walch et al., 1998). It is notable that adverse effects may not be seen until years following treatment (Redd, 1994; Rubenstein et al., 1990).

## POSTTRAUMATIC SYMPTOMS RELATED TO CANCER TREATMENT

Many articles have been published recently about PTSD in pediatric cancer patients (Barakat et al., 1997; Butler et al., 1996; Hill and Stuber, 1998; Kazak et al., 1997; Manne et al., 1998; Smith et al., 1999; Stuber et al., 1997; Wintgens et al., 1997). This was in part because the stressor criterion was modified in DSM-IV to include life-threatening illnesses (Smith et al., 1999). The incidence of PTSD in pediatric cancer survivors in one study was 14.7% (Barakat et al., 1997), with parents also having significantly elevated PTSD symptoms associated with child symptoms and mediated by perceived life threat and family and social support resources. Stuber and associates (1997, p. 958) found that predictors of PTSD in 437 child and adolescent pediatric cancer survivors included the following:

1. The survivor's retrospective subjective appraisal of life threat at the time of treatment, and the degree to which the survivor experienced the treatment as "hard" or "scary" (This was mediated by the mother's perception of both the threat to life and the severity of treatment.)
2. The child's general level of anxiety
3. A history of other stressful experiences
4. The time since the termination of treatment (negative association)
5. Female gender
6. Family and social support

Stuber noted child-specific PTSD symptoms, including somatic indications such as stomachaches, nightmares, and repetitive play. The majority of survivors experience mild PTSD symptoms, although it was noted that these might not necessarily be maladaptive in all cases. Interestingly, neither exposure itself to life-threatening procedures such as bone marrow transplantation, nor the age of the child, nor the medical sequelae of treatment were predictive of the development of PTSD symptoms in and of themselves. The role of the mother's perception of life threat to the child and her anxiety level were found to be quite significant, which supports the importance of psychological interventions directed toward the mother. The social support finding was interesting from the point of view of how the survivor's perception of an upsetting response on the part of other family members and friends could impact on PTSD symptoms, underscoring the need for further work to help delineate how this effect is mediated, such as through empathy or guilt in the survivor as Stuber and associates (1997) suggest. Interventions during the acute phase of treatment could impact on the child and family's perception of life threat, help mold this view in a realistic but hopeful manner, and offer acute anxiety-reduction interventions for the child (Stuber et al., 1997).

In a literature review of PTSD studies of pediatric cancer patients and their parents, the children and adults involved in studies using a validated PTSD diagnostic tool experienced PTSD symptoms in all but one case (Smith et al., 1999). In one study, 6.2% of mothers of children who survived cancer had PTSD, and an additional 20% had subclinical PTSD (Manne et al., 1998). Twenty-five percent of mothers with PTSD also had an anxiety or depressive disorder. Reviews on this topic have noted research documenting that parents of adolescent survivors of cancer also have a heightened incidence of PTSD (Kazak et al., 1998; Pelcovitz et al., 1996; Stuber et al., 1997); the latter study noted the important predictive power of parental anxiety.

## OTHER BEHAVIORAL AND PSYCHOSOCIAL INTERVENTIONS

Behavioral interventions in this population have been reviewed recently (DuHamel et al., 1998). In addition to the specific interventions suggested in the preceding by research on PTSD and the cognitive-behavioral interventions designed specifically for pain, distraction and relaxation in particular have been found to be effective treatments in this population, especially for chemotherapy side effects such as nausea and vomiting (McQuaid and Nassau, 1999). Psychoeducation has been found to reduce distress levels in parents in this population (Hoekstra-Weebers et al., 1998). The potential positive impact of both individual and group psychotherapy has been reviewed recently (Sourkes, 1998; Woolverton and Ostroff, 1998), both from preventative and posttreatment perspectives. In particular, play therapy with younger children can be valuable in facilitating a sense of mastery and desensitizing children from procedure-related distress. School-based interventions, peer groups, and other interventions to address psychosexual concerns can be valuable for older children and adolescents (Woolverton and Ostroff, 1998).

Up to 29% of pediatric cancer patients receiving chemotherapy have been reported to experience nausea, and 20% develop vomiting in anticipation of chemotherapy, mediated by a classical conditioning model, and correlated with emetic potential of the offending agent. Children with greater anticipatory nausea and vomiting also tend to have parents who reassure and model healthy coping strategies less, and rely more on threats. The response to Ondansetron, an antiemetic agent, in controlling anticipatory nausea and vomiting prior to chemotherapy, is mediated by psychological factors such as expectations of vomiting following chemotherapy. This also implies that psychological intervention addressed at the cognitive component that interplays with the classical conditioning model could be helpful (Tyc et al., 1997).

Clowning has been investigated as a tool in reducing distress in children undergoing invasive medical procedures (Oppenheim et al., 1997). Two pilot studies examined the effect of specially trained clowns on distress in children undergoing medical procedures (Slater et al., 1998). In Study I, 27 6- to 20-year-old heart transplant recipients (mean age = 14.06 years) were observed during three consecutive biopsies during cardiac catheterization. Subjects were randomly assigned to clown versus no-clown conditions during their second visit. Baseline data were collected without the clowns present. Children and their caregivers completed self-report measures of distress and anxiety. Trained observers, caregivers, nurses, and physicians rated child distress. Results show a decrease in observed child distress ( $P = 0.054$ ), and significant decreases in child self-reported distress ( $P = 0.011$ ) and parent-rated child distress ( $P = 0.049$ ) with the clowns present. Doctors found the procedure easier to perform with the clowns present ( $P = 0.011$ ).

Study II was a crossover design, with 32 3- to 18-year-old children (mean age = 10.22 years) in a cancer day hospital who were randomly assigned to exposure to clown treatment at either visit 1 or visit 2 during invasive procedures. Children's distress was rated as in Study I. Results of Study II show no significant difference in child distress with clowns present. Doctors and nurses found procedures easier to perform with clowns present ( $P = 0.066$ ). The presence of specially trained clowns during invasive medical procedures significantly reduced children's distress during cardiac catheterization. Less robust findings in the oncology setting may be owing to the small number and pain-control measures already available in the oncology clinic. Future studies might focus on benefits of clowns with sick children in less frightening medical settings.

Support groups or family conferences for patients, their siblings, and parents, along with educational material disseminated to the patients, families, and schools also can be helpful (Whitsett et al., 1999). One-day programs for families of children with cancer also have been constructed and systematically evaluated, showing that they can reduce levels of anxiety and PTSD symptoms at 6-month follow-up (Kazak et al., 1999).

These issues have been written about in a multicultural context as well, stressing the importance of the relationship between family members and health care workers, as well as the relationship between families as a group and the health care team as a group (Masera et al., 1998). Salient points include the importance of the delivery of adequate psychosocial care, mutual respect, education, regular psychosocial team meetings, dealing with reentry issues following treatment (in school, work, and with friends), liaisons between medical teams, parent associations (which should be organized and directed by parents), and community-based efforts; as well, education must occur on a national and international level to heighten awareness of these issues. The emerging message is clear: Psychosocial interventions for these children and their families are imperative and *do* work (Kazak et al., 1995).

Interventions designed to reduce stress in pediatric oncology staff also have received recent attention (Grootenhuis et al., 1996). In this study, none of the staff underestimated the parents' need for support, which was even higher than the parents' estimated. There were gender effects, with female health care staff rating pain during three medical procedures higher than male staff, and the ratings of staff who had worked longer in oncology being less than those working for a lesser period of time. More experienced staff tended to see the patients in a more positive light, for example, perceiving them as stronger, which may represent a coping strategy for the staff. This study highlights the importance of health care workers being attuned to the evolution of their own attitudes regarding their patients.

The importance of an awareness of psychosocial issues in these children is also relevant to the radiologist (Gunderman, 2000). Recommendations include that



support staff not discuss radiologic findings with patients and families, and that the radiologist set aside time to meet with patients and families, certainly before the procedure or imaging study, to explain what will happen, and answer questions, although the radiologist probably should not be the physician who informs the patient of a diagnosis of cancer. However, the author makes the point that questions should be answered directly (in a private setting), and may even communicate concern about radiologic findings, although discussed in a general fashion, leaving the role of definitive diagnosis to the pediatric oncologist. The author underscores the importance of understanding negative reactions of patients and families to radiologic procedures and revisits (e.g., which may be accompanied by a fear of relapse), and to look for ways to help parents feel more in control during these procedures. The radiologist may allow parents to assist in positioning their children, administer oral contrast, and remain close by to give support. The use of developmentally appropriate explanations about the procedure to children, such as using dolls in younger children, is also essential. It is also important for the radiologist to administer sedation in a liberal fashion (as opposed to physically restraining the child), develop a relationship with the family, and be sensitive to the physical changes associated with illness and treatment.

The relevance of social support as a construct in pediatric cancer patients also has been reviewed recently ( [Woodgate, 1999](#)). Increased social support facilitates adjustment in these patients and their families, although there is an inexact appreciation for how these experiences are protective. The author concludes that there is a need for more comprehensive research efforts looking at social support from a family, sociocultural, and developmental perspective. Treatment implications proceed from this perspective as well as the understanding of social support needs as dynamic, time- and context-dependent, and patient-specific over the course of treatment.

The importance of spiritual care in pediatric cancer patients also has received comment in the literature ( [Hart and Schneider, 1997](#)). They reference spirituality in children as “the ability of a child through relationships with others to derive personal value and empowerment” ( [Taylor et al., 1995](#)). Such relationships occur both on a vertical (higher being) and horizontal level (self, others, and environment) ( [Fulton and Moore, 1995](#)). The approach to spiritual care first involves an assessment of religious needs (religious preference and activities) and receptivity to support groups, as well as the spiritual needs of the child. Using developmental theory, such as that espoused by Piaget ( [Ginsburg and Oppen, 1998](#)) and Erikson (1963), can allow nurses to intervene successfully to reduce distress, within a religious framework as well. An awareness of and sensitivity to losses specific to each child, the importance of interventions designed to facilitate a sense of normalcy, and the continuing role of nursing in the terminal stages of illness are also underscored.

Psychotherapy in this population has been reviewed, with three domains of focus identified: effects of cancer and its treatment on body image related to procedures and so on; effects on “normal” social, academic, and family functioning; and life, death, and grief issues ( [Sourkes, 1998](#)). Individual psychotherapy can address issues related to the trauma of the threat against survival and experience of illness, loss and helplessness, and need to rebuild one's life following treatment ( [Sourkes, 1998](#)). Sourkes stresses the importance of considerations of the cognitive, affective, and social impact of cancer on the child, and reviews the “props” needed, such as a dollhouse or stuffed animals. He also reviews the tools employed by the therapist, such as art techniques, lists made by the patient that identify areas of focus, mutual storytelling, or writing. Play psychotherapy can play a vital restorative function in these patients and facilitate a sense of mastery and partnership with the therapist.

Normalization of the emotional experience (common to cancer patients) can facilitate acceptance of the therapy; the goals are to enhance coping strategies, autonomy, and control ( [Sourkes, 1998](#)). Issues of confidentiality must be respected, because trust is the cornerstone of treatment. The importance for the therapist of developing a liaison with parents and other professionals caring for the patient and being attuned to countertransference is also underscored. The latter can include the impact of unresolved issues of loss in the therapist's life, and the need to walk a fine line between the intense suffering these children experience and the need to maintain a healthy distance that allows preservation of the therapist's emotional stability without compromising an empathic connection with the uniquely demanding nature of the child's emotional needs. Guilt over being healthy and anxiety about the child's life are common in therapists working with these children ( [Sourkes, 1998](#)). A therapist may develop displaced anxiety about her or his own health or the health of her or his own children as part of a countertransference phenomenon.

Brief psychotherapy in children with cancer also has received comment in the literature ( [Oppenheim and Hartmann, 2000](#)). In the four cases presented, the importance of the effects of parents' relationships with their own parents; the psychological status and ambivalent feelings of siblings; the symbolic aspects of disfigurement, body image, and self-concept; and the relevance of intense fears and fantasies were material ripe for psychotherapeutic intervention.

On a systems level, particularly in areas where there is a lack of academic medical centers doing oncologic research, “shared management” of pediatric cancer patients has been instituted, which divides care between the university medical center and community-based physicians ( [Kisker et al., 1997](#)). [Kisker and colleagues \(1997\)](#) reviewed the initial evaluation of this system, which indicated that treatment protocols were followed rigorously, that there were both direct (medical costs) and indirect (loss of work) savings, and no significantly higher rates of medical morbidity. The 15-year follow-up of this program had similar results. Comparable survival rates in the children and improved relationships between the academic center and primary care physicians were found. Such benefits can have potential “trickle down” benefits on the patients and families (personal comment).

The Children's Cancer Group (CCG), created in 1955 with 116 member institutions, is a pediatric clinical trials cooperative oncology group that is unparalleled in scope in North America ( [Kodish et al., 1998](#)). Informed consent in pediatric patients and their families has been researched ( [Kodish et al., 1998](#)), and it was found that clinicians tend to underestimate the amount of information parents prefer to know; the role of the child, equally important, was not studied in this instance. Informed consent is most accurately described as a process, not a singular occurrence. In this study, although parents generally were satisfied with the informed consent process, clinician-investigators were not, perhaps because of the overpowering nature of the informed consent process and its weight on parents.

## PSYCHOPHARMACOLOGY IN THE PEDIATRIC CANCER PATIENT

Psychopharmacology in this population was recently reviewed by [Spiegel \(1998\)](#), who noted a paucity of published work in this area. Use of tricyclic antidepressants for children with anxiety and depression and the use of alprazolam for anticipatory anxiety have been reported ( [Maisami et al., 1985](#); [Pfefferbaum et al., 1987](#); [Pfefferbaum-Levine et al., 1983](#)). [Spiegel \(1998\)](#) stresses the role of medication as complementing psychological and behavioral interventions, with a focus on treating specific symptoms as opposed to psychiatric disorders. The use of psychotropic medication in this population is hampered by our limited understanding of their interactions with oncologic medications and FDA approval for such usage, and the need to extrapolate from adult literature. It is essential that consideration of cytochrome P450 drug interactions be studied when using psychiatric medication in this population ( [Flockhart and Oosterheld, 2000](#)).

### Anxiety

[Spiegel \(1998\)](#) reviewed the literature documenting anxious and phobic symptoms in this population. Procedural phobias, separation anxiety, and PTSD have been described in the pediatric cancer patient. Emotional symptoms must be viewed within a developmental context and are intimately intertwined with physical illness, often making differentiation of the two challenging. For example, a procedural phobia can be a representation of incompletely treated pain, and anxiety can be a sequelae of sleep deprivation or represent a premorbid psychiatric condition that becomes exacerbated in the context of cancer and its treatment; all three phenomena could occur simultaneously. Anxiety also can occur secondary to delirium, medication side effects, infection, or other metabolic causes. The use of specific agents such as antidepressants, antihistamines, benzodiazepines, neuroleptics, and narcotic analgesics is guided by the intensity and time course of the anxiety and comorbid symptoms ( [Spiegel, 1998](#)). Posttraumatic stress disorder may be treated acutely with benzodiazepines and chronically with antidepressants. Antihistamines can be used for insomnia, sedation, and generalized anxiety, although they present risks because of anticholinergic toxicity in cognitively impaired or delirious patients. Benzodiazepines can be used for anxiety related to procedures, insomnia, and generalized anxiety, although side effects can include impaired cognition and disinhibition, especially in the presence of CNS disease or delirium. Routes of administration depend on the specific agent, with alprazolam given sublingually; lorazepam given orally; and lorazepam, midazolam, and diazepam given intramuscularly. Diazepam can be given intravenously; and clonazepam may be the most appropriate benzodiazepine for chronic use because of its long duration of action and twice-daily dosing.

Tricyclic antidepressants are effective in the treatment of insomnia, panic attacks, or generalized anxiety. Selective serotonin reuptake inhibitors have not been studied systematically, but anecdotally seem to be effective for both depression and anxiety in this population.

### Depressive Disorders

[Spiegel \(1998\)](#) underscored the difficulty of making a depression diagnosis in this population, given the preponderance of somatic symptoms (insomnia, anorexia, diminished energy) and the psychological responses to illness in children being treated for cancer. Undertreatment of pain, subsyndromal delirium, and PTSD can all present with depressive symptoms as well.

Low-dose tricyclic antidepressants are potentially valuable in treating specific depressive symptoms in this population, such as insomnia, and may increase appetite in these physically compromised patients. Side effects include anticholinergic effects (urinary retention, dry mouth, constipation, delirium) and effects on cardiac function

(hypotension, hypertension, EKG changes, and arrhythmias), although these are less likely on lower doses. Choice of agent is dependent on desired effect and route of administration, with amitriptyline being the most sedating, and nortriptyline available as an oral elixir.

Selective serotonin reuptake inhibitors, which have now been shown to be effective in pediatric depression in a controlled trial ( [Emslie et al., 1997](#)) and open trials ( [Rey-Sanchez and Gutierrez-Casares, 1997](#)), seem to be tolerated well in this population and appear to be superior to tricyclic antidepressants for the treatment of depression in this population, and may as well be effective for the treatment of PTSD ( [Spiegel, 1998](#)). Paroxetine may have the advantage of occasionally enhancing appetite. It is essential to monitor for pharmacologic interactions between selective serotonin reuptake inhibitors (SSRIs) and other medications pediatric cancer patients may be taking, given the potential for effects on the P450 enzyme system.

### Stimulants

Stimulant medications, the hallmark of treatment of attention deficit hyperactivity disorder (ADHD), are effective in this population in lower doses for enhancing energy state or counteracting the sedating effects of narcotic analgesics. Important side effects to monitor include insomnia and anorexia. Stimulants may be used in conjunction with anxiolytics, SSRIs, or tricyclic antidepressants.

### Delirium and Psychosis

Pediatric cancer patients are at risk for delirium owing to multiple factors, including the following ( [Spiegel, 1998](#)):

1. Primary effects of CNS tumors
2. Metabolic derangement
3. Drug withdrawal
4. Infection
5. Inadequately treated pain
6. Fever
7. Effects of radiation
8. Effects of chemotherapy and other medication (especially narcotics, medications with anticholinergic side effects, or drug-induced mania)

Delirium frequently goes unrecognized, and must be considered in the differential diagnosis of any acute change in a pediatric cancer patient's behavior, which might be manifested as anxiety, psychosis, aggression, or confusion. The mini-mental status examination, or more formal neuropsychological testing (if time permits) can be useful in formally assessing and following alterations in mental status; family recognition of a change in a child's behavior can be invaluable.

Low-dose treatment with high-potency neuroleptics, such as haloperidol or risperidone, can be quite effective in treating delirium and the accompanying behavioral agitation, so long as the underlying etiologies are also being investigated and treated. Intravenous haloperidol, although not an FDA-approved treatment, has been reported to be quite effective in appropriate circumstances ( [Spiegel, 1998](#)). Low-potency neuroleptics (e.g., chlorpromazine, thioridazine) may be used to treat insomnia or for sedation. Neuroleptics also may be used to treat aggressive behavior in this population, which may represent a symptom of delirium, an underlying psychiatric disorder, or a medication side effect.

Neuroleptics, when used in low dosages, tend to be fairly well tolerated in this population, although the treating clinician should be astute to extrapyramidal side effects such as dystonic reactions, Parkinsonism, and akathisia. Of note is that neuroleptics, including chlorpromazine, haloperidol, and risperidone, can all potentially prolong the QTc interval ( [Welch and Chue, 2000](#)). Risperidone has calcium-channel blocking properties. Other serious potential side effects include neuroleptic malignant syndrome.

### Pain

Tricyclic antidepressants (TCAs) and benzodiazepines can be useful adjuncts to the pharmacologic management of pain, particularly to treat comorbid anxiety and/or insomnia, or as premedication for procedures. Tricyclic antidepressants actually may potentiate the analgesic effects of narcotics, and psychostimulants may counteract the sedating effects of narcotics ( [Spiegel, 1998](#)).

### Comorbid Psychiatric Disorders

Unfortunately, some pediatric cancer patients may have preexisting psychiatric disorders that may require continued treatment with psychostimulant medication (ADHD), antidepressants, or mood stabilizers, although these treatments have not been systematically studied in this population. It is imperative to monitor for drug interactions with other medications the patient may be on. [Spiegel \(1998\)](#) notes the vulnerability of survivors to affective disorders, learning and attentional difficulties, and PTSD, to which the clinician must be attuned, and which may require pharmacologic treatment.

The treatment of the terminal patient also must not be overlooked. Longer-acting benzodiazepines (e.g., diazepam, clonazepam) minimize rebound anxiety and are effective in delirium, and neuroleptics are less likely to cause confusion in toxic states. High-potency neuroleptics may be used to preserve alertness, or low-potency neuroleptics may be used to enhance sedation, depending on the clinical situation ( [Spiegel, 1998](#)).

## HOSPICE CARE AND THE TERMINAL PATIENT

The issue of hospice care and death unfortunately cannot be ignored in pediatric oncology, and a familiarity with published material on these issues is essential to any professional working with these children ( [Davies, 1996](#); [Farrell, 1996](#); [Gillance et al., 1997](#); [Goldman, 1995](#); [Goldman and Christie, 1993](#); [Hill and Stuber, 1998](#); [Martinson, 1995a,b, 1996](#); [Pfund, 1998](#); [Thompson, 1998](#); [Wakai et al., 1996](#); [Wallace and Jackson, 1995](#); [Whiting, 1997](#)). The underlying premise of hospice care is that terminally ill children and their families can benefit from interventions designed to improve quality of life even in this phase ( [Davies, 1996](#)), most effectively delivered by a psychosocial team ( [Wallace and Jackson, 1995](#)). Hospice care has been written about from a developmental perspective, with the terminally ill adolescent patient presenting unique challenges to hospice staff owing to the physical changes associated with cancer and its treatment, and the need for the adolescent to maintain independence, which can lead to refusal of care in the setting of anger and denial ( [Klopfenstein, 1999](#)). Training programs in pediatric hospice care are felt to be a necessary component of a comprehensive treatment program for pediatric cancer by many authors ( [Papadatou, 1997](#)).

The issue of discussing death itself with children and adolescents also has been written about, with a direct, honest but sensitive approach generally advocated ( [Goldman and Christie, 1993](#)), although, only 19% of families openly discussed the child's approaching death with them in this study, and staff generally overestimated the extent to which such discussion was happening. Both families and staff may lack the requisite communication skills to have such a discussion, which could be enhanced. [Goldman and Christie \(1993\)](#) reviewed literature suggesting that even 3- or 4-year-old children can discuss death, and note that open communication about death is advantageous for families and children overall.

Pediatric palliative care recently was reviewed ( [Frager and Shapiro, 1998](#)); it was pointed out that the concepts of "cure" and "care" should not be mutually exclusive. A comprehensive model that allows for palliative care (including pain management and psychological services) and bereavement work must focus on issues related to loss and be present throughout the treatment of a child, and not merely follow the active phase of treatment. The central ethical orientation involving the care of the terminal child is "to consider all actual and potential benefits and burdens from the perspective of the child as patient" ( [Frager and Shapiro, 1998](#), p. 908; [Solomon et al., 1993](#)).

## CONCLUSION

Pediatric cancer has a myriad of psychological effects on the patient and his or her family, with the evolution of an understanding of this phenomenon encompassing the concept of quality of life when broadly defined. The diagnosis of cancer has acute psychological effects on the child and family system, with ripple effects that influence school and community. As cancer incidence has risen, so has cancer survival, resulting in a large population of survivors whose psychological, social, educational, and occupational needs must be addressed by society at large, and especially by those delivering psychological and behavioral care.



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# 97 PSYCHIATRIC ISSUES IN PEDIATRIC BONE MARROW, STEM CELL, AND SOLID ORGAN TRANSPLANTATION

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Both bone marrow transplantation (BMT) and solid organ transplantation (SOT) have undergone a revolution in the past 20 to 30 years, evolving from primarily experimental procedures to accepted treatment for a variety of diseases and varieties of end-stage organ failure ( House and Thompson, 1988; Surman, 1989). The history of pediatric cardiac transplantation was reviewed by Addonizio (1990). Christiaan Barnard performed the first adult cardiac transplantation in 1967, although the patient lived only 17 days. In 1967, Kantrowitz transplanted a heart into a 2-week-old infant who survived for several hours, and in 1968, Denton Cooley transplanted a new heart and lungs into a 2-month-old, who also survived for several hours. Although government-sponsored treatment of end-stage renal disease was instituted in 1972, almost 20 years after the first successful renal transplantation in 1954 (using an identical twin as donor), poor success rates during the 1970s, due primarily to infection, rejection, and donor shortages, caused a relative moratorium on transplantation of other organs during this time ( Surman, 1989).

In the late 1970s, however, positive results at Stanford University, which had persisted with its heart transplant program, led to a resurgence of interest in heart transplantation. The experience with children and adolescents, however, remained quite limited. With the introduction of a new immunosuppressant, cyclosporine, in 1980, success rates began to improve because of both a decreased rate of infection and improved management of rejection episodes. After this, the number of centers performing heart transplantation soared, and the experiences with greater numbers of patients led to efforts at heart transplantation in infants and children. The progression from the laboratory into mainstream medicine thus was facilitated by the development of immunosuppressant medications, improvements in operative technique, more careful immunologic subtyping and matching, greater refinement in the candidate selection process, and growing experience in the medical management of transplant recipients (Parkman, 1986; Surman, 1989).

The historic “Baby Fae” xenograft (nonhuman donor) received much media attention in 1984. In 1985, Leonard Bailey successfully performed a neonatal cardiac allotransplantation (human heart as donor), and during the mid-1980s, the number of transplantations in children increased exponentially. Between 1984 and 1988, the International Heart Transplantation Registry recorded 583 cardiac transplantations in patients younger than 20 years of age; before 1980, there were fewer than five transplantations a year performed on patients in this age group ( Addonizio, 1990).

The first allogeneic BMT was reported in 1969, in a 5-year-old with severe combined immunodeficiency syndrome ( Meuwissen et al., 1969). For BMTs in particular, statistics have been cited that indicate that approximately half of all BMTs have taken place in the pediatric population ( Wiley and House, 1988). The number of BMTs rose as chemotherapy and immunologic techniques improved, and the types of illnesses treated extended to soft tissue tumors, immunologic and metabolic diseases, and leukemia (Stuber, 1993a). BMT is a mainstay of treatment for early acute lymphoblastic leukemia or acute myelogenous leukemia, chronic myelogenous leukemia, and some solid tumors in children (Parkman, 1986; Trigg, 1988). The application of transplant technology to the pediatric population thus often has been center stage in this process for both SOT and BMT, with an exponential rise in the number of transplantations performed ( Table 97.1A, Table 97.1B, Table 97.1C, Table 97.1D and Table 97.1E and Table 97.2).

Year	Age (yr) of Recipient										
	<1	1-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-49	Total
1989	13 (1.3)	20 (2.0)	17 (1.7)	4 (0.4)	16 (1.6)	32 (3.2)	63 (6.3)	41 (4.1)	0	0	135
1990	14 (1.4)	22 (2.2)	18 (1.8)	5 (0.5)	17 (1.7)	35 (3.5)	65 (6.5)	42 (4.2)	0	0	143
1991	15 (1.5)	23 (2.3)	19 (1.9)	6 (0.6)	18 (1.8)	36 (3.6)	66 (6.6)	43 (4.3)	0	0	146
1992	16 (1.6)	24 (2.4)	20 (2.0)	7 (0.7)	19 (1.9)	37 (3.7)	67 (6.7)	44 (4.4)	0	0	149
1993	17 (1.7)	25 (2.5)	21 (2.1)	8 (0.8)	20 (2.0)	38 (3.8)	68 (6.8)	45 (4.5)	0	0	152
1994	18 (1.8)	26 (2.6)	22 (2.2)	9 (0.9)	21 (2.1)	39 (3.9)	69 (6.9)	46 (4.6)	0	0	155
1995	19 (1.9)	27 (2.7)	23 (2.3)	10 (1.0)	22 (2.2)	40 (4.0)	70 (7.0)	47 (4.7)	0	0	158
1996	20 (2.0)	28 (2.8)	24 (2.4)	11 (1.1)	23 (2.3)	41 (4.1)	71 (7.1)	48 (4.8)	0	0	161
1997	21 (2.1)	29 (2.9)	25 (2.5)	12 (1.2)	24 (2.4)	42 (4.2)	72 (7.2)	49 (4.9)	0	0	164
1998	22 (2.2)	30 (3.0)	26 (2.6)	13 (1.3)	25 (2.5)	43 (4.3)	73 (7.3)	50 (5.0)	0	0	167

These data include multiple recipients after the first listing in which country is not specified. Percentages are based on total recipient population.  
From: *ISHT Registry Report*, 1998.

**Table 97.1A. Transplant Recipient Characteristics—1989 to 1998: Heart Recipients (n (%))**

Year	Age (yr) of Recipient										
	<1	1-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-49	Total
1989	1 (0.1)	0 (0.0)	1 (0.1)	1 (0.1)	2 (0.2)	7 (0.7)	13 (1.3)	10 (1.0)	0	0	24
1990	1 (0.1)	1 (0.1)	1 (0.1)	1 (0.1)	2 (0.2)	8 (0.8)	14 (1.4)	11 (1.1)	0	0	28
1991	1 (0.1)	1 (0.1)	1 (0.1)	1 (0.1)	2 (0.2)	9 (0.9)	15 (1.5)	12 (1.2)	0	0	31
1992	1 (0.1)	1 (0.1)	1 (0.1)	1 (0.1)	2 (0.2)	10 (1.0)	16 (1.6)	13 (1.3)	0	0	34
1993	1 (0.1)	1 (0.1)	1 (0.1)	1 (0.1)	2 (0.2)	11 (1.1)	17 (1.7)	14 (1.4)	0	0	37
1994	1 (0.1)	1 (0.1)	1 (0.1)	1 (0.1)	2 (0.2)	12 (1.2)	18 (1.8)	15 (1.5)	0	0	40
1995	1 (0.1)	1 (0.1)	1 (0.1)	1 (0.1)	2 (0.2)	13 (1.3)	19 (1.9)	16 (1.6)	0	0	43
1996	1 (0.1)	1 (0.1)	1 (0.1)	1 (0.1)	2 (0.2)	14 (1.4)	20 (2.0)	17 (1.7)	0	0	46
1997	1 (0.1)	1 (0.1)	1 (0.1)	1 (0.1)	2 (0.2)	15 (1.5)	21 (2.1)	18 (1.8)	0	0	49
1998	1 (0.1)	1 (0.1)	1 (0.1)	1 (0.1)	2 (0.2)	16 (1.6)	22 (2.2)	19 (1.9)	0	0	52

These data include multiple recipients after the first listing in which country is not specified. Percentages are based on total recipient population.  
From: *ISHT Registry Report*, 1998.

**Table 97.1B. Transplant Recipient Characteristics—1989 to 1998 Lung Recipients (n (%))**

Year	Age (y) of live of recipient										
	<1	1-4	5-9	10-17	18-24	25-34	35-44	45-54	55-64	65-74	75-84
1989	5.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
1990	5.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
1991	5.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
1992	5.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
1993	5.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
1994	5.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
1995	5.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
1996	5.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
1997	5.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
1998	5.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0

These data include multiple recipients after the same procedure in which living donor live was one of the organs transplanted. Percentages are based on total kidney transplants.  
 From NIDDK Scientific Registry, Data as of September 1, 1998.

**Table 97.1C. Transplant Recipient Characteristics—1989 to 1998: Living Donor Kidney Recipients (n (%))**

Year	Age (y) of live of recipient										
	<1	1-4	5-9	10-17	18-24	25-34	35-44	45-54	55-64	65-74	75-84
1989	0.0	2.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1990	0.1	4.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1991	0.4	9.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1992	0.4	14.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1993	0.4	17.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1994	0.5	2.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1995	0.5	14.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1996	0.5	16.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1997	0.5	25.9	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1998	0.5	22.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

These data include multiple recipients after the same procedure in which living donor live was one of the organs transplanted. Percentages are based on total kidney transplants.  
 From NIDDK Scientific Registry, Data as of September 1, 1998.

**Table 97.1D. Transplant Recipient Characteristics—1989 to 1998: Living Donor Liver Recipients (n (%))**

Year	Age (y) of live of recipient										
	<1	1-4	5-9	10-17	18-24	25-34	35-44	45-54	55-64	65-74	75-84
1989	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1990	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1991	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1992	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1993	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1994	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1995	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1996	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1997	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1998	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

These data include multiple recipients after the same procedure in which living donor live was one of the organs transplanted. Percentages are based on total kidney transplants.  
 From NIDDK Scientific Registry, Data as of September 1, 1998.

**Table 97.1E. Transplant Recipient Characteristics—1989 to 1998: Lung Recipients (n(%))**

Approximate 5-year survival rates after HLA-identified sibling allogeneic stem cell transplantation, age <20 y:  
 AML (first remission), 60%  
 AML (second remission), 45%  
 ALL (second or subsequent remission), 40%  
 Severe aplastic anemia, 75%  
 Approximate 5-year survival rates after autologous stem cell transplantation, age <20 y:  
 Hodgkin's Disease (second remission), 60%  
 Neuroblastoma (first remission), 35%

HLA, human leukocyte antigen.  
 Data reprinted with permission from James Garvin, M.D., Ph.D., New York Presbyterian Hospital, NY, NY.

**Table 97.2. Five-Year Survival Rates After Stem Cell Transplantation**

Research into the quality of posttransplantation life, which was reviewed by [Surman \(1989\)](#), helped push SOT transplantation into the forefront of accepted medical technology for end-stage or incurable conditions. The role of psychiatric involvement developed in the setting of the implicit ethical issues surrounding allocation of a resource (the donated organ) in which the demand far exceeds the supply; as psychosocial factors in candidate selection (such as the potential for nonadherence) became identified as important in affecting outcome; and as neuropsychiatric complications such as delirium, anxiety, depression, and the psychological impact of the transplantation process on the patient and family became recognizable.

The United Network for Organ Sharing (UNOS), an outgrowth of the National Transplant Act of 1984, is under the directorship of the Health Resources and Service Administration, which sets standards about transplantation success rates, criteria for review, and application to function as a transplantation center ( [Surman, 1989](#)). UNOS directs the national Organ Procurement and Transplantation Network (OPTN) and United States Scientific Registry on Organ Transplantation. All transplantation centers, organ procurement agencies, and tissue typing laboratories in the country participate. UNOS administers the allocation and distribution of organs using a computerized list that is managed by strict scientific criteria, including organ type, tissue match, blood type, length of time on the waiting list, geographic distance if applicable (heart, heart–lung, lung, liver, pancreas), and immune status.

Research into the psychiatric aspects of transplantation in children has been limited, although child psychiatric involvement in a consultation–liaison role represents a standard component of the transplantation team. It has become increasingly evident, however, that the psychiatric ramifications in the world of SOT and BMT represent paradigms of modern, high-technology medicine today that are generalizable to many pediatric medical conditions and treatments ( [Stuber et al., 1991](#)). Issues involving informed consent/assent, ethical and quality-of-life concerns, developmental considerations, pain and anxiety management, nonadherence, and the effects of acute and chronic illness on the patient and family transcend this particular field, although perhaps nowhere else are they all found in such a compelling, poignant manner.

In a similar way, the child psychiatrist's task in consulting to a transplantation team requires the use of all of the practitioner's professional skills in a setting where individual and family psychotherapy, psychopharmacologic intervention, behavior therapy, liaison to other staff, and the understanding of medical illness and procedures are required. This chapter reviews the psychiatric issues involved in pediatric SOT and BMT, with the overall focus being the role of the consultation–liaison child and adolescent psychiatrist. In the 6 years since the first edition of this chapter:

- The relative percentage of pediatric donors has increased from 19% of cadaveric donors in 1989 to 26% of donors in 1998.
- Survival rates for pediatric organ transplant recipients are comparable with adult survival rates.



- Pediatric patients usually have shorter waiting list times than adults because UNOS allocation policies tend to favor patients younger than 18 years of age (UNOS, 1999).

## GENERAL ISSUES IN THE PEDIATRIC TRANSPLANTATION POPULATION

The literature on the psychiatric aspects of transplantation in the pediatric population has been reviewed ( Sexson and Rubenow, 1992; Slater, 1994; Stuber, 1993a). Stuber (1993a) notes many of the salient aspects specific to transplantation in the pediatric population, including effects on donor supply secondary to size, longer waiting periods, and the ethical implications of using living-related donors or organs from anencephalic infants. The effects of transplantation on identity and self-concept formation are just beginning to be studied, and pediatric transplant recipients differ from many of their adult counterparts in that they are not regarded as having "caused" the conditions that necessitate transplantation ( Stuber, 1993a). Caring for these patients also has been reviewed from a primary care perspective (Kosmach et al., 1998). The effects of transplantation on maternal stress has been studied ( Rodrigue et al., 1997). It was found that after bone marrow, liver, kidney, or heart transplantation, more than one-half of mothers experienced significant stress at 1-month follow-up, and over a third still experienced significant stress at 6-month follow-up. These findings indicate that the psychological response to transplantation represents an ongoing stressful experience, even when the child is recovering.

Posttransplantation issues for mothers, who usually are the primary caretakers, include dealing with the feelings of well siblings who may feel neglected, care and potential noncompliance in the patient, reentry issues and social isolation, being responsible for appointments, and overseeing limitations placed on the children. Financial strain may increase months after transplantation when bills and prescription costs mount, and mothers with fewer financial resources exhibited greater levels of stress, greater family conflict, and less family support, indicating the need for special attention to this population. Extended family supports may be less available once the initial phase after transplantation is over. Medical factors seemed not to be associated with psychological functioning of mothers in this study. Specific programs to assist parents in development of coping strategies, increasing resources, and bolstering family stability are essential. The latter includes support groups, which have also been found to be valuable at this author's institution, where a multifamily heart transplant group is run monthly.

The authors point out that high stress levels may predispose to anxiety and depression in mothers, which can have potential effects on child care and compliance (Rodrigue et al., 1997). Successful coping strategies exercised by parents included information gathering, especially in the form of support groups and liaison with members of the transplantation team.

Solid organ transplantation has become recognized as a legitimate treatment for many types of end-stage organ failure ( Table 97.3). The number of children on waiting lists at the time of writing, the number of transplantations performed annually by organ, the number of children on the waiting lists for organs, and survival rates are detailed in Table 97.1A, Table 97.1B, Table 97.1C, Table 97.1D and Table 97.1E, Table 97.4 and Table 97.5A, Table 97.5B, Table 97.5C, Table 97.5D, respectively. Analogous data on BMT recipients are presented in Table 97.2. The dilemma in allocation of solid organ donations is presented in Table 97.6, which indicates how many children die on the waiting list.

Heart	Liver (Donor n, Recipient n, %)	Kidney (Donor n, Recipient n, %)	Lung (Donor n, Recipient n, %)
Congenital anomalies	End-stage liver disease	Ischemic cardiomyopathy	Cystic fibrosis
Cardiomyopathy	α-1-Antitrypsin deficiency	End-stage renal disease	Primary biliary cirrhosis
	End-stage liver disease	Chronic hepatitis	Interstitial disease
	Alcoholic cirrhosis	Hemochromatosis	Congenital heart disease
	Hemochromatosis	Hereditary spherocytosis	
	Chronic active hepatitis	Hereditary spherocytosis	
	Systemic	Hereditary spherocytosis	
	Primary biliary cirrhosis	Hereditary spherocytosis	
	Hemochromatosis	Hereditary spherocytosis	

From Slater, J. Psychiatric aspects of transplantation in children and adolescents. *Child Health: Pediatric Clin North Am* 1992;39:199-210.

**Table 97.3. Possible Etiologies for End-Stage Organ Disease in Children**

Age Group (y)	Heart	Liver	Kidney	Other	Heart/Lung	Lung	Total
0-4	10 (0.2)	48 (0.7)	10 (0.2)	5 (0.1)	10 (0.2)	9 (0.1)	92
5-10	10 (0.2)	25 (0.4)	10 (0.2)	10 (0.2)	10 (0.2)	10 (0.2)	80
11-17	41 (0.8)	118 (1.9)	5 (0.1)	10 (0.2)	10 (0.2)	10 (0.2)	194
18-49	248 (4.6)	1,100 (18.5)	1,000 (17.0)	1,000 (17.0)	1,000 (17.0)	1,000 (17.0)	5,348
50-64	1,174 (21.7)	1,200 (20.0)	1,000 (17.0)	1,000 (17.0)	1,000 (17.0)	1,000 (17.0)	5,374
65+	5,302 (96.4)	1,400 (23.5)	1,000 (17.0)	1,000 (17.0)	1,000 (17.0)	1,000 (17.0)	10,102
Total	6,837 (125.3)	16,206 (283.5)	1,075 (19.1)	1,075 (19.1)	1,075 (19.1)	1,075 (19.1)	26,360

Some patients are listed with more than one transplantable organ listed and the total number of registrations may be greater than the total number of patients.  
From UNOS Scientific Registry Data.

**Table 97.4. Number of Patient Registrations on the National Transplant Waiting List by Age as of July 31, 2000 (n [%])**

Age of Recipient	Liver		Kidney		Heart		Lung	
	n	%	n	%	n	%	n	%
<1	10	0.1	29	0.3	11	0.1	15	0.2
1-5	20	0.2	18	0.2	11	0.1	12	0.2
6-10	10	0.1	29	0.3	11	0.1	15	0.2
11-17	10	0.1	29	0.3	11	0.1	15	0.2
18-24	40	0.4	13	0.1	11	0.1	11	0.1
25-49	2,077	21.5	10	0.1	11	0.1	11	0.1
50-64	2,077	21.5	10	0.1	11	0.1	11	0.1
65+	480	5.0	17	0.2	11	0.1	11	0.1
Unknown	0	0.0	0	0.0	0	0.0	0	0.0
Total	5,077	53.2	64	0.7	22	0.2	22	0.2

n = The number of registrants in the UNOS Scientific Registry for Organ Donation (SRJD) as of December 31, 2000. The total number of registrants in the SRJD as of December 31, 2000 was 9,527. The total number of registrants in the SRJD as of December 31, 2000 was 9,527. The total number of registrants in the SRJD as of December 31, 2000 was 9,527.  
From UNOS Scientific Registry Data as of December 31, 2000.

**Table 97.5A. Patient Survival Rates at 3 Months and at 1, 3, and 5 Years: Liver Transplants**





transplant is one probably mediated less by psychiatric factors. A multidisciplinary transplantation team represented by pediatrics, surgery, psychiatry/psychology, nursing, and social work is oriented more toward identifying risk factors for psychopathology and nonadherence, and ensuring that there are adequate social supports. The child and adolescent psychiatrist performs a dual function. As a consultant, he or she conducts a thorough psychiatric evaluation of the patient and family, with an emphasis on identifying psychopathology or potential risk factors for adjustment reactions or nonadherence. The consultant role also includes periodic neuropsychiatric assessment and the brief treatment of patients and families with a variety of modalities that may extend into the posttransplantation period, and perhaps should in many circumstances ([Stuber et al., 1991](#)).

As the psychiatric liaison, the child and adolescent psychiatrist performs an equally important function as an intermediary, communicating a level of understanding about the patient's and family's experience to the rest of the medical staff. In addition to this, however, the psychiatrist may serve a supportive or directive function for the other members of the transplantation team, who may struggle with feelings triggered by particular patients, clinical situations, or ethical dilemmas. He or she is very much a part of the "family" composed of the transplantation team, patient, and patient's family.

Transplantation teams comprise individuals who may conceptualize the psychiatrist's role in different ways, and expectations or priorities should be specified before evaluation or suggested interventions. For instance, lung transplantation in an adolescent patient with cystic fibrosis and a history of nonadherence will carry a particular focus. The evaluation of the family consisting of a 2-year-old and 5-month-old twins, where one of the twins has a serious cardiomyopathy requiring transplantation, will be different. The pediatrician's focus in the first case may be to try to develop a formulation of the causes of nonadherence and the likelihood of successful intervention to address this problem. In the latter case, the availability of family supports and the psychological state of the mother may be of more concern. Few data are available on parental factors that may correlate with postoperative morbidity, and more are needed ([Stuber, 1993a](#)).

Transplantation is all the more compelling because, with the exception of kidney transplantation, SOT and BMT are treatments of last resort, without which the patient would likely die. There is less urgency on the pediatric nephrology service because these children can survive on dialysis without transplantation, which therefore may be considered more electively.

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#### CASE ILLUSTRATION

Psychiatric consultation was requested when the father of a child who received a cardiac transplant punched a hole in the wall and repeatedly threatened nursing staff in the aftermath of his son's cardiac arrest. Psychotherapy with this man was not possible because of what appeared to be his limited ability to gain insight into the underlying causes for his anger, which were believed to be a sense of helplessness and tremendous desperation, as well as the wish to fix blame on somebody or something for what had happened to his son. Although it was important to help the staff understand what this man was going through, it was perhaps more important to ensure they felt protected. Security was called, and definitive limits were set for this man with regard to verbal and nonverbal threats.

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The pretransplantation evaluation should be part of the multidisciplinary assessment of the transplantation candidate, the purpose of which should be explained to the family by the primary physician. Psychiatric consultants may best be conceptualized as experts in child development, pain and anxiety management, and the psychological responses to transplantation. He or she is an important member of the transplantation team who can provide emotional support to the patient and family during an unusual crisis that anyone would find extremely distressing and help prepare them emotionally for transplantation. The child and adolescent psychiatrist also can serve a vital function in helping to communicate the child's needs to his or her family and the rest of the transplantation team. If framed in this manner by the primary physician, the evaluation rarely will be met with resistance by the family. It is important that there be continuity of care with respect to psychiatric services because the pretransplantation evaluation can form the basis for a trusting relationship that will affect the receptivity of future interventions and allow a greater depth of understanding in the patient's and family's experience.

The pretransplantation evaluation must encompass the standard elements of child psychiatric evaluation. In addition, however, the consultant must be familiar with the medical problems for which transplantation is being considered, as well as have a working understanding of transplantation itself from a medical and surgical perspective. In so doing, he or she will more likely be seen as an integral part of the transplantation team. The consultant should ascertain whether other members of the transplantation team have specific concerns about a patient or family.

The child or adolescent patient and his or her family have reactions simply to the fact that they are being evaluated for transplantation (see section on [Diagnosis](#), later). Such reactions may cover the range from denial to panic, with different family members often reacting differently. In some cases, extended family members or siblings are not told about the evaluation. In others, grandparents and siblings are present at the evaluation and clearly have an impact on the process itself. Evaluation often is complicated by the fact that it is occurring in an urgent setting with a critically ill child, and parents are required to digest relatively quickly a great deal of complicated information about a subject about which they know nothing, in the setting of acute anxiety and fear, leaving them overwhelmed, which often makes true informed consent impossible.

In other cases, small children with cardiomyopathies may not appear that ill, taxing the capabilities of parents to overcome the understandable inclination to use denial. These children nevertheless may have borderline cardiac function and be subject to fairly rapid decompensation. They therefore may be listed for transplantation because the relatively long waiting periods for small children could make organ procurement difficult. Parents later may look back on videotaped footage of their child before transplantation and wonder how they did not realize the child was sick. Listing the child early also may give the transplantation team more time to familiarize themselves with the family, and vice versa. Before meeting with the family, the psychiatric consultant can benefit from information concerning the reactions of the family to the evaluation procedure.

Parents and patients may ask the psychiatric consultant questions about medical or surgical aspects of transplantation, such as survival rates, that are best referred back to the pediatrician or surgeon. With prepubertal children, it often is helpful to meet with the parents first, as in a standard child psychiatric evaluation. Adolescents may wish to meet with the psychiatric consultant first, or with his or her parents; at some point, adolescents and children should be interviewed alone because they often will reveal feelings not expressed in the presence of their parents. The child and adolescent psychiatrist should begin the consultation by asking the parent or patient about conceptions and expectations about the psychiatric evaluation, to allow appropriate clarifications to be made.

The psychiatric pretransplantation evaluation can be diagnostic as well as educational and potentially therapeutic. Elements of this evaluation should include:

1. Getting to know the patient and family, and vice versa: Will they be able to function as part of a "team?"
2. How is the patient/family experiencing the pretransplantation evaluation and the prospect of transplantation? Significant "psychoeducation" may take place by helping to "normalize" the patient/family's reactions to the transplantation experience.
3. Obtaining a history of the medical/surgical problem, prior illness experience, and the "parallel" emotional or psychiatric history. This includes:
  - a. An assessment of the patient's and family's coping styles. Certain coping styles may be predictive of fewer adjustment problems in the setting of a chronic illness and include "positive self-talk, attention diversion, relaxation, thought stopping, task orientation, talk(ing) with someone, and good problem solving activity," as opposed to "catastrophizing" strategies such as the "focus on negative affect or fear, anxious anticipation, escape or avoidance, worry/rumination, self-denigration and self-blame, [and] fear of unlikely consequence" ([Olson et al., 1993](#)).
  - b. Obtaining a description of the temperament of the child/adolescent
  - c. Past and present psychopathology in patient, parents, siblings
  - d. Nature of past procedures, hospitalizations, and surgeries, and reactions to same, including "secondary" psychopathology, giving consideration to affective (depression, anxiety), cognitive (confusion, distortion), and behavioral (tantrums, oppositional behavior, nonadherence, posttraumatic phenomena) parameters
  - e. Risk factors for nonadherence
  - f. History of drug or alcohol abuse in the adolescent
4. Assessing the quality of the child/adolescent-caregiver(s) relationship and family dynamics
5. Detailed developmental, neuropsychiatric, social, and school history
6. Mental status examination and neurocognitive assessment of the child/adolescent
7. Assessment of how socioeconomic factors may have affected the management or course of the child/adolescent's illness, and how these factors may be of concern regarding life after transplantation. Are psychosocial supports adequate?
8. What the patient/family understand about the process of transplantation, from being listed, to the surgery itself, postoperative course, and immunosuppressant medication. Much of this information may not be given routinely to the patient until there is consideration for formal listing, but the family often has many misconceptions about this process, perhaps from what they have read or heard from other families. Are parents able to give informed consent, and is the child/adolescent able to give informed assent/consent? Is the family and adolescent accepting of long-term medication administration and care? [Modified from

[Slater \(1994\)](#), with permission.]

Other factors influence the response to hospitalization at the time of transplantation:

1. The age of the child and developmental level. Younger children are more prone to cognitive distortions common to the Piagetian preoperative stage, regression, fantasies of intended harm, mutilation, and separation fears. They are less likely to understand the rationale of procedures, nature of the pathophysiologic process, and course of an illness.
2. The number of prior hospitalizations early in life. Repeated hospitalizations between the ages of 6 months and 4 years may increase the risk of later psychiatric disorder ([Mrazek, 1984](#); [Quinton and Rutter, 1976](#)).
3. Hospital characteristics, such as visitation policies and quality of the child life department. [From [Slater JA \(1994\)](#), with permission.]

At the conclusion of the evaluation, which may take several interviews, a concise formulation should integrate data, which have been gathered into a form easily “digestible” by nonpsychiatric colleagues. This formulation should identify psychopathology, describe coping styles, and address possible predictors of resilience or nonadherence and the adequacy of social supports. The psychiatric consultant should indicate areas where misconceptions exist regarding information presented to the family by other members of the transplantation team and relay this back to his or her colleagues.

The diagnostic impressions and therapeutic recommendations should be communicated to both the referring physician as well as to the patient and family. This should be done both verbally and in the form of a written note, a copy of which may be given to the parents as well, if appropriate, which may help “demystify” the psychiatrist's role as well as educate. Recommendations should correlate with problem areas identified in the formulation and may include, for example, preparation for transplantation with play or videotapes, the teaching of relaxation techniques for anxiety reduction, social service intervention to clarify medical insurance, or contact with other transplant families.

Families commonly look forward to hearing suggestions from the psychiatrist for helping them through the arduous process of transplantation. If immediate follow-up is not thought to be necessary by the consultant and family, the family should be told how the consultant can be reached. If the patient is in the hospital, continued follow-up is advised. When the psychiatric consultant communicates with colleagues, judgment should be exercised with respect to patient confidentiality about issues not relevant to the transplantation process; this should be discussed with the patient and family. Care should be exercised about what is written in the hospital record because in many ways this is a public document.

## DIAGNOSIS

The reactions of the patient and family to the diagnosis of a medical problem serious enough to warrant transplantation occur in the context of the prior medical history. A chronically ill child who already has had several surgeries for congenital heart disease presents a different scenario from a previously well child who noticed a lump in his cheek initially assumed to be an abscess that turns out to be a Burkitt's lymphoma (see [Case Illustration](#), later). These differences may seem obvious, but what all patients and families come to share is an extreme fear of uncertainty associated with the unknown, fears of pain—emotional and physical—and on some level the fear of death.

Parents may react differently ([Gold et al., 1986](#)), some in more of an assertive, information-seeking mode, acting proactively for their children, and others with more passivity and increased reliance on medical staff for direction. The psychiatric consultant can be of great assistance in helping parents to act in their own (and the child's) best interests and to become more aware of how their individual coping styles affect them.

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### CASE ILLUSTRATION

Adolescents themselves may have a powerful sense of denial. An adolescent, on being told that he needed a heart transplant, said: “Really how to figure how I felt back then, basically a thirteen year old kid is invincible in his own mind. I remember I was playing football and my dad pulled me over. He didn't want to tell me, but then he finally told me, and I'm like, ‘All right dad, can I go back in the game?’ And he was like, ‘Yeah, go ahead.’ Because at thirteen you don't think of what a transplant means. You don't know the dangers, the risks, or anything.” [From [Slater \(1994\)](#), with permission.] One adolescent with severe heart disease confided that when she was around her parents, she used to bite her lips to make them turn red, so they would not realize she was [cSlater.](#) ( [1994](#)).

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### CASE ILLUSTRATION

The consultation–liaison child and adolescent psychiatrist was called by a senior colleague to see his relative, an 8-year-old boy with newly diagnosed Burkitt's lymphoma. The patient's father was in a state of shock, unable to believe that what he thought was a simple abscess was in actuality a malignancy for which his son would receive emergent BMT. The fact that, like many chemotherapy protocols, the one his son would receive was experimental further overwhelmed this man. In desperation, he began frantically calling different centers that do BMTs to compare protocols, and in fact he began to wonder if a mistake had been made when the pathology slides were read or that they had somehow been switched. He became irritable and sleep deprived, and to complicate matters further, he did not want his son told that he had a malignancy, fearing it would overwhelm and terrify him. The staff reacted powerfully to this scenario, and many meetings with the family and staff in various combinations were needed.

The psychiatrist was buffeted by many stresses, including the feelings associated with referral from a senior colleague, the need accurately to assess the father's mental status and intervene appropriately, the need to consider the best interests of the boy, and the need to liaison with other staff. All of this happened rapidly in the setting of a diagnosis leading to BMT within several days.

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Diagnosis itself may thus represent a trauma that manifests posttraumatic symptoms, requiring intervention. Parental misconceptions about or lack of exposure to the process of transplantation, such as donor allocation and wait listing, further complicate matters. In this setting, parents are asked to organize themselves cognitively and make terribly difficult decisions in a short time that will affect the survival of their child; it often is an impossible quandary. They may find it difficult to communicate with one another openly about fears, owing to concerns over destabilizing the spouse, and may suffer in solitude. Parents may focus on concrete details like survival statistics to attempt to center themselves in the face of a feeling of overwhelming helplessness and anxiety over entrusting their child's life to people whom they barely know, who are recommending procedures about which parents may be completely ignorant. Illusions of control over the events of one's life seem to be obliterated.

Alternative treatments may be sought that potentially may delay medical intervention, such as the case of a family who made phone calls around the world: “Maybe somebody will tell us that there's a doctor in the Philippines who has found a papaya serum that can cure cardiomyopathy” ( [Slater, 1994](#)).

## BEING LISTED

If recommended, informed consent for transplantation is obtained after evaluation, when families are educated about the indications, risks and benefits, and process of transplantation. Being listed represents a “point of no return” ( [Slater, 1994](#)), and families typically experience great anxiety about the future that may include confusion over how UNOS manages the wait list for organs. Parents are in a sense formally committing their child's future to events and personnel over which they have little control, often with a feeling of blind faith that things will work out positively, knowing full well they might not. The “roller coaster” metaphor has been used by many families in describing the process of a building up of tension, followed by a fearful, perhaps exhilarating, but out-of-control descent ( [Slater, 1994](#)).

Parents of young children wonder how they should explain to their children what will be happening to them, and how to prepare them. Here, the child and adolescent psychiatrist can be of great assistance.

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### CASE ILLUSTRATION

Steven, a 3½-year-old boy who had a cardiomyopathy, was listed for heart transplantation. The consulting psychiatrist, using dolls and stuffed animals, engaged Steven in a story about a little boy named “Chris” who had become ill. Using some elementary drawings and a heart cut from red construction paper, he explained what the heart did, and how it had become sick in this little boy. In the play, he told how the boy's heart was too sick even for medicine to help, but that he had to somehow be helped.

“What could we do?” asked the psychiatrist.

“I don't know,” responded Steven.

“Well, we could get him a new one,” said the psychiatrist.

After that, Steven and the psychiatrist acted out the heart operation many, many times over a period of days, using an actual anesthesia mask and as many “real” props as possible, including miniature intravenous poles, operating room tables, tape, bags of



intravenous solution, masks, gloves, and so forth. Each time Steven would become more involved, insisting on the operation, and emphasizing specific aspects of it, such as the use of the mask or saying good-bye to the mommy or daddy in the operating room.

At one point, the psychiatrist said, "You know, Steven, you are like the little boy in our story in some ways."

"How?"

"Well, you are in the hospital because your heart is sick too. And we have tried to give you medicine to make you better, but it isn't working, so, like Chris, we are going to have to give you a new heart."

Steven's reactions to the preceding play intervention were carefully discussed with his mother before the sessions with him, and at an appropriate pace, more details about the story were layered on in successive versions, incorporating aspects of Steven's experience, all within the play. Chris, for example, was so tired of waiting in the hospital for so long, but this was explained to be because he was too sick to go home. His new heart could come at any moment! He had an especially hard time saying good-bye to his mommy when she went home for Saturday night and his father stayed over with him, because he feared his mommy might never come back—but this was not true, for she always did.

Additional observations were made about Steven's negative reaction toward his father on these occasions, and his father's anger at this treatment, which his father took personally. Counseling with the father was directed at explaining how beneath Steven's rejecting behavior was anxiety over his mother's absence and his failing to understand and accept her need for a night's sleep. Specific suggestions were given to facilitate the "transfer" between parents, which included the father bringing something special with him on visits, phone contact with his mother on the nights when she was absent, coaching his mother to address Steven's underlying fears that she would not return, and helping his father adopt more of a "holding environment" for the boy's anger and frustration.

In the play, much of Steven's underlying anxieties were fleshed out in the character of Chris, and all of the details of surgery and postoperative care, including pain management and medication administration, were added. Steven would react excitedly to the psychiatrist's entrance, eagerly gathering his stuffed animals and recreating the stories over and over. In another version, it was "ET, the Extraterrestrial" who had the heart problem and was far from home, in the clutches of doctors who were trying to do him harm. Even ET eventually was successfully transplanted and returned home.

He often would ask the psychiatrist to supply voices:

"Have him [Chris] say 'good-bye' to his parents again," or "have his mommy tell him she will be coming back."

When the actual time of Steven's transplantation approached, after some initial reluctance, and to his mother's surprise, he said "Okay, let's go!" In the operating room, he calmly held the mask to his face himself, said good-bye to his parents, and was successfully transplanted.

Children and adolescents may react with mixtures of fear, anxiety, depression, confusion, and existential angst, which is mediated by cognitive level and the tendency to regress. This may take the form of oppositional behavior and irritability. They often wonder "Why is this happening to me? What did I do wrong?" (Slater, 1994).

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#### CASE ILLUSTRATION

One 12-year-old girl was constantly in tears whenever the subject of transplantation came up: "I had never had surgery before, any kind. And just the thought of having your heart taken out. There were just so many questions: 'Am I going to live?' 'Is it going to hurt?' 'How long is it going to take?' 'How am I going to recover?' 'Am I going to be normal?'"

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#### CASE ILLUSTRATION

An 8-year-old boy who came from a religious family and was refusing transplantation put his parents in an extremely difficult position. His mother felt tormented that if she signed the consent against his will and he did not survive, that she would never be able to forgive herself. After considerable work with a child psychiatrist, the underlying fear was revealed. The boy said that under no circumstances would he allow someone else to be killed so that he could have a new heart. With the help of a hospital chaplain, this boy's misconceptions were corrected, and he was successfully transplanted. [Both the preceding illustrations from Slater (1994), with permission.]

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Older children and families may benefit from viewing a videotape about children and adolescents who have undergone transplantation. One such film about cardiac transplantation in children includes interviews with donor families, as well as a family who lost a child after transplantation (Slater, 1992). Seven families have watched the film before cardiac transplantation; all have had positive reactions. Samples have included:

"The first reaction was relief that these kids existed, that they were apparently healthy. Our son could survive. It really worked. When we actually saw the donor families talking, it was enormously sad and moving, but in a positive way that somehow helped me."

"I felt reassured that I had no choice and that it was the only decision possible, as I listened to the parents of the child who died wrestle with their guilt over having put her through the surgery, knowing she would die without it."

"It made me feel better even though I cried during certain parts. It helps but it hurts at the same time."

"It was a helpful film which confirmed the emotions we went through...it also provided firsthand knowledge of what we would go through...you realize that no matter who you are or what walk of life you come from, you all have the same concerns, fears, anxieties and questions."

"It was hard to see the donor parents' sadness, talking about their son. It is so hard to think about the donor family, yet it's something I always find myself thinking of."

#### WAITING

The waiting period, between the time a child is listed for a transplant and the arrival of the donor organ, can last from days to months. This can be tantamount to psychological torture because the transplantation can happen at any moment or not at all. Either alternative can be devastating. Intervening complications such as infection can temporarily remove the child from the waiting list. Mean waiting times to transplantation by organ are depicted in Table 97.8.

Waiting list	Age Group (yr)	Mean Waiting Time
Kidney registrants	6-10	300 days
	18-34	891 days
Liver registrants	6-10	237 days
	18-34	392 days
Heart registrants	6-10	51 days
	18-34	214 days

From UNOS Scientific Registry Data, 1999.

**Table 97.8. Mean Waiting Times to Transplantation**

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#### CASE ILLUSTRATION

One parent commented that "waiting for the heart is like sitting in a tunnel with no light in either direction, there's nowhere to go."

Another described this period: "Waiting for the heart is a very helpless and scary feeling. You can't do anything. As a mother and as a parent, you're used to being able to take care of your children. If they have a fever you take them to the doctor, you put on cold soaks, you give them Tylenol. If they have colds you give them fluids. You can do a lot of things to cure little illnesses that they have. You can't do anything when you're waiting for a heart" (Slater, 1994, with permission.)

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Parents may consider interfacing with the community, either to request assistance in meeting the extraordinary costs of transplantation or in an effort to publicize the need for a donor organ with the hope that this will make a difference. Such exposure can have costs for the family in terms of loss of privacy, but may be seen as necessary.

Parents often regard the issue of organ procurement with ambivalence, as they wrestle with conflicting feelings that arise from survivor guilt or identification with the family who lost their child. On an emotional level, many parents fear they are “wishing” for another child to die, although they “rationally” know they have no control over this event. On occasion, two children in the same hospital might need an organ of the same size and blood type, with one listed higher than the other because he or she is more critically ill, for example. Such a situation can cause tremendously conflicting feelings for parents who might feel they are in a sense “competing” for the same organ; guilty feelings often ensue.

As time goes by, the waiting period often seems to wear patients and families down. If the child is hospitalized, mothers often literally move into the hospital for weeks or months at a time, such that marital relationships are entirely disrupted. Emotional and physical reserves of parents often become so depleted during a turbulent medical course that their own basic needs are neglected. As they respond to day-to-day events, often in turbulent cycles, the parents have little time to reflect on what has happened or replenish their emotional and physical reserves. The psychiatrist often can detect this trend and advise accordingly with simple suggestions that may act as “permission” for a guilt-ridden parent to leave the hospital temporarily, for example, to get a night's sleep or a decent meal. Parents may experience a sense of support and validation of their experience from speaking with the psychiatrist.

Parents commonly feel torn between the responsibilities of other children, work, and the child in the hospital, and may feel guilty about “failing” to meet all these demands, although the task may be impossible to carry out completely. If the family does not live near the hospital, these feelings can be exacerbated, as siblings are shunted off to other family members, often leading to resentment toward the parents and sometimes toward the sibling in the hospital. Siblings may have a variety of adjustment reactions, and the psychiatric consultant's duties may extend to counseling parents about issues with their other children and, on occasion, the psychiatric evaluation of siblings.

Parents may become resentful of what has happened to their lives and describe being short-tempered with one another. The parent in the hospital with the child may become resentful toward the working parent, who is not with the child on a day-to-day basis, dealing with events in the hospital. Marital problems that were present before transplantation obviously are worsened, and parents may become clinically depressed or develop anxiety disorders, such as one parent who began to experience panic attacks that required treatment.

“False alarms” may add to the feeling of unpredictability and helplessness, when parents receive word about a possible donor organ that does not come to fruition. Such instances can be even more anticlimactic and potentially traumatizing if the parent has already said good-bye to the child, who is moved to the operating room. In one instance, a mother became seriously depressed, returned home, and could not leave her bed for a number of days.

The patients themselves may become progressively more irritable and obstinate, straining their relationships with parents, who often are the objects of angry outbursts that test their abilities both to empathize and set limits with their children. In some cases, the parents cannot take extended leave from work, and the child may spend much of his or her time in the hospital without visitors.

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#### CASE ILLUSTRATION

In one such case, a 12-year-old boy developed strong, somewhat flirtatious attachments to some of the intensive care unit nurses, which became problematic when he began to engage in inappropriate behaviors, such as suddenly cutting the hair of one of the nurses with a scissors when she turned around. He later bit off the end of a glass thermometer, saying he was just “kidding around” (Slater, 1992). The consulting psychiatrist felt that fueling these behaviors was the boy's sense of frustration and helplessness at the long waiting period, exacerbated by his parents being unable to visit him during the daytime, and his being semiconscious during a witnessed cardiac arrest and resuscitation several days before these behaviors. Psychotherapy with the boy allowed him to discuss his lack of feeling safe in the intensive care unit, even in the presence of all the high-tech monitoring equipment. As a developing male adolescent, he felt his body becoming weaker and weaker, which severely affected his self-image and emerging sexual identity. The psychiatrist also assisted the intensive care unit staff in setting limits with this young man, which led to an abatement in these behaviors.

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#### SURGERY

When parents are notified that a suitable organ donor has been found, many report ambivalent feelings. One parent thought “Oh my God, it's here. Well, I don't want to go. I just felt like saying, ‘Give it to the next person, we're not ready’” (Slater, 1994). On the way into the hospital, she and her husband felt the impulse to turn off the exit and return home with their daughter, who was listed for a heart transplant. Parents struggle with the responsibility they bear for committing their child to transplantation, knowing that there are operative and postoperative risks, and sometimes are tormented by the thought that somehow the child could almost magically survive without the transplant. The ambivalence usually is worse if the child is waiting for a transplant as an outpatient and is not critically ill at the time of transplantation. In either case, parents experience guilt over making decisions that expose the child to the potential trauma of hospitalization and surgery, despite knowing it is the only way to save their child. If the child does not fare well, parents need maximal support to help them cope with their sense of responsibility for the outcome, however “irrational” that might be.

The moment of separation just before surgery is a moment many parents recall as an indelible mark in their minds. As one parent described:

I kissed her good-bye, turned toward the door to the operating room, and never turned around. It was because you make that separation, even though you still hear sound... You just have to do it. If you turned around, I think the impulse would be so strong they'd probably have to restrain you and drag you out (Slater, 1992).

Another recalled: “We certainly felt that we were leaving her in very capable hands, but there's all of the unknown questions, you know: ‘Are you going to see this child again? Is everything going to go through okay?’ And just simply walking away from it was a hard thing to do. Somehow I felt if I could just stay there it would be okay” (Slater, 1992).

During surgery, time often is telescoped for these parents who sit waiting to hear word on their child's progress. A visit from the psychiatrist at this time can be quite supportive.

Parents should be prepared both visually and descriptively for what they will see in the intensive care unit after surgery. A preoperative visit and detailed description often can help defuse parents' acute shock at witnessing their child with an endotracheal tube, intravenous lines, indwelling chest tubes, and high-tech monitoring devices. Despite explanations before surgery, they often are overwhelmed and confused by the complicated medical management in the immediate postoperative setting, and this anxiety may be exacerbated by the presence of other critically ill children (Bradford, 1990).

The conduct and sensitivity of the intensive care unit staff can greatly affect parents' emotional state during this period, when postoperative sedation, analgesia, and the presence of an endotracheal tube make communication with their child difficult. As the child's level of awareness returns, he or she may be irritable, combative, or withdrawn, and the psychiatrist may assist in recommending pharmacologic intervention or other behavioral techniques to help the child. The psychiatrist can be invaluable in helping to explain the nature of the child's experience to parents, as well as to staff (Bradford, 1990), and should conduct serial neuropsychiatric assessments. Strokes, metabolic derangement, medication effects, delirium, and hypoxia are among the many events that may compromise mental status in the perioperative period.

A common tendency among families is to compare their postoperative course with that of other families whom they have met. Unfortunately, although kidney transplant recipients often can be discharged within a week (Stuber, 1993a), other SOT recipients may remain in the intensive care unit for that long and spend weeks or even months in the hospital, subject to unpredictable complications. In the often elated atmosphere of a successful transplantation, it is easy for parents to think ahead to discharge, setting themselves up for inevitable disappointment at the first postoperative complication. Such events, like mild rejection episodes and postoperative fevers, are common, and can “burst the bubble” of the “honeymoon” phase described by some families immediately after transplantation. This deflation may herald continuation of the “roller coaster” ride, with cycles of high hopes and expectations alternating with periods of intense fear and worry. As the sense of reality sets in that the transplant is really not a “cure,” patients and families may feel quite disillusioned. The psychiatrist can both help prepare them for this process, as well as allow expression of these feelings in a supportive setting.

Thus, speaking with other transplant recipients can have both positive and negative aspects. In one respect, feelings can be validated by others who have gone through the same experience. On the other hand, because these experiences inevitably are individualized and subject to so many variables, patients may experience



a sense of frustration, failure, or disappointment if their expectations, which are modeled by the experiences of others, are not met.

As tubes and lines are withdrawn, and analgesia reduced, children can benefit from more contact with visitors and the child life department. Parents can read to their children, and videotapes can be viewed. Behavioral interventions such as guided imagery, story telling, and self-relaxation, and games such as hand-held computer games can continue to play a role during this period and be used to help children tolerate postoperative procedures. Such practices may decrease the need for sedation ([Bradford, 1990](#); [Bullock and Shaddy, 1993](#)).

### Developmental Considerations

As [Stuber \(1993a 1993b\)](#) describes, developmental issues affect the child's and adolescent's experience of and response to transplantation in a manner different from that in adults. Parents must be involved in the decision-making process, and adolescent assent or consent also is essential. Developmental regressions compete with the demands for autonomy common to all phases of childhood. The impact of developmental factors on illness has been approached from many different models, including ego psychology, self-psychology, and cognitive psychology, and delineated by many, including [Anna Freud \(1952\)](#) and others ([Bauman, 1981](#); [Nagera, 1978](#); [Schowalter, 1971](#)). Other writers have applied theories of affective development in the infant-caregiver dyad ([Emde, 1989](#)) to help understand how illness can affect relationship formation ([Parmelee, 1989](#)). The application of Piagetian theory to the progression of the cognitive understanding of illness has been reviewed by [Schonfeld \(1991\)](#). [Thompson and Vernon \(1993\)](#) and [Vernon and Thompson \(1993\)](#) have reviewed the literature on the effects of hospitalization and directed interventions on children. Developmental theory has been applied specifically to transplantation as well ([Sexson and Rubenow, 1992](#); [Slater, 1994](#)) ([Table 97.9](#)).

**Table 97.9. Developmental Aspects of Transplantation**

The patient-family system is exposed to the multiple stresses of acute and chronic illness, intrusive high-tech procedures, surgery, and hospitalization, often in an unrelenting fashion. Ethical issues, involving solid organ or bone marrow donations by family members, and quality-of-life issues may further complicate this picture. A fundamental purpose of the child and adolescent psychiatrist is to translate childhood experience into terms adults can understand and therefore respond to empathically.

When transplantation occurs in the infancy or early childhood, children are more likely to incorporate physical sequelae such as the scar and medical management (chronic medication and medical follow-up) into their developing identity and self-image. The subject of transplantation may be dealt with in a similar manner to adoption, explaining to the child from an early age what has happened to him or her and helping the child develop a narrative and more complex understanding about who he or she is as time goes by. This openness and acceptance on the part of the parents that the transplant is not a “secret,” but an integral part of the child's identity, may help facilitate adjustment and the child's acceptance of the requirements of life as a transplant recipient. Such issues apply to BMT as well, which is covered later in the chapter.

Although children may experience cognitive distortions, such as using immanent justice theorizing to explain their illness as a punishment for misdeeds ([Schonfeld, 1991](#)), it is continually surprising to physicians and parents alike how much children do understand. For example, the parents of a 4-year-old who had undergone cardiac transplantation at 2 years of age felt that she had little recollection of the events and were amazed when she played out features of the hospitalization in exact detail when given miniature hospital props.

In communicating with a child or adolescent about his or her illness, transplantation, procedures, or other aspect of medical care, the accepted approach is to be honest within a developmental orientation. In the pediatric transplantation population, these distortions often occur alongside normal childhood curiosity. In the setting of heart transplantation, cultural symbols and metaphors invest this organ with tremendous meaning:

We tend, in the medical world, to think of a heart as a pump. We've got to be reminded, sometimes by the patient, that the heart is an organ with a lot of mystery to it, if you will, a lot of history to it. The heart loves, the heart represents not simply that organ which sends the blood around, but that organ which we associate with some of our most wonderful human attributes. I mean love, heart as courage. We'll talk about an athlete who has heart; identifying the heart with valiant, courageous impulses. The heart with sentiment, with affection. Do we really think that one can take out a heart, and replace it with another heart, and not set off an extraordinary chain of associations on the part of the recipient? Will my new heart be valiant? Will my new heart be loving? Am I the same person now that I have a new heart? Am I that other person? [From [Slater \(1994\)](#), with permission.]

Children may wonder, for example, if it matters whether the donated organ comes from a male or female, black or white donor. Others queries have included children asking “how they will know whom to love, for example, when they receive their new heart: Should they still love their parents, or the family from which the donor heart came? They think of the sayings, ‘I love you from the bottom of my heart,’ or that someone's heart can ‘break’ because they miss someone, and apply this to the transplant experience and become understandably confused” ([Slater, 1994](#)).

Under the influence of tremendous anxiety, cognitive distortions can be exacerbated. This interface became apparent when a child began refusing her endocardial biopsies because she believed that each time, more “pieces” of her heart were being removed, such that eventually it would fall into her stomach. In her play, she repeatedly tied her stuffed animals down to perform “biopsies” on them and soothed them by saying “it's going to be all right.” This posttraumatic repetition perhaps could be understood as helping the child master her helpless feelings during catheterization. In ego psychological terms, one might characterize her as “identifying with the aggressor” or “turning passive into active.”

Another child told his mother he was worried that other children in the playground would start coughing, as he did, if they overexerted themselves. This projection of one's own experience on that of others is typical of the egocentrism in the preoperational Piagetian phase.

Latency or school-age children also respond to transplantation within a developmental frame. Interference with physical prowess in boys, and relationship development with peers in girls, can result from repeated hospitalization and illness. Academic performance can be affected by school absence or the neurocognitive sequelae of treatment, such as cranial irradiation in BMT. The development of self-esteem can be affected by the decreased opportunities for mastery that normally are associated with important affirming responses from role models.

Adolescent transplant recipients present unique challenges to the physicians working with them because of the seriousness of nonadherence, which is more prevalent in this age group. Adolescents react to contingencies of their illness, which undermine developmental needs for independence and control. The enforced dependency, although in line with regressive forces brought about by serious illness, may result in more demanding and boisterous behavior.

The psychological issues in this population have been reviewed by [Stuber \(1993b\)](#), who addresses several important points, including ethical concerns about the adolescent's right to refuse treatment, the need for systematic research to identify psychosocial predictors for outcome, specific developmental considerations such as the parents' involvement in the adolescent's “personal body functions and decisions,” and the psychological reactions to implantation of a foreign organ. As Stuber notes, hearts may be imbued with more philosophical meaning. An adolescent who was approaching his birthday, for example, said that he felt strange that he would be turning 16, but his heart, which came from a donor 2 years younger, would be only 14. Over time he came to experience his new heart as an integral part of his

identity and body.

Illness and medication side effects can dramatically affect appearance and physical and sexual development, having a significant impact on the adolescent's identity and relationships, and making him or her more self-conscious. Prednisone can cause weight gain, acne, and a cushingoid appearance. Cyclosporine causes hirsutism. BMT is associated with hair loss and cachexia. Adolescents may desperately struggle with the need to be "normal" like their peers, while at the same time accepting that he or she is different. Their sense of a possible foreshortened future may interfere with plans for college, a career, and intimate relationships.

Adolescents may struggle with decisions about who to tell about the transplant. This question may have antecedents in failure to confide in friends about the illness leading to transplantation, such as an adolescent with cystic fibrosis who is listed for lung transplantation and has told her friends over the years that she has asthma. The issue of who to tell usually centers around concerns over peer acceptance and may have painful aspects, such as one adolescent whose boyfriend's mother told her son not to get involved with the patient because she was a transplant recipient and might not live a long life.

Making it "public knowledge" that one is a transplant recipient can lead to celebrity status in school initially for an adolescent, but also can lead to intrusive questions about sexual functioning and other naive questions that must be fielded about life as a transplant recipient. An adolescent reported that he was initially treated like an invalid, and one classmate asked whether he had to carry oxygen with him on a date. These questions reflect the transplant candidate's concerns early on as well, but often are not addressed unless the consultant raises them in a sensitive way.

Adolescents often describe feeling out of step with their contemporaries in terms of both concrete issues like missed social events, as well as in a sense that many of them describe that they have grown up too quickly and have lost a carefree quality they see in their friends:

I guess at that point I just felt they weren't taking life serious, as seriously as they should. But I think in reality I was just taking it too seriously, like I felt like every minute was my last minute. And I was trying to be the best I could be because I thought maybe it was because I was bad that it happened to me. And I found that I was given this second life and I was making a mess of it because I was so tight and wound up about everything ( [Slater, 1992](#)).

Transplant recipients often talk about entering a health-related profession or other field where they can feel useful to others. One adolescent joined the volunteer fire department and relished in the idea of carrying a beeper with him; in fact, he used his fire fighting "on-call" schedule as a rationale for missing clinic appointments at times. Several have confided in me that they would rather be nurses because they cannot see themselves as the one actually "causing" the pain, but understand what it is like to be the patient, and feel they could more sensitively soothe children in pain and fear. Many of their aspirations and specific commendations reflect on the importance of supportive care in the hospital and the important role of hospital staff at crucial junctures in their recovery.

### **Pain and Anxiety Management**

The topic of pain and anxiety management in the pediatric transplant population has not been well studied or described ( [Green, 1994](#)). There is a literature on this subject in the pediatric population, especially in the psychology and pediatrics journals ( [Abu-Saad, 1984](#); [Bush, 1987](#); [Kuttner et al., 1988](#); [Manne et al., 1988, 1990](#); [McGrath and Hillier, 1989](#); [Siegel and Smith, 1989](#); [Varni et al., 1987](#)). [Bush \(1987\)](#) reviewed the literature on analgesic use in children, which suggests that children are undermedicated for pain compared with adults undergoing similar procedures. Undermedication can exacerbate pain behaviors and disrupt the trust between the child and physician.

In addition, there is good reason to expect that this population can benefit greatly from cognitive-behavioral techniques designed to diminish pain and anxiety associated with biopsies, catheterizations, venipunctures and central line placement, surgery, bone marrow aspirations, lumbar punctures, and many other procedures that may occur in these patients. These techniques have been used for such procedures in different populations ( [Bush, 1987](#)), especially in pediatric patients with cancer ( [Kuttner et al., 1988](#); [Manne et al., 1990](#); [McGrath and Hillier, 1989](#)). [Manne et al. \(1990\)](#) described a behavioral approach designed to decrease anxiety in children with cancer who undergo repeated venipunctures (see also [Cardona, 1994](#)). A combination of parent coaching, distraction, and positive reinforcement was associated with a decrease in parental anxiety and report of the child's pain, and less distress for both child and parent.

One relevant study used such techniques during right ventricular endocardial biopsies in pediatric patients ( [Bullock and Shaddy, 1993](#)), which are used to monitor for episodes of rejection in the cardiac transplant population. The techniques described included preparation, reassurance, deep breathing, imagery, distraction, and muscle relaxation. By their sixth biopsy, all patients discontinued the use of benzodiazepines, felt more in control, and reported less pain (which was associated with decreased autonomic measures). In addition, the length of time required to do the biopsies was diminished.

[Bush \(1987\)](#) and [McGrath and Hillier \(1989\)](#) have reviewed this literature from a developmental point of view, recommending an interdisciplinary approach. Early impressions that infants do not experience pain have been refuted, and it is clear that from several weeks of age, infants react to pain in gradually more differentiated patterns of response. Specific responses to painful stimuli as well as anticipatory anxiety and avoidance behavior follow a typical developmental sequence along Piagetian lines. Cognitive factors affect pain intervention in multiple areas that include the subjective pain experience, how to describe pain intervention measures, how to evaluate pain, how to implement the pain reduction techniques, and how much children will retain after being taught techniques to help them manage pain ( [Bush, 1987](#)). Prior experiences with pain also are modulators of later pain response ( [McGrath and Hillier, 1989](#)).

Pain behaviors may serve communicative functions in families. Parental responses, including anxiety before to a procedure and expectation of the child's fear or uncooperativeness, can significantly affect the child's experience of a painful stimulus and subsequent behavior. Heightened parental anxiety thus can exacerbate distress for a child undergoing a procedure ( [Bush, 1987](#); [Manne et al., 1988](#)). Family patterns of pain response are learned through modeling and reinforcement, and operant conditioning models also have been used to describe responses that reinforce pain behaviors as predominant coping strategies. In line with a learning model, instruments or even people that are associated with pain induction can later independently elicit anxiety, fear, avoidance, and complaints of pain. Nevertheless, most clinicians working in this area oppose a differentiation of pain between "psychogenic" versus "organic." Such a distinction can disrupt therapeutic alliances and usually is not relevant or able to be substantiated diagnostically or therapeutically.

In assessing pediatric pain, [Bush \(1987\)](#) suggests focusing on diminishing anticipatory anxiety, the prior history of pain experiences, and the environmental responses to pain behaviors. [Varni et al. \(1987\)](#) underscore the importance of combining cognitive, behavioral, and physiologic data in evaluating pain. In children, self-reported distress during a procedure may not habituate after repeated exposure, although it may appear to lessen to an observer. Developmental and psychological factors, such as age and fearfulness, may be more operative ( [Manne et al., 1988](#)). Adaptive coping mechanisms may include distraction, desensitization, information seeking, and parental presence. Visual analog scales ( [Bush, 1987](#); [Varni et al., 1987](#)) or "word descriptors" ( [Abu-Saad, 1984](#)) have been used successfully in pain assessment in children to help quantify the fear or pain experience.

Psychological techniques in pain management have included hypnosis, distraction, and relaxation with the help of breathing exercises. Other interventions may address family factors that are operative in the child's pain experience. The preparation aspect of intervention may use a modeling principle in exposing children to educational videotapes or other children who are using adaptive coping mechanisms. Younger children may benefit more from "imaginative involvement," a technique that takes advantage of their active fantasy life in creating stories and using hypnotic suggestions, than distraction ( [Kuttner et al., 1988](#)).

In summary, the BMT, peripheral blood stem cell transplant, and SOT pediatric population undoubtedly can benefit from psychological techniques to assist with anxiety, fear, and pain associated with the myriad of procedures these patients experience. There may be long-term benefits as well, such as improved compliance and the possible decrease in potential posttraumatic symptomatology.

### **Posttransplantation Life and Nonadherence**

#### *THE FIRST YEAR*

Lifestyle is irrevocably changed after transplantation and evolves in parallel with the medical course and nature of follow-up. There seems to be a differentiation with respect to issues involved in early follow-up and late follow-up, and the end of the first year after transplantation may be an important marker.

Initially, there are frequent clinic visits and a necessary immersion in becoming versed with the requirements of transplant life. Complications may occur during the first year, as the immunosuppressant regimen is adjusted. One parent summed up this period by saying: "Life then was centered around the hospital. We lived



transplant, we ate, we slept transplant. We were totally removed from the rest of our lives" ([Slater, 1994](#)).

Patients and families are more closely followed and monitored during the first year and live in the aftermath of having survived an extraordinary ordeal; they often describe having more powerful family bonds and pride in having weathered the ordeal. Being closer in time to the pretransplantation and transplantation experiences, they may be more likely to treat their bodies and the new organ inside with respect. They are much more task focused as they learn about administering medication and recognizing signs and symptoms of illness. Many of the social sequelae may not yet be apparent because children and adolescents are just beginning to reenter their social milieu and still might be treated as quite special by peers, a time-limited but perhaps supportive influence on self-esteem. In addition, many of the physical side effects may not be fully in evidence. For all these reasons, adolescents may be less likely to be noncompliant during the first year.

Biopsies and catheterizations are more frequent in the early period after heart transplantation and tax the skills of most parents, who are used to having more latitude with respect to setting limits with their children. In the world of transplantation, there often is little room for negotiation: medication must be given, doctor's appointments must be kept, biopsies and catheterizations must be done.

#### LATE FOLLOW-UP POSTTRANSPLANTATION

The end of the first year often is marked by an anniversary celebration by patients and families, and as routine follow-up diminishes in frequency, many families describe a discomfort with being more on their own, "waiting for the other shoe to drop" ([Slater, 1994](#)).

The uncertainty of posttransplantation life is a background anxiety that some patients and families are better able to suppress than others, and unexpected complications, which can include rejection, seizures, alterations in renal function, infection, and posttransplantation lymphoproliferative disorder, for example, can be powerful reinforcers of a constant background dread that may seriously affect patients and families. Other potential chronic effects of immunosuppressive therapy include hypertension, coronary artery disease (cardiac transplant recipients), bone disease, cataracts, and growth reduction ([Moskop, 1989](#)).

[Stuber \(1993b\)](#) underscores salient clinical considerations in the adolescent population, such as anxiety, impaired self-esteem, nonadherence, depression, and posttraumatic stress disorder (PTSD). One adolescent told me that he was troubled by the discontinuity in his conscious experience of the transplantation because he did not "see" the actual operation, but simply awoke with a scar on his chest and a new heart inside. He developed intrusive images of what he felt the actual operation looked like, seeing his heart repeatedly lifted out of his chest and replaced by another. Eventually, the treating psychiatrist obtained some videotaped footage of an actual heart transplantation, which the adolescent eagerly watched, and later reported his intrusive thoughts had abated. He later brought this tape to school to show his classmates in health class.

Major depression can be differentiated from organic mental syndromes or constitutional symptoms secondary to depleting medical illness. Useful criteria in making this differentiation include the presence of cognitive correlates of depression, including hopelessness, guilt, low self-worth, and suicidal ideation, and the absence of signs and symptoms of delirium or encephalopathy on formal mental status examination ([Stuber, 1993b](#)).

Parents also may exhibit posttraumatic phenomena, such as a mother of a transplant recipient who nearly died waiting for an organ, who was tormented for months by intrusive images of her daughter's funeral.

Over time, parents struggle with normal developmental issues in relinquishing control and responsibility to their adolescent children, but this is exacerbated by heightened anxiety over whether the adolescent will be compliant and recognize physical signs and symptoms. Events such as the adolescent leaving for college can have profound impact on a parent. As one mother described: "I think that this whole transplant life, is a life, and you can get lost in it. When my daughter went to college, she said, 'Mom, you know, like get a life.'

"I said, 'But you are my life. I've been taking you back and forth, doing things with you, and watching you all this time—you have been my life.'

"She said, 'Yeah but mom, now you have to get a life'" ([Slater, 1992](#)).

There may continue to be residual issues among siblings and between healthy siblings and parents that originated in the need for the parents to be so preoccupied with the ill child. These feelings may have distorted relationship development in the family to such a degree that psychiatric treatment is warranted.

Outpatient individual or family psychotherapy can be of great assistance, especially during the first year after transplantation, when most of the acute adjustment to transplant life takes place, and psychiatric follow-up may be made easier by patients' frequent visits to the transplantation clinic. During rehospitalizations for complications or routine biopsies or catheterizations, the psychiatric consultant should reevaluate, looking for the presence of any intercurrent psychopathologic process in the patient or family members or risk factors for nonadherence. The consultant should assess social adaptation, compliance behaviors, and the overall level of family and patient functioning, including school attendance and presence of any cognitive impairment. Appropriate referrals for neuropsychological testing or Committee on Special Education evaluation can be made.

#### NONADHERENCE IN TRANSPLANT RECIPIENTS

Most of the literature on nonadherence in the transplant population involves adult renal transplant recipients or pooled data from adults and children ([DeLone et al., 1989](#); [Dunn et al., 1990](#); [Kiley et al., 1993](#); [Rovelli et al., 1989](#), [Rovelli et al., 1989b](#); [Schweizer, 1990](#)), although some studies have looked specifically at the pediatric population ([Beck et al., 1980](#); [Bunchman, 1999](#); [Foulkes et al., 1993](#); [Korsch et al., 1973, 1978](#); [Phipps and DeCuir-Whalley, 1990](#)); [Bunchman \(1999\)](#) cited data ([Blowey et al., 1997](#)) suggesting that lack of adherence in the pediatric renal population is responsible for 93% of the loss of all allografts, with 20% to 40% of children nonadherent with their medical regimens. Possible interventions to reduce nonadherence include the simplification of therapy, support groups, techniques to remind patients to take medication (alarms, pill boxes), once-daily dosing, transdermal preparations, and small pills or liquid preparations with reasonable taste ([Bunchman, 1999](#)). Diaries, logs, and frequent visits with health care specialists who can troubleshoot and monitor compliance also can be valuable. There is literature on general nonadherence issues in medically ill adolescents that has been reviewed ([Cromer and Tarnowski, 1989](#)).

Shapiro and Fingerth (unpublished data) reviewed the literature on nonadherence in the adult cardiac transplant population and found a robust association between nonadherence and both global psychiatric risk and personality disorders. Global psychiatric risk was estimated clinically as low, moderate, or high by the psychiatrist after reviewing the prior psychiatric history. Included among the items scrutinized were any history of substance abuse, cognitive impairment, personality disorder, social stressors, prior level of functioning, previous history of nonadherence, social supports, interpersonal problems with treating physicians, and knowledge/expectations about transplantation. For those patients with high global psychiatric risk, 50% were noncompliant. Personality disorders were diagnosed clinically as being nonexistent, mild, moderate, and severe, with patients having severe personality disorders noncompliant 100% of the time.

[Korsch et al. \(1973\)](#) examined the psychosocial outcome of 35 pediatric renal transplant recipients and found that, within a year, most normal family patterns had returned if the child was healthy, with most of the disruption in family functioning occurring during this year. Ten of the 35 patients had scores indicating maladjustment on personality measures, most apparent in the anxiety and self-esteem scales. Eight patients had psychopathology, with depression the "most specific test finding." Transplant recipients with personality disturbances were more likely to be noncompliant. Seventeen of 19 older adolescents were in college or working. Eleven of 16 younger patients were in school. Leisure activities were age appropriate. Anxiety over rejection and side effects of steroids emerged as significant clinical issues.

[Korsch et al. \(1978\)](#) found that risk factors for nonadherence with immunosuppressive medication included sex (female greater than male), adolescence, premorbid deviance on personality scores, absence of the father, and lack of community support and other resources.

[Beck et al. \(1980\)](#) reported a 43% prevalence of nonadherence in a sample of pediatric renal transplant recipients and found that all of the noncompliant patients were adolescent. The noncompliant patients seemed to have less parental involvement in supervising their medication and attending clinic visits. An intervention program including counseling and instruction resulted in five of the noncompliant patients becoming compliant. None of this latter group of patients lost their grafts, as was the case with six other noncompliant patients. Notebooks in which medication administration and laboratory results were recorded have facilitated compliance.

[Rovelli et al. \(1989a\)](#) found nonadherence in renal, heart, and liver transplant recipients to be higher in the age group younger than 20 years. After the initial 3-month period posttransplantation, it was reported that nonadherence caused graft loss more frequently than unmanageable rejection episodes in compliant patients.

[Schweizer \(1990\)](#) reported that clinic visit nonadherence was associated with medication nonadherence.

Factors that predict compliance in the outpatient setting also have not been well studied in the pediatric BMT population. One study in outpatient adolescents with cancer (not specific for BMT recipients) showed that nearly 50% were noncompliant with oral medication regimens ([McConville et al., 1990](#)). Risk factors for nonadherence included less developed concepts of illness, less perceived vulnerability, greater denial, and relatively decreased orientation toward the future ([Tamaroff et al., 1992](#)). A study that looked specifically at compliance in inpatient pediatric BMT recipients reported greater than 50% nonadherence rates as inpatients, most commonly found with oral antibiotics, and most prevalent in the preschool or school-age population ([Phipps and DeCuir-Whalley, 1990](#)). More research in this area is needed.

[Hesse et al. \(1990\)](#) suggested that nonadherence with corticosteroids may be more prevalent in the pediatric and adolescent population. [Foulkes et al. \(1993\)](#) found that compliance with different medications may vary within an individual's regimen and underscored the importance of assessing compliance to each medication.

[Bradford \(1990\)](#) reviewed other risk factors for nonadherence in patients with chronic renal failure, which included "side effects, duration, and complexity (of medication); the doctor-patient relationship; family factors: instability or discord, poor communication, lack of support from family members, maternal mood, poor maternal understanding of treatment, and lack of supervision; poor understanding, perceived vulnerability and doubts about the effectiveness of the treatment" ([Bradford, 1990](#)). Periodic psychosocial assessment of transplant recipients as outpatients was recommended.

In one series of 28 pediatric liver transplant recipients, 34 instances of nonadherence were documented, with a mean of 2.19 episodes of rejection before the nonadherence was identified ([Molmenti et al., 1999](#)). The mean age of transplantation was 9 years, with the mean age of nonadherence being 14.8 years. The authors conclude that the clinician should have a high index of suspicion for lack of adherence when adolescent patients present with repetitive episodes of rejection associated with low immunosuppressant levels.

Preliminary results from a study of nonadherence in pediatric heart transplant recipients found nonadherence in 13 of 64 (20%) patients and was significantly associated with adolescent age and "late" rejection occurring greater than 1 year after cardiac transplantation. In noncompliant patients, graft survival dropped off precipitously in stepwise fashion from 1 year posttransplantation, such that only 30% graft survival was present at 5 years in these patients ([Douglas et al., 1993](#)). This is alarming because eight of these patients lost their grafts (two retransplantations and six deaths). One of these patients maintained a powerful sense of denial and invincibility throughout the entire transplantation experience. He later became secretly noncompliant with his medication, rejected the first heart, and almost died before being retransplanted. He eventually died several years later. Because of this alarming statistic, in 1993, a noncompliance treatment plan was instituted that included:

- Pretransplantation and posttransplantation teaching, specifically about noncompliance
- Assignment of patients to a primary pharmacist who worked directly with the transplantation coordinator and family
- Demonstration of adherence to treatment while awaiting transplantation if there was a preoperative noncompliant history
- Mandatory psychiatric referral and follow-up
- Inpatient and outpatient retraining of patient and family
- Frequent (weekly or biweekly) immunotherapy drug monitoring and clinic visits
- Monthly peer support group meetings
- Daily home visiting nurse if indicated and feasible
- Paid transportation for visits
- Liaison with school nurse
- Transplantation coordinator pouring medications weekly
- Child welfare agency intervention

When these data were updated in 2000, it was found that of 156 children who had had heart transplantations since 1984, there were 129 survivors at a mean follow-up age of  $5.8 \pm 3.9$  years, with 23 surviving more than 10 years. Seventy-seven were attending school or college, 6 had graduated from college, and 11 were working. Despite the noncompliance treatment plan, 31 patients died after discharge, and noncompliance was the primary factor in the death of 50% of these patients. In these follow-up data, which looked at patients both before 1993 and after 1993:

- Compliant patients had a >90% overall survival rate after transplantation.
- The initial incidence of nonadherence had not changed despite an intensive prevention and treatment regimen.
- The frequency of nonadherence increases with time in long-term survivors, particularly when the young child reaches adolescence.
- Noncompliance is significantly associated with adolescent age.
- Rejection frequency and episodes of late rejection and hemodynamic compromise were significantly higher in all nonadherent patients.
- Although survival of nonadherent patients has improved with focused therapy (60%), it is still significantly less than in compliant patients (90%).
- Nonadherence continues to be the leading cause of late graft loss and death in pediatric cardiac transplant recipients.
- Adolescents are at constant risk despite previous compliance, and a high index of suspicion is warranted.
- Prevention and treatment of nonadherence is labor intensive but may improve survival.

Contrary to expectations, a significant number of patients who were transplanted before 1993, when they were preadolescents, and who were initially compliant, later as adolescents became noncompliant. This phenomenon seemed to counter the prevailing belief that children who "grew up" ill would be less likely to be noncompliant as teenagers. Based on this author's experience, some patients continued to have significant morbidity and mortality from nonadherence for the following reasons:

- They continued, despite the transplantation team's best efforts, to miss medical and psychiatric clinic visits and blood drawing for immunosuppressant blood levels, and in our experience, the transplantation team has little leverage to "force" patients to be compliant. Social service can be helpful at times to younger patients, although the adolescent sample remains the most difficult to control. Psychiatric hospitalization usually was an ineffective option in the treatment of such patients, even if a case could be made for the suicidal potential of nonadherence. This is because psychiatric hospitals in general are uncomfortable managing medical problems, or the patient's insurance plan had limited psychiatric inpatient coverage. In several instances in which patients could be admitted, the limited length of psychiatric admissions (1 to 2 weeks) did little to solve the problems causing nonadherence.
- Peer support groups were voluntary, and although viewed as quite necessary by the transplantation team, clearly are not sufficient to prevent nonadherence.
- It was difficult to mobilize appropriate psychiatric and social services that potentially would address factors contributing to nonadherence, such as conduct problems, family psychopathology, patient psychopathology, and psychosocial discord, especially when such factors occurred in combination. In the author's opinion, the type of intensive psychiatric service, either extended psychiatric partial hospitalization, or a residential facility with psychiatric, nursing, and medical follow-up, that might have a chance of treating these patients successfully *simply does not exist*. Intensive efforts to find such placements by our transplantation team, or even to create them, have not yet been met with success.

In a sample of 53 pediatric heart and heart-lung transplant recipients (mean age, 10.3 years), 30% were found prospectively to be nonadherent to the treatment regimen ([Serrano-Ikkos et al., 1998](#)). Interestingly, the heart-lung transplant group was significantly less adherent than the heart transplant group. A greater number of adolescents in the latter group may have explained this finding. However, pretransplantation adherence was not studied, and different chronic illnesses before transplantation in the heart-lung transplant group (e.g., cystic fibrosis) also may have contributed to this finding. Children living with both biological parents had higher adherence than children living in single-parent or blended families, suggesting the possible protective factor of support. These findings may help target specific interventions to families and patients who may be at higher risk for nonadherence.

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#### CASE ILLUSTRATION

An adolescent young man who was secretly noncompliant as a 13-year-old later confided: "I guess when you're that age you think you're invincible, especially after going through a transplant. I mean hey, you were close to death and you made it. You're even more invincible now. I mean you were just a normal invincible thirteen year old before, now you kind of feel I guess, for lack of a better *immorta*. Yeah, you laughed in the face of death, big deal. But in the end, if you don't take your medicine, he's going to laugh back really hard. Probably why I needed a second transplant is one very big reason, stupidity: I was too proud to take my medicine in front of other kids. I had that, you know, the peer pressure, the look of someone turning around while I'm taking it. And it really got to me, it did. The reason I spoke up about not taking my medicine is that I realized I was hurting myself. I realized whether consciously or not I was committing suicide. Whether I actually thought of it that way or not, I was killing myself. So, while the doctors were going crazy trying to research every angle on this of why I could be getting sick, I just said, "Look, I wasn't taking my medicine, that's why I've been getting sick." I paid the price, losing a transplanted heart and having to go through it again was painful, not to mention I lost the trust of a lot of doctors." [From [Slater \(1994\)](#), with permission.]

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Based on available data, it would appear that risk factors for nonadherence in the pediatric transplant population include:

1. Adolescence
2. Conduct disorder, sociopathy, or other significant personality disorder
3. Affective disorder, including major depression and perhaps other, more chronic, indolent depression with impaired self-esteem such as dysthymia; possibly the presence of another psychiatric disorder such as PTSD, which has been receiving more attention as an identifiable sequela in pediatric patients with cancer, and is reviewed in the [Chapter 96](#) in this volume
4. Family psychopathology, possibly an absent father, and poor supervision by parents
5. Prior history of nonadherence
6. Cognitive limitations and problems understanding the medication regimen by adolescents or parents
7. Low perceived vulnerability and expectation of benefit from treatment, including a sense of a foreshortened future the patient feels powerless to affect
8. Poor physician–patient relationship or continuity of care
9. Inadequate resources to meet treatment requirements (e.g., transportation, financial)
10. Poor understanding or acceptance of the need for commitment to treatment and follow-up

Warning signs for nonadherence in this population may include ( [Douglas et al., 1993](#)):

1. Late rejection
2. Variations in cyclosporine levels or reluctance to have them drawn
3. Casual reporting of missed doses of immunosuppressants
4. Missed or delayed clinic appointments
5. Not reporting illness

Management issues should include treating nonadherence as a phenomenon with a differential diagnosis ( [Cromer and Tarnowski, 1989](#)):

1. Direct physical assays should be used when possible to monitor compliance.
2. Interventions should be directed at presumed underlying causes and risk factors.
3. Nonadherence should be treated as an issue “shared” by the patient, family, and physicians and approached from a task-oriented, problem-solving point of view.
4. The clinician should combine interventions that use closer monitoring (more frequent clinic visits), and help the patient with organization strategies (use of alarm watch, pill boxes, carrying extra doses of medication at all times).
5. Psychiatric involvement and individual and/or family psychotherapy and pharmacotherapy should be used if appropriate.
6. Pain should be managed adequately.
7. Increased involvement of parents or parental surrogates should be considered.

Adolescent issues affected by transplantation should be anticipated even before surgery and discussed with the patient in an appropriate fashion to help identify potential problem areas. Nonadherence should be addressed early on with the patient and family as a major side effect of treatment and serious cause of graft loss and death. This discussion should continue after transplantation. Specific areas that might be addressed include concrete help with side effects (e.g., helping to establish diet and exercise regimens, advice on removing excess hair) or peer issues. Adolescents as young as 10 years of age, depending on their level of maturity, often can be taught to manage their own medication from the time they are in the hospital and should be put in touch with other transplant recipients who can provide opportunities for modeling coping behaviors.

For adolescent patients in particular, outpatient follow-up should be considered as a routine measure. An early alliance between an adolescent and mental health specialist may be of great value in facilitating work later, when the adolescent is at higher risk for nonadherence (see later), and allow early identification of risk factors and intervention.

In a study that looked at brittle diabetic patients ( [Moran et al., 1991](#)), many of whom are presumed to have psychosocial factors that exacerbate their medical course, intensive inpatient psychotherapy three to four times weekly, for an average duration of 15 weeks, was associated with improved diabetic control, which was maintained at 1-year follow-up. One might extrapolate from such a preliminary study and suggest that severely noncompliant adolescent transplant recipients should be hospitalized for a period of weeks to months in a medical facility that can provide a multidisciplinary approach to treatment that includes a strong psychiatric component. It is hoped that insurance companies will reimburse such services, which may have a strong impact on patient survival.

## SPECIFIC ORGAN SYSTEMS

### Bone Marrow Transplantation and Peripheral Blood Stem Cell Transplantation

#### OVERVIEW

The psychiatric aspects of BMT were reviewed in two articles by [Andrykowski \(1994\)](#) and [Wolcott and Stuber \(1992\)](#), and in a more recent paper by [Andrykowski and McQuellon \(1998\)](#). The use of BMT has undergone a rapid increase since the late 1970s because of better survival rates, the expansion of its use to treat a greater variety of illnesses, and the growth of alternative donor pools besides an identical twin sibling.

The procedure itself is offered only as a final option in diseases where conventional treatments will not or have not sufficed and that include leukemias, lymphomas, breast cancer, neuroblastoma, ovarian cancer, germ cell tumors, melanoma, multiple myeloma, and malignant gliomas ( [Abramowicz, 1992](#); [Andrykowski, 1994](#); [Trigg, 1988](#); [Wolcott and Stuber, 1992](#)). In the pediatric population, BMTs also have been used to treat aplastic anemia and congenital metabolic and immunologic disorders. Cytotoxic drugs, often in combination with total-body irradiation (TBI), are used to wipe out the patient's own bone marrow over a 4- to 10-day period while the patient remains in relative isolation, often in specialized laminar air-flow rooms, with visitors usually required to wear gowns, gloves, and masks. Healthy marrow is then delivered by intravenous infusion. BMTs are categorized by the source of the marrow to be transplanted, which is either autologous, allogeneic, or syngeneic.

Autologous BMTs use the patient's own marrow or peripheral blood progenitor (stem) cells, which are harvested before BMT and reinfused after high-dose chemotherapy, and is used in lymphomas, sarcomas, and advanced breast cancer ( [Andrykowski, 1994](#); [Andrykowski and McQuellon, 1998](#)). Stem cells are harvested on an outpatient basis by passing peripheral blood through a machine that collects white blood cells, which have progenitor cell function, with the remaining blood returned to the patient, and blood growth factors sometimes are used to stimulate production of white blood cells before the collecting of stem cells ( [Andrykowski and McQuellon, 1998](#)). Immunosuppressant medication is not necessary, and there is less risk of graft-versus-host disease (GVHD), where the infused white blood cells react to the host as being foreign and mount an immune reaction. Ninety percent of children receiving BMT undergo significant pain secondary to the mucosal damage and desquamation after high-dose chemotherapy, which produces oropharyngeal inflammation, ulceration, and oral mucositis ( [Pederson and Parran, 1999](#)). Research indicates that continuous infusion of opioids at milligram per kilogram dosage levels that may need to exceed that administered to adults ( [Pederson and Parran, 1999](#)). This higher dosing may be secondary to the more rapid tolerance that develops in children compared with adults, and the relatively higher doses of mucotoxic chemotherapy regimens used in children, as well as the greater attention paid to behavioral indicators of pain; the importance of using self-reports was emphasized. These authors reviewed literature suggesting that patients receiving peripheral blood stem cell transplants may undergo mucositis for shorter periods because of more rapid bone marrow recovery, possibly due to the use of different chemotherapeutic regimens than in BMT.

Allogeneic transplants are used to treat hematologic malignancies and immune disorders ( [Andrykowski, 1994](#)) and use human lymphocyte antigen (HLA)–matched marrow from a family member or other donor, with the benefit that tumor cells are not part of the infused marrow ( [Abramowicz, 1992](#)). GVHD is more common than with autologous BMTs. Syngeneic transplants use marrow from an identical sibling donor.

Bone marrow transplantation typically involves a hospitalization of at least 5 to 6 weeks and can require a much more lengthy hospitalization of several months because of the high rate of complications, with a mortality rate of 5% to 20% with autologous transplants and 10% to 30% with allogeneic transplants ( [Abramowicz, 1992](#); [Andrykowski, 1994](#)). In general, patients with leukemia fare better than those with solid tumors, with disease-free survival rate at 2 years approaching 60% with early leukemia ( [Table 97.2](#)). Many factors have been associated with survival rates, including the type of tumor, stage of tumor before treatment, age and physical

state of the recipient, origin of the marrow, type of chemotherapy used before transplantation, the actual transplantation protocol used, and the experience of the transplantation center itself ([Abramowicz, 1992](#)).

After discharge, patients often are in an immunocompromised state, with normal immune function not returning for up to 1 to 2 years posttransplantation, thus placing the patient at moderate risk for dangerous infections and requiring a strict care regimen on the part of the patient and family. Acute GVHD occurs and runs its course within 30 to 100 days posttransplantation and commonly affects the liver, gastrointestinal tract, and skin. GVHD is differentiated from rejection in SOT, where the host is mounting the immune response. Chronic GVHD occurs later and does not resolve, although it can be managed medically ([Andrykowski, 1994](#)).

Thus, even though the patient may survive the initial malignancy, postoperative morbidity may include infections, GVHD, graft failure, primary malignancy relapse, secondary malignancies, and a multitude of treatment-associated side effects that may affect a variety of organ systems, including cognitive function ([Andrykowski, 1994](#); [Wolcott and Stuber, 1992](#)). Recovery thus can be broken down into the acute phase in the hospital, a slower, subacute phase that may last months after discharge, and a significantly prolonged stage of "adjustment and rehabilitation" ([Wolcott and Stuber, 1992](#)). Conceptualization of the process in phases, as in SOT, can lend itself to a better understanding of the psychological sequelae.

Psychosocial or psychiatric evaluation of BMT candidates may play a less crucial role in the selection process because bone marrow is a "renewable resource" whose supply is not limited as in SOT. Nevertheless, because of the expense of the procedure, its significant medical and psychological morbidity, and debate over the allocation of health care resources, there may be increasing pressure to establish priorities along guidelines that take into account survival rates and quality of life ([Andrykowski, 1994](#)). Research has been extremely limited, especially in the pediatric population, in looking at psychosocial variables that may predict compliance and medical/psychological outcome in patients and families.

#### PSYCHIATRIC SEQUELAE AND INTERVENTIONS

A body of literature addresses the neuropsychiatric sequelae and psychological adjustments in pediatric BMT recipients and their families ([Andrykowski, 1994](#); [Arvidson et al., 1999](#); [Dermatis and Lesko, 1990](#); [Gardner et al., 1977](#); [Kramer et al., 1992](#); [McConville et al., 1990](#); [Parkman, 1986](#); [Patenaude et al., 1979](#); [Pfefferbaum et al., 1977, 1978](#); [Pot-Mees, 1987](#); [Smedler et al., 1990](#); [Stuber, 1993b](#); [Stuber et al., 1991](#); [Trigg, 1988](#); [Wiley and House, 1988](#); [Wolcott and Stuber, 1992](#); [Wolcott et al., 1987](#)).

Pediatric patients receiving BMT are thought to be at higher risk for cognitive deficits than children receiving standard cancer therapy because they typically receive initial chemotherapy, followed by a conditioning regimen consisting of TBI, chemotherapy, or both ([Arvidson et al., 1999](#)). [Arvidson et al. \(1999\)](#) noted that although children treated with autologous BMT tested for general intelligence in the average range, such testing was insufficient to categorize learning problems whose identification required more specific neuropsychological testing. They found more learning difficulties in children receiving cranial radiation with young age at diagnosis and that a long follow-up time correlated with lower performance on neuropsychological testing, and recommended that specific tests evaluating attention and memory be administered.

[Arvidson et al. \(1999\)](#) cite a study ([Kramer et al., 1997](#)) that found that there was a 5-point decline in IQ 1 year after BMT, in which the authors suggested that psychosocial factors may be important. Long-term follow-up studies are few, although one found deficits in perceptual function and fine motor speed ([Smedler et al., 1995](#)). An interesting study in the adult population ([Gregurek et al., 1996](#)) suggests that anxiety may be related to the incidence and severity of acute GVHD through potential immunomodulatory mechanisms.

A study ([Cool, 1996](#)) examining the effects of BMT on neuropsychological function in children, found that prior cranial radiation therapy, although improving survival rates in acute lymphoblastic leukemia, caused effects on cognition (lower IQ scores and effects on memory) even before BMT. The TBI and chemotherapy regimens that prepare a child for BMT seemed not to have significant independent effects on cognition at 1-year follow-up post-BMT, although as a whole, the BMT group showed lower achievement, and a subgroup of children did evidence declines in verbal IQ. Longer-term follow-up of BMT recipients found declining IQ scores over time, with greater effects seen in younger children and those with prior cranial radiation therapy. This has implications for remedial services, which should target memory and organization skills during school reentry, as well as for in-hospital or at-home schooling before the child is able to return to school. Follow-up testing should go beyond 5 years because many effects occur late.

In a neuropsychological study of patients after BMT, children who had received chemotherapy only did not differ significantly from those receiving chemotherapy and TBI ([Simms et al., 1998](#)). These authors note, however, that there does appear to be a subset of children 1 to 5 years after BMT who appear to have significant declines in neuropsychological function. Cognitive effects on children younger than 3 years of age tend to be more variable across studies.

The first year after BMT may be a time when children are especially vulnerable to emotional difficulties, with effects on social competence and self-esteem, and may represent an opportunity for psychological intervention ([Phipps et al., 1995](#)); in this particular study, no significant declines in neuropsychological function were found, although 21 of the 25 posttransplantation survivors in the sample were 6 years of age or older.

The experience of pediatric BMT can be conceptualized in terms of emotional stages ([Patenaude et al., 1979](#); [Pfefferbaum et al., 1978](#)). [Pfefferbaum et al. \(1978\)](#) describe a 10-stage process, with implications for the need for psychological management. Many of these stages have correlates in SOT, as discussed previously. Chronologic hallmarks include:

1. The exigencies of the informed consent/assent process and living donor considerations such as putting a healthy sibling at risk. Many authors note the unrealistic expectations on the part of the patient and family that often accompany this stage ([Patenaude et al., 1979](#); [Pfefferbaum et al., 1978](#)). Psychological distress in parents during this stage has been studied ([Dermatis and Lesko, 1990](#)) and suggests that parents experience a variety of psychiatric symptoms that may inversely correlate with the quality of the communication between parents and the physician. Parents may not fully grasp all aspects of the informed consent process, and single parents may be more at risk for psychiatric symptoms. Psychological intervention may be especially important during this stage to facilitate communication and the use of coping strategies.
2. Pretransplantation evaluation (similar to that in SOT).
3. Effects of separation from school, home, and friends.
4. The intense feelings of separation and helplessness during the child's isolation for several hours for TBI and the inevitable chills, nausea/vomiting, and diarrhea that follow.
5. Transplantation day, with the specific risks of general anesthesia and bleeding for the donor. The bone marrow infusion itself may be a somewhat "anticlimactic event, greater in symbolic than immediate medical significance" ([Patenaude et al., 1979](#)). The isolation of the sterile room may pose a significant stressor.
6. The waiting period, during which time the family "settles in" to the hospital, and parents may attempt to counter feelings of helplessness by carefully following blood counts with attendant anxieties.
7. Watching for the donated marrow to "take," approximately 2 weeks after the infusion, with children sometimes feeling a sense of "responsibility for the outcome,... assuming blame for infiltrated intravenous lines, unsuccessful venipunctures, nausea, diarrhea, and even results of blood counts." [Patenaude et al. \(1979\)](#) describe the "vacillation between...fear and hope."
8. Medical complications and psychological reactions to "waiting again" for marrow activity to stabilize, with children perhaps most prone to withdrawal, depression, and irritability; and parents feeling "rejected and embarrassed"; and the possible decrease in support from family members, who may become less available, as well as from the staff itself.
9. Anxiety over reentering the social milieu as discharge approaches, owing to the change in physical appearance (hair loss, weight loss, cushingoid changes), and the inevitable disappointment that the child is not "well."
10. The relative isolation, dietary restrictions, chronic medication administration, and uncertainty that characterizes life after discharge ([Pfefferbaum, et al., 1978](#)). BMT survivors may be even more compromised physically than SOT recipients. The enforced dependency and reliance of the child on a single figure, often the mother, poses a significant stressor for the entire family ([Patenaude et al., 1979](#)).

The child and adolescent psychiatrist is perhaps best used as an integral member of the BMT team who makes contact with the family during their initial evaluation and serves as a liaison to the rest of the hematology–oncology team. Pretransplantation evaluation of the patient, family, and living related donor, as well as neuropsychiatric assessment and family support during the procedure, also are important avenues for both treatment and prevention of psychiatric morbidity. Attention should be paid to the relationship between the referring physician or institution and the hospital administering the BMT because the patient may likely return to the original hospital at some point, and families may experience complicated feelings about the two institutions, with potential feelings of abandonment, issues over



transferring care, or possible splitting mechanisms ([Atkins and Patenaude, 1987](#)).

A waiting period between the decision to transplant and the BMT procedure itself may occur because of the availability of hospital beds, financial factors, or failure to identify suitable unrelated or related donors. Like the waiting period in SOT, this can be associated with ambivalence about the procedure, significant anxiety over intercurrent survival or progression of illness, and the psychological consequences of possibly not living near the transplantation center ([Andrykowski, 1994](#)). In the case of a living related donor, donor-related feelings may be less ambivalent because an organ is not being "sacrificed" ([Gardner et al., 1977](#)), compared with kidney donation, for example, where recipient guilt is common. In BMTs, the donors nevertheless may experience guilt and perhaps added pressure because the request for marrow donation does not involve the irretrievable loss of an organ. Solid organ donation may represent a sacrifice that makes absolution from further responsibility, or ambivalence about donating, more "justifiable" ([Gardner et al., 1977](#)). Other siblings may feel "relief" as well as feeling "left out" ([Patenaude et al., 1979](#)). All these factors have an impact on parents, siblings, and patients alike ([Pot-Mees, 1987](#)), and mothers of BMT recipients may be particularly prone to these sequelae because they may spend an extraordinary amount of time in the hospital with their child.

Simply identifying and assisting the family in coping with the extensive array of individual stressors can represent tremendously supportive interventions. These stressors may alter family roles and finances and include long geographic distance from home, the tremendous fear of death, and the tendency for attention to center around the BMT recipient to the exclusion of other family members. The latter can evoke feelings of both guilt and resentment over individual needs not being met ([Andrykowski, 1994](#)). Addressing these problems is paramount because psychiatric sequelae can affect the relationships between the members of the team working with the child and his or her family network, which can have deleterious effects on the BMT recipient ([Andrykowski, 1994](#)). The psychiatric consultant can be of great help in assisting the rest of the medical staff in their understanding and management of the patient and family. He or she also may be of great assistance to parents in helping them to manage both their own feelings and the reactions of their children. [Nelson et al. \(1997\)](#) studied the effects of stem cell transplantation on the mothers of pediatric recipients, concluding that maternal anxiety during this treatment approximates anxiety in the unstable, acutely ill patient with cancer, and surmising that this is the second time mothers experience an acute risk to their child's life (the first being the diagnosis). An awareness of maternal anxiety is paramount in assisting clinicians in intervening because such anxiety may affect supportive functions for the child, as well as the reporting of childhood problems by the mother. Heightened depressive scores in mothers also were noted, and this phenomenon had an equivalent impact on care of the child. Intrusion coping, consisting of the parent reviewing feelings and information associated with the transplantation, seemed to be adaptive and was encouraged by the authors. Perception of adequate social support seemed to correlate inversely with depressive symptoms.

Behavioral and affective responses in pediatric BMT recipients may include depression, sleep difficulties, anxiety, apathy, withdrawal, irritability, and regression, as well as organic mental syndromes from neurotoxic central nervous system irradiation, chemotherapy, and infectious and metabolic effects ([Andrykowski, 1994](#)). Children often experience a gradually decreasing tolerance of and increasing opposition to procedures, along with increasing fearfulness and separation anxiety, all of which may cause havoc for parents ([Gardner et al., 1977](#); [McConville et al., 1990](#)). For the child, the need for intravenous hyperalimentation due to decreased oral intake, the denial of symptoms due to the (legitimate) fear of further intervention, and pronounced helplessness and dependency may further complicate the medical aspects of the procedure ([McConville et al., 1990](#)).

One study suggests that, over the long term, pediatric patients who undergo allogeneic stem cell transplantation are at risk for emotional difficulties ([Felder-Puig et al., 1999](#)). The sample consisted of patients between 14 and 30 years of age who had undergone stem cell transplantation at least 2 years before the assessment. Thirty-five percent had significant levels of anxiety, with 62% showing elevated levels of sensitivity and vulnerability (both perhaps etiologically related to the history of multiple invasive procedures and the fear of relapse), which were higher than those of the bone cancer survivor control group. Thirty-five percent of stem cell transplantation survivors reported dissatisfaction with their romantic lives (perhaps related to effects of their treatment on sexual function, diminished opportunities for meeting people, or negative effects of treatment on body image). Only 12% had major problems academically or occupationally, although the 12% were more dissatisfied with their performance compared with a control group of bone cancer survivors. Stem cell transplantation survivors did not differ from control subjects in multiple other domains that were evaluated, nor was the maladjustment rate greater than in the general population. Eighty-five percent of stem cell transplantation survivors were able to return to their schools or jobs. However, restrictions in physical activities were the main problems reported, and were greater than those in the bone cancer survivor group.

One study looked at children's perception of pain during BMT ([Pederson et al., 2000](#)). Important points included the ability of children accurately to self-report pain, the need for frequent serial assessments, the need specifically for nursing to use analog pain scales that assess for the intensity, type, and location of pain, as well as the need to include children in the choice of intervention strategies to help empower them. Despite receiving preparatory information about the pain they might experience before BMT, few children were able either to recall or assimilate this information. This fact suggested the need for feedback from children after their being given information to determine what they have retained, as well as a continuing teaching effort. Children noted the value of nonpharmacologic interventions, which emphasizes the need for staff to pay attention to these interventions even before BMT to help orient them to what will be most effective for an individual child. The practice of the child self-reporting pain should be fostered by staff.

In a series of 15 children undergoing BMT, the children appeared to adapt to the demands of BMT ([Gunter et al., 1999](#)). On a conscious level, they tended to become mildly depressed and generally to avoid the issue of what they were experiencing, with mild regressive trends, referred to by the authors as a type of "overnormalization." Projective testing, on the other hand, suggested that the children were dealing with more intense issues of death, depression, loneliness, anger, and feelings of guilt and punishment. More significant adjustment reactions, which were in the minority, were characterized by three varieties of reactions, including temper tantrums, regression, and depression reactions with noncompliance; such reactions seemed to be predicted by a history of "neurotic developments" or family conflict. The importance of recognizing the adaptive defensive structure of the individual child, as well as stressing the acceptance of expressed feelings of depression and anger, was underscored.

Another study noted that family cohesion and family expressiveness were protective factors for resilience in this population ([Phipps and Mulhern, 1995](#)), underscoring the importance of the family milieu in these patients. These findings potentially can provide a focus for the necessary psychosocial intervention for these children and their families. Mental status assessment often may be complicated by the presence of a dynamic interplay of these factors. BMT recipients are acutely affected by the medical course of other, similar patients in the hospital and, in the setting of another patient's death, may undergo a complex series of reactions that include denial and distancing, a heightened fear of death due to identification, and survivor guilt ([Patenaude and Rapoport, 1982](#)); psychological intervention is indicated in such circumstances. Decompensation and death provide unique challenges to all involved in the care of the child and adolescent. Concerns over quality of life and minimizing the suffering of a child compete with the wish to save the child and may present conflicts among staff and family members.

There also are psychological effects on the patient and family that relate to the source of the marrow and the subsequent success or failure of the graft, which may resemble analogous issues in living related organ donation in kidney or liver transplantation ([Wolcott and Stuber, 1992](#)). Death of the BMT recipient presents unique psychological sequelae for a family member who has donated marrow and may feel guilty that somehow his or her marrow was faulty ([Freund and Siegel, 1986](#)) or that he or she failed to save the recipient.

Recently, the psychological effects of BMT on siblings have become better appreciated ([Packman, 1999](#); [Packman et al., 1997](#); [Spinetta et al., 1999](#)). Sibling donors exhibited greater anxiety, lower self-esteem, and more adaptive skills in the academic setting, compared with nondonor siblings ([Packman et al., 1997](#)). One-third of the siblings in either group exhibited a significant degree of PTSD symptoms, which was a critical observation, and these symptoms occurred at 3- and 6-month follow-ups, suggesting the continuing impact of these experiences over time. Donor siblings experience the effects of an invasive procedure and the subsequent impact of continuing stresses; they may experience pressure to donate and feelings of loneliness and a tendency to withdraw emotionally after donation, along with a closer bond to the recipient after BMT. Packman notes that even a sibling who does not donate marrow must deal with two family members undergoing a medical procedure (assuming another family member is the donor), and that these symptoms could be better understood if seen as arising through a mechanism of "contagion," or simply by viewing the posttraumatic exposure as consisting of what the victim "felt" rather than what they experienced first-hand. Repeated interventions over time with siblings, rather than single interventions, should prove to be more effective, and should use multiple strategies, including sibling support groups. The effects of psychological interventions on siblings should be systematically studied ([Packman, 1999](#)). Guidelines for assistance to siblings of children with cancer have been published by the SIOP Working Committee on Psychosocial Issues in Pediatric Oncology ([Spinetta et al., 1999](#)). These guidelines stress:

- The importance of the involvement and education of siblings, including explanations about testing for potential bone marrow donation
- That siblings are not responsible for the cancer
- The need for sibling visits to the patient
- The value of support groups
- The need for siblings to remain informed about effects of cancer treatment during different phases

In the author's experience, BMT may be more stressful for the transplantation team, with guilty feelings perhaps more easily aroused because the children necessarily



are made so visibly sick by this extended procedure. Many SOT recipients, in contrast, may look significantly better soon after transplantation. Heart transplant recipients, for example, no longer appear cyanotic and may be out of the intensive care unit in several days, soon enjoying relatively greater physical activity compared with what may have been possible in a fairly compromised pretransplantation state. The physical changes secondary to immunosuppressant medication, so distressing to the adolescent patient, appear later.

Psychopharmacologic and psychotherapeutic intervention in the pediatric cancer population was reviewed by [McConville et al. \(1990\)](#) and [Spiegel \(1998\)](#). Medications have included anxiolytics, antidepressants, and low-dose neuroleptics to manage anxiety and depressive symptomatology during both the acute and terminal phases of illness; pharmacologic intervention in the pediatric patient with cancer is reviewed in another chapter in this volume.

Psychotherapeutic techniques range from working with the family, to teaching hypnosis and behavioral relaxation exercises to help patients tolerate painful procedures and decrease overall anxiety levels. Especially with respect to the latter, supportive collaboration with other medical staff is an essential aspect of the psychiatrist's role. Therapeutic play has been anecdotally reported to be quite helpful for pediatric BMT recipients, particularly when specific issues and goals are identified and used to guide the interventions ([Kuntz et al., 1996](#)).

Hospital staff can play a significant role in helping the patient and family prepare for discharge realistically and assist in concrete planning about resuming activities, helping parents understand the likely reactions of siblings and anticipate the effects of changes in marital roles ([Freund and Siegel, 1986](#)). Discharge from the hospital also is accompanied by stressors that are associated with the return home and reentry into the social milieu. These issues are similar to those confronted by SOT recipients, but complicated by the fact that recovery may be slower in the BMT population. Patients are not physically well and often exhibit the continuation of adjustment reactions, with consequent effects on siblings. There is a required preoccupation with cleanliness and minimizing exposure to potential pathogens that represents a constant reminder of the precarious line that is being traversed. These concerns often take on a compulsive quality.

Disruption of the marital relationship is common owing to the enforced separation during hospitalization, the reformulation of roles, and potentially conflicting coping styles, and preexisting difficulties are magnified tremendously ([Freund and Siegel, 1986](#)). A sense of "anger and betrayal" may be evoked in the patient and family when the transplant has not provided a cure ([Atkins and Patenaude, 1987](#)). This feeling may be exacerbated in the setting of post-BMT complications. Depression and impaired self-esteem may result when expectations meet with disappointment ([Freund and Siegel, 1986](#)). Individual psychotherapy, including play therapy with younger children, may facilitate psychological adjustment and the expression of feelings associated with the illness and procedure ([Atkins and Patenaude, 1987](#)). "Open communication, maintenance of hope, and the positive use of denial" may predict a more positive outcome, although clinical course may be a powerful predictor of psychopathology ([McConville et al., 1990](#)).

Although denial may be adaptive in children and adolescents in helping to maintain self-esteem, caregivers can play a significant role in addressing issues that would not be raised voluntarily and provide guidance as to the timing of education concerning these issues for the child/adolescent and family ([Wolcott and Stuber, 1992](#)).

Denial, however, may not necessarily be adaptive. [Stuber et al. \(1991\)](#) noted persistent symptoms of posttraumatic stress in 3- to 7-year-olds for more than 1 year post-BMT. Symptomatology appeared most pronounced around the time immediately after discharge from the hospital, when anxiety over survival is high. Denial and avoidance were the most prominent PTSD symptoms. Stuber et al. theorize that the more "chronic, nonimminent" nature of a threat that is internal and not external may be more likely to lead to denial and avoidance. This reaction "may seem the only possible response when the traumatic event is perpetrated by adults who are said to be acting in the child's interest, with the permission and assistance of parents." They also suggest that the simple fact of asking children about symptoms may be therapeutic. Intrusive, repetitive preoccupation may be evident only in the child's play. If the psychiatrist does not ask about symptoms or observe the play of these children, necessary treatment may be delayed because of the nature of this phenomenon, where symptoms are less dramatic and more insidious, and early intervention thus will be compromised.

These data should guide future early psychiatric treatment in this patient population and probably others who experience life-threatening illness with high-tech, dramatic, invasive interventions. It also has implications for follow-up care because of the probability that denial will affect how and when medical help is accessed. The occurrence of late psychological effects of BMT calls into question the view expressed by some to put "the BMT experience behind them," and suggests a more direct approach to dealing with issues surrounding the illness and its sequelae.

Specific sequelae for pediatric BMT recipients may include learning disabilities, growth retardation, and infertility. Significant motor delays and subtle impairments of performance in perceptual and fine motor tasks have been reported in children between 3 and 11 years of age treated with BMT accompanied by TBI ([Smedler et al., 1990](#)). Children in this age group were more significantly subject to these effects than were older children and adolescents, and cranial irradiation may be the significant factor in predisposing to neurocognitive deficits ([Kramer et al., 1992](#)). Cognitive deficits seem to be related to the dose of cranial irradiation or intrathecal methotrexate, and such sequelae can have significant effects on the developing child's school performance and self-esteem. Low-dose cranial irradiation may be associated with fewer cognitive sequelae ([Kramer et al., 1992](#)). Other long-term sequelae include sterility in most patients after BMT, with consequent effects on sexual identity and romantic relationships, as well as self-esteem. Sexual dysfunction also is common, such as problems in vaginal lubrication in women and impotence or retarded ejaculation in men ([Andrykowski, 1994](#)). Psychological intervention may address these effects and provide appropriate counseling, which may mitigate and help differentiate between effects that are secondary to treatment/illness and social or psychological factors such as affective illness ([Andrykowski, 1994](#)).

## **Kidney Transplantation**

Adjustment after kidney transplantation seems to be influenced by developmental factors. Preschool children exhibit the most rapid rates of growth and improved social functioning, with adolescents experiencing the greatest degree of social and emotional problems. [Bernstein \(1977\)](#) reported risk-taking behavior, school phobia, and depression in 4 adolescents of the 32 renal transplant recipients he studied. [Korsch \(1973\)](#) examined outcome in pediatric renal transplant recipients and found that most negative effects on family functioning occurred during the first year. A subset of patients (10/35) had maladjustment on personality measures that consisted of problems with anxiety and self-esteem; these patients were at higher risk for nonadherence. Depression was the "most specific test finding." Seventeen of 19 adolescents were working or in college, and 11 of 16 younger patients were in school. Important concerns in this population included steroid-induced side effects and apprehension about rejection.

One study of 19 adolescent renal transplant recipients found that 21% of the patients took less than 80% of their prescribed medication ([Blowey et al., 1997](#)). A review noted that lack of adherence, or noncompliance, has a mean prevalence across studies of approximately 40% in children and adolescents, with one-third of the authors' sample reporting "psychologically meaningful" reasons for noncompliance; the authors recommended that, rather than emphasizing individual patient characteristics when addressing noncompliance, that one should look specifically at the patient's psychological experience of chronic illness ([Wolff et al., 1998](#)). Patient satisfaction, or how the patient's expectations correlate with reality, was suggested to be a major factor in compliance. In this series of adolescents, there was a 69% incidence of medication noncompliance, with 24% of the patients showing improved compliance after the resolution of crises in their lives that were found to be influencing compliance. Lack of adherence usually was found to have multiple causative factors, including the burden of the illness, lack of understanding, and feelings of dependency, not being taken seriously, or being uninfluential; a significant number of patients also tended to blame the health care providers, feeling disappointment, a lack of trust or insufficient time spent with them, a sense of not having been given enough information, or that health care providers had "given up" on the patients, for example. An important concept in this article was the view that the medical requirements of transplantation conflicted with the needs of self-determination—to some degree, inevitably—and that noncompliance often was precipitated by crises in the patients' lives. The approach to dealing with noncompliance should be less judgmental, with a greater attempt to communicate with the patient and to understand the psychological underpinnings for not complying with treatment.

There have been attempts to quantify the relative risk for nonadherence. The score on a psychological scale based on assessments of IQ, schooling, psychomotor development, emotional development, and social development was found to correlate with renal graft survival during the first year after transplantation in one study ([Mongeau et al., 1997](#)).

Several studies have indicated that quality of life improves after renal transplantation in the pediatric population ([Almond et al., 1991](#); [Brownbridge and Fielding, 1991](#); [Cole, 1991](#); [Khan et al., 1971](#); [Krmr et al., 1997](#); [Poznanski et al., 1978](#); [Reynolds et al., 1991](#)). Denial seems operative as a defense against fears of rejection and may lessen as patients enter their third decade. Disturbances in autonomous functioning associated with enforced dependency on adults may lead to social isolation, impaired self-esteem, and depression. In particular, impaired social functioning may put children at risk and may be exacerbated by prolonged school absence ([Khan et al., 1971](#)). The continuation of peer relationships is especially important in adolescents, and specific interventions might focus on the maintenance of "social networks" ([Melzer et al., 1989](#)). One follow-up study of 17 kidney transplant recipients who retained their grafts found that 59% were "completely satisfied" with their lives, with 94% reporting no interference of their health with family life 10 or more years after transplantation in childhood ([Krmr et al., 1997](#)); of note, however, in this



sample, was that 21 of the original 40 children in the sample eventually lost their grafts.

There may be a role for preemptive transplantation before the progression to end-stage renal disease requiring dialysis, to attempt to avoid the metabolic and psychological impact of chronic renal disease (Cole, 1991), and transplantation has been advocated as the preferred treatment in end-stage organ failure (Almond et al., 1991; Brownbridge and Fielding, 1991). Improved physical health after transplantation has been postulated to have a positive effect on psychosocial outcome in the patient and family (Reynolds et al., 1991). Brownbridge and Fielding (1991) found psychosocial adjustment in transplant recipients to be superior to that in patients on hemodialysis or peritoneal dialysis.

However, despite reports of improved physical, emotional, and cognitive functioning, renal transplant recipients continue to experience the problems associated with chronic immunosuppression, impaired growth, anxiety over rejection, and delayed sexual maturation (Reinhart and Kempf, 1988). Cognitive deficits may persist, especially if the onset of disease was at an earlier age, and there was a longer time with end-stage organ failure before transplantation (Sexson and Rubenow, 1992), as is the case with liver failure (see later). Child psychiatry has been described as having a significant role in addressing these issues and in helping to facilitate adjustment.

One study found that there were improvements in mental processing speed and sustained attention in children after renal transplantation when individual children's pretransplantation and posttransplantation performance was compared (Mendley and Zelko, 1999). The authors emphasized the importance of "within-subject comparisons," noting that if they had merely compared groups, there would not have been any positive findings.

Family cohesion was found to correlate with daily living skills in a study of pediatric renal transplant recipients (Davis et al., 1996), suggesting the importance of family support for the functioning of these children. Impaired socialization skills also were correlated with lack of adherence to medication regimens. Eighty-two percent of the transplant recipients in this study exhibited maladaptive behavior (defined by frequency of temper tantrums). However, these patients, although skewed in age toward early adolescence, were found to be compliant with medication and diet much of the time.

### Liver and Intestinal Transplantation

The role of the consultation–liaison psychiatrist in liver transplantation cases has been described by Pinard and Minde (1991), Krener (1987), and Surman (1994). Psychosocial effects have been discussed by Bradford (1991), who noted that competition for organs, maternal overprotectiveness, and enhanced dependency by the child on parents may have a significant impact on the family. Cognitive deficits may persist, although posttransplantation life in general is marked by fewer hospitalizations, shorter lengths of stay, and fewer medications. Outcome has been commented on by Stone et al. (1997), who noted that 93% of transplanted children in a series of 20 patients experienced an acceleration in growth velocity that allowed them to catch up after transplantation. At 5- to 10-year follow-ups posttransplantation, 100% attended school, with 70% not in special education, 80% participating in sports, and 85% in other extracurricular activities. Children's Global Assessment Scale (C-GAS) scores indicated 75% were functioning in the normal range, and Global Assessment of Relational Functioning scale (GARF) scores indicated that 70% of families were functioning normally. The authors note that a comprehensive psychosocial support program throughout all stages of the transplantation experience, along with specific efforts at reintegration (with few restrictions on the children), limiting visits to the transplantation center, and using the lowest possible effective immunosuppressant dosages were important factors in the aforementioned statistics. Windsorova et al. (1991) studied the emotional adaptation of pediatric liver transplant recipients and found that, as a group, they did not display any more behavioral disturbance, anxiety, depression, or effects on self-concept than did a control group of diabetic children. Compared with normative data, however, the liver transplant recipients showed indications of greater anxiety and depression on projective testing. Multidimensional assessments of psychosocial functioning were deemed more sensitive for identifying such phenomena than was parental or patient report.

Others have assessed quality of life in liver transplant recipients (Chin et al., 1991; Windsorova et al., 1991). In Chin's sample (1991), all but one school-age child had returned to school, with quality of life described as good or excellent in all. However, in 7 of 12 children older than 5 years of age, maladaptive behavior was reported, which may have been related to the degree of hepatic encephalopathy that persisted after transplantation. Zamberlan (1992) identified problems in socialization and peer relations, which were associated with feelings of loneliness and vulnerability, in the 20 school-age liver transplant recipients she studied. Psychiatric intervention, especially during the first year posttransplantation, has been recommended (Sexson and Rubenow, 1992; Zamberlan, 1992).

Mastroyannopoulou et al. (1998) addressed the prevailing trend in consultation–liaison to begin to look more at factors predisposing to a greater ability to cope and adapt, rather than risk factors for poor psychological outcome. The use of varied coping strategies is thought to be adaptive and is based on an increased capacity for flexibility and change. Overall, normal children, children with chronic liver disease, and liver transplant recipients used similar coping strategies. It was found that, compared with children with chronic liver disease who had not been transplanted, transplant recipients tended to have a greater sense of control over their illness; in addition, the transplanted group saw themselves as less "ill," and in fact many viewed themselves as "cured." Both of these groups seemed to use wishful thinking and distraction as effective coping strategies, defenses that might be considered more maladaptive in an otherwise healthy population, highlighting the need to appreciate differences in what constitutes adaptive psychological functioning in this population.

The most comprehensive data on neuropsychological outcome in pediatric liver transplant recipients have been published by Stewart et al. (1989, 1991a, 1991b). In one series (Stewart et al., 1989), 15 of 29 children had delays in cognitive or motor function before transplantation that were associated with earlier onset of liver disease. After transplantation, 4 of 11 patients with cognitive delays and 5 of 10 with motor delays had improvements great enough to place them within the normal range at 1-year follow-up. As a group, improvement in cognitive function did not reach significance. Improvement on the Social Scale of the Child Behavior Checklist (Achenbach and Edelbrock, 1983) was found at 1-year follow-up in children who were transplanted at 4 years of age or older. Enhanced functioning was reported with peers, in sports, and in the ability to take on greater home responsibilities. Prednisone exerted robust effects on linear growth, and gains in head circumference were most notable in children younger in chronologic age.

Neuropsychological deficits were found in these patients Stewart et al., (1991a, 1991b). When matched against a control group of patients with cystic fibrosis (Stewart et al., 1991b), the liver transplant recipients had greater impairments in intellectual/academic function, learning and memory, abstraction and concept formation, visual-spatial function, and motor skills. Significantly more liver transplant recipients were receiving special services in school, although less than a third were receiving the services they needed. The greater impairment in functioning in the liver transplant recipients was postulated to be related to the effects of hepatotoxins on the developing brain, although other factors, such as acute illness requiring invasive surgery, were not controlled for. Liver transplantation can potentially prevent the irreversible effects of hyperammonemia on cerebral function in children with arginosuccinic acid synthetase deficiency (citrullinemia), a urea cycle defect (Fletcher et al., 1999).

More recently, the academic functioning of 50 pediatric liver transplant recipients was studied 3 or more years after transplantation (Kennard et al., 1999). This study found that these patients did not make statistically significant gains in intellectual functioning after transplantation, highlighting the effects of illness on mental functioning. In addition, compared with the normal population, academic achievement was lower in this population, although three subgroups were identified: 56% of the children who were performing on standardized academic tests within 1 standard deviation (SD) of their full-scale IQ (FSIQ) scores, and greater than half of this subgroup were performing above expectation, indicating the presence of facilitating factors that would be useful to identify; 26% of children performing greater than 1 SD below their FSIQ scores; and 18% performing in the mentally deficient range. Although 48% of the sample received special education services, only 38% of the children with learning difficulties were recipients of these services, which is quite problematic. The most common difficulties in children who were not intellectually impaired were in the areas of math and written language, which is interesting in light of the visual-spatial difficulties cited in earlier studies. Potential factors that might account for the variability in intellectual function include those related to the underlying disease itself (patients with biliary atresia tended to do more poorly) and the relationship of encephalopathy to the child's developmental status; the severity of pretransplantation illness and duration of posttransplantation immunosuppression; and "facilitative" factors such as motivation and persistence. Several important conclusions by these authors include the need for routine psychoeducational evaluations and the awareness that achievement can be enhanced with special education services.

Ethical factors involving living related liver donation, often from a parent to the child, were reviewed by Singer et al. (1989). Emond (1993) points out that approximately 40% of children in need of a liver transplant might be appropriate for a living related graft, and notes the significant advantage of not being dependent on the cadaver waiting list. UNOS Scientific Registry Data indicate that the number of living liver donors has been increasing since 1989, when 2 were reported, to 1993, when 35 such donors were recorded. Emond (1993) also comments on the ethical issues surrounding living related liver donation. Even though the risk of donor morbidity is relatively low, there have been reported cases of bile duct injury, nerve apraxia, or wound infection. He also noted several cases of donation made by unrelated living donors, which raises additional ethical issues. In an additional case evaluated by this author's consultation–liaison service, a school teacher who knew the recipient intimately agreed to be the donor, although it appeared as if her wish to donate was motivated greatly by issues of guilt in her own life deriving from the loss of her own child. Her investment in the donation as a means of personal psychological reparation seemed to make the consequences of an unsuccessful

outcome potentially devastating for this teacher.

Intestinal transplantation in children was reviewed by [Reyes et al. \(1996\)](#) and cited as having a 70% survival rate, with a 60% survival rate for combined liver–intestinal transplants. The psychosocial effects on parents of liver or intestinal transplantation also has been reviewed ( [Tarbell and Kosmach, 1998](#)), with significant psychological distress in both parents found perioperatively, and with fathers exhibiting greater distress than mothers. Stress appeared to be higher when the transplant recipient was younger or male, or there were fewer people in the household. The study underscores the importance of including fathers in psychosocial assessments.

### Heart Transplantation

Neuropsychological function in cardiac transplant recipients has been reviewed in the adult population ( [Nussbaum and Goldstein, 1992](#)), but few studies exist in the pediatric population. In one series of 54 infants with congenital heart disease who were transplanted at 4 months of age or younger, 80% of these infants survived the first year, and 89% were neurologically normal at 14 months posttransplantation ( [Trimm, 1991](#)). Normal language functioning was found in the 77% old enough to be assessed, and normal audiologic function was found in 90%. The Bayley Scale of Infant Development was administered sequentially, and when their scores were averaged, all but two infants fell within 2 SD of the mean on the Mental Developmental Index, and all but two infants fell within 2 SD of the mean on the Psychomotor Developmental Index.

Like other chronically ill children, adolescents with congenital heart disease that was surgically corrected earlier in childhood may be at higher risk for later behavior and adjustment problems ( [Utens et al., 1993](#)). Studies examining the psychosocial outcome of pediatric cardiac transplantation are limited, but in a multicenter study, [Uzark et al. \(1992\)](#) reported that these children's level of function approaches that of their peers, with 93% attending school and able to participate in athletics. Nevertheless, this sample of 49 children, who averaged 21 months posttransplantation and whose mean age was 10.4 years, showed less social competence and more frequent behavior problems relative to the norm on the Child Behavior Checklist ( [Achenbach and Edelbrock, 1983](#)). Such problems were associated with impaired self-concept and higher anxiety levels, as well as the inclination not to discuss feelings. The authors hypothesized that depression and behavior problems were related to personality and interpersonal factors, such as the adolescent's not feeling accepted by or communicating well with peers. Families experienced relatively high stress levels without a concomitant increase in resources to assist in coping with the stress. Psychosocial assessments, enhancing communication between family members, and cognitive work with adolescents were suggested.

Other authors have looked at psychosocial adaptation in this population ( [DeMaso et al., 1995](#); [Gold et al., 1986](#); [Hanton, 1998](#); [Lawrence and Fricker, 1987](#); [Suszycki, 1988](#); [Uzark and Crowley, 1989](#)) or addressed clinical issues more descriptively ( [Slater, 1994](#)). [Lawrence and Fricker \(1987\)](#) found that quality of life in seven children who had received heart transplants was characterized by a return to normality. [Uzark and Crowley \(1989\)](#) note that pretransplantation stress levels secondary to chronic and sometimes life-threatening illness are superimposed on the transplantation scenario, resulting in powerful effects on family function that relate to doubts about the child's vitality, financial pressure, marital tension, and potential distance from the social milieu. Multidisciplinary intervention from social work, nursing, and mental health workers can be of enormous help. [Hanton \(1998\)](#) commented specifically on the role of nursing care in the transplantation of a preadolescent girl.

[DeMaso et al. \(1995\)](#) found that 78% of pediatric heart transplant recipients who were transplanted between 3 and 20 years of age had improved psychological functioning in the normal range posttransplantation, with pretransplantation and posttransplantation scores correlating. Children with higher pretransplantation and posttransplantation emotional functioning had fewer posttransplantation hospitalizations. From a diagnostic standpoint, Psychological Symptoms Affecting the Medical Condition were diagnosed in 35% of the pretransplantation patients, as opposed to 4% of the posttransplantation patients. Mood disorders secondary to medical status were found in 13% of pretransplantation patients and 0% of posttransplantation patients. As well, after transplantation, children with congenital heart disease adjusted as successfully as children with acquired cardiac illness. Family functioning correlated with the child's pretransplantation and posttransplantation psychological functioning. DeMaso and colleagues underscore the importance of psychological intervention before transplantation, given these findings.

### Heart–Lung and Lung Transplantation

[Warner \(1991\)](#) published data from a series of pediatric patients in end-stage respiratory failure referred for heart–lung transplantation and found that two-thirds died at some point during the referral, evaluation, or surgery process. He emphasized the necessity for careful screening of these patients to avoid putting them through additional trauma that would be associated only with a poor outcome, although for some children heart–lung transplantation represents a desperate but viable final option. Five children of 10 transplanted had returned to school and other age-appropriate activities. Psychosocial morbidity in children awaiting heart or heart–lung transplantation was studied prospectively ( [Serrano-Ikkos et al., 1997](#)). Twenty-five percent of children met criteria for a psychiatric diagnosis, and over 50% exhibited impaired psychosocial functioning, which the author attributes to the effects of chronic illness rather than the specific effects of awaiting transplantation. Interestingly, the well siblings displayed the same amount of emotional or behavioral difficulties as the ill children. Severely ill children had higher depression scores compared with their well siblings. Marital and family difficulties were common, with 57% of families being “moderately” or “poorly” adjusted.

Research on psychosocial or neuropsychiatric outcome in lung or double-lung transplantation is quite sparse. In one series, four of the seven pediatric patients who underwent heart–lung or lung transplantation currently are alive, with all school-age children back at school and doing well emotionally ( [Quitell, personal communication, 1993](#)). Indications for transplantation included decompensation in the context of pseudotruncus type 6, pulmonary hypertension, pulmonary fibrosis, and pulmonary atresia.

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#### CASE ILLUSTRATION

One of the children who received a single-lung transplant at 16 months of age is currently approximately 18 months postsurgery. He was reportedly delayed neurodevelopmentally before transplantation, but has made significant gains since his surgery. After his birth, he had spent only 2 months at home before being admitted to the hospital, where he spent the next 14 months until he was transferred to another medical center and transplanted.

At that time, the medical staff was concerned about the lack of time that his parents spent with him. It almost seemed as if they had “dropped him off” to have a transplant, and there were large stretches of time that the baby was alone in his hospital room, unlike many of the other children whose parents literally hovered by the child's bedside and never left. The staff became concerned about whether this constituted neglect, but after transplantation, it became apparent that the parents had been completely misjudged because they became model parents, “never skipping a beat,” according to their pediatrician.

In retrospect, on further evaluation, it became apparent that these parents had never had the opportunity to bond with their child because he had been admitted to the hospital in an extremely compromised state so soon after birth. His mother had been completely overwhelmed and had never had the opportunity to “feel like a mother.” With the multitude of nurses who were necessarily caring for her baby, she felt without a role. A follow-up conversation with this family disclosed a beaming father, proud of and loving toward his son, who was reported as “just like any other 3-year old” except for some speech and language problems. [[Slater \(1994\)](#), with permission.]

#### Comment

This case illustrates the importance of impaired maternal/paternal–infant bonding in the case of some severely impaired infant transplant candidates, and the need both to help the parents develop some role in their care, and to use this knowledge in assessing the relationship between caregivers and their child.

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In summary, available neuropsychiatric and psychosocial follow-up studies in this population indicate that the quality of life usually is improved after transplantation; many of these children, in fact, would not be alive without transplantation. After surgery, many have increased levels of physical activity and school attendance, and cognitive function may be improved in some. Nevertheless, many have persistent cognitive deficits because of the effects of chronic illness and circulating neurotoxins during brain development. Transplantation is another stressor superimposed on a stressed family system, with sequelae for siblings and marital relationships. For the transplant recipient, studies suggest that they are at risk for depression, behavioral problems, and impaired self-esteem, which probably relate to the social problems for which these patients also are at risk. Anxiety symptomatology, including school refusal and posttraumatic symptomatology, also may be more common in this population. For these reasons, neuropsychological testing and early psychiatric evaluation and intervention are recommended.

### PSYCHOPHARMACOLOGIC ASPECTS OF TRANSPLANTATION

Neuropsychiatric side effects of immunosuppressant and antiinfectious agents used in transplantation and aspects of psychopharmacologic intervention in this population were reviewed by [Trzepacz \(1993a, 1993b\)](#) ([Table 97.10](#) and [Table 97.11](#)). Important considerations of drug use in this population include altered pharmacokinetics due to drug interactions and impaired hepatic, renal, or cardiac function. Children have altered pharmacodynamics because of both developmental





Cognitive factors obviously play a powerful role with respect to informed consent earlier in childhood. However, as children mature, an “absence of dissent” can be determined, and the level of “assent,” or “emotional consent,” the nature of the child’s emotional response to what is being asked, can be ascertained ( [Slater, 1994](#)).

Ethical issues surround solid organ or bone marrow donation. Psychologically, all transplant recipients have reactions to the donation process, and specific feelings about the donor have been studied in the adult population ( [Bunzel et al., 1992](#)). From the point of view of the recipient, feelings include guilt, a sense of responsibility, and identification with the donor family.

The wish for contact between donor and recipient is a phenomenon that occurs from both directions. Psychologically, the donor–recipient connection is imbued with tremendous meaning. One adolescent organ recipient felt that “they [the donor’s family] are my family inside. I have their blood running through my veins” ( [Slater, 1994](#)).

The parent of a child who had received a cardiac transplant reflected: “It’s really impossible to put into words the way you feel about what they went through. I mean it’s a true, deep empathy, except you know you didn’t quite cross that line. And you’re grateful you didn’t, but you feel badly enough getting as close to that line as you did. I can’t imagine what it would be like to lose a child, or maybe the problem is that I can, and that’s what’s so frightening, and that’s what’s so hard.” [From [Slater \(1994\)](#), with permission.]

Although the right to privacy is protected, anonymous contact is possible under certain circumstances. Sympathy cards, thank-you notes, and photographs have been sent. Many donor families simply wish to know that the recipient is doing well, and often the recipient is curious about from where the organ came. Nevertheless, the two parties may not share the same wishes for giving or receiving information. For the donor’s family, it may be too painful to be exposed to the child that has lived while theirs has died. For the recipient, it might be too difficult to have contact with the family who donated their child’s organs to allow him to live and is mourning their own child’s death.

As [Rothman \(1992\)](#) notes, one stands in awe of donor families, who in the midst of such tragedy and traumatic loss are able to think of the welfare of another child and family. A mother who donated the organs of her deceased son recounted her feelings:

At first there was a little apprehension, but the apprehension is more than compensated with compassion, and that any of your fears that you do have, which are right to feel, would really be smoothed over tremendously by making your child available in a sense to being a donor; and that it is such a wonderful gift. There is no greater gift of giving life to someone else. And its rewards are not just—in a sense, they will never be monetary—but it’s more precious than gold. And it’s the wisdom to be able to ... you’re not really giving up anything. That’s what love really is about, it’s being able to give. [From [Slater \(1994\)](#), with permission.]

With respect to living-related organ or bone marrow donation, can the decision to donate ever be “voluntary” ( [Lewis, 1974](#); [Lowy and Martin, 1992](#); [Singer et al., 1989](#))? What about situations where two children in the same family require donor kidneys but only one parent is immunologically compatible? Other considerations include the morbidity and risk associated with making the donation, and the effect on family relationships for both the sibling who donates and the siblings who do not ( [Lewis, 1974](#); [Sexson and Rubenow, 1992](#)). The feelings surrounding the death of a child reached dramatic proportions when a parent once pleaded with me to allow her to donate her own heart to her dying daughter.

[Lewis \(1974\)](#) wrote extensively on the case of an identical twin confronting the decision to donate a kidney to her sister, where it appeared that overt refusal was an impossibility. In this situation, the child psychiatrist had to consider the impact of this decision on the relationship between the sisters, the donor’s current state and later development, and the emotional state of the family. All of this was felt difficult to predict ( [Lewis, 1974](#)):

She might have a nagging sense of loss, emptiness, and resentment at losing her kidney; but she might feel a greater sense of loss if she were to lose her sister. She might develop a fantasy of being anatomically united with her sister; on the other hand, she might experience a precipitous increase in individuation if her sister were to become more and more different and eventually die. She might feel a sense of increased self-esteem from giving her kidney, and might feel guilty later if she had not given her kidney.

Both girls initially did well, although persistent somatic anxiety developed in the donor, and somatization symptomatology developed in other family members as well. When [Lewis \(1993\)](#) reported anecdotally on follow-up 14 years later, the twins had drifted apart both geographically and emotionally. The donor twin continued to have a fantasy that because she and her sister were twins, each having one functioning kidney, the donor symbolically still had two kidneys.

Bereavement work is an inevitable consequence for the child and adolescent consultation–liaison psychiatrist in this setting and represents perhaps the most difficult task the clinician could face. Feelings of tremendous loss, sadness, and impotence often are engendered in the psychiatrist, who nevertheless can play an enormously supportive role simply by his or her presence. One family later thanked the child psychiatrist for being at the bedside so their child did not die alone. Involvement with these families is different from the typical work in child psychiatry. I have personally attended funerals and received cards from such families years later.

Last, consideration given to transplanting a child while he or she is still relatively stable and not in dire need of the organ has ethical implications as well. Transplanting a child earlier in the course of what appears to be a disease that will result in end-stage organ failure also may decrease the persistent cognitive deficits presumably caused by circulating neurotoxins, by shortening the time that the child is exposed to them. It also lessens the chances that the child will die waiting for an organ.

## CONCLUDING COMMENT

Transplantation inexorably alters life for the patient and family who endure it. A change in perspective or philosophy inevitably accompanies adjustment to posttransplantation life. Many adolescents describe a change in priorities, such that they are not as troubled by trivial matters and are better able to appreciate life. At the same time, however, these patients and their families often live life day-by-day, with an omnipresent uncertainty lurking in the background. As one adolescent described, “I was worried about what to wear, and now, you know, I’m worried about whether I’m going to live” ( [Slater, 1994](#)).

The role of the child and adolescent psychiatrist in this drama may be as deep as he or she is willing to allow. There exists the opportunity for much collaboration with physician colleagues and a flexibility of roles in the physician–patient relationship that may be unique to this type of work, in which the psychiatrist truly can function as a member of a “family” composed of the transplantation team, patient, and patient’s family. Being around this “family” can be awe-inspiring at times and quite painful at others. In a sense, it is a privilege to be involved with these children and adolescents. More systematic research obviously is needed to help identify risk factors for nonadherence and other secondary psychiatric morbidity, to develop a clearer understanding of psychosocial and neuropsychiatric sequelae, and to help formulate intervention strategies.

“There is a strange heroism and poignancy to their stories, some sense that a miracle has taken place, but also the lament of two childhoods lost in the act of one being saved. Perhaps this is the ultimate paradox of organ transplantation in children” ( [Slater, 1994](#)).

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# 98 NEUROBIOLOGICAL AND PSYCHOSOCIAL SEQUELAE OF HIV DISEASE IN CHILDREN AND ADOLESCENTS

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The epidemic of acquired immunodeficiency syndrome (AIDS) represents a major world crisis affecting the health and psychological well being of tens of millions of persons, causing devastation among families, and threatening the social welfare of communities. There is a widening gap between the developed and developing countries of the world. In countries such as the United States, where newer, expensive therapies are available, there have been significant decreases in morbidity and mortality; countries with more limited resources are experiencing horrifying escalations in the spread and effects of the epidemic. This contrast is particularly true for children. In the developed world, where interventions aimed at reducing perinatal transmission are readily affordable, there has been a dramatic decrease in the number of perinatally infected children. In the developing world, where resources are lacking, reduction in perinatal transmission has not yet become a reality. However, throughout the world, the increasing focus is on adolescents and youth, because they are the age cohorts experiencing the highest rate of new infections.

## WORLD VIEW OF THE EPIDEMIC

According to estimates from the Joint United Nations Programme on HIV/AIDS (UNAIDS), by the end of 1999, approximately 34.3 million people were living with human immunodeficiency virus (HIV) infection, of whom 95% were in the developing world. Approximately 5.4 million persons were infected in 1999 alone, and one-tenth of them were less than 15 years of age ([UNAIDS, 2000](#)). Since the epidemic began, 19 million adults and children have already died. In 1998, AIDS rose to become the fourth leading cause of death worldwide, after being ranked seventh just 1 year previously ([UNAIDS, 1999b](#)).

Sub-Saharan Africa continues to dominate the world AIDS epidemic in sheer numbers. Approximately 75% of all new infections in 1999 occurred in the region, with 2.2 million AIDS-related deaths. In the face of this devastation, another 23.4 million adults and 1 million children are expected to die prematurely if they are not provided access to therapy. Southern Africa is particularly hard hit: in most countries, the rates of infection are between 20% and 26% of those aged 15 to 49 years; in Botswana, the infection rate among young adults is as high as 36%. It is predicted that more than one-third of all youth now aged 15 years will eventually die of AIDS in many sub-Saharan countries ([UNAIDS, 2000](#)). Amid such dire statistics, there is promise that some countries may be successfully turning the tide of the epidemic. Most prominent is Uganda, in which a strong national prevention campaign has reduced the rate of infection to slightly more than 8%, from a peak of 14% in the early 1990s.

Although rates of infection are relatively lower in other parts of the world, the numbers remain significant and show a rapid increase in some areas. By the end of 1999, there were approximately 5.6 million infected people in South and Southeast Asia, and approximately one-fifth of these were infected within the last year alone ([UNAIDS, 1999a](#); [UNAIDS, 2000](#)). Spread of the epidemic is affected by changing economics, with people moving from rural to urban areas as economies expand and then moving back to rural areas as unemployment rises. In addition, the growing numbers of young women who engage in prostitution for their livelihood are affecting the increased rate of transmission. In some of the major cities in India, nearly half of the prostitutes are younger than 18 years of age, and more than one-fourth of them have been found to be HIV infected.

The HIV epidemic in Latin America is highly diverse; heterosexual transmission predominates in some countries, particularly those in Central America and on the Caribbean coast, whereas in other countries, such as Mexico, Argentina, and Colombia, injecting drug use and homosexual transmission are significant contributing factors. Rates of infection in some Caribbean islands are the highest of any area outside of sub-Saharan Africa. For example, in Haiti, more than 5% of adults are infected, and in the Bahamas the rate of infection exceeds 4% ([UNAIDS, 2000](#)).

Countries in Eastern Europe and Central Asia have rapidly emerging epidemics that are largely related to injecting drug use ([UNAIDS, 1999c](#)). The number of cases in Eastern Europe increased ninefold in just 3 years, from fewer than 30,000 cases in 1995 to 270,000 cases by the end of 1998. The spread of the epidemic in these countries is related to serious economic failure and concomitant increases in poverty and the disenfranchisement of young people, which lead to higher rates of injecting drug use and greater reliance on prostitution as a means of livelihood ([Dehne et al., 1999](#)).

## Epidemic in the United States

In the United States, 1996 marked a major turning point in the epidemic when the introduction of new therapies slowed the progression of HIV disease and led to a reduction in the number of cases of AIDS and a dramatic decrease in HIV-related deaths. AIDS was the leading cause of death for those aged 25 to 44 years in 1995, but by 1997, it had fallen to the fifth leading cause of death in this age group.

There continue to be other changes in the epidemic as it progresses, and these changes disproportionately affect women and minorities. In 1999, women represented 32% of all those reported with HIV, compared with 7% of those reported with AIDS in the first 5 years of the epidemic (Centers for Disease Control and Prevention, 2000). Blacks and Hispanics have had markedly higher rates of infection than whites. Since 1996, blacks have outnumbered whites in new AIDS diagnoses. Statistics on AIDS fail to portray the true nature of the epidemic; with the advent of more effective therapies that delay or prevent the onset of AIDS, many HIV-positive persons may go unreported because they do not meet criteria for an AIDS diagnosis. As a result, tracking cases of HIV infection is now a more accurate way of monitoring the spread of the epidemic than tracking statistics on AIDS. In particular, because of the delay between the time of infection and the development of AIDS, data on HIV infection provide more pertinent and potentially useful information on the spread of the disease among youth. It is significant that, unlike the declining rates of AIDS, the rate of spread of HIV infection remains fairly constant.

## CHILDREN AND AIDS

Changes in the incidence of AIDS have been even more dramatic among children than among adults. For example, in 1999, only 263 children were reported to the Centers for Disease Control and Prevention with perinatally acquired AIDS, less than one-third of the number of cases reported in 1992 (Centers for Disease Control and Prevention, 2000). Although some of this reduction is the result of therapeutic advances, the greater contribution to this decline is the success of decreasing perinatal transmission from mother to child through treatment of HIV-infected pregnant women with antiretroviral therapy ([Connor et al., 1994](#)). This approach, together with delivery by elective cesarean section, has had a rapid and dramatic effect on decreasing rates of HIV transmission ([European Mode of Delivery Collaboration, 1999](#); [International Perinatal HIV Group, 1999](#)). Before 1994, the rate of transmission of HIV infection from an infected mother to her child was approximately 25%; this rate has now fallen to less than 5% ([Blattner et al., 2000](#)). Estimates from one study suggest that fewer than 250 children were born with HIV infection in the United States in 1997, compared with approximately 1,260 cases of infection just 5 years previously ([Lindgren et al., 2000](#)).

Similar successes have not been seen in poorer countries, where the protocols used for treatment of pregnant women in developed countries are unaffordable. This picture is further complicated by the finding that HIV is also transmitted by breast-feeding, and this results in infection of an additional 10% to 20% of infants ([Dunn et](#)

al., 1992). Substituting infant formula for breast-feeding not only is too expensive in many countries, but also it is associated with an increased risk of morbidity and mortality from other infectious causes (Butz et al., 1984; Kuhn and Stein, 1997). Some studies have demonstrated that shorter, less expensive courses of treatment with antiretroviral medications can be efficacious in decreasing transmission of infection from mother to child ( Dabis et al., 1999; De Cock et al., 2000; Saba et al., 1999; Shaffer et al., 1999; Wiktor et al., 1999). Among these, perhaps the most important is the HIVNET 012 Trial, which demonstrated that just two doses of the antiretroviral medication, nevirapine, one given to the mother at the beginning of labor and one given to the baby after birth, had the effect of almost halving the rate of transmission (Guay et al., 1999).

Whether the countries that have the highest rates of HIV disease can successfully introduce programs to prevent perinatal transmission remains to be seen. Implementation of such interventions requires access to affordable medications and a sound system for providing prenatal and perinatal care, as well as HIV testing and counseling. Even when women agree to be tested, many may not return for the results (Cartoux et al., 1998). Fears of stigmatization and, even worse, fear of abuse or being thrown out of their homes prevent women from being tested or returning for test results. Even when formula feeding is a viable option for an infected woman, she may choose to breast-feed to avoid suspicion that she is HIV infected ( Farquhar et al., 2000).

## Orphans

Although a real opportunity exists to decrease the number of children being infected with HIV, the number of infected adults worldwide continues to increase, resulting in a rapidly escalating number of children orphaned by the epidemic. According to UNAIDS statistics, by the end of 1999, approximately 13.2 million children less than 15 years of age had lost their mothers, or possibly both parents, to AIDS, and one-third of these children were less than 5 years old. More than 95% of these orphans live in sub-Saharan Africa (UNAIDS, 2000). Before AIDS, about 2% of children in developing countries were orphans, but by 1997, this figure had risen to 7% in most African countries and in some cases had reached as high as 11%. Orphaned children are often looked after by family members, frequently aging grandparents, and sometimes adolescent siblings. However, as the infection rate in a community increases, there are fewer working adults, and the result is a decrease in the resources available to care for orphaned children. In the United States, early projections that the number of motherless children and adolescents would exceed 80,000 by the end of the 20th century (Michaels and Levine, 1992) have not been completely borne out because HIV-infected parents are living longer. Nonetheless, many children are living in families seriously affected by the disease.

## Adolescents and Youth

It is now estimated that about half of all new HIV infections worldwide occur in people less than 25 years old ( UNAIDS, 1998; UNAIDS, 2000). The infection rate is increasing more rapidly among youth than it is among older persons. In the United States, even though there is a decline in AIDS incidence, there has not been a comparable decline in the number of newly diagnosed cases of HIV infection among young people ( Centers for Disease Control and Prevention, 1998c; 1999). HIV infection in adolescence tends to be more prevalent among girls than among boys because young women tend to have sex with older men. Among young men with HIV infection, homosexual transmission accounts for the single largest risk group. Evidence indicates that the advent of antiretroviral therapies sparked a resurgence of unsafe sexual practices among gay men, particularly among those less than 25 years of age ( UNAIDS, 2000).

Rates of infection with other sexually transmitted diseases (STDs) continue to be very high among youth and serve as indicators of their elevated risk of HIV infection. Of the 12 million cases of STDs that occur each year in the United States, one-fourth occur among teenagers, and two-thirds are acquired by 25 years of age (Chabon and Futterman, 1999). Drug use among adolescents can also increase the risk of HIV infection, not only through direct infection from injecting drug use, but also through increased risk for unsafe sex when inhibitions are lowered by alcohol or drugs. There is some evidence, however, that young people may be more likely to adopt safer sex practices than older persons. Studies carried out in Chile and Brazil show that compared with older adults, more young people use condoms, and in the United States, the proportion of high school students reporting either abstinence or the use of condoms has increased ( Centers for Disease Control and Prevention, 1998b).

## Clinical Manifestations of HIV in Children

Because of newer therapies, the prognosis of HIV-infected children is significantly improved compared with just a few years ago and can no longer be predicted with any certainty. Early studies documented that, unlike adults, children become symptomatic very quickly. For example, one study found that approximately 70% of infected children were symptomatic by 1 year of age (European Collaborative Study, 1991; Forsyth et al., 1996; Scott et al., 1989). The presentation of illness tends to be bimodal; some children become seriously ill in infancy (referred to as *rapid progressors*), whereas others remain relatively healthy for a prolonged period (referred to as *slow progressors*) (Blanche et al., 1990; Commenges et al., 1992).

In general, the clinical characteristics of HIV disease in children are similar to those in adults, although there are several important differences. Regarding opportunistic infections, the most significant difference relates to the timing of infection with *Pneumocystis carinii* pneumonia (PCP). Unlike in adults, in whom PCP generally occurs late in the course of disease, HIV-infected children tend to present with PCP very early (median age, 5 months). PCP used to be the leading cause of death in HIV-infected children; however, with present-day management, PCP is now less prevalent, and mortality from PCP has decreased significantly. Of the other opportunistic infections, *Mycobacterium avium* complex disease is the most prevalent among children. This usually presents as a generalized illness, often with fever, night sweats, weight loss, and diarrhea.

HIV-infected children also have a higher incidence of serious bacterial infections, such as sepsis, pneumonia, and meningitis. *Lymphoid interstitial pneumonia*, a condition affecting children, occurs only rarely in adults. This chronic interstitial lung disease usually presents in the second or third year of life and is reported to result from the coexistence of infection with Epstein-Barr virus and HIV. The course is often relapsing, but acute exacerbations generally respond well to therapy with steroids. Other manifestations of HIV disease are extremely varied and can affect all organ systems ( Forsyth, 1995). These include the lymphoreticular system, hematologic abnormalities, gastrointestinal and hepatobiliary disease, cardiomyopathies, and renal disease. The malignant diseases most frequently associated with HIV disease in children are lymphomas; Kaposi's sarcoma occurs only rarely. Growth stunting is one of the central effects of HIV disease ( Center for Disease Control and Prevention, 1999; McKinney et al., 1993); the result is that many infected children approaching adolescence are delayed in their physical development and are smaller than their peers, and they struggle to adapt to their unusual appearance and their physical immaturity. It is expected that, with improvement in disease management, growth problems will become less prevalent.

## Central Nervous System Disease

Early studies of central nervous system (CNS) manifestations of HIV disease suggested that between 40% and 90% of infected children had some degree of neurologic involvement (Belman, 1988; Epstein, 1986). These studies, however, were conducted in cohorts of children with more advanced disease. Later prospective studies documented rates of serious neurologic signs of 8% to 13% in HIV-infected children and of 19% to 31% in children diagnosed with AIDS ( Blanche et al., 1989; European Collaborative Group, 1990; Lobato et al., 1995; Msellati et al. 1993). The occurrence of CNS disease tends to parallel the bimodal progression described previously; children who develop severe disease and have opportunistic infections in infancy are those who are most likely to develop severe encephalopathy, whereas those who are *slow progressors* tend to be relatively spared and present with milder forms of CNS disease. Now that HIV-infected children are growing into their teenage years, there is likely a trimodal progression of disease. Some children, who were spared symptomatic disease earlier in life, may later develop CNS disease that is more comparable to HIV disease in adults ( Belman, 1992).

### EARLY-ONSET ENCEPHALOPATHY

Three-fourths of children with encephalopathy are diagnosed before the age of 36 months ( Lobato et al., 1995). Clinical manifestations include a decrease in head growth, loss of developmental milestones or failure to achieve new milestones, and progressive development of CNS signs. These signs include weakness and flaccidity, which, in time, progress to spastic paraparesis or quadriplegia, possibly with pseudobulbar palsy. Disorders of movement such as dystonic posturing or extrapyramidal tremors are less frequent, as are cerebellar signs ( Belman, 1992). There is often a characteristic facial expression marked by a paucity of facial movements, despite the child's appearing alert and wide eyed. The progression of disease is often rapid, occurring over 1 to 2 months, or it may fluctuate with periods of relative stability. If death does not occur from another cause, there is generally a decreasing responsiveness to the environment, and, in the end, the child is mute and quadriplegic.

Another group of infected children includes those who have more static encephalopathy. They do not lose milestones, but as they grow older, deficits in development become more evident, and new skills are acquired at a slower rate than normal. There can be abnormalities in either motor or cognitive development, or both. Some



children may show improvement over time, whereas others have little change.

#### LATE-ONSET ENCEPHALOPATHY

The risk of developing HIV-related signs of CNS disease later in childhood has not been adequately assessed. Early signs of CNS disease in older children include a decline in academic performance, emotional lability, problems with attention or worsening of attention deficit hyperactivity disorder, and possibly conduct disorders, although these same symptoms may represent manifestations of multiple causes unrelated to HIV (Belman, 1992). Cognitive deficits are more frequent than motor deficits, and impairments may become evident on tests involving quantitative, verbal, memory, visual-spatial, and time-orientation abilities (Exhenry and Nadal, 1996). Within the realm of language, expressive skills are more likely to be impaired than receptive skills (Wolters et al., 1995).

#### OTHER CENTRAL NERVOUS SYSTEM DISORDERS IN HIV DISEASE

Other manifestations of CNS disease in HIV-infected children include neoplasms, opportunistic infections, and strokes. Primary brain lymphoma or lymphoma arising elsewhere and metastasizing to the brain may occur at all ages. Presenting signs may include seizures, changes in behavior, or the appearance of focal deficits.

HIV-infected children are at increased risk of CNS infection with the usual childhood pathogens that cause bacterial meningitis. The most frequent opportunistic infections affecting the CNS in children are cytomegaloviral encephalitis, *Candida albicans* meningitis, and infection with *Mycobacterium avium-intracellulare* (Belman, 1992). Opportunistic infections of the CNS with *Cryptococcus*, *Toxoplasma gondii*, and cytomegalovirus occur less frequently in children than they do in HIV-infected adults, likely because in adults these infections are most often a reactivation of latent infection, which is not true for children. Congenitally acquired toxoplasmosis and cytomegaloviral infection have been described, but only rarely. Progressive multifocal leukoencephalopathy, a progressive demyelinating disease of the brain caused by the JC virus, which has been reported in adults with HIV disease, is now being reported in children and may occur more commonly as the population of HIV-infected children ages (Vandersteenhoven et al. 1992).

Children with HIV disease have an increased incidence of strokes which include both hemorrhagic and nonhemorrhagic causes. Hemorrhage is usually associated with immune-mediated thrombocytopenia, whereas nonhemorrhagic causes include abnormalities of cerebral blood vessels, meningeal infections, and cardiomyopathy (Belman, 1992; Park et al., 1988).

#### EVALUATION OF CENTRAL NERVOUS SYSTEM ABNORMALITIES IN HIV-INFECTED CHILDREN

An extensive history focusing on developmental milestones and a detailed neurologic examination are most important in alerting the clinician to the possibility of progressive encephalopathy. A full neurodevelopmental assessment is warranted when there is any suggestion of developmental abnormalities or sign of neurologic disease. Neuropsychological testing can be useful in establishing an initial baseline, monitoring subsequent alterations in cognitive processing secondary to CNS involvement, and devising appropriate rehabilitative interventions.

In general, results of cerebrospinal fluid studies are normal in HIV encephalopathy, although there may be slightly elevated protein and a mild, predominantly lymphocytic pleocytosis. Abnormalities associated with HIV encephalopathy are nonspecific and include enlargement of the ventricles, cortical atrophy, attenuation of periventricular white matter, and cerebral calcifications (Exhenry and Nadal, 1996). The calcifications, when they occur, are usually symmetrical and are located in the basal ganglia and periventricular frontal white matter, or occasionally in the cerebellar regions. Computed tomography scanning is most helpful in demonstrating cerebral calcifications, whereas magnetic resonance imaging is better at detecting the abnormalities in white matter. Abnormalities can be seen on neuroimaging studies, even in the absence of other signs of encephalopathy; however, repeated assessments are usually helpful in assessing progression of disease in an individual patient.

#### Treatment of HIV Infection

In the mid 1990s, because of a greater understanding of the pathophysiology of HIV disease and the advent of protease inhibitors, a class of antiretroviral medications, there were changes in the approach to treatment of HIV disease and a dramatic improvement in associated mortality and morbidity. Formerly, the approach had been to reserve the use of antiretroviral medications for patients who already had fairly advanced disease, as evidenced by a decline in CD4 cells or the development of symptoms of their disease. However, once it was understood that the period that preceded clinical signs of disease was not a period of latency, but, in fact, a period characterized by continuous replication of the virus and destruction of the immune system, newer recommendations for treatment were developed (Perelson et al., 1996; Saag et al., 1996). It is now recommended that the level of viral replication be used to inform decisions regarding treatment, such as when best to start treatment and when to change pharmacologic agents because a therapeutic regimen is not sufficiently effective. The extent of viral replication, commonly referred to as the *viral load*, is quantified by measuring the HIV ribonucleic acid (RNA) by polymerase chain reaction amplification.

The present treatment approach, referred to as *HAART* (highly active antiretroviral therapy), includes starting treatment early and achieving maximal suppression of viral replication using a combination of at least three different antiretroviral medications. Unfortunately, there is a potential for the virus to develop resistance to antiretroviral medications, particularly if someone has already been treated with a single medication before starting triple therapy. In such cases, once there is a rebound in the viral load, the virus is likely to be resistant to all three medications, and all three will need to be changed. In addition, within the different classes of medications is a tendency for cross-resistance to develop: if the virus is resistant to one medication in a class, it may also be resistant to others within the same class. Thus, even in this era of multiple medications from which to choose, the choices become limited once viral resistance has developed. Despite this limitation, some evidence indicates that although patients may have a rebound in viral load measurements on triple therapy, they continue to do better and have a slower progression of disease than would otherwise be expected (Kaufmann et al., 1998). As with other manifestations of HIV disease in children, advances in management are almost certainly having an effect on decreasing the prevalence and severity of CNS disease among children. Such a decrease in CNS manifestations of HIV disease has been well documented in adults, but it awaits further study in children (Mascke et al., 2000).

#### ADVERSE EFFECTS OF MEDICATIONS

All the antiretroviral medications have adverse effects, some of which may be potentiated by the use of other medications, including psychotropic medications. The finding that antiretroviral medications, particularly the protease inhibitors, can cause significant derangements in metabolic processes resulting in abnormal lipid profiles and glucose levels, and alterations in body composition are among the most important adverse effects (Carr et al., 1999). This latter, referred to as the *lipodystrophy syndrome*, may be very distressing to patients because of the changes in physical appearance. There is wasting and disappearance of fat from the face and limbs but an increase in fat in the abdominal region and over the lower part of posterior neck, referred to as a *buffalo hump*.

#### ADHERENCE TO MEDICATIONS

A major challenge posed by the advances in therapy of HIV disease has been the need to ensure that patients are able to take their medicines. Treatment regimens often call for a large number of medications, taken at regular intervals each day. Some of the medications are available only as large capsules that are difficult to swallow; others have a particularly bad taste. Meanwhile, studies have shown that missing doses of medications can lead to the faster development of viral resistance. One study reported a linear association between self-reported adherence and level of HIV viral suppression. Patients taking fewer than 80% of their prescribed doses of antiretroviral medications had a significant increase in viral load measurements compared with those who took more of their medications (Haubrich et al., 1999).

#### Development, Psychopathology, and Pediatric HIV

Intervention for children infected with or affected by HIV disease requires a comprehensive approach in which medical interventions are delivered with attention to the complex psychological and social needs of these children and their families and are provided within a developmental frame of reference. As with their overall cognitive development and capacity for logical reasoning, children's understanding of HIV and AIDS follows a predictable sequence. Preschool and early school-age children explain AIDS in terms of contiguous events. At this age, children may recognize that symptoms are the result of an underlying illness, but they are likely unable to explain the cause of HIV as a virus that is contracted in specific ways. With development, the causation of AIDS can be described as a sequential process related to internal sources, including sexual behavior, blood exposure, or external sources such as drug use. Typically, these explanations may provide later school-age children with a factually accurate account of how HIV and AIDS may be contracted; yet the explanation remains based on a series of events culminating in illness. The final stage of understanding is based on the expanded capacity for abstract reasoning that accompanies adolescence. Youth become able to describe the origins of

HIV and AIDS in terms of its underlying disease origin ([Walsh and Bibace, 1991](#)). Findings of an expectable developmental progression in children's understanding of HIV and AIDS should inform pediatric prevention and intervention efforts, because they highlight the importance of practices that are tailored to children's psychological, emotional, and cognitive capacities.

### *INFANTS AND TODDLERS*

Theorists from a variety of perspectives agree that development undergoes its most intensive period within the first few years of life and in the context of the child's relationship with a primary caregiver. The quality of attachment between infant and caregiver sets the stage for later development and influences how children progress in terms of their sense of self, their capacity for relationships with others, and their emotional resilience.

Preoccupation with the medical and psychological demands of coping with HIV disease may deplete families of the energy and psychological resources available to attend to normative aspects of early childhood development. For example, parents of an HIV-infected infant may be overcome with anxiety and remorse at having transmitted the virus to their child, and they may, in turn, become less responsive to the infant's cues related to basic needs involving nurturance, warmth, or sustenance. Although most caregivers remain able to provide for their infant's basic needs, for others, HIV may interfere with the establishment of a predictable and reciprocal pattern of interaction between caregiver and their infected or affected babies, and the result may be episodic and irregular dyadic engagement.

Infancy is a time of intimate contact between child and caregiver. Bodily fluids are routinely exchanged, although HIV-positive mothers may be advised to forego the intimacy of breast feeding, a problematic issue in developing nations, where a mother's breast milk represents a needed source of nutrition that cannot be easily replaced with costly formula that is difficult to procure. The expectable acts of defiance and desires for separation that mark toddlerhood may heighten unconscious fears of loss and permanent separation for affected children. The anger and defiance of the toddler represent developmentally appropriate and necessary assertions of autonomy and distinctness from a parent or primary caregiver ([Mayes and Cohen, 1993](#)). Infected, symptomatic young children require an enhanced degree of caretaking and protection that may constrict the expression of their normal drives toward the external world.

### *EARLY CHILDHOOD AND PRESCHOOL YEARS*

Arguably the most important developmental accomplishment of the young child involves the capacity for imaginative or pretense play ([Marans and Cohen, 1996](#)), which forms the leading edge of symbolic development and predates the use of complex language to mediate between internal states and action. At this age, play, which is intimately connected to language and cognitive development, represents a primary means for integrating internal thoughts and feelings with external experiences. The play of a child affected by HIV may involve repeated scenarios related to illness or loss, even though the same child may never be able to verbalize these concerns. Although children may gradually be able to understand basic facts about HIV illness and death, their ideas will likely remain concrete and specific to their life experiences. Metaphors about death may engender greater confusion, because young children are unable to abstract the basic concept that death represents a final cessation of life. For these reasons, the therapeutic use of play represents a significant means of assisting young children to cope with the feelings of guilt, loss, and abandonment that they cannot express.

Relationships with parents become intensified by a youngster's heightened feelings of power and pleasure in bodily sensations. When HIV becomes involved at this stage of development, emotional conflicts involving the simultaneous experience of holding both loving and rivalrous or aggressive feelings that may become especially threatening and upsetting. Parents with HIV or AIDS may be too psychologically and physically fragile for a child to express these concerns freely. The egocentric orientation of this stage may potentiate feelings of guilt and responsibility, because children may assume that their actions play a causal role in a parent's illness. Emotions may be undifferentiated, and the ability to recognize the possibility of holding varied and perhaps conflicting feelings contemporaneously will be compromised. When efforts to emulate the parent or caregiver are usually internalized as values and standards for personal behavior that are extended into a broader social world, strivings for independence based on these parental identifications may be unbearable for a child struggling with the implications of familial HIV. Independence and engagement with peers and a broader social world may be experienced as a rejection of the already fragile caregiver, and some children may find themselves unable to negotiate this expectable developmental step.

### *MIDDLE CHILDHOOD AND SCHOOL-AGE CHILDREN*

[Erikson \(1968\)](#) describes the stage of "industry versus inferiority" at which time children's sense of self becomes tied to their accomplishments involving school, peer groups, and age-appropriate activities. To develop a sense of "industry," children must negotiate earlier developmental tasks involving the development of a distinct sense of self that is connected to, yet relatively less dependent on, their primary caregivers. Children who have been unable to resolve conflicting feelings concerning medically compromised parents may remain tied to their caregivers in a manner that precludes their developmentally appropriate ventures into the world of peers and school. Although far from the physical and psychological maturity of adolescence and adulthood with its expectations of caring for oneself and others, the school-age child, one hopes, becomes an "agent" of his or her own actions. However, HIV-affected child may be unable to take a stance that carries an implicit rejection of a parent who may be ill.

Entry into elementary school, with its expectations for applied problem solving and learning, coincides with the child's enhanced capacity for problem solving and logical thought. Mastery of basic logical principles allows a child to infer cause-and-effect relationships, to devise alternate solutions to problems, and to comprehend reciprocal relationships. These advances may be invaluable as children attempt to understand their own illness or that of their parent. Language serves a regulatory as well as a communicative function; a child who can put words to his or her feelings is able to refrain from acting on those feelings and acquires greater mastery over his or her experiences. Unresolved anxiety about separation from a parent or caregiver who is ill not only may compromise these capacities but also may interfere with the development of relationships with peers and teachers. Preoccupation with illness may interfere with problem solving, learning, and academic performance.

### *ADOLESCENCE*

HIV infection has become an increasing problem among adolescents, particularly those who engage in high levels of risk-taking behaviors. In the 1 year from July 1995 to July 1996, more than 2,600 new cases of AIDS were reported among 13 to 24 year olds ([Centers for Disease Control and Prevention, 1999](#)). Because existing prevention and intervention programs have resulted in significant decrements in prenatal and perinatal HIV infection, many of these new cases may involve persons who contracted HIV during adolescence. As in the general population, HIV infection among adolescents is expected to affect young women and youth of African-American and Latino descent disproportionately ([Anderson and Morris, 1993](#)).

Many adolescents experiment with new behaviors in an attempt to fashion a separate identity and to establish their own autonomy from their families. Among affected youth for whom childhood has not been a normative process, the tasks of adolescence are still more challenging. These adolescents appear at greater risk of poor health outcomes as well as maladaptive behaviors leading to truancy, high-risk sexual behavior, criminal activity, substance abuse, and psychiatric disturbances ([Brown et al., 1997](#)). This cohort of adolescents rarely seeks needed medical treatment, thus increasing their own risk of contracting both STDs and HIV. Moreover, youth who are truant or who withdraw from school prematurely miss receiving information promoting healthy behaviors and preventing STDs ([Slonim-Nevo et al., 1995](#)). Adolescents within the criminal justice system, who present with high reported rates of infectious diseases, including HIV infection and AIDS, and limited histories of primary prevention and health care are thought to be among those at highest risk ([Hammett et al., 1998](#)).

For youth who are either infected with or affected by HIV or AIDS, the "second individuation process" ([Blos, 1962](#)) of adolescence may go awry as a result of chronic concerns about isolation, illness, and death. Although most adolescents strive to loosen the ties of childhood dependency by substituting intimate and romantic relationships with peers ([Freud, 1965](#)), those who must contend with personal or familial HIV may be unable to engage in these behaviors in the service of adolescent individuation and the formation of an independent sense of self. For these youth, the expectable rule transgressions and rebellions against parental strictures also become fraught with danger, real and imagined, about the potential consequences of their emerging sexuality and the strength of their angry, hostile, or defiant feelings.

As adolescents contend with their desire for adult sexuality and relationships, they now do so in the context of the potential for HIV infection. Peers have been found to exert a strong influence on adolescents' attitudes toward the practice of safer sex, and peer attitudes that favor or discourage condom use are strong predictors of their actual use ([Brown et al., 1997](#)). Adolescents, out of their own discomfort, may be unlikely to approach parents with these concerns, especially if these relationships are already marked by conflict, dispute, or unexpressed concerns about HIV disease. Although avoidance of topics related to HIV may reduce immediate feelings of discomfort and may prolong a wish to protect teenagers through an extended and protected childhood, adolescents continue to engage in and experiment with sexual activity at ever younger ages. Initial sexual encounters among teens are rarely planned, and this spontaneity decreases the likelihood of their using birth control or engaging in sexual practices aimed at reducing the chances of HIV transmission. Normative sexual exploration with same-sex and opposite-sex peers and



questions about gender orientation may also heighten the risk of HIV, as well as other STDs, and may limit an adolescent's attention to issues of risk amelioration ([Brown et al., 1997](#); [Hein et al., 1995](#)).

### **Psychopathology and HIV Disease**

Adolescents diagnosed with psychiatric disorders may engage in a range of behaviors that potentiates their risk of contracting HIV ([Brown et al., 1997](#)). Compared with nonpsychiatrically disordered peers adolescents with comorbid psychiatric disorders engage in more frequent substance abuse, including injecting drug abuse, unprotected sexual intercourse, and sexual activity with partners of unknown risk history. They also contract STDs with greater frequency than nonpsychiatrically impaired youth. [Brown and colleagues \(1997\)](#) note the lack of prevalence estimates for HIV infection among adolescents with psychiatric disorders but suggest seroprevalence rates in excess of the 0.2% to 2.2% rates among adolescents of indeterminate psychiatric status. This has been the case for psychiatrically compromised adults, in whom prevalence rates have ranged from 5.5% to 14%, a finding suggesting that psychiatric symptoms increase the likelihood of engaging in activities that pose particular risks for HIV infection.

Whether they arise from separate causes or result from the associated stress of HIV infection, comorbid psychiatric disorders further compromise the functioning and quality of life for youth infected with HIV ([Sherbourne et al., 2000](#)). Sherbourne and her colleagues conducted a psychiatric screening in a large national probability sample of 2,864 adults with HIV infection and found elevated 12-month incidence rates of major depression (36%), dysthymic disorder (26%), generalized anxiety disorder (16%), substance dependence (12%), and panic disorder (11%). Those with comorbid mood and anxiety disorders reported a poorer quality of life in terms of psychological functioning and medical status than HIV infected persons without evidence of a psychiatric disorder, a finding that warrants verification in youth samples.

Among HIV-positive adults, depression has been consistently associated with continued practice of unprotected sexual intercourse. HIV-positive adults who engage in high-risk sexual behavior appear more pessimistic and hopeless about their future and possess fewer strategies for coping with stress. As a result, they appear less likely to disclose their serostatus to potential sex partners or to initiate discussion of safer sex practices. Both negative affect and positive affect have been associated with unsafe sexual practices among HIV-positive adults, a finding suggesting to some investigators that high-risk sexual practices may represent an attempt to modulate affective experiences. From this perspective, positive moods may be associated with the decision to engage in unprotected sexual activity, perhaps fueled by denial and a wish to minimize awareness of personal HIV status. Subsequent negative moods then increase the likelihood that sexual activity may be used to ameliorate affective distress.

Studies have also shown that adolescents diagnosed with conduct disorder or a depressive disorder engage in higher rates of substance abuse, including intravenous drug abuse, and unprotected sexual activity, including prostitution ([Stiffman et al., 1992](#)). Efforts at primary prevention among depressed adolescents who may have acquired knowledge of HIV transmission may be confounded by their compromised sense of self-worth. Their pessimistic outlook may interfere with their ability to apply the information they have received to their behaviors ([Brown et al., 1997](#)). High-risk sexual behavior or drug abuse may also be symptomatic of a depressive disorder, in which the behavior represents a self-destructive act or an attempt to escape from an intolerable affective disturbance, a finding that has received empirical support among adult samples ([Brown et al., 1997](#)). Disorders associated with impulsivity or sexual preoccupation, such as bipolar disorder, borderline personality disorder, or impulse control disorders, may also interfere with the use of safer sexual practices ([Kalichman, 2000](#); [Sherbourne et al., 2000](#)).

Children with attention deficit hyperactivity disorder or conduct disorder are more likely to engage in impulsive actions that increase their risk of HIV infection ([Booth and Zhang, 1997](#)). Adolescents with conduct disorder abuse substances at higher levels, engage in sexual activity with a greater number of partners, and more frequently contract STDs relative to nondisordered youth. Even among runaway and homeless adolescents, who are much more likely than most adolescents to engage in high-risk sexual activity and injecting drug use, those with a comorbid diagnosis of conduct disorder are still more likely to have engaged in an exchange of sexual activity in return for money or drugs and to have unprotected sex with multiple partners. A diagnosis of conduct disorder increases the likelihood of injecting drug use by more than a 2:1 margin (odds ratio, 2.28) and the likelihood of exchanging sex for drugs or money by almost a 3:1 margin (odds ratio, 2.82). Adolescents who know someone infected with HIV are also at greater risk of drug injection (odds ratio, 1.40) or sexual exchange (odds ratio, 1.29) ([Brown et al., 1997](#)).

In addition to coping with the normative press of adolescent sexuality and identity formation, adolescents with cognitive deficits may also be at heightened risk of contracting HIV. Youth with mental retardation or borderline intellectual functioning may lack important skills related to judgment and logical decision making that would allow them to conduct accurate appraisals of the risks associated with a range of behaviors, including those related to sexual activity and substance use. They may lack accurate information about prevention and routes of HIV transmission, which may lead to erroneous conclusions about HIV. Their judgments may mirror those of younger children, who may overgeneralize their risk to common situations such as physical touching and sharing utensils ([Brown et al., 1997](#)).

### **SEXUAL ABUSE AND HIV TRANSMISSION**

Although HIV infection has been found to be a sequela of sexual abuse, the extent to which this has occurred is difficult to quantify. For example, a study of 9,136 cases of HIV infection reported to the Centers for Disease Control and Prevention through 1996 identified only 26 cases attributed to sexual abuse ([Lindgren et al., 1998](#)). In contrast, 14 (14.6%) of 96 children diagnosed with HIV infection at a single institution were confirmed to have been sexually abused ([Gutman et al., 1991](#)). Factors that put children at greater risk of HIV disease, such as substance abuse, social and economic marginalization, and dysfunctional lifestyle, are the same factors that put them at greater risk of sexual abuse. Some evidence indicates that the experience of sexual abuse in childhood may result in increased sexual risk taking and drug use in adolescence and adulthood, thus increasing a person's lifetime risk of HIV infection ([Allers et al., 1993](#); [Gellert et al., 1993](#); [Johnsen and Herlow, 1996](#); [Mullings et al., 2000](#)).

There are certain barriers to HIV testing of children who are being evaluated for sexual abuse, and recommendations vary from testing all children undergoing evaluation to testing only those for whom information suggests an increased risk of exposure to HIV ([Gellert and Berkowitz, 1994](#); [Gutman et al., 1993](#)). Risk of transmission of HIV should be a particular consideration if a child is evaluated immediately after an incident of sexual abuse, because of the possibility of providing prophylactic antiretroviral medications to prevent transmission. Such postexposure prophylaxis has been shown to be beneficial in needlesticks and other types of occupational accidents ([Henderson, 1999](#)), and it is now being used with persons who have been raped or have put themselves at risk of sexual transmission ([Centers for Disease Control and Prevention, 1998a](#)).

### **EFFECTS OF HIV/AIDS ON FAMILY FUNCTIONING**

HIV affects families across generations, as children, parents, and grandparents confront illness and premature loss of loved ones. HIV-infected children must contend not only with multiple losses and relationship disruptions resulting from the illness of their parents, but also with effects of their own illness that may remove them from normative childhood activities and may destabilize their world. Families with HIV-infected children tend to be socially isolated, with fewer available supportive relationships at times of crisis or acute illness. Uninfected siblings, grandparents, and other family members must cope with uncertainty, guilt, and the fear of anticipated and premature loss, as well as the social and psychological burden of caring for chronically or terminally ill relatives ([Mellins and Ehrhardt, 1994](#)). Family structures are often disrupted by parental illness, and children may be forced to change caregivers frequently as they move within or beyond their family networks.

### **CHILDREN AFFECTED BY HIV AND AIDS**

The numbers of children affected by parents or caregivers with HIV disease will continue to rise, with substantial rates of HIV infection among young, minority women, 50% of whom are estimated to bear children ([Family Health Project Research Group, 1998](#); [Pilowsky et al., 2000](#)). Unfortunately, family members as well as professionals may fail to recognize the unique concerns and obstacles that threaten the development of these children. As witnesses to repeated periods of acute, incapacitating parental illness, these children anticipate the death of one or both parents and worry about who will take care of them when their parents are no longer available. They may be left to contend with feelings of sorrow, anger, guilt, and confusion in isolation as other family members turn their attention to the infected adult or struggle with their own histories of loss ([Gewirtz and Gossart-Walker, 2000](#); [Ickovics et al., 1998](#)). Children in HIV-affected families face additional burdens related to parental incarceration, family and residential instability, exposure to violence, caregiver substance abuse, and social isolation ([Gewirtz and Gossart-Walker, 2000](#); [Maman et al., 2000](#); [Nagler et al., 1995](#)). Family and caregiver relationships, including those with fathers, may be unstable, marked by erratic and unpredictable contact ([Wright and Draimin, 2000](#)).

In an effort to redress the paucity of data on the functioning of HIV-affected children, Forehand and his colleagues developed a multidisciplinary, longitudinal study of HIV-infected mothers and their affected, HIV-negative children ([Family Health Project Research Group, 1998](#)). Researchers followed an urban sample of 107 6- to 11-year-old HIV-affected African-American children and a matched comparison group of 150 mothers and children from the same locale. After controlling for the

severity of AIDS symptoms, 3-year results indicate higher levels of internalizing and externalizing psychiatric symptoms among the HIV-infected mothers and their affected children, as well as greater impairments in children's social competence.

The unpredictable nature of HIV disease, marked by multiple episodes of relapse and recovery and a chronic course of illness, limits parents' ability to provide consistent and stable care and perpetuates a sense of anxiety and dread among their children ( [Gossart-Walker and Moss, 1998](#)) ( [Rotheram-Borus et al., 1998](#)). Ironically, many parents may neglect their own health as they focus on the pressing needs of their children, and children's longer-term needs for permanency and security may be sacrificed by their parent's unwitting neglect of their own important health issues ( [Wiener et al., 1994](#)).

Children who confront HIV disease in a parent or caregiver are often unable to express their expectable feelings of anger and guilt because of an unrecognized fear that they may become overwhelmed by their distress or may hasten the death of their loved one ( [Gossart-Walker and Moss, 1998](#)). Many HIV-affected children contend with a sense of foreboding about their future and that of their caregivers that can neither be expressed nor anticipated. Adolescents affected by parental HIV infection and AIDS are particularly vulnerable. As [Zayas and Romano \(1994\)](#) noted, "Perhaps no group of children in modern time has been more battered by the combination of social and familial decay and a devastating illness, coupled with the normal storms of adolescence." Adolescents residing with a parent diagnosed with AIDS consistently report high levels of parent-child conflict, academic failure, peer relationship problems, and criminal behavior, with estimates that 25% to 73% of HIV-affected adolescents present with clinically significant difficulties ( [Rotheram-Borus et al., 1998](#)).

#### STIGMA AND SECRECY

The psychological disability secondary to the guilt, shame, and anxiety of living with a family member whose illness may not be discussed or even revealed represents a notable challenge to the development of HIV-affected youth ( [Nagler et al., 1995](#)). The secrecy and stigma surrounding an HIV diagnosis, as well as its uncertain course, compromise a developmental trajectory already affected by inconsistent caregiving and the specter of loss and abandonment. Psychological forces of denial and shame conspire to leave parents and children isolated and overwhelmed. Although children know of the parent's illness, they may not be provided with opportunities for identifying the illness and discussing its ramifications, processes that allow them to organize their ideas, fears, and feelings ( [Nagler et al., 1995](#)). Should death occur in the absence of disclosure about the illness, children may be unable to metabolize their complex reactions and may be left to struggle with their poorly understood and painful memories and feelings. Ironically, even though many parents are reluctant to inform their children of their HIV infection, some adolescents become caregivers to sick parents and younger siblings without ever being able to name the illness that has debilitated them.

Societal ramifications in the form of stigma and shame combine to marginalize affected families further and to reinforce their need for secrecy. Families must manage a chronic, incurable disease in the context of a society that adopts a punitive stance toward the behaviors that result in HIV infection ( [Nagler et al., 1995](#)). In the presence of this social disapprobation, many parents elect to keep their diagnosis secret from family, friends, and society as a whole. Self-imposed secrecy and reactions to social stigma may preclude families from procuring necessary treatment, seeking assistance with permanency planning for affected children, and obtaining needed forms of social support ( [Gewirtz and Gossart-Walker, 2000](#)). Although children's development may go awry in the face of the inexorable stressors associated with the illness, these psychiatric complications cannot be addressed adequately when they represent manifestations of unacknowledged HIV infection or AIDS.

#### LOSS AND BEREAVEMENT

Children, whether infected or affected by HIV, anticipate and experience bereavement on a continual basis. As with other types of trauma, the adjustment of children who lose one or both parents to HIV infection or AIDS remains heavily influenced by premorbid functioning and the stability of subsequent caregiving relationships ( [Forehand et al., 1999](#)). Although debates about children's ability to mourn continue among psychological theorists, most agree that mourning, particularly in relation to the loss of a parent, requires the prior establishment of object constancy, a sustained mental representation of the caregiving relationship. For many HIV-affected children and youth, the death of a parent has been preceded by multiple disruptions of attachments because of the instability and relapses attributable to the progression of the disease. As a result, the internal representations held by these children related to caregiving lack the necessary features of predictability and responsiveness that allow mourning to take place and serve to promote healthy adaptation to loss.

#### DISCLOSURE

Despite emerging findings for the benefits of disclosure, many parents are unable to reveal an HIV diagnosis to an affected or infected child. As [Nagler, Adnopolz, and Forsyth \(1995\)](#) observed, children "represent parents' replacements and hope for immortality," yet these aspirations become untenable in the presence of HIV disease and AIDS. Parents may be overcome by guilt and beset by self-recrimination at having transmitted the virus to their children. Disclosure forces parents to confront their personal responsibility and to acknowledge the negatively sanctioned behaviors related to substance abuse or sexual activity ( [Lipson, 1993](#); [Lipson, 1994](#)). Unable to tolerate their own remorse and psychological pain, parents may withdraw and may deny an illness that is evident to their children and loved ones ( [Faithfull, 1997](#)). Other parents struggle with their knowledge of HIV but equate disclosure with harming their children and instead attempt to protect children from this painful knowledge, to preserve the mystique of childhood innocence. [Lipson \(1993\)](#) suggests that the conscious fear that children cannot understand the ramifications of HIV masks a deeper fear that children will indeed grasp its fatal implications.

#### Prevalence of Disclosure

Despite interest in disclosure and its potential implications for intervention with HIV-infected and affected children, few studies have examined parental decisions about revealing their diagnosis to their children. In the early 1990s, relatively few HIV-infected children were informed of their diagnosis ( [Lipson, 1993](#)). Several years later, [Rotheram-Borus and her colleagues \(1997\)](#) reported a disclosure rate of approximately 75% by HIV-infected parents. Older children were more likely to be told of a personal or parental diagnosis of HIV infection or AIDS; infected parents tended to disclose increased information as they became more symptomatic ( [Armistead et al., 1997](#); [Ledlie, 1999](#)).

The issues of disclosure and permanency planning become still more important with the ongoing evolution of the HIV and AIDS epidemic from an acute, fatal illness to a chronic condition that nevertheless portends a foreshortened life span as children experience the painful consequences regardless of their specific knowledge about the disease ( [Murphy, 2000](#)). Clinicians have come to accept the axiom that knowledge is preferable to the fantasies that children may construct about their illness or their parent's illness. For some children, disclosure may dispel erroneous beliefs about personal responsibility for the illness, yet it may also heighten fears and anxieties related to abandonment. Although secrecy can represent a burden for child and parent alike, openness may accentuate their shared anxiety because of the unpredictable trajectory of the disease ( [Nagler et al., 1995](#)). Thus, disclosure should represent a process that neither begins nor ends with the actual revelation of HIV infection.

#### Stages of Disclosure

A model of disclosure, based on more than a decade of clinical intervention, has emerged from the work of the Yale Child Study Center Program for HIV Affected Children and Families ( [Adnopolz, 2000](#); [Geballe et al., 1995](#); [Murphy, 2000](#)). The program informed and enriched Tasker's five-stage model of disclosure ( [Tasker, 1995](#)) with a developmental perspective that clarifies and integrates factors favoring and mitigating against disclosure.

As previously noted, many parents, whether through conscious or unconscious choice, do not inform their children of an HIV diagnosis. Tasker has identified a *secrecy phase* during which parents struggle with their own acceptance of the illness and their wish to deny its implications. Their unwillingness to reveal their health status may also be influenced by the negative associations among HIV and AIDS, promiscuous sexual activity, and the use of illicit substances. Rather than share any knowledge of their illness, parents may look to their health and mental health providers for repeated reassurance that their serostatus remains confidential. Children whose parents are unable to move beyond this phase are left to cope with reactions to their overwhelming sense of loss, guilt, and abandonment in secrecy and isolation.

Parents in the second, *exploratory phase* demonstrate ambivalence about disclosing their HIV diagnosis, although the need to guard against revelation diminishes over time. The Yale program found that the average time between parent's receipt of the diagnosis of HIV and actual disclosure of their status to their children is 2½ years. The exploratory phase generally progresses from secrecy and denial of HIV to tentative attempts to name the disease through the use of euphemisms. Some parents may admit to being "sick," without further amplification, whereas others may report secondary illnesses associated with HIV. For example, one youngster was told that he had a heart condition serious enough to require frequent medical visits, but he was not told of his HIV diagnosis.



As parents move closer to revealing an HIV diagnosis, they enter the *readiness phase* of disclosure in which they rehearse possible disclosure scenarios, either in fantasy or explicitly, with a trusted friend or professional helping to guide them through the process. Practicing the form, timing, and content of disclosure provides opportunities for parents to consider the possible reactions of their children and to identify strategies to assist their coping responses.

The *disclosure phase* begins at the time children are told of an HIV or AIDS diagnosis. Disclosure provides the child with the words to name a condition that is pervasive in its effects but cannot be discussed openly. It allows menacing secrets to be replaced by knowledge, and it shifts the developmental tasks toward coping with the painful reality (Nagler et al., 1995). The following case vignette from the Yale program illustrates the importance of disclosure.

The patient was a 16-year-old perinatally infected boy who had never been told his diagnosis even though he had lost both his parents and several other relatives to AIDS and he had received his medical care from a pediatric HIV specialty clinic since birth. Disclosure was prompted by a period of medication noncompliance and resulted initially in feelings of shock and despair. However, in time he was able to develop an important therapeutic alliance in which he was able to address the tumultuous feelings associated with his own illness and begin the process of grieving his multiple losses.

Although the American Academy of Pediatrics has recommended that HIV-infected children be informed of their diagnosis (American Academy of Pediatrics, 1999), there are few protocols to guide the clinician and family through the ambivalence and regret preceding the actual disclosure. Studies of HIV-infected children report diminished parental depression and improved family functioning after disclosure (Wiener et al., 1998). The effects on children's psychological functioning appear more equivocal. Although disclosure of other life-threatening illnesses, such as cancer, appears to alleviate anxiety (Lipson, 1993; Reeve and Kline, 1995), the increased burden of social stigma and isolation associated with HIV infection may cause some children to experience heightened levels of distress. Symptoms may abate as children are afforded continued opportunities for discussion and expression with family members, mental health workers, and pediatric providers. Once additional stressors are accounted for, disclosure of an HIV diagnosis does not appear to result in further decrements in children's emotional or social functioning. Instead, disclosure may alleviate the burden of unspoken fears and may become a basis for further integration of painful knowledge and a foundation for permanency planning efforts. However, clinical experience suggests that external support before and after disclosure is essential for the adults providing the information and the children who receive it.

With the burgeoning rates of HIV infection among adolescents, disclosure will increasingly become a decision that will be made by youth as well as their adult caregivers. A comparison of mother-child dyads in which the child had a positive HIV status found that children who had disclosed their diagnosis to similarly aged friends reported no decrements in their behavior or self-concept. Importantly, children who disclosed their status to their peers experienced a statistically significant increase in their immune response (CD4 percent) relative to their nondisclosing, HIV-positive peers, a difference that was apparent regardless of age or medication regimen (Sherman et al., 2000). Although both content and context dictate responses to disclosure by children, families, and friends, the study by Sherman and her colleagues adds weight to recommendations stressing the importance that children become aware of their diagnosis and exert control over the management of their illness, when possible.

#### PERMANENCY PLANNING

For many families, disclosure of HIV can be seen as preparation for the task of permanency planning for children who will be orphaned by the disease. Long considered the most appropriate mechanisms for child rearing, families offer children the nurturance, protection, and guidance essential for healthy development. Within a stable, secure, and consistent family environment, children acquire a set of beliefs and values, learn standards of behavior, develop expectations for the future, and gain knowledge about themselves and the world (Adnopolz, 2000). According to Solnit and his colleagues (Goldstein et al., 1996; Solnit, 1976), the family serves as "the bridge from the past to the future," offering children a sense of being rooted in time and place. This process, which suffuses development, underscores the centrality of long-term family stability and coherence.

Permanency planning with affected families includes efforts to assist parents to identify a familiar and accepted surrogate caregiver able to offer children affective support, reassurance, and understanding during periods of the parent's acute illness and after their deaths. However, it is not surprising to find that most parents have great difficulty making realistic plans for the future of their children. Parents may approach the possibility of premature death with untested assumptions about their children's future. They may assume that a relative or older sibling will take custody of younger children, yet they may be unaware that without specific planning these assumptions easily go awry. The process of developing a workable plan that names adults who will serve as parental replacements and calls into view a future for children in which parents will not participate is long and painful. Although efforts can be made to identify a potential guardian earlier in the disease process, most frequently this work does not begin in earnest until the terminal stages of the disease. Many states now provide legal remedies designed to ease the strain on parents of making custodial decisions and to allow for a more flexible approach to guardianship. It is now possible for children to be cared for by a temporary guardian who is responsible only when parents are too ill to fulfill their familial obligations. Guardianship reverts to parents as their acute symptoms remit and they are able to resume responsibility. Standby guardianship and coguardianship are additional legal options that have been developed in response to the special needs of HIV-affected children and families and as AIDS has come to resemble other chronic illnesses with unpredictable periods of relapse and recovery lasting over time.

The caregivers selected by parents most frequently are members of the extended family who represent the most realistic option for supporting the psychological and physical development of affected children and providing continuity in the midst of loss (Adnopolz, 2000; Geballe, 2000). The multiple functions served by extended families include maintenance of the child's ties to family history, culture, and beliefs (Wilson, 1989). Although strenuous attempts may be made to place children who lose a parent to AIDS in the care of relatives or others known to them through their family's social networks, siblings remain together in fewer than 50% of these cases (Draimin, 1995). The separation from other siblings can be experienced as a replication of the loss of the parent, a situation that could be avoided with careful planning and a commitment to maintaining the integrity of sibling groups.

The interrelationship between disclosure and permanency planning was demonstrated in a study of 151 HIV-infected parents and their 171 adolescent children (Rotheram-Borus et al., 1997). Almost three-fourths of the teens in the study had been told of their parent's HIV diagnosis, and most were aware of plans for their future custody. Virtually all parents who made a permanency plan consulted the potential guardian, and almost all plans were agreed to by the chosen guardian. Legal custody arrangements were rare, however, occurring in less than one-fourth of cases, a statistic attesting to the difficulty parents experience in taking the final step toward the release of their custodial interest in their children. The high rates of permanency planning attempts are encouraging, because they indicate that many parents are able to address their children's needs in a planned and thoughtful manner. However, the relative lack of legally sanctioned custody arrangements raises concern. Without legal intervention, it is quite possible that the plan developed by the parent may be jeopardized by the trauma, stress, anger, frustration, and disorganization that frequently follow the trauma of the parent's death. Helping parents to embed their custodial preferences in law remains an important aspect of permanency planning.

#### Mental Health Interventions

HIV disease exacts a dire toll on families and often compromises their ability to use available mental health care. Traditional modalities of office and clinic based care may be ill suited to families who must contend with multiple medical appointments and complex treatment regimens within a sometimes disorganized and overstressed family environment. The myriad burdens associated with HIV infection and AIDS may overwhelm even the most concerned parents. In light of these obstacles to traditional service use, we advocate a comprehensive, home- and community-based approach to the mental health care of children and families beset by HIV infection and AIDS.

The Yale Child Study Center Program for HIV-Affected Children and Families was created to provide home-based psychiatric services to children and their families affected by HIV and AIDS (Adnopolz, 2000; Adnopolz et al., 1994; Gewirtz and Gossart-Walker, 2000; Murphy, 2000; Nagler et al., 1995). The program serves children, adolescents, and families who are either infected with or affected by HIV or AIDS. Clinical services are distinguished by their comprehensive and flexible nature; duration, intensity, and location of treatment are determined based on a thorough evaluation of the needs of each child and family. Specific interventions include home- and community-based clinically informed case management, psychotherapy, psychiatric consultation and medication management, psychological assessment, support groups, family stabilization, 24-hour emergency consultation, and coordination of medical providers for biological, extended, and foster care families. Clients are referred from hospital- and community-based programs, including pediatric and adult AIDS providers, social service agencies, juvenile justice personnel, and community police officers; caseloads consist of families with multiple medical, psychological, social, and economic problems related to HIV disease. A staff of pediatric AIDS providers, psychiatrists, psychologists, social workers, and family support workers provides home- and community-based treatment to more than 70 families each year. Since its inception in 1989, the program has served more than 475 children and families.

A clinical team consisting of a social worker or psychologist, paired with a case manager with experience within the target community, coordinates services for each

family. Teams provide in-home individual, group, and family therapy to children and their families, as well as concrete assistance in the form of case management, parent guidance, and practical assistance with housing, entitlements, and coordination with other systems of care. The treatment team is able to work with families for extended periods, ranging from several months to many years. In addition to the variable length of intervention, which is tailored to the ongoing treatment needs of children and families and includes attention to medical compliance, family stabilization, and permanency planning, clinical providers remain available to families on a 24-hour basis through an emergency pager service. Significantly, the team is able to respond directly to families at times of medical or psychological crisis, thus ensuring continuity of treatment relationships and overall care.

The intensity of intervention is reviewed through formal supervision and case presentation, as well as through regular review of treatment plans with the families. Some families may have daily contact with the team at time of acute crisis and need, whereas for others, contact is maintained at a minimum to solidify already achieved therapeutic gains. Beyond the utility of coordinated therapeutic and case management services, the use of a clinical team serves an important role in terms of the professional development of its members. The sharing of their professional experience provides an ongoing source of support and consolation in the face of the inevitable losses that confront them.

#### *SPECIALIZED INTERVENTION FOR HIGH-RISK ADOLESCENTS*

Adolescents have been cited as a high-risk group for HIV infection and AIDS, with concerns about their welfare moving to the forefront as rates of prenatal and perinatal HIV infection have diminished. From limited research, the relationship of knowledge about HIV and AIDS and risk behavior remains unclear; investigators report contradictory results about the application of relevant knowledge to adolescent risk behaviors. [Rotheram-Borus et al. \(1997\)](#) note an increase in high-risk behavior among adolescents who have been informed of their parents' HIV infection. Clinicians in the Yale program have observed similar situations in which affected teens engage in multiple instances of unprotected sexual activity, a finding suggesting that denial may be a predominant mechanism by which adolescents cope with the complex emotional sequelae of HIV infection in themselves or in loved ones. In these instances, detachment and avoidance of emotion provide defenses against the overwhelming chaos and anxiety that multiple losses present. Reenactment of the very behaviors that led their parents to contract HIV may emerge from the age-appropriate feelings of invincibility and may represent a means to control the internal distress and external chaos experienced by adolescents who themselves must confront their own heightened sexual drives. These behaviors may also represent expressions of internalized rage against parents for their impending abandonment.

Adolescents as a group may engage in additional activities that increase their risk of contracting HIV. Recognized risk factors include relationships with multiple sexual partners, teenage pregnancy, prostitution, substance abuse, criminal activity, and school truancy. The presence of several known risk factors has been found to be more detrimental to psychological and developmental outcomes than the presence of a single risk factor, with an exponential relationship between number of risk factors and severity of psychosocial impairment. This documented comorbidity of HIV and other risk factors in vulnerable families implies a greater likelihood of finding adolescents affected by HIV within impoverished, marginalized urban neighborhoods or nonnormative settings, such as within the juvenile justice and mental health systems.

Despite predictions of a rising incidence of HIV infection among adolescents, appropriate prevention efforts and clinical responses have often been lacking ([Kalichman, 2000](#)). Prevention curricula based on a psychoeducational and problem-solving approach to sexual and other risk behaviors have demonstrated a range of positive outcomes in general school and community settings, including increased condom use during sexual intercourse, decreased high-risk (unprotected) sexual behavior, decreased sexual activity with multiple partners, increased age at time of first intercourse, and increased discussion of sexual practices between parents and children ([Jemmott et al., 1992](#); [Lefkowitz et al., 2000](#); [Main et al., 1994](#); [St. Lawrence et al., 1995](#)). The extent to which primary preventive approaches may generalize to high-risk, psychiatrically impaired, or juvenile delinquent cohorts outside these mainstream settings remains untested ([Schoeberlein et al., 2000](#); [Schonfeld, 2000](#)), yet those at greatest risk of contracting HIV or AIDS tend to be these same adolescents whose isolation from traditional health and mental health treatment systems complicates their receptivity to primary and secondary prevention efforts.

Clinical and case management services provided in collaboration with the juvenile justice system, in which other prevention efforts have made little headway, have the potential for decreasing the behaviors of adolescents that place them at risk of contracting HIV. Given the treatment-refractory nature of this population, the goals of ameliorating psychological impairment, reducing the incidence of high-risk behaviors, and providing access to appropriate medical and mental health care can be best accomplished through home- and community-based services. Changes in the juvenile justice system toward community-based probation, with its emphasis on alternative sanctions, have made inroads in providing structured, therapeutic programming as a component of court sanctions. Home-based providers with existing collaborative relationships with law enforcement, juvenile justice, school, and medical personnel can serve as a focal point for coordinated psychosocial and medical interventions for youth who are most likely to engage in the high-risk behaviors that may culminate in HIV infection.

At the Yale Child Study Center, home-based clinical services have been integrated with community juvenile probation in an effort to provide acute community psychiatric care to youth who appear at greatest risk of contracting HIV through their own risk behavior. Risk factors that result in a referral from a community-based probation officer include: (a) a caregiver with HIV infection or AIDS, (b) high-risk sexual behavior (teen pregnancy, prostitution, diagnosis of an STD, multiple sexual partners, unprotected sexual contact), (c) significant drug abuse, or (d) any history of injecting drug use. Youth and families are assigned to a clinical team consisting of a psychiatric social worker and a paraprofessional case manager with extensive community experience. The core of the intervention consists of the coordinated efforts of the clinical team who forges a consistent and supportive relationship with the family. A focus on specific risk behaviors and their amelioration occurs in the context of the clinical relationship, which itself serves a catalytic function in promoting family change. Further pragmatic and discrete interventions assist the family in supporting changes in the adolescent's behavior by providing adolescents and their families with access to affordable health insurance and ongoing medical and mental health care. The collaboration between the clinical staff and personnel from juvenile probation is critical to the intervention. For delinquent youth, who may present with little intrinsic motivation to alter their behavior or to comply with necessary medical or psychiatric treatment, the probation officer represents an important figure of benevolent authority and containment. Thus, probation officers reduce delinquent youths' feelings of anonymity in their communities and can complement community-based clinical interventions that would be unlikely to succeed in a clinic-based setting.

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#### CASE ILLUSTRATION

Raymond is a 16-year-old youngster who lives with his mother and her boyfriend. His mother contracted HIV during his early childhood through injecting drug abuse. Her continued drug use and sporadic compliance with HIV medications compromised her health to the extent that she often lacks the energy and motivation to leave the house or participate in daily activities. Despite the encouragement of the home-based clinical team, she has not been able to discuss her illness with Raymond, nor has she addressed questions related to permanency planning. In the meantime, Raymond has become increasingly depressed. He is often truant from school and instead remains in his bedroom throughout the day. He has withdrawn from friendships and has become increasingly hostile to his mother. Coordinated responses by the clinician-family support worker team and the community probation officer have resulted in improved school attendance this year. His clinician conducts regular sessions with Raymond during the school day, facilitates school attendance and educational planning, and attempts to address his increasing despondency through individual and family interventions. The family support worker works with his mother to address her own illness and her capacity to plan for her son's future. Meanwhile, the probation officer has become a benign authority figure known to Raymond, his family, and school personnel as committed to his school attendance, treatment participation, and involvement in prosocial activities.

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#### CONCLUSIONS

In the decades since the identification of HIV disease, advances in medical treatments, particularly antiretroviral pharmacotherapy, have transformed the disease from an acute illness with a vastly foreshortened life span to an entity with the characteristics of a serious chronic illness. These hard-won gains have improved the quality of life for many infected persons, yet for others throughout the world, HIV infection continues to proliferate at truly epidemic proportions. Within the United States, perinatally acquired HIV has diminished from approximately 25% to less than 2% of births to HIV-infected women. Like the virus itself, the demographics of HIV have mutated and evolved. Most new HIV infections appear among persons less than 25 years of age, with women of childbearing age and minority communities experiencing a disproportionate share of newly diagnosed infections. Although fewer American infants and children will be infected with HIV infection or AIDS, increasing numbers will be affected by the illness of their parents or others who play significant roles in their lives. Data now suggest that affected children are at increased risk of becoming infected as adolescents because of their own risk-taking behaviors.

Reports issued by the Institute of Medicine ([Institute of Medicine, 2000](#)) and the Office of National AIDS Policy in conjunction with the White House ([Office of National AIDS Policy and the White House, 2000](#)) highlight these worrisome developments in the HIV and AIDS epidemic that require a coordinated national and international effort toward prevention of new HIV infection and provision of care to those who are already infected. A comprehensive strategy to ameliorate the effects of HIV disease on children and adolescents should arise from a concerted, national strategy focusing on both prevention of new HIV infection and treatment provision to HIV-positive youth. Research will be needed to evaluate the transportability of prevention programs from school and clinic settings to community settings, such as the juvenile justice system, in which youth at greatest risk are likely to be found. The availability of effective treatment for HIV disease does not, at present, portend a cure. Nonetheless, counseling and testing remain crucial ingredients in a public health approach to HIV infection and service provision. Given the myriad



psychosocial stressors encountered by HIV-infected and affected children and youth, comprehensive and effective medical and mental health services remain crucial. Access to care must be extended to children, adolescents, and families who have traditionally existed at the margins of the health care delivery system. Psychiatric treatment must address children's well-being in the context of their families, so interventions address children's need for stability of caregiving relationships and safety through a comprehensive strategy that addresses the medical and mental health needs of individual children, supports family functioning, addresses permanency planning for infected and affected youth, and coordinates care among psychiatric, medical, and social service providers ( [Burr and Lewis, 2000](#); [Casey Family Services, 1999](#)).

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# 99 CARE OF INFANTS, CHILDREN, AND ADOLESCENTS WITH BURN INJURIES

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Fire over the ages has inspired awe, wonder, fear, allegory, and myth from Hephaestus, the Greek god of fire, peaceful but ugly and deformed and wed to the beautiful Aphrodite, to Daedalus, flying too near to the sun, losing his wings, and plunging to his death. The Jews escaped Egypt through a miracle: “the Lord went before them by day in a pillar of a cloud, to lead them the way; and by night in a pillar of fire, to give them light” ( [Exodus 13:21](#)). A Mother Goose nursery rhyme catches the child’s wish for a mother’s rescue with “Ladybug, ladybug, fly away home. Your house is on fire, and your children will burn.” For authors and poets, fire can be an image of mental awakening, as in *A World Lit Only by Fire* ([Manchester, 1992](#)) and “The Poets light but lamps...” ( [Dickinson, No. 883](#)), and feelings, “Some say the world will end in fire, some say in ice. From what I’ve tasted of desire, I hold with those who favor fire...” ( [Frost, 1923](#)). Also pertinent is Tennyson, as he catches the power of language to assuage with “But for the unquiet heart and brain/A use in measured language lies; The sad mechanic exercise, Like dull narcotics numbing pain” ( *In Memoriam*, 5, st 2). We feel these evocative images, such as fear and rescue, ugliness and beauty, death and miracle, each day in the care of burned children.

## HISTORY

The era of intensive research and treatment of burns began during World War II, at the Massachusetts General Hospital after the Cocoman Grove Fire in Boston on November 28, 1942. Cobb and Lindemann chronicled the posttraumatic reactions of the survivors of that fire ( [Cobb and Lindemann, 1943](#)). Lindemann, in a classic article about them, also described the symptoms and management of their and their relatives acute grief ( [Lindemann, 1944](#)). Since then, images of war have made plain for all to see the catastrophe wrought by fire. Drawings by child and adult survivors of Hiroshima, televised images from Vietnam, films depicting a nuclear holocaust, and the terrorist attacks of Sept. 11, 2001, have riveted the world’s attention. Such images evoke terror but also compassion, and a positive benefit of such compassion is the near-miraculous care now available for children with burns.

In Boston, the Shriners Hospitals joined with the Massachusetts General Hospital and Harvard Medical School in 1968 to build the Shriners Burns Hospital, recently rebuilt, where care is provided at no cost to burned children and their families. The Shriners have also built pediatric burn hospitals in Cincinnati, Galveston, and at the University of California at Davis. Awareness in the 1960s and 1970s of the high rate of burns to children led to the founding of trauma centers including pediatric burn units in children’s hospitals and general hospitals throughout the United States and Canada. There are now designated burn centers for children in most major cities of the United States, Canada, Europe, and many other countries. Research has led to improved methods of resuscitation and transport, pain management (Stoddard et al., 2002), excision and grafting ( [Sheridan et al., 1994c](#)), cardiovascular and infection control, artificial skin and skin substitutes ( [Sheridan et al., 1994a](#)), plastic surgical techniques ( [Salisbury, 1992](#)), and other advances. Together, these have improved survival rates and outcomes ( [Blakeney, et al., 1998](#); [Ryan et al., 1998](#); [Sheridan et al., 2000](#)).

Child psychiatrists, such as Norman Bernstein ( [Bernstein, 1976](#)) and Richard Galdston ( [Galdston, 1972](#)), a child psychoanalyst, were pioneers in the psychiatric care of burned children, their families, and in work with the “burn team.” [Stoddard \(1982a\)](#) initially highlighted a developmental approach to pain of burned children, and an adult psychiatrist, Samuel Perry, and colleagues spearheaded the focus on undertreatment of pain in children and adults through a national survey of burn units ( [Perry and Heidrich, 1982](#)). Trauma surgeons and plastic surgeons, nurses, anesthesiologists, pediatricians, psychologists, social workers, physical therapists, burn survivors, and many others have contributed to major advances in prevention, education, research, and treatment. Although acute care of serious burns is specialized, most mental health professionals, at some time in their careers, provide or supervise treatment for patients who have been burned, as well as caring for their siblings and families.

## ORGANIZATIONAL AND RESEARCH ISSUES

The organization of care differs from some other pediatric areas: in large centers, burns tend to be a subspecialty in many of the fields mentioned earlier. As managed care forces increased generalization of services, more acutely burned children are transferred to specialized burn centers from hospitals without these services. After recovery, follow-up services are provided where the child lives, with periodic reevaluation or surgery at the burn center. The American Burn Association and the International Society for Burn Injuries are the principal scientific organizations sponsoring meetings to educate physicians, nurses, other professionals, and the public. The Phoenix Society, the international self-help group for children and adults with burns and their families, has been instrumental in advancing the care and understanding of burned patients.

Psychological aspects of burns are an active research area, especially pain outcome studies. As surgical research and improved treatment increased survival rates, there was new focus on pain as a crucial variable increasing stress and adversely affecting psychological outcome ( [Patterson et al., 1993](#)). Improving pain relief (U.S. Department of Health and Human Services, 1992) and increasing focus on psychological interventions provide increased hope to children and their families and may be improving outcomes, but pain remains undertreated in many centers ( [Marvin et al., 1991](#)). As comprehensive care improves, burn care personnel and the families are helping disfigured children and adolescents to resume normal lives and may enable them to adapt to handicaps. Research with burned children can pose ethical and human studies dilemmas because the research benefits to them and others are balanced against risks of their age, critical condition, and exposure to many procedures. Levine ( [Chapter 131](#)) wisely presents an ethical overview pertinent to such research with children, including sections on informed consent, on a cognitive basis for assent by children aged 7 to 14 years, and on parental permission.

## EPIDEMIOLOGY

There were an estimated 1.25 million burn injuries in the United States in 1992 and an estimated 5,500 deaths in 1991 ( [Brigham and McLoughlin, 1996](#)), of which about 35% were in children. Time trends reveal significant decreases from 1971 to 1991, with decreases in deaths and hospitalizations for burn injury both about 50%. Although in the previous edition of this text, I wrote that the United States had one of the highest rates of burn injury in the world, it has decreased into the midrange. This progress coincides with major efforts in burn prevention including smoke detectors, sprinkler systems, burn prevention education, improved regulation of consumer product and occupational safety, and other societal changes such as reduced smoking and alcohol abuse. In addition, regional burn treatment centers have been established in major population areas, and these centers improve outcomes after burn injury. Fire and burn injuries are the third leading cause of



accidental death in children 1 to 4 years old and the fourth leading cause of death in those 1 to 19 years old ( [Guyer et al., 1998](#)). Most burns are preventable. Preventive public health laws for fire-retardant sleepwear and lowered hot water temperatures in public housing have reduced burns to children. However, further laws are needed. Many infants and young children are burned by scalds by extremely hot water. Other common types of burns are flame, electrical, ingestion, and chemical ( [McLaughlin and Crawford, 1985](#)). These may be the result of poor housing, overcrowding, child abuse or neglect, sensorimotor deficits ( [Ramirez et al., 1998](#)), parental substance abuse or depression, risk-taking behavior, smoking, match play or firesetting ( [Chapter 57](#)), suicide attempts ( [Table 99.1](#)), or war.

**Table 99.1. Adolescent Survivors of Self-Inflicted Burns**

Child neglect or abuse ( [Chapter 100](#)) is a frequent cause of burns. The child psychiatrist collaborates with the surgeon, nurse, and social worker in suspected cases, and psychiatric assessment skills may confirm neglect or abuse when other methods fail to do so. This type of burn is common; neglect or abuse accounts for 6% to 20% of pediatric burns in different units, including some fatalities. Hight et al. ( [Renz and Sherman, 1993](#)) found 13 to 24 months to be the period of maximum risk of abuse. The most common type of inflicted burn is a scald, with other types (cigarette, hot iron, or radiator) much less common. [Renz and Sherman \(1993\)](#) prospectively studied abusive scald burns and found that all 30 children, at a mean age of 22.5 months, had buttocks burns, 80% had complications, and four died of sepsis. Indicators suggesting abuse include a changing history of what occurred, a past history of “accidents,” an inattentive parent, a consistently passive, withdrawn child who is “numb” to pain, and physical findings suggestive of abuse, such as a burn distribution not consistent with the history, other physical signs of abuse, and fractures. When abuse is suspected, reporting to the appropriate state social agency is mandated, and an investigation follows, with subsequent interventions as indicated. When support and explanation are provided to the parents or caretakers regarding why filing a report is required, they often accept counseling as part of burn care, although in some cases they withdraw from unit staff. Recognition of neglect or abuse is very important because these children are at increased risk of subsequent injury and death.

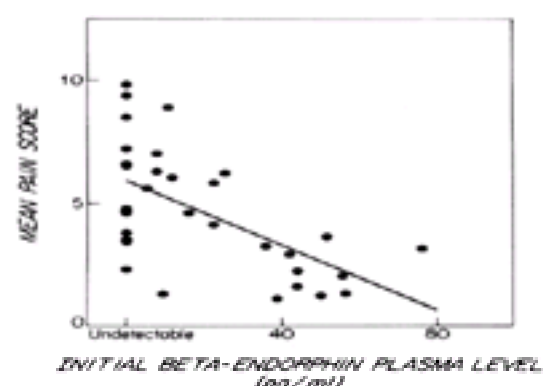
## UNDERMEDICATION FOR PAIN

Pain is very severe during acute treatment, especially during débridement of the burn wound (dressing changes, “tubbing” or “tanking”), but pain management and protocols have improved reducing staff conflict. Although the reasons are not fully understood, some reasons for undermedication by doctors and nurses seem to be (a) lack of education about pain management, (b) fear of respiratory depression and other side effects, (c) reluctance to obtain self-ratings of pain systematically, (d) false confidence (“I know what pain in children looks like”), and (e) lack of attention to adequate doses of opiates required with curariform drugs. [Perry \(1984\)](#), in a reflective article about this issue with adult burn unit staff, found that (a) preservation of the patient’s pain unconsciously preserved the boundary of the patient as a definable being, separate from staff, and (b) pain responses helped to confirm for the staff that the patient was alive; it was *not* a sadistic wish to cause pain but, rather, a reluctance to eliminate it. He suggested that consultants be aware of possible unconscious factors, identify and differentiate them from pain management, and encourage appropriate levels of analgesic use. Hospital pain protocols ( [Table 99.2](#)), collaboratively developed, and monitored, and revised regularly, may reduce this problem.

**Table 99.2. Pain Management Protocol from Shriners Burns Hospital, Boston**

## PAIN MANAGEMENT

The pain problems for burn patients are mainly acute rather than chronic. Before treatment of pain, physicians and nurses identify the location and source of pain, its usual intensity, course, and duration, and its response to prior treatment. Pain does correlate with endorphin levels ( [Fig. 99.1](#)) and with the extent and depth of burn in children ( [Carr et al., 1993](#)). Several years ago, analgesics were withheld from a hateful adolescent girl with massive self-inflicted burns and a history of drug abuse, because her constant complaining about pain was viewed as manipulative; a research study later revealed that her endorphin levels had been negligible and that she should have been receiving high doses of morphine. Unfortunately, endorphin levels are not rapidly available, but self-ratings are usually sufficient. For infants, behavioral measures (facial expression, body movement, behavioral state, and cry), as well as physiologic parameters (heart rate, blood pressure, respiratory rate, and oxygen saturation, if available, and epinephrine, norepinephrine, growth hormone, and cortisol), are used to monitor pain response ( [Porter, 1993](#)). Pain relief in infants improves surgical outcomes (not yet studied in burns), and it is enhanced with the use of pain rating scales ( [Anand et al., 1997](#)).



**Figure 99.1.** Mean pain score versus initial b-endorphin level during burn dressing change. The relationship of the initial (before analgesic and burn dressing change) b-endorphin plasma immunoactivity and mean pain score for the time of burn dressing change. (From [Szyfelbein SK, Osgood PF, Carr DB](#): The assessment of pain and plasma b-endorphin immunoactivity in burned children. *Pain* 22:173, 1985, with permission.)

For any child who cannot communicate, for example, because of the use of curariform drugs or intubation, estimates of maximum analgesic requirement for body weight are made. The only easily used assessment for pain and pain relief for conscious patients is self-rated pain ratings, preferably recorded on each shift and at each dressing change. When such ratings or pain questionnaires, performed consistently and reliably, are not present, there is little basis for evaluation of pain treatment, although clinical judgment and nurses' ratings are still used. [Matthews et al. \(1993\)](#) point to limitations of each but indicate particular strengths of self-report measures such as the Faces Pain Scale, Visual Analog Scale, and Oucher Scale. Of observational scales, these authors favor the Childrens Hospital of Eastern Ontario Pain Scale (CHEOPS). The most easily used self-rating scale is a simple 1 to 10 visual analog scale rating or asking the child to rate the pain from 0 to 10, with 0 as none and 10 as the most severe pain ([Szyfelbein et al., 1985](#)). Various scales are in use for young children, such as the Faces Pain Scale, which pictures faces rated from 0, a happy face that "doesn't hurt at all," through 5, a sad crying face "hurting as much as you can imagine." The range of developmentally targeted approaches for use in managing pain is shown in [Table 99.3](#).

**Table 99.3. Developmentally Targeted Approaches to Pain and Anxiety Treatment**

Of the psychological methods, the cognitive-behavioral method involving the child's participation was designed to increase the "locus of control" in burned children themselves ([Kavanaugh et al., 1991](#)). The method involves preparation for each step of the burn dressing change and gives the children greater psychologic control over their pain experience by encouraging their help in removing bandages and in other steps. It leads to fewer maladaptive behaviors, improved outcomes, and lower dosages of narcotics. [Kuttner \(1993\)](#), following Olness and Gardner, highlights the effectiveness and practicality of *hypnosis* for burn pain in children and notes that education of parents and encouraging their presence are desirable. She describes practical hypnotic methods in 3 to 6 year olds and in older children, states that maximum hypnotizability occurs in children, advocates its rapid, short-term use, and indicates that capacity for self-hypnosis is the ideal end point, to give maximal control. This method is underused, given its relative ease with children. [Foertsch et al. \(1998\)](#) and [Martin-Herz et al. \(2000\)](#) address hypnosis with burned children in major burn centers. Their findings suggest that although effective, hypnosis is less generally reliable for rapid relief of severe burn or procedural pain than are opiates and other drugs.

### Pharmacologic Treatment

Pharmacologic treatment is the primary intervention to relieve pain in burned children. Pain treatment is important for biological as well as psychological reasons: the stress pain causes disrupts metabolic, autonomic, and thermoregulatory as well as immune functions; it also can "adversely affect morbidity and mortality" ([Carr et al., 1993](#)). One study of burned children found that those receiving more morphine developed significantly less burn-induced posttraumatic stress disorder (PTSD) ([Saxe et al., submitted for publication](#)). Studies have clarified the effectiveness of morphine in relieving pediatric pain, by using either intravenous or oral administration of the drug ([Carr et al., 1993](#)). The doses used are much higher than doses used for most other purposes, as demonstrated in [Table 99.2](#), which is a protocol incorporating current research on pharmacologic pain and anxiety management. However, other pediatric centers have different protocols and often lower dosages. [Gaukroger \(1993\)](#) reviewed *patient-controlled analgesia* (PCA) and noted its unsuitability for preschool and mentally retarded children. [Berde et al. \(1991\)](#) evaluated PCA in a randomized, prospective comparison with intramuscular administration of morphine for postoperative analgesia and found excellent results for PCA with children 6 to 18 years old. [Doyle et al. \(1994\)](#) studied children after surgery (not burns), with the findings that those treated with higher-dose morphine had less pain and the lower-dose group had more hypoxemic episodes. [Carr et al. \(1993\)](#) state that "although PCA has been successfully used in acutely burned children, it lends itself more easily to the more manageable postoperative pain of patients returning for reconstructive surgery."

Haloperidol, droperidol, and the newer short-acting agent, propofol, potentiate the analgesic and sedative effects of morphine, but the latter two require monitoring by an anesthesiologist because of potential serious side effects and toxicity. These drugs can be very helpful for children in severe pain who are agitated ([Stoddard et al., 1997](#)).

Adjuvants, which enhance the effect of opiates, can make the difference between the presence and absence of pain relief. Most common in current use are low-dose benzodiazepines, acetaminophen ([Meyer et al., 1997](#)), and nonsteroidal antiinflammatory drugs, and less common are low-dose methylphenidate, amphetamine, tricyclic antidepressants, and haloperidol. Except for benzodiazepines, adjuvants have had no formal study in treatment of pain in burned children.

The benzodiazepines in common use, depending on pain severity and duration of action desired, include the following: of the *short-acting* agents, midazolam ([Sheridan et al., 1994b](#)), lorazepam, oxazepam, and triazolam; and of the *long-acting* agents, chlordiazepoxide, diazepam, clonazepam, and flurazepam. In a study of three acutely burned children at our center, clonazepam caused excessive sedation and anorexia in combination with morphine in two children but was effective in the third, a child with a family history of panic disorder.

There are few indications for use of psychotropic drugs with acutely burned children other than management of pain, anxiety ([Table 99.2](#)), or delirium. Using an algorithm to differentiate complex comorbid psychiatric disorders is essential to care. These include delirium or other psychosis, intractable pain, phobia or panic, sleep deprivation or sleep disorder, acute stress disorder (ASD), acute depression, medication side effects, or the toxic effects of sepsis, alcohol, or drugs.

On rare occasions, major tranquilizers are necessary to control severe burn delirium, agitation, aggression, or severe insomnia. In the rare instances when benzodiazepines are unsuccessful, or paradoxically cause disinhibition or agitation, the drug of choice for acutely burned children who are more than 12 years old is intravenous haloperidol on a short-term basis, usually in low doses (0.5 to 2.0 mg slow push) for only 24 to 48 hours, while the cause (e.g., sepsis, metabolic change, etc.) is being identified and treated. This drug has few cardiovascular effects and is rapidly effective. However, it may have adverse effects. In my experience in two of approximately 75 cases, rapid infusion of haloperidol appeared to cause brief reversible hypotension in children, and these two children were less than 10 years old. Continuous electrocardiographic and blood pressure monitoring at the time of administration is indicated if this drug is used in young children. Although dystonia can occur with continued use and may require an antiparkinsonian agent, that has not occurred with short periods of intravenous use, whereas the therapeutic benefits are marked. One study of haloperidol use in children with burns found it to be useful ([Brown et al., 1996](#)). Risperidone, a newer antipsychotic, is preferable if the child can take oral medication, and it may also assist weight gain, which is important in burn recovery.

Despite wide use in acutely burned children, the major studies to date of symptom response to psychotropic agents have been with analgesics and benzodiazepines. Occasional children with attention deficit hyperactivity disorder who were previously taking a stimulant may require it in the course of burn treatment; a side benefit is that it can enhance analgesics as well. The reasons for generally not using other psychotropic agents are possible interactions or toxic effects, such as from lithium, further compromising acute burn treatment.

During early acute burn care, other psychotropic agents should be used with great caution because of potential interactions or toxic effects. When these drugs are needed, they should be carefully monitored with serum levels, blood pressure, and cardiac monitoring. Special considerations that should influence the psychotropic drug selected and dosing are pharmacokinetics and the presence of common complications such as fluid and electrolyte shifts, acute respiratory distress syndrome from pulmonary burns, and hepatic or renal impairment. With drugs such as lithium or tricyclic antidepressants, there may be risk of intoxication or cardiovascular complications. The selective serotonin reuptake inhibitors (SSRIs) interact with a number of other drugs and reduce excretion of many through hepatic P-450 enzyme



blockade. Despite these caveats and those that follow, judiciously selected and monitored psychotropic agents may assist in management of the acute burn patient.

Drug side effects and, rarely, toxicity occur, especially with opiates, benzodiazepines, droperidol, haloperidol, and propofol ( [Kane and Lieberman, 1992](#)). Neuroleptic malignant syndrome may occur, and symptoms can be almost the same as malignant hyperthermia from anesthetics. Opiate or benzodiazepine tolerance, dependence, and withdrawal do occur. Some patients remain sedated and minimally responsive for too long because of high doses, impaired excretion, or a combination. The withdrawal syndrome from opiates includes abdominal cramps, vomiting, diarrhea, tachycardia, hypertension, diaphoresis, restlessness, and insomnia; it is reversed by opiate agonists. Abrupt withdrawal from benzodiazepines may cause anxiety, dysphoria, or insomnia but also full-scale withdrawal with abdominal cramps, vomiting, sweating, tremors, and convulsions. Some symptoms occur even several days later after gradual withdrawal. Resuming and then slowly tapering the benzodiazepine usually reverse the withdrawal symptoms. Although some patients become dependent on opiates and benzodiazepines, *addiction (i.e., severe drug abuse) is hardly ever caused by analgesia for burn pain*, despite the concerns of many staff members about this, especially with adolescents. Dependence is effectively managed, once the patient no longer needs the medication, by gradual tapering, including with occasional adolescent substance abusers who may have required higher doses of opiates at first than others. The use of a clonidine patch has been effective in eliminating moderate opiate withdrawal symptoms. In children, the combination of opiates and benzodiazepines may contribute to respiratory depression, delirium, anorexia, and excess sedation ( [American Psychiatric Association, 1990](#)). Severe opiate side effects can be reversed by naloxone, and benzodiazepine toxicity can be reversed by flumazenil, but this drug may cause seizures. Taking these risks into account, the judicious use of medications and careful monitoring normally result in predictable and reliable reductions in pain and anxiety.

## PHASES OF BURN RECOVERY

Three recovery phases occur after a burn, and they have both neurobiological and psychological features ( [Watkins et al., 1988](#)). These are *acute*, *intermediate*, and *rehabilitative*. Psychiatric assessment and treatment are adapted to each phase. The full range of psychiatric knowledge and skills is used in work with burned infants, children, adolescents, and their families.

### Acute Phase

#### DIAGNOSTIC ASSESSMENT

From the time of admission, assessment and treatment proceed together. Orientation, explanation, and preparation for what has occurred and is to come are linked with assessment of the child's and parent's responses to these interventions. Communication is best when it is at the child's stage of development ( [Gaffney, 1993](#); [Stoddard, 1982a](#)) and in the child's and parents' language ( [Table 99.3](#)). Because the child is often confused in the strange burn unit setting, is afraid of dying regardless of wound severity, and is anxious about pain, the initial history may be obtained from others, but it is important to elicit it from the child when possible. The child responds positively to reassurance about his or her condition and efforts to create familiarity and comfort. Relief of pain lessens fear and confusion. When parents understand and feel support and reassurance, they can assist and be successful in calming their child. Once an initial secure and trusting staff–parent alliance is established, additional history emerges that may clarify preburn risk factors such as a toddler's new motor skills, homelessness or poverty, firesetting, child neglect or abuse, and parental substance abuse, mental disorder, or criminality ( [Kolko and Kazdin, 1992](#)). Similarly, postburn emotional reactions such as fear, guilt, denial, grief, or withdrawal become evident.

An important early diagnosis to be alert for is *delirium* because the confused, agitated, or aggressive patient can cause self-injury by abrupt events such as unplanned pulmonary extubation or pulling out of intravenous lines. Electroencephalographic abnormalities associated with burn delirium have been demonstrated in adults ( [Andreasen et al., 1977](#)), and [Antoon et al. \(1972\)](#) described seizures in 10 of 20 burned children. Delirium in burned children can be common and “alerts staff to the patient's unstable medical condition and can be the first indicator of opiate side effects, sepsis, metabolic disturbance, CNS injury, or a combination of these. Prompt diagnostic evaluation of the delirious child, treatment of the causative factors, symptomatic relief, environmental change, and increased personal contact with staff and family are indicated” ( [Stoddard, 1990](#)). [Antoon et al. \(1972\)](#) studied the origin of burn encephalopathy in 20 children and found the cause to be hypoxia from smoke inhalation in six cases, sepsis in four, a combination of the two in two, and hyponatremia in four cases; of the survivors, only one had residual neurologic impairment; 10 patients died, and five had complete neuropathologic examinations, one with *Pseudomonas* meningitis, one with sepsis, and three with minimal findings. Although delirium remains a sign of complications, modern treatment has reduced the mortality rate to a fraction of what it was in most burn centers.

The stress of an acute moderate or severe burn is extraordinary, a shock that may involve loss of possessions and of loved ones, as well as injury to the child's own body. Many children with burns manifest ASD. This was found in nearly 40% of burned children by [Saxe et al. \(1999\)](#). The burn unit offers an opportunity for case identification, assessment, and intervention including referrals for developmental, neurologic, and educational evaluation of at risk children who would otherwise not receive them. For this reason, it is important to complete an initial assessment even during brief hospitalizations when indicated.

The basis for psychiatric diagnosis is the text revision of the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR) ( [American Psychiatric Association, 2000](#)), as well as the classification set forth in the *Child and Adolescent Mental Disorders—Primary Care* ( [American Psychiatric Association, 1995](#)).

#### DEVELOPMENTAL NEUROBIOLOGY

The child's body may be able to respond remarkably to trauma such as a massive burn, but it is becoming clear that the child's neurobiological response leaves him or her vulnerable emotionally. “A massive catecholamine release occurs immediately after burn injury both from the adrenal medulla and the autonomic nervous system” ( [Demling, 1990](#)). The body goes through three phases. First, an acute shock or resuscitation phase occurs, with decreased metabolism, dulling of severe pain, and many other acute changes requiring intensive treatment, especially fluid replacement. Second, after a few days, is a hypermetabolic or recovery phase, with an increase in the metabolic rate of about twice normal, during which the hypothalamus seems to select a higher temperature set point and that is associated with increases in plasma catecholamines, cortisol, glucagon, and growth hormone. In the presence of pain and anxiety, the brain can increase the hypermetabolic response ( [Demling, 1990](#); [Wilmore et al., 1974](#)). In infants and children today, unless complications occur, the excision of burn-damaged tissue and coverage of the burn wounds with skin grafts result in healing often in 1 to 3 months, even with large wounds. The acute and intermediate treatments are often accompanied by severe chronobiological changes, including for some a disruption of normal sleep stage distribution ( [Gottschlich et al., 1994](#)) and reversal of the day–night sleep–wake cycle because of around-the-clock treatments, pain, itching, and other factors, although the cause of disordered sleep in burn patients is not yet understood fully. As healing progresses, the sleep–wake cycle gradually returns to its preburn pattern, although sleep disorders, day napping, and enuresis are common postburn symptoms ( [Kravitz et al., 1993](#); [Lawrence et al., 1998a](#); [Murphy et al., 1989](#)). Third, during the rehabilitative phase, wound healing is completed, and metabolism gradually returns to normal a few months after discharge.

#### NEUROBIOLOGICAL POSTBURN PHASES: ACUTE STRESS DISORDER

The physiologic changes briefly outlined earlier appear to be mirrored by changes in brain function and mental status in those patients who are not so sedated that they cannot be evaluated. Some of the mental states commonly seen during acute treatment, which may reflect or relate to the systemic phases described earlier, include anxiety, confusion, pain syndromes, insomnia, depression, and recovery, which may be rapid or gradual.

After a burn, stress disorder symptoms are very common. The differences between symptoms expressed in the proximal and distal aftermath of a trauma are formalized in the DSM-IV-TR, with the distinction between ASD and PTSD. ASD describes the psychopathologic response in the immediate aftermath occurring up until 1 month after the trauma, whereas PTSD describes the psychopathology that persists after 1 month. ASD and PTSD are also distinguished by prominent dissociative symptoms. Children with ASD often have significant dissociative symptoms, numbness, a decrease in emotional responsiveness, and difficulty in recalling elements of the traumatic event. These children tend to reexperience the traumatic event, avoid reminders, and become hyperaroused to reminders of the event. Newer findings indicate that burned children who manifest the dissociative symptoms of ASD after the injury are at high risk to develop PTSD ( [Daviss et al., 2000](#); [Saxe et al., 1999](#)).

Sleep disorders are also common after burns, and they may be a symptom of stress or represent a comorbid disorder ( [Stoddard et al., 1996](#)). Sleep terror disorder is not rare, acutely, after burns, including reexperiencing but without recall of the burn experience. Insomnia and nightmares are often related to awakenings for dressing changes, medications, itching, and prior history of sleep disorders or family history. Sleep deprivation in burned children can be a cause of other psychopathology including stress disorders, depression, and disruptive behavior.

Neuroimaging studies provide evidence of lasting neurobiological abnormalities, such as reduced hippocampal size associated with PTSD, although this has not been studied in patients with burns ([Carr, 1998](#); [Rauch et al., 1996](#)). The hypermetabolic response may appear as a pseudomaniac delirious state with confusion, anxiety, and agitation; later, associated with catecholamine depletion, a state of depression usually develops, with depressive mood, interpersonal withdrawal, and decreased appetite ([Gold et al., 1988](#)). This is not true in all cases, and some children, whose pain and anxiety are controlled, are mentally clear and conversant as deep fascial excisions or amputations of burned areas are about to be or have just been done; in all cases, psychiatric assessment, surgical preparation, and psychotherapeutic support are essential.

In the child, adequate relief of pain and anxiety, together with rapid healing, may lessen the probability of ASD or depression. [Charney et al. \(1993\)](#) describe a model that shows a central role for the locus ceruleus, ventral tegmental area, and especially the amygdala in facilitating the “encoding of traumatic memories” through “fear conditioning, extinction, and sensitization.” Interestingly, the postulated neurotransmitter systems involve norepinephrine, dopamine, opioids, and corticotropin-releasing factor. These are some of the same systems activated in severe burns, and drugs affecting these systems are frequently required for complications arising during burn treatment: cardiovascular, renal, pain, and metabolic disturbances. However, studies reflecting central neurobiological effects in burned children have included mainly endorphins and cortisol. Current research is attempting to discover ways of preempting the processes resulting in PTSD ([Stoddard and Todres, 2001](#)).

Some of the common phenomena in acute burn treatment affecting and being affected by neurobiological systems include the following: neurotoxins (anoxia, carbon monoxide); delirium; catecholamine depletion and depression; opiate dependence, side effects, and withdrawal; and benzodiazepine dependence, side effects, and withdrawal. Endorphins, and probably endogenous benzodiazepines ([Hyman and Nestler, 1996](#); [Rothstein et al., 1992](#)), and the receptor systems involved affect the response of the amygdala to fear, to anxiety, and to medications ([Davis, 1992](#)). It is not known whether residual neurodevelopmental abnormalities follow burn trauma treated by modern methods.

## Intermediate Phase

### NEUROBIOLOGY

This phase, after the patient has survived and is healing, is only somewhat less stressful. Physiologic responses such as a conservation-withdrawal reaction or postacute stress depression seem to be normal after burn trauma. Hospitalization is not free of stress, and it is this period for which the term *continuous traumatic stress* may be most fitting ([Gilboa et al., 1994](#)). Stress occurs throughout burn treatment, not only acutely and then after it is all over (PTSD), but continuously to varying degrees. Vulnerable children with prior comorbid conditions, such as anxiety, affective, or disruptive behavior disorders, may have special difficulty in adapting to the frequent stresses of burn care and may benefit from resumption of psychotropic medications if they were temporarily stopped. Children requiring limb amputations benefit from diagnostic and therapeutic care to cope with severe or intractable pain, acknowledgment and adaptation to the loss, phantom limb, and posttraumatic stress ([Atalar and Carter, 1992](#); [Billig and Weaver, 1996](#); [Thomas et al., 2000](#); [Varni et al., 1991](#)). Other neurobiological changes occur commonly in burn care secondary to acute surgical use of potent agents such as dopamine, curare, analgesics, or ketamine. Complications may emerge including infection, organ failure, or metabolic disturbance, any of which can contribute to the occurrence of delirium.

A special problem in burn care is human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome ([Blumenfeld and Schoeps, 1993](#)). Children are less commonly infected than adult burn patients, but when a burned child is infected, it is doubly tragic. The improved antiviral treatments and overall case management are major advances offering hope. It is important for staff members to adhere strictly to universal precautions. Generally, acute burn treatment takes precedence over HIV interventions. Adverse events such as needlesticks may occur; such events and staff fears are addressed using standard hospital policies and procedures.

### POSTTRAUMATIC STRESS DISORDER AND COMORBID DISORDERS

In the assessment of neurobiological sequelae of burns, the child psychiatrist's medical expertise and skills in diagnosis and treatment become important. There are only a few psychiatric studies of injured children ([Stoddard and Saxe, 2001](#)), but research is progressively delineating the risks to neurobiological development and resulting developmental psychopathology, as well as evidence of resilience. Studies assessing physical or gross developmental but not psychopathologic outcomes, as is true of some outcome studies, may provide falsely optimistic results.

PTSD and other disorders are beginning to be studied in young burned children, but they are well defined in older children. It is not yet proven just what the symptoms of PTSD are in very young children because there are only a few clinical studies ([Scheeringa et al., 1995](#); [Terr, 1988](#)). Based on these studies, the symptoms of PTSD in children less than 4 years old seem to include reexperiencing of the burn demonstrated in posttraumatic play, nightmares, numbing of responsiveness, constriction of play or feelings, loss of acquired skills such as language regression or toilet training if achieved, increased arousal including night terrors and exaggerated startle response, and new fears (e.g., fear of the dark or separation anxiety) or new aggression. Children may also develop language delays ([Gorga et al., 1999](#)) or attachment disorders because the traumatic event may threaten their sense of security. Some more recent studies ([Alfred, 1999](#); [Johnson et al., 1998](#); [Meyer et al., 1999](#)), but not all past studies ([Sawyer et al., 1983](#)), of very young burned children support these findings. DSM-IV-TR criteria include “disorganized or agitated behavior” in the response to trauma, and posttraumatic play, “frightening dreams without recognizable content,” and “trauma-specific reenactment” as evidence of reexperiencing ([American Psychiatric Association, 2000](#)).

PTSD has been little studied with burned children; the largest study is a follow-up of 60 children aged 7 to 19 years with mean burn size of 38% body surface area (F.J. Stoddard, J.M. Murphy, L. Rizzone, 1994, unpublished data). The findings are consistent with the investigators' earlier study demonstrating that for children with burns at various ages who were assessed when older, between 25% and 33% of children eventually develop PTSD, and more than 50% manifest “partial” PTSD ([Stoddard et al., 1989a](#)). Although it may be surprising that the rates are not higher, these prevalence rates are consistent with other studies of burned children and adults. [Kravitz et al. \(1993\)](#) have consistent findings, with a similar incidence of sleep disorders. They studied sleep disorders in 82 children (mean duration after burn, 7.3 years), 41 boys and 41 girls, with a mean burn size of 44% (range, 2% to 91% of total body surface). They found that a subgroup of 30 children reported nightmares. Of the 55 reporting dreams, 45 reported normal childhood topics, six reported burn injury–related topics, and five related burn treatment–related topics. Some of the “normal” topics may be burn related also, such as monsters, kidnapping, being killed, scary movies, dying, being locked up, parents dying, or a sibling harmed. Topics clearly related to the burn were reliving of the event, smelling smoke, dreaming of others who died in the fire, and screaming “Fire.” Topics related to treatment included pain from dressing changes, anesthesia induction, fear of dying in the operating room, dreaming that scars are gone, and fearing a burned hand will be amputated. In that study, nightmares involved clear disorders of arousal with screaming, thrashing, sweating, and yelling. They found sleepwalking in 7% of the children. Similar to [Stoddard et al. \(1989a\)](#) and [\(1989b\)](#), these investigators found a history of enuresis in 24% of the children studied. The outcome of untreated PTSD after burns is not yet known, although studies suggest some attenuation of symptoms after the first few months.

### Psychotherapy

Ongoing psychotherapeutic intervention, with brief focused visits, is directed toward emotional adaptation to the burn and posttraumatic symptoms. This treatment may reduce symptoms without medication. Parents and family are essential participants. Psychotherapy should combine developmental psychodynamic principles, eliciting the patient's narrative when feasible, educating about what to expect in burn treatment, and supporting positive coping. Although cognitive–behavioral therapy has been used for pain ([Lasoff and McEttrick, 1986](#); [Kavanaugh et al., 1991](#)) but not yet formally for PTSD in burned children, the principles of cognitive–behavioral therapy for PTSD such as anxiety management, exposure-based interventions, coping with anger, and cognitive restructuring form the core for the practice of cognitive–behavioral therapy with burned children with PTSD ([Deblinger et al., 1990](#); [March et al., 1998](#); [Solomon et al., 1992](#)). PTSD is not the only mental disorder afflicting burned children, and psychotherapy may focus on comorbid disorders, such as phobia, depression ([Stoddard et al., 1992](#)), enuresis, or behavior disorders. Sleep disorders may be aggravated by itching, and in addition to antihistamines, massage may have a useful role. Massage therapy relieved pain, anxiety, and itching in an outpatient study of 20 adults by [Field et al. \(2000\)](#), but this has not been formally studied in children.

### Psychopharmacology

In addition to pain management, psychopharmacologic treatment may begin unless there are contraindications because of the patient's critical condition ([Marshall and Pierce, 2000](#)). Several agents are in common use in patients with burn injuries, particularly benzodiazepines. The introduction of short-acting parenteral agents, such as midazolam and lorazepam, made it feasible to target anxiety symptoms without the chronic sedation caused by diazepam and to monitor symptom ratings in response acute interventions. As a result of treating anxiety as well as pain, it is clear that one can trigger, reinforce, or exacerbate the other. Although some



researchers question the use of benzodiazepines for patients with PTSD, both the critical care literature and broad experience with injured patients suggest that short-acting benzodiazepines are very effective acutely in reducing stress, and they may improve outcomes ( [Stoddard et al., 1997](#)). For very severe confusion, anxiety, agitation, or aggression that can accompany delirium, panic, or PTSD, antipsychotic agents, such as haloperidol or (if oral medication is possible) risperidone, may rarely be indicated. Depression is often comorbid with PTSD in injured patients. Most acute depressive symptoms are responsive to supportive psychotherapy alone. SSRIs are often contraindicated because of drug interactions (P-450 enzyme blockade) in early acute care, but not always for smaller burns. Some patients are diagnosed with acute depression early in treatment, and delay in instituting psychopharmacologic treatment may slow recovery and participation in physical rehabilitation. Although a pilot study demonstrating effectiveness of imipramine for ASD is one of the few psychopharmacologic studies in acutely burned children, indications for its use are limited ( [Robert et al., 1999](#)). Strengths of imipramine are that this one agent may reduce pain, ASD, PTSD, insomnia, and enuresis. Liabilities include risks of hypotension, which we have seen in young children, and cardiac arrhythmias ( [Biederman, 1991](#); [Biederman et al., 1995](#)). Although the risks may be low, SSRIs offer many benefits with little risk, and for this reason they have displaced tricyclic antidepressants in psychopharmacology. The SSRIs in common use for depressive diagnoses or PTSD include sertraline, fluoxetine, and others ( [Hughes et al., 1999](#)). Other potentially beneficial agents include clonidine and  $\beta$ -blockers.

## EFFECTS ON PSYCHOLOGICAL DEVELOPMENT

### *Psychological Interventions*

**Individual Therapy.** There is a shift from the acute into the intermediate phase once survival is ensured and most burn wounds are grafted, although the patient may not be ambulatory. During this period, it is possible to assess mental status more fully and to begin differentiating issues of mourning the prior body image, grief over loss of loved ones, depression, and PTSD. In addition, assessment and diagnosis of neurologic or preexisting psychiatric impairment become feasible. The child's or adolescent's emerging awareness of their functional losses and cosmetic disfigurement are eased by responsive staff and supportive, informed family members. "Healing well" or "looking good" surgically does not mean that the child or parent will see the raw wounds or new grafts in the same way, especially if they expected a return to previous appearance. When a child requests a mirror, the curiosity and emerging self-awareness about facial burns and appearance can unsettle a nurse or parent and can lead to the question whether the child is ready or may react with too much anxiety or depression. Although each situation must be individually assessed, in most cases children benefit when such requests are honored, but with exploration of their expectation and adequate support and explanation nearby when they first view themselves. Modes of intervention usually include brief consultative checks, and, for those in most distress, several sessions of 15 to 30 minutes per week focused on the issues mentioned earlier as well as preparation for forthcoming surgery, return to home and school, and rehabilitation.

**Family Therapy.** Adaptation of the parents and family usually follows the course of the child's recovery. Remarkably similar feelings and defensive responses are observed in the child and family. Psychotherapeutic support, especially regarding guilt feelings, and grief work with parents, often in several sessions per week, during the intermediate phase assist the parents and enable them to support the child's coping through this phase ( [Kaslow et al., 1994](#)).

**Group Therapy.** Some hospitals with many burned children provide groups varying from brief psychotherapy to education to rehabilitation. At the Shriners Burns Hospital in Boston, three types of group interventions have been used. I have conducted an inpatient *children's group* with a resident, nurse, or teacher, for children 2 to 12 years old, which varies from preschoolers to school-age children to preadolescents, depending on which children are in the hospital. This is a semistructured play group in which children introduce themselves, say why they are in the hospital and how they were burned, play with puppets and toys, draw, and then clean up the toys and prepare to say good-bye. The children readily express their feelings and fantasies in displacement to the consistent "Curious George" puppet, who, while a troublemaker, is very curious about the children's puppets' understanding of their burns, pain, surgery, dreams, scars, peer ridicule, and other issues. Although children are hesitant to participate at first, they become attached to the group. When they are again readmitted, they ask for the "children's group"; once they become adolescents, they remember it with a smile because they usually enjoyed it despite occasional painful topics. An *adolescent group* for hospitalized adolescents and a *parent group* for parents of acute patients are also provided, to focus on education about treatment, grief, response to hospitalization, surgery, stigmatization, family and school issues, and discharge and reentry issues. In England, [Rivlin et al. \(1986\)](#) conducted similar groups, which the parents rated as helpful, using a multidisciplinary approach.

### *Indications for Transfer to a Psychiatric Inpatient Unit*

The hospitalization for burns may be the only opportunity for skilled psychiatric diagnosis and the initiation of appropriate psychiatric treatment. Transfer or commitment to an inpatient psychiatric treatment unit is rarely necessary for children and adolescents (after acute and intermediate treatment) who are suicidal, violent, severely depressed, or who fail to respond to consultative psychiatric treatment in the surgical setting. Psychiatric inpatient units require consultation regarding burn-related psychopathology, and they benefit from burn surgical and nursing consultation regarding any remaining wound care and surgical follow-up.

## Rehabilitative Phase

### *BODY IMAGE AND PLASTIC AND RECONSTRUCTIVE SURGERY*

After the child has survived and healed from the burn, the child's surgical follow-up care is transferred to the plastic and reconstructive surgeon, if not previously involved. The acute surgical service and plastic and reconstructive surgical services are often linked, and this assists transition from one to the other ( [Chedekel et al., 1998](#)). The child psychiatrist and mental health team learn through their patients and the plastic surgeons about the various surgical and nonsurgical options available for scars, disfigurement, functional impairment or loss, and amputations. Plastic surgeons of necessity develop psychological skills in evaluating burned patients and may seek psychiatric consultation for some cases ( [Goin and Goin, 1981](#)). Because much of acute treatment, even with recent changes, is outside the control of the child and family, a central psychological goal at this point in care is to increase the child's and parents' roles in the choice of treatment, its timing, and the long-term plan. Although staff responses assist the patient in seeing himself or herself anew ( [Solnit and Priel, 1975](#)), the consultant in turn, through the eyes of the child, becomes aware of the experience of being burned.

### *Seeing Through the Eyes of a Burned Child: The Developing Body Image*

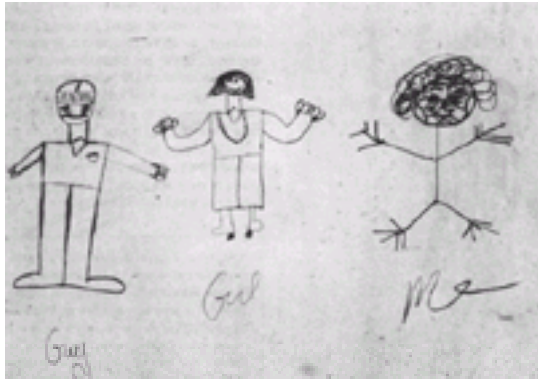
Interviewing a burned child and observing the child's play provide a window on the child's body image development, the interference in that development caused by the burn and painful treatments, and, usually, the psychological adaptation and recovery that follow. The early concept was summarized by Fenichel as "the sum of the mental representations of the body and its organs, the so-called body image, [which] constitutes the idea of I and is of basic importance for the future formation of the ego" ( [Stoddard, 1982b](#)). A more contemporary definition derives from the functions of body image and the developmental levels at which it operates ( [Shontz, 1974](#)). The functions it serves are sensory register, instrument for actions, source of drives, a stimulus to the self (as in arousal, pain, and proprioceptive experiences), a stimulus to others (appearance identifies attractiveness or can be a stigma), an expressive instrument, and a private world. Disorders may occur at any cognitive level of body image experience including body schemata, body self, body fantasy, or body concept, and there are associated defense patterns and symptoms. Parental and peer attitudes toward the child's physical defects help to shape body image development. Some scarred persons have no disorder of body image, whereas others with little or no scar may have significant body image disorder (body dysmorphic disorder, in which there is no necessary relationship between external appearance and internalized body image).

The issue of "how first was the body imaged?" is of practical relevance in the care of burned children. Research on infancy ( [Chapter 22](#)) pertains to the impact of burns on children from birth to 3 years old and on older children. Intricate attachment processes begin prenatally and continue seamlessly in the mother-child relationship after birth. Observations reveal that a burned infant is emotionally traumatized in the following ways: pain is felt and localized, the sense of safety and security in attachment to the mother is threatened or even shattered, and the recovery from that experience or those experiences depends on the reestablishment of the attachment and the signs of responsivity present before the burn. What does this imply for body image development? A disturbance results from disruption of the protective mother-infant dyad for development of body image with its attendant affects and relational interactions. Such a disturbance may constitute an early disorder of body image. That appears in behavior as follows: increased helplessness, sleep and feeding disturbances, and affective withdrawal in infants; social restriction and loss of acquired skills in the toddler; and problems in development of control over new fears and other feelings in 2- to 3-year-old children, such as aggression, problems with excretory functions, nightmares, play constriction, and play reenactment of the burn trauma or burn treatments ( [Scheeringa et al., 1995](#)).

Hospitalization for a burn is often an early separation experience, which reveals the child's capacity to form and be consoled bodily by transitional objects, including a father or nurse who may represent a transitional relationship or surrogate. The capacity to create such relationships bodes well for the prognosis of the child and resembles hypnosis in its intensity and capacity to soothe. Body image is a relatively stable part of the self-concept by age 2 to 3 years and is modified by growth, puberty, trauma, and aging. Burns in childhood and adolescence may leave an emotional scar as well as the physical one and may profoundly alter the subsequent

body image, self-esteem, and interpersonal relations as well as ego functioning. Body image development is affected by gender, parental support, medical and surgical care, and developmental changes in adolescence ( [McQuaid et al., 2000](#)). However, body image disturbance may not be inevitable, as suggested by a study of children ages 5 to 15 years with matched controls using human figure drawings, which did not find significant differences in body image, except for increasing body image disturbance in both burned and unburned adolescents ( [Jessee et al., 1992](#)).

A change in the child's body image and associated feelings such as sadness, shame, or pride may be seen in self-portraits ( [Fig. 99.2](#), [Fig. 99.3](#) and [Fig. 99.4](#)). In addition, body image revision occurs through plastic surgery and often allows another stage of reintegration of body image and optimally a degree of healing of the prior damage to appearance and self-image. Focused short-term supportive and educational psychotherapy is helpful, and there are protocols for short-term therapy addressing body image issues such as coping with stigma and learning to manage staring or teasing and other reactions to facial disfigurement ( [Pruzinsky and Doctor, 1994](#); [Robinson et al., 1996](#)). Scales to assess satisfaction with appearance are available ( [Lawrence et al., 1998](#)).



**Figure 99.2.** Portraits, including self, by a facially burned girl.



**Figure 99.3.** Accurate self-portrait by boy with severe burns with his hand surgically attached to his groin during a phase of a reconstructive procedure.



**Figure 99.4.** Self-portrait by boy with facial and hand burns.

#### *Psychological Trauma: Burns, Body Image, and Reintegration*

Facially burned persons have become commonplace on television, in movies (*Nightmare on Elm Street*), and in plays (*Phantom of the Opera*), but only occasionally with compassion or explanation, and most commonly in horror films. An exception is an award-winning pediatric film by Andrew Maguire, *Here's Looking at You, Kid*, a documentary about a boy with facial burns and his and his mother's experiences afterward. Television and film have familiarized the public with scarring from burns. Notwithstanding, or sometimes because of, publicity, children are subjected to ridicule with epithets such as "monster," "French fry," and "Freddie Krueger." Clear explanation by children with burns to peers about their injuries and unequivocal leadership and support by teachers and parents directly addressing such problems may reduce the frequency of these attacks.

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#### CASE ILLUSTRATION: Sibling Stigmatization Reduced by Outreach to the School

Paul is a Caucasian boy who sustained deep, full-thickness burns over 90% of his body, an inhalation injury, and renal failure at age 19 months in a house fire, and he survived only because of heroic measures and 6 months of hospitalization. Three years later, his older brother, Bobby, was brought to a local clinic by his mother because Bobby was being isolated by his peers and had been beaten up severely by neighborhood boys who ridiculed him because his disfigured little brother "was a monster." Bobby was fighting with his peers about this and at the same time deeply resenting Paul and all the special attention he had received. Their mother felt helpless and sought child psychiatric assistance after Bobby slammed the door on Paul to prevent children who were passing by from seeing him. In addition to caring for Paul, this single mother on welfare was caring for younger twins requiring constant medical care for respiratory distress syndrome. They lived in a tough, low-income, suburban neighborhood.

The clinic child psychiatrist, together with the special education outreach team from the hospital and Bobby's school, arranged a school outreach presentation about burns, disfigurement, and burn prevention. The hospital teacher, who conducted the presentation, told a special assembly of 300 first and second graders and teachers how Paul had survived almost miraculously through use of skin grafts. She also explained about "same and different" using *We're Different We're the Same* and illustrated that M & M candies are different colors but taste the same. She emphasized how different each child's feelings may be and "what it feels like to be teased." She reported that the faculty and students were very receptive to her presentation and that Bobby's class looked forward to helping Paul feel welcome when he entered school next year. The boys' mother soon reported that children were now coming to their house to visit the boys, and they looked forward to coming to Bobby's birthday party, whereas before, none of the children had socialized with them at all.

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MacGregor and Bernstein reported that social isolation or *social death* was common among the severely disfigured ([Bernstein, 1976](#)). More recent studies do not confirm this finding, at least in the United States, and this may mean that they and others succeeded in helping to lessen the stigma of burns by stimulating improved surgical and psychological techniques and by advocating vigorously for equal social, educational, and occupational opportunity. However, social stigmatization is prevalent in cultures where disfigurement is seen "as a form of punishment. The individual with disfigurement, his or her family, or an ancestor, according to the particular belief system, has been either cursed by God or the Gods, sinned or violated a taboo. . . . Others may seek to distance themselves from those who have incurred such 'evil' " ([Groce and Zola, 1993](#)).



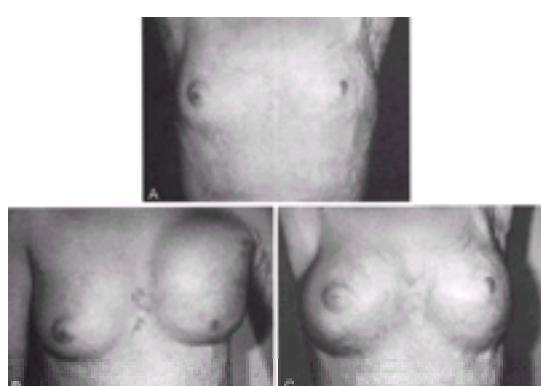
*Note to the Reader: Figure 99.5, Figure 99.6, Figure 99.7, Figure 99.8 and Figure 99.9 are included not to offend but to inform and usefully prepare the consultant for feelings associated with seeing and talking with burned children. Most burns are much less severe. Although such images are seen in surgical texts or on rounds, they are rare in psychiatric writing. The reader may progress through feelings of shock, revulsion, disgust, sadness, and even despair on viewing them—all normal reactions of new staff on ward rounds, and these are reactions that scarred children and adolescents also experience from others at home, school, and, at times, in intimate relationships. Consultants may feel at first unable to comprehend or discuss these kinds of injuries or disfigurements, which many children and families survive and cope with effectively. Being able to discuss the appearance of burn wounds and scars is a key consultative step in helping burned children to find words for their suffering and how they look. These cases all demonstrate positive emotional recoveries that are common today after severe burns. Such pictures are “dehumanized,” standing alone, and, therefore, more upsetting than talking with the child. A child psychiatry fellow, completing his 3-month rotation, observed thoughtfully that he no longer “sees,” that is, reacts to, the scarring because he relates to the children as people. This gradual adaptation is a common experience among burn unit staff.*



**Figure 99.5.** Facial burns before and after reconstructive surgery in a girl at ages 6 and 16 years.



**Figure 99.6.** An 8-year-old girl, who became a pianist, with healed hand burns, amputated digits, but no reconstruction.



**Figure 99.7. A:** Healed breast burns in a 14-year-old patient. **B:** Breasts being reconstructed through use of tissue expanders (balloons inflated with saline). **C:** Postoperative result at age 15 years, after reconstruction.



**Figure 99.8. A:** Burn to the penis in a 3-year-old boy. **B:** His healed perineal scar at 15 years, with full recovery of function.



**Figure 99.9.** Burn causing severe genital and leg scarring in an 8-year-old girl who had no reconstruction, accepted her scars with the help of supportive parents, and later married and gave birth to a child.

**Figure 99.9.** Burn causing severe genital and leg scarring in an 8-year-old girl who had no reconstruction, accepted her scars with the help of supportive parents, and later married and gave birth to a child.

One need only see a child with facial burns ([Fig. 99.5](#)), amputated fingers ([Fig. 99.6](#)), and breast ([Fig. 99.7](#)) or genital scars ([Fig. 99.8](#) and [Fig. 99.9](#)) to appreciate some of the damage to appearance and to internalized body image, as well as the suffering endured, and to realize how irrational thinking and prejudice can be elicited in response. Yet burn treatment and subsequent plastic and reconstructive surgery are remarkably effective in preserving life, in reducing suffering, and in restoring appearance and function. Burns do not inevitably cause psychopathology, but they can, especially in vulnerable children whose burn care and familial support do not meet their emotional needs.

#### HOSPITAL READMISSION FOR PLASTIC AND RECONSTRUCTIVE SURGERY

What are the key questions in child psychiatric evaluation before plastic and reconstructive surgery? What issues are involved perioperatively? Of primary importance are *secure relationships* with the family and with the doctor and treatment team. Another factor is the *capacity to tolerate fear and anxiety* associated with rehospitalization. Other factors involve the *results of preparation and consent for surgery*: (a) realistic awareness of scars and functional impairments, that is, the indications for surgery; (b) the child's wish or willingness to have the surgery at this time; (c) the parents' wish for the child to have surgery at this time; and (d) understanding of the procedure and the capacity to communicate it. It is essential for the consulting psychiatrist to ask the surgeon what the surgical procedure will be, how much time it requires, and what the expected usual emotional and physical sequelae may be. Anxiety often blocks a child's understanding, and multiple explanations may be necessary. If there is significant psychopathology ([Table 99.1](#)), review of records and preoperative assessment are indicated to reduce complications. Suicide is very rare in patients who were not suicidal before the burn injury ([Stoddard, 1993](#); [Wiechman et al., 2000](#)), and the rate appears to be the same or less than in the general population. However, screening for suicide risk is important in all cases of depression and also for those enduring long, stressful, or potentially disfiguring procedures or complications. On occasion, a child will refuse surgery at the operating room door because of acute anticipatory anxiety. This will often have a basis in reality, such as prior serious operative complications or having witnessed those of another patient. Emotional support and benzodiazepines given early often relieve this anxiety, while one makes certain that the child participates in choosing "if, when, and what" surgical procedure to have if the child's wishes were not a part of the original decision. Other preoperative problems include unrealistic expectations of "perfect" surgical results, embarrassment or shame related to severe disfigurement, and resurgence of PTSD symptoms such as flashbacks or nightmares. Supportive, reality-oriented preoperative psychotherapeutic interventions usually allow the patient to cope and progress with a reasonable and hopeful attitude toward surgery.

To perform a psychiatric consultation, some understanding of the surgical procedure—whether it is standard or newly developed—is essential ([Feldman, 1984](#); [May and Moses, 1988](#); [Salisbury, 1992](#)). This includes split-thickness skin grafts and releases of contractures and such dramatic advances as facial resurfacing, restoration of scalp and hair with scalp expanders and flaps, laser surgery, microsurgery to the hand with reconstruction of usable opposition and other digits, breast reconstruction with or without expanders or implants, amputations and prosthetic placement, and even penile or scrotal or vaginal repair or reconstruction. The reason for a consultation, although seemingly broad, often narrowly concerns the patient's reactions to a recent surgical procedure and its success or failure in the child's, parent's, nurses or doctor's eyes. Children undergoing such procedures benefit from psychiatric consultation, reassessment, support, and, at times, treatment.

#### SEXUAL DEVELOPMENT

As children with burns approach and pass puberty, they, like others their age, become more concerned with attractiveness, loving and being loved, and sexuality. Their sense of self is revised, and some become very self-conscious, covering their scars and avoiding being seen in public such as in locker rooms and beaches. Others become counterphobic and may be promiscuous or abuse substances in an effort to cope with conflict over self-image and self-esteem. Their requests for surgery involve wishes to appear normal, such as to repair scalp scars and have normal hair, to have asymmetrical breasts repaired and made symmetrical ([Fig. 99.7](#)), or to repair a facial scar that may cause peer ridicule. Most adolescents, including those with genital burns, which usually heal well ([Fig. 99.8](#) and [Fig. 99.9](#)), become sexually active, and many become parents. Finding a supportive partner, accepting of their burn injuries, aids in sexual adjustment ([Fig. 99.9](#)). Some studies indicate that women with burns have a lower level of sexual satisfaction than men, but this was not found in a small study of adolescents ([Robert et al., 1998](#)). Continued individual, family, and group psychotherapy assists with difficult personal, sexual, social, educational, and occupational transitions. Those who have not had such services or who live far from the centers where they are available may benefit from burn camps in their region ([Doctor, 1992](#)). Although some of these adolescents are followed in medical centers for plastic and reconstructive surgery, many do not receive counseling to assist them emotionally or with education about drugs, contraception, HIV, childbirth education, or parenting skills.

Since the 1980s, most centers have provided "reentry programs" after discharge through the hospital social service, special education, and other departments to assist with prompt return to school of burned children after discharge, rather than home tutoring, which was formerly common and increased morbidity and social isolation. This often involves school outreach educational programs to reduce peers', parents', and teachers' fears of disfigured children and to support reentry. This support is essential for some children, especially those with facial disfigurement, to return to school and to begin effective rehabilitation.

### LONG-TERM TREATMENT

#### Psychological Interventions

##### INDIVIDUAL THERAPY

Many children are not seen for psychological follow-up after their acute burn injury. This group may be most likely to have unexpressed feelings that may shape development, influencing avoidance behavior, and, in some cases, learning problems, phobia, or depression. The most common psychiatric consultations are in plastic surgical clinics, during rehospitalization, or in groups. These medically oriented follow-ups address specific burn-related concerns and encourage the child's and family's mastery over new, often developmentally related issues. Collaborative long-term monitoring with plastic surgeons is indicated for at-risk children. Referral to outside agencies is necessary for those with emotional difficulties or who require psychopharmacologic treatment. A few patients receive long-term weekly insight-oriented psychotherapy. Those therapies are directed toward enhancing self-esteem, body image, interpersonal relatedness, and autonomy, and this often occurs for children who are separated from or have lost their parents. On request, mental health teams at regional burn centers can provide supervisory guidance to therapists in distant locations who are unfamiliar or uncomfortable treating children who have been burned.

##### FAMILY THERAPY

Parents and families may be at greater risk of impairment than the children themselves. Siblings are at increased risk because attention is diverted from them to the burned child, and some siblings share responsibility for the burn. Parental guilt for the injury is a most painful, persistent emotion. Parents often blame themselves rather than attributing a burn to fate or the improbable. Although this defends against feelings of helplessness, it creates a diminished sense of worth as a parent ([Cahners and Kartiganer, 1990](#)). Guilt impedes coping and parental emotional recovery. [Cella et al. \(1988\)](#) find a high rate of depression in mothers years after the burn injury. [Rizzone et al. \(1994\)](#) find an incidence of PTSD of 52% in mothers of burned children, which is higher than that in any study of children or adults with burns. [Meyer et al. \(1994\)](#) find increased stress in mothers, a finding correlating with increased stress in their children who survived burns and in earlier studies that family support and the family value of autonomy are critical to the child's adjustment. [Fukunishi \(1998\)](#) has similar findings with mothers of scalded children. Over many years, parents seen during follow-up clinic visits in group are able to benefit from catharsis and mutual support in outpatient parent groups focused on relief of anxiety and guilt feelings, grief work, feelings about the scars, and improvement of family coping skills. Parents who are in fact responsible for the burn may suffer severe guilt and may be even more difficult to help through this trauma.

##### GROUP THERAPY

In some clinics and camps, groups are led by social workers, psychologists, or child psychiatrists. For instance, a weekly outpatient *group* for 13- to 21-year-old patients provides support for teenagers as they return and focuses on their family lives, body image, peer relations, school, drugs, sexuality, and choices for surgical reconstruction or the specialized cosmetics available. Social workers conduct *groups for parents* addressing how to parent a burned child, guilt, grief, assistance with feelings about plastic surgery, rehabilitation issues, resolution of unrealistic expectations from surgery, and help with referrals to social, educational, or occupational resources in their areas. Salient information from the groups is shared in weekly psychosocial rounds. Social workers and volunteers have also used self-help groups by mail and by telephone.



## Psychopharmacologic Treatment

During readmission for plastic and reconstructive surgery, other indicators for psychopharmacologic treatment are anxiety disorders, anticipatory phobic reactions preoperatively, PTSD, depression, attention deficit hyperactivity disorder, and enuresis. Treatment is based on the diagnosis differs little from that in outpatient psychopharmacology clinics. Agents include the full range of stimulants, anxiolytics, antidepressants, mood stabilizers, and antipsychotics. Because enuresis is common among burned children, behavioral interventions and the bell and pad are the initial interventions, but intranasal aqueous vasopressin and imipramine are also treatment options. Skin rashes, which can be caused by any drug, have the potential of complicating burn care and should be monitored.

## OUTCOME AFTER CHILDHOOD BURN INJURIES

This is a challenging subject for review. There are many small studies varying in quality and a few larger ones. The quality of burn and reconstructive treatment has improved since the mid-1970s, and this makes earlier studies invalid in predicting outcomes for recently treated children and young adults. There are two viewpoints on the outcome data: [Tarnowski et al. \(1991\)](#), in their review, assert that “collectively, findings indicate that there exists little empirical data to support the contention that the majority of pediatric burn victims exhibit severe poor post-burn adjustment,” and [Blakeney et al. \(1993\)](#) essentially agree. The current longitudinal study of [Saxe et al. \(1999\)](#) confirms and extends our earlier findings regarding high lifetime occurrence of ASD and PTSD. My colleagues and I ([Murphy et al., 1989](#); [Stoddard et al. 1989a, 1989b](#)), in a cross-sectional follow-up study of severely burned children, found that a majority of children manifested mental disorders (anxiety including PTSD, depression, disruptive behavior, enuresis), and 20% had no disorders; although some disorders existed before the burn injury, the study's aim was to identify disorders at the time and not to differentiate preburn and postburn psychopathology. In this study, although lifetime prevalence of psychopathology was high, it was low at long-term follow-up. No other study of burned children has used comprehensive structural diagnostic interviews. The longitudinal follow-up study by [Blakeney et al. \(1998\)](#) of children surviving burns over 80% of their body surface area found adjustment within the “norms” for most of the group, but not all. Even using the review by Tarnowski et al., these authors' assertion is premature that few data support severe postburn adjustment, and it may depend on what is considered “severe.” Many studies ([Table 99.4](#)) have found symptoms of postburn anxiety, depression, and behavior problems. Several found burned adolescents to be especially vulnerable to depression, but this is true of the adolescent population at large as well. Several, but not all studies, have found positive correlations between psychopathologic symptoms and visible disfigurement (especially facial) and the size of burn injury. Although several studies are satisfactory, few are excellent. There is need for longitudinal research with structured diagnostic interviews, not only mailed surveys, and large sample sizes including large and small burns, matched controls, and especially inclusion of surgical, psychological, and other treatment variables.

**Table 99.4. Outcome Studies of Burned Children**

What are the likely explanations for somewhat conflicting findings regarding outcomes? First, *preburn psychopathology* does not disappear after the burn injury, but it is difficult to measure reliably retrospectively: It is rarely reported. Second, *selection bias* is present in many studies, and it is difficult to eliminate in tertiary referral centers. Third, *many common instruments do not provide diagnostic data on mental disorders*. Fourth, *other pediatric handicaps and chronic illnesses* have been shown to be associated with increased psychopathology ([Perrin and MacLean, 1988](#)), and this could be true for burns, as our outcome study suggested. Fifth, *treatment interventions of all kinds are rarely described and their effects analyzed*.

## DEATH OF A CHILD

A child's death from burns is a most tragic loss of life resulting from a usually preventable injury. End-of-life care should be planned with the burn team for any critically burned child ([Schnitzer et al., 2000](#); [Stoddard and Saxe, 2001](#)) ([Chapter 104](#)). Parents and family members may have little preparation if the death occurs quickly, as it can from massive burns, respiratory burns, or sepsis. Their grief can be almost unbearable, and the presence of empathic staff members is especially consoling. When there is more time for parental participation and preparation, such as in cases of slow brain death, the grieving process can proceed in stages. Sensitive emotional and ethical ([Chapter 104](#)) issues arise with consent for do-not-resuscitate orders and organ donation, as reuse of body tissues for transplants increases ([Fratianne et al., 1992](#); [King et al., 1993](#); [Hammond and Ward, 1989](#); [Petro and Salzberg, 1992](#)). Such decisions are made with the burn team, led by the surgeon, together with the family. The burn team, supported by the mental health team, provides much needed support after the death to the grieving family and assists with their transition to gaining support from friends and community. Follow-up, by the attending surgeon and social worker, is sustaining to the family both soon after the death and on monthly follow-up consultations and anniversaries. Other children and their parents present in the burn unit may react with alarm, sadness, somatization, posttraumatic play, and fear of their own death. Sensitivity and developmentally appropriate responses to the child aid their coping with yet another unexpected trauma.

## STAFF SUPPORT

The burn unit is a most challenging place to work, and psychiatric consultative skills are highly valued. An understanding of consultative principles, models, impediments, and process is useful before initiating consultation ([Lewis, 1994](#)). Witnessing horribly injured children, seeing, smelling, and touching their burned flesh, and causing pain as part of treatment for an infant or child may evoke anxiety, sadness, and guilt, but also satisfaction and pride in providing needed treatment to an injured child ([Ravenscroft, 1982](#)). Turnover can be frequent in stressed units, but it is reduced by appropriate administrative leadership and staff support, and loyalty develops. Although some units may appear focused on technology to the exclusion of feelings, women physicians in all specialties as well as women in allied professions have contributed to a greater focus on emotional sustenance of both children and staff. The burn unit offers unparalleled opportunities to learn, provide treatment, and teach in areas such as pain, stress, grief, healing, consultation, and diagnosis and treatment in the acute care setting.

The aura of the burn unit is special and, for many, intimidating at first. Strong staff cohesion may make the burn team like the elite “Marine Corps” or battlefield station of the medical center, with a staff “tough enough” to treat such severe, frightening injuries. The image of the patient in the burn unit is of a “monster,” frightening to see and whose handicaps can be crippling—an image at great variance from the real child who speaks, feels, creates, and has relationships, whether with minimal or extensive scarring. Other scary images associated with the burn unit are of death and burning in hell, but also more hopeful ones of rebirth, as in the phoenix rising up from the ashes. The surgeon and others on the team may be both pitied (“how can you stand to work there?”) and idealized as a superhero, savior, or god invested with the magical powers of healing science.

## Entry by New Staff

Arrival at the burn unit may be a culture shock for doctor, nurse, patient, and family alike. New staff, trainees, medical students, and graduate students alternately tend naturally to withdraw emotionally or to identify with the child's suffering, at times overly so. For example, a new researcher said of a boy with massive healed burns, “He must be in so much pain.” On being asked about it later, the boy said that he had no pain and was only concerned with his computer game. There is a usual sequence of emotional adaptation after arrival on a burn unit: shock and dismay, curiosity and fear, frustration and helplessness, and sadness with often persistent feelings and images on return home, appearing as anxiety dreams, and even for some, a questioning of professional identity and goals ([Jellinek et al., 1993](#)). [Bernstein \(1976\)](#) pointed out how the feelings of shock are often adaptive and mobilize staff to use their skills on behalf of the patients and thereby to master their

fear, frustration, and sadness.

When new staff members arrive, it is helpful when they are prepared to expect feelings such as those mentioned earlier. One resident said how it had helped to have heard from me that I, too, even after many years there, feel the stress of the burn unit. It is also helpful to them to know that the feelings of horror wane as they come to know the children and their families and join the burn team, enhancing feelings of competence. Knowing about the usual course of the three phases postburn (acute, intermediate, recovery or rehabilitative) is a hopeful approach in the presence of such stress. They learn, too, that preparation of patients and families for these phases assists in their transitions and hopefulness. Further, it is helpful to provide assurance that mentors are available to listen and provide validation or advice when needed.

### Teaching Empathy

The psychiatrist and other mental health professionals demonstrate listening skills, communication, compassion, reflection, and constructive therapeutic action. Sensitivity to shame and embarrassment ([Lazare, 1987](#)) during rounds when the children's or adolescents' bodies are exposed to groups of staff, or where discussions may be overheard, is crucial to prevent humiliation or involuntary exposure to unwelcome information. It is important to encourage empathy in those "stepped" against feelings and also to convey understanding of inevitable overidentifications with children or their relatives, who evoke feelings reminiscent of their own children, families, or of their own life experience. Such transference and countertransference experiences are frequent, and supervision and experience can assist with these intimate feelings. For instance, a primary nurse transiently wept with grief over the suffering of a child to whom she had to cause pain. This child reminded her of her own, and she was grateful to share her feelings off the unit and then return to her work. In another instance, a doctor became the target of much fear, blame, and rage over a child's disruptive behavior and had to voice his feelings of frustration, despair, and inadequacy before resuming his work. Others can be less aware of their feelings and may act them out within the clinical situation or at home. Such feelings are easily aroused when parents' rage is evoked, when disruptive boys resist helpful nurses and insult them, or when staff members are in conflict over what course of action to take, such as with critically ill or dying children. In such situations, group consultation is most effective ([Jellinek et al., 1993](#)).

### Staff Consultation

The presence of the psychiatrist validates attention to emotional and diagnostic and treatment issues that may otherwise be avoided, often with adverse effects on patient and family care and staff morale. An overall goal is to support staff cohesion and prevent burnout, which may be more likely for staff in burn units and may relate to individual coping skills or "hardiness" ([DePew et al., 1999](#)). A working alliance and frequent communication with the surgical and nursing directors are important for effective consultation. Scrubbing and gowning and entering isolation areas or the operating room convey a willingness to talk with the patient or surgical staff "where they are." The consultative approach is ideally informed, consistent, flexible, and responsive to the situation. Admitting ignorance, reviewing the cases, and helping implement constructive recommendations are effective ways to join the burn team. The consultant models developmental and family assessment skills, and respect and advocacy for children, because surgical trainees may have limited experience with infants and children.

In the situations of staff conflict, various interventions are possible. The psychiatrist reflects on his or her own feelings and realizes that another team member reacts similarly, sometimes also carrying home feelings of fear, helplessness, or dread at returning the next day. Although the ultimate responsibility for treatment is the surgeon's, the spoken or unspoken burdens are shared with nurses, who are often "in the middle" ([Jameton, 1992](#)), and with all other staff. The nurse is more likely to know the child's situation but may feel conflict between duty and irritation when decisions are made or not made that do not agree with her or his sense of what is good care. Routine teaching rounds, psychosocial rounds, or informal or formal acute or plastic surgical rounds, separately or in combination, may be effective for "venting" feelings and working through the issues. Support groups for nurses or other staff members are held to discuss and process stress and morale issues together. The primary nursing management system seeks to achieve that aim within the nursing care team.

Communication is enhanced in many ways. Because consultation is rarely continuously available, staff is sustained by knowing when the consultant is available and the times of next meetings. Crises call for special meetings, including surgeon, nurse, and consultant, the other involved team members, and at times the family and patient. Respect for defenses, which can interfere with empathy, but which are necessary for staff to cope, is important. Defenses include denial, transient regressions, distancing from the situation, and overinvolvement and are often bridged by more senior members of the burn team who help master the situation. Having the psychiatrist on the team "puts behavior into a positive developmental and group-process perspective. Through emphasis on the normal and expectable phases of burn trauma recovery, the usual course of burn hospitalization for different age groups, and the impact of various burn treatments on children and families with different backgrounds and styles," the psychiatrist increases levels of mental health expertise among burn team members ([Ravenscroft, 1982](#)).

### Departure from the Burn Team

When staff members leave the burn unit, their leavings demonstrate their attachment and sense of involvement. The collected staff members often tell residents and trainees in rounds how they will be missed. When seasoned senior staff members leave, the departure can be so painful that those in the unit may react wondering "how will we manage without them?" The farewells and testimonials are proof of their dedication and the appreciation for their work and convey to those who continue that their efforts are worth the hardships. Sensitivity to administrative changes, team cohesion, losses, and the staff's adaptation is useful in maintaining the consultative alliance.

## CONCLUSIONS

Burn injuries are prevalent worldwide among infants, children, and adolescents. Treatment of burns has progressed since the 1940s to a point where most children currently treated in the best burn units survive even massive burn injuries. The priorities in psychiatric care throughout the postburn phases are pain management, diagnosis and treatment of delirium, stress disorders and depression, coping with disfigurement, psychotherapeutic support for the child and family through later plastic and reconstructive surgery, and end-of-life care, including family follow-up. A reflective attitude assists the consultant's gradual adaptation to the stresses of being in the burn unit. Critical psychological interventions are infant-parent support, patient and parent education, encouraging participation by the child in burn dressing changes, aiding in school reentry, aiding in adaptation to body image change, and aiding in social mastery during stressful developmental periods such as adolescence. Rehospitalization for plastic and reconstructive surgery, although stressful, is also an opportunity for emotional processing and reintegration. The child psychiatrist and mental health team experience, along with the other burn team members, significant emotional stress and also the many satisfactions from care of children and their families in the burn unit.

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# 100 CHILD PHYSICAL ABUSE AND NEGLECT

Sandra J. Kaplan, M.D.

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The physical abuse and neglect of children and adolescents continue to be major public health problems. The latest child maltreatment prevalence data were reported by the National Incidence Study-3, which sampled child protective services, law enforcement, juvenile probation, public health, hospital, school, day care, and mental health and social services agencies for a 3-month period during 1993 ( [Sedlak and Broadhurst, 1996](#) ) and by *Child Maltreatment 1998: Reports from the States to the National Child Abuse and Neglect Data System* ( [U.S. Department of Health and Human Services, 2000](#) ), which compiled summary case data from state child protective services.

The Third National Incidence Study ( [Sedlak and Broadhurst, 1996](#) ), using a harm (injury of child documented) standard, found that physical abuse had increased to 5.7 per 1,000 (381,700) children from the First National Incidence Study (1980; [U.S. Department of Health and Human Services, 1981](#) ), when the incidence was 3.1 per 1,000 (199,100) children, and from the Second National Incidence Study (1986; [U.S. Department of Health and Human Services, 1988](#) ), when the incidence was 4.3 per 1,000 (269,700) children.

The abuse of children has been associated with subsequent emotional and behavioral problems, including violent behavior by victims. Aggressive behaviors by victims that have been reported include those directed toward others ( [Alfaro, 1978](#); [Dodge et al., 1990](#); [Herrenkohl et al., 1997](#); [Kashani et al., 1992](#); [Klimes-Dougan and Kistner, 1990](#); [Lewis, 1992](#); [Lewis, 1985](#); [Lewis et al., 1979](#); [Widom, 1989](#) ), toward other family members ( [Kaufman and Zigler, 1987](#); [Oliver, 1993](#); [Straus and Gelles, 1986](#); [Straus et al., 1980](#) ), and toward themselves ( [Deykin et al., 1985](#); [Garnefski et al., 1992](#); [Green, 1978b](#); [Livingston et al., 1993](#); [Pfeffer, 1986](#); [Rotheram and Bradley, 1987](#) ).

Cruelty toward children has been described throughout history, with children viewed as parental property ( [Wolfe, 1987](#) ). However, in the United States, medicine has focused on child maltreatment only since 1946, when Caffey described fractures of the long bones in multiple stages of healing in children with subdural hematomas ( [Caffey, 1946](#) ). Early efforts on behalf of maltreated children focused on advocating for and implementing legislation to protect children. Child maltreatment reporting laws became instituted in all 50 states.

Kempe, a pediatrician, and his colleague Steele, a child and adolescent psychiatrist, both at the University of Colorado, were largely responsible for the current focus on child maltreatment by American medicine, including psychiatry ( [Steele and Pollack, 1968](#) ). They with their colleagues Silverman, Droegenmueller, and Silver reported the *battered child syndrome* in 1962 ( [Kempe et al., 1962](#) ). However, psychiatric studies of and services for maltreated children and their families continue to remain in the early stages of development.

## PHYSICAL ABUSE

### Definitions

*Physical abuse* definitions vary according to (a) cultural practices regarding corporal punishment, (b) cultural values (i.e., privacy versus interdependence), and (c) biological predispositions, such as the higher activity levels of U.S. neonates versus Chinese neonates ( [Gabarino and Etaba, 1983](#) ). Straus and Gelles, the principal investigators of the National Incidence Studies of Violence in America, define child abuse as the use by a parent of any of the acts of the Severe Violence Index of the Conflict Tactics Scale. *Severe violence* is defined by this scale as kicking, biting, hitting with a fist, hitting or trying to hit with something, beating up, threatening with a gun or a knife, or using a gun or a knife ( [Straus, Gelles, and Steinmetz, 1980](#) ). Using this definition, [Straus and Gelles \(1986\)](#) found that 6.9 million children are physically abused each year. They also reported that 97% of children from 0 to 3 years old are physically punished.

Physical abuse has been defined by the Child Abuse Prevention, Adoption, and Family Services Act of 1988 (Public Law 100-294) as “the physical injury of a child under 18 years of age by a person who is responsible for the child’s welfare, under circumstances which indicate that the child’s health or welfare is harmed or threatened thereby, as determined in accordance with regulations prescribed by the Secretary of Health and Human Services.” Persons responsible for a child’s welfare include employees of a residential facility, any staff of a facility, or any staff providing out-of-home care. The New York State Family Court Act (1976) defines physical abuse as “the situation which results when a parent or other person legally responsible for a child less than 18 inflicts or allows to be inflicted upon such child physical injury by other than accidental means” ( [Family Court Act, 1971](#) ).

### Incidence

During 1998, there were an estimated 903,000 victims of child maltreatment nationwide. The 1998 rate of victimization was 12.9 per 1,000 children. Neglect involved 53.5%, physical abuse involved 22.7%, and sexual abuse involved 11.5% of child maltreatment victims. One-fourth (25.3%) of victims were reported as being victims



of more than one type of maltreatment ([U.S. Department of Health and Human Services, 2000](#)).

The exact incidence of child abuse in the United States is unknown, as a result of reporting biases and investigatory procedural constraints. The probability that an abuse report will be filed is increased for ethnic minorities, the poor, urban residents, and those who use public rather than private sources of health care. The ability of a child protective service to document or substantiate abuse also varies with the source of the report.

In 1998, 29.2% of investigated reports were legally documented as involving child maltreatment. Physicians' reports have been found to be most likely to be substantiated by investigatory agencies ([Eckenrode et al., 1988](#)). Sources of 1998 child maltreatment information included, in descending order, educators, legal law enforcement and criminal justice personnel, anonymous reporters, social services, relatives, physical health personnel, and mental health personnel ([U.S. Department of Health and Human Services, 2000](#)).

### Age of Onset of Abuse

Severity of abuse found by the 1993 Child Abuse and Neglect Incidence and Prevalence Study varied inversely with the age of the child victim. Most fatalities occurred in younger children ([U.S. Department of Health and Human Services, 1998](#)). [Daro and Mitchel \(1990\)](#) reported that more than 50% of fatalities involved children less than 1 year old. Child abuse involving prepubertal children is most often reported in single-parent, ethnic minority, low-income families, whereas the majority of adolescents reported as abused in the United States during 1977 were white and from intact two-parent families who earned at that time more than \$11,000 per year ([U.S. Department of Health and Human Services, 1981](#)). Fathers are more often indicated as the perpetrators of physical abuse of adolescents, whereas mothers are more often indicated as perpetrators of physical abuse of prepubertal children ([Straus et al., 1980](#)). Most studies of abuse involving adolescents report that, although such abuse sometimes begins in early childhood and is continued into adolescence, in the majority of cases the onset of abuse begins during adolescence ([Libbey and Bybee, 1979](#); [Lourie, 1979](#); [Straus et al., 1980](#)).

The onset of abuse during adolescence has been explained by [Lourie \(1979\)](#), [Libbey and Bybee \(1979\)](#), [Garbarino et al. \(1984\)](#), and [Pelcovitz et al. \(1984\)](#) to relate most often to interpersonal conflicts around adolescent developmental tasks and parental midlife crises, rather than as a result of socioeconomic stress, as is often the case with the abuse of younger children. Parental psychopathology, as hypothesized in study by the Pelcovitz et al., is an additional risk factor for abuse in families of abused adolescents ([Pelcovitz et al., 1984](#)).

### Physical Examination Findings

Every child suspected of being physically abused or neglected should be given a physical examination ([American Medical Association, 1992](#); [Kessler and Hayden, 1991](#)). The American Medical Association *Diagnostic and Treatment Guidelines Concerning Child Abuse and Neglect* ([Council on Scientific Affairs, American Medical Association, 1985](#)) state the following in terms of diagnostic physical findings: "Characteristically, the injuries are more severe than those that could reasonably be attributed to the claimed cause" ([Council on Scientific Affairs, American Medical Association, 1985](#)).

Physical signs of abuse include the following: bruises and welts on the face, lips, mouth, ears, eyes, neck, head, trunk, back, buttocks, thighs, or extremities that form regular patterns often resembling the shape of the article used to inflict the injury (e.g., hand, teeth, belt buckle, or electrical cord); burns inflicted with cigars or cigarettes, especially on the soles, palms, back, or buttocks; immersion burns (stockinglike or glovelike on extremities, doughnut shaped on buttocks or genitals) or patterned burns resembling an electrical appliance (e.g., iron, burner, or grill); fractures of the skull, ribs, nose, facial structures, or long bones, frequently with multiple or spiral fractures in various stages of healing; lacerations or abrasions; rope burns on wrists, ankles, neck, torso, palate, mouth, gums, lips, eyes, ears, or external genitalia; bruises of the abdominal wall; intramural hematoma of the duodenum or proximal jejunum; intestinal perforation; ruptured liver, pancreas, or spleen; ruptured blood vessels, kidney, or bladder; and central nervous system injuries, including subdural hematoma (often a product of blunt trauma or violent shaking), retinal hemorrhage, or subarachnoid hemorrhage (often a product of shaking) ([Council on Scientific Affairs, American Medical Association, 1985](#)).

### Etiology

The prevailing model of the origin of abuse is the ecologic model ([Belsky, 1980](#)), which views child abuse as the consequence of the interactions of parental vulnerabilities (mental illness, substance abuse), child vulnerabilities (low birth weight, difficult temperament), a particular developmental stage (adolescence, toddler), and social stressors (lack of social supports, poverty, single parenthood, minority ethnicity, lack of acculturation, presence of four or more children in a family, young parental age, stressful events, exposure to family violence) ([Belsky, 1980](#); [U.S. Department of Health and Human Services, 1988](#)).

### Physically Abused Children and Adolescents

Studies of psychiatric disturbance in victims of child abuse who are referred for treatment have found the children to be impulsive, hyperactive ([Martin and Beezley, 1977](#)), depressed ([Green, 1978a](#); [Kaplan et al., 1986](#)), conduct disordered ([Kaplan et al., 1986](#); [Kinard, 1980](#)), learning impaired (Kline and Christiansen, 1975; [Salzinger et al., 1984](#)), and, frequently, to abuse substances ([Kaplan et al., 1986](#)). Studies of children and adolescents recruited directly from child protective services (state agencies responsible for investigating and documenting abuse) have found depression, conduct disorders, and substance abuse in victims ([Kaplan et al., 1998](#)). Studies of child and adolescent psychiatric populations have often found these children to have histories of physical child abuse ([Kashani et al., 1987a](#); [Kashani et al., 1987b](#)). Lewis and associates ([Lewis, 1985](#); [Lewis et al., 1979](#); [Lewis et al., 1992](#)) and [Alfaro \(1978\)](#) find that delinquent and violent adolescents also frequently, have histories of physical abuse. More specifically, children with symptoms of depression are more likely to have been physically abused than nondepressive children ([Allen and Tarnowski, 1989](#); [Kazdin et al., 1985](#)). [Kaufman \(1991\)](#) finds that 18% of her sample of maltreated children (abused or neglected) meet criteria of major depression and 25% meet criteria for dysthymia.

[Salzinger et al. \(1984\)](#) found that victims of maltreatment who were referred for treatment, and to a lesser extent their siblings, showed significantly more conduct disturbance, hyperactivity, tension, and anxiety than did a nonmaltreated comparison group. In an analysis of data using the Diagnostic Interview for Children and Adolescents ([Herjanic and Reich, 1982](#); [Reich et al., 1982](#)) on a referred sample, investigators found that abused children and adolescents were significantly more often diagnosed as having depression, alcohol abuse, conduct disorders, and attention deficit disorders than were a comparison group of nonmaltreated children and adolescents ([Kaplan et al., 1986](#)).

[Kaufman \(1991\)](#) found a prevalence of 18% of 56 maltreated children being diagnosed as having major depressive disorder and 25% as having dysthymic disorder. [Kaplan et al. \(1998\)](#) reported in a comparison study of physically abused adolescents and nonabused adolescents, who were not referred for treatment, that the abused adolescents were more often diagnosed as having depressive disorders and conduct disorders than were the nonabused adolescents.

None of the previously mentioned studies of behavioral dysfunction include any analyses of commonly suggested risk factors for child psychopathology, such as parental psychopathology ([Weissman et al., 1984](#); [Wolkind and Rutter, 1985](#)), lack of family cohesion and adaptability ([Wolkind and Rutter, 1985](#)), lack of perceived parental supportiveness ([Keller et al., 1986](#)), marital discord ([Illfeld, 1977](#)), prolonged separation of a child from a parent ([Rutter, 1971](#); [Wolkind and Rutter, 1973](#)), or head trauma ([Rutter et al., 1983](#)). The studies by [Kaplan et al. \(1986, 1998\)](#) are the only studies that did analyses of psychiatric illness in parents of abused children as risk factors. In their 1986 study, abused children of psychiatrically disturbed parents were more often diagnosed as having a psychiatric disorder than were nonabused children with a psychiatrically disturbed parent. Because these risk factors are frequently present in child abuse, research on the association of child mental illness and abuse will be improved by adjusting for the presence of these risk factors.

### Suicide and Abuse

An association between abuse and suicide has been found in studies of abused children and adolescents, of adolescent suicide attempters, and of mothers who attempt suicide. Self-mutilation is reported in abused and neglected children by [Green \(1978b\)](#). Deykin has reported that adolescents who attempt suicide have more often been reported as abuse victims than have adolescents who have not attempted suicide ([Deykin et al., 1985](#)). [Pfeffer \(1986\)](#) reports frequent child abuse in families of children who attempted suicide, and [Farber et al. \(1984\)](#), [Kaplan \(1986\)](#), and [Rotheram and Bradley \(1987\)](#) report high rates of suicide attempts in adolescent runaways. Garbarino and Farber report high rates of adolescent abuse in runaway youth ([Farber et al., 1984](#); [Garbarino et al., 1984](#)).

In a preliminary report on adolescent suicide of New York City metropolitan area adolescents, [Shaffer \(1987\)](#) reported that conduct disorders, substance abuse, and, less frequently, depression, as well as family discord and family histories of suicide and other exposure to suicide appear to be risk factors for adolescent suicide. These same psychiatric disorders have been reported in child and adolescent victims of abuse ([Kaplan et al., 1986](#); [Kaplan et al., 1998](#)). Child-abusive behaviors

were also found more often in mothers who attempted suicide than in a comparison group of nonsuicidal mothers ([Hawton et al., 1985](#)). Parental suicide attempts are known to be major risk factors for adolescent suicide ([Shaffer, 1989](#)).

The origin of the association between suicidal behavior and physical abuse remains to be studied. It may be secondary to modeling of aggressive behavior within the family or to exposure to the suicidal behavior of family members. It may also be secondary to increased biological risk in these families for disorders highly associated with suicide: affective disorders, substance abuse, and impulsive conduct disorders ([Kaplan et al., 1983](#); [Kaplan et al., 1986](#); [Shaffer, 1987](#)). Finally, the adolescent as well as his or her parents and family may be socially isolated and therefore at increased risk of suicide ([Salzinger et al., 1983](#); [Shaffer and Fisher, 1974](#); [Spinetta and Rigler, 1972](#)). [Kaplan et al. \(1997\)](#) find that physically abused adolescents have significantly more risk factors for suicide than do nonabused adolescents.

### Psychopathology of Parents of Maltreated Children

Comparative studies, although reporting an increased incidence of psychopathology in abusive parents, have been limited by the failure to use structured diagnostic interviews ([Estroff et al., 1985](#); [Paulson et al., 1976](#); [Smith et al., 1973](#)), by study populations inadequately defined as abusive ([Bland and Orn, 1986](#)), by relying on cases referred for treatment, and by indiscriminately combining parents of child and adolescent abuse victims. These studies have reported maltreating parents as depressed ([Bland and Orn, 1986](#); [Kaplan et al., 1983](#); [Wolfe, 1985](#)), aggressive ([Kaplan et al., 1983](#); [Wolfe, 1985](#)), having increased somatic concerns, exhibiting an imbalance in the proportion of negative to positive and aversive control behaviors when interacting with the target child, having more physical and verbal aggressive behaviors when interacting with the child, and having increased arousal and reactivity to any aversive child stimuli when compared with nonmaltreating parents ([Wolfe, 1985](#)).

In a study of a referred sample that used diagnostic structured interviews and distinguished between parents of abused children and parents of abused adolescents, investigators found significantly more diagnosed psychopathology in maltreating parents than in nonmaltreating parents. Mothers in abusive families were more frequently diagnosed as having a depressive disorder, whereas fathers, usually the perpetrating parent, were more often diagnosed as having alcoholism, antisocial personality disorders, or labile personalities. Mothers of maltreated children were more often diagnosed as having drug abuse than were mothers of maltreated adolescents ([Kaplan et al., 1983](#)).

The temporal relationship of the onset of child maltreatment with that of the onset of parental mental disorders has been reported in a study of physically abused adolescents. This pilot study suggests the importance of increased focus on supporting the parenting of adults with mental illness as a child maltreatment prevention strategy. Very few treatment programs for adults with mental disorders include any services specifically designed to enhance parent-child dyadic functioning.

### Intergenerational Transmission of Abuse

Abuse during childhood has been associated with increased risk that its victims will abuse their own children ([Kaufman and Zigler, 1987](#); [Oliver, 1993](#); [Straus et al., 1980](#)). In a critical review of this area, [Kaufman and Zigler \(1987\)](#) suggest that approximately one-third of those who are physically abused, sexually abused, or severely neglected will maltreat their offspring. They report that the presence of one supportive parent during childhood, a supportive spousal relationship, and fewer stressful events during adulthood may buffer against the intergenerational transmission of abuse.

### Social Isolation of Abusive Families

Social competence has been found to be impaired both in child abuse victims and in their parents ([Salzinger et al., 1983](#); [Salzinger et al., 1984](#)). Mothers of abused children and adolescents are less often employed outside their homes and are more socially isolated, particularly with respect to peers. As a result, outsiders are found to have less access to the family interactions of abusive families than to those of comparison families ([Salzinger et al., 1983](#)). This type of social isolation may be hypothesized to lead to a lack of child-rearing acculturation of the abusive parent ([Salzinger et al., 1983](#)). In addition, maltreated children have been demonstrated to have impaired social functioning and to be less popular when compared with their nonabused peers. ([Salzinger et al., 1993](#))

## EMOTIONAL ABUSE

*Emotional abuse* has been defined in the Study of the National Incidence and Prevalence of Child Abuse and Neglect ([U.S. Department of Health and Human Services, 1988](#)) as described in the following sections:

### Close Confinement (Tying or Binding and Other Forms)

This consists of tortuous restriction of movement, as by tying a child's arms or legs together or binding a child to a chair, bed, or other object or confining a child to an enclosed area (e.g., a closet) as a means of punishment.

### Verbal or Emotional Assault

This involves habitual patterns of belittling, denigrating, scapegoating, or other nonphysical forms of overtly hostile or rejecting treatment, as well as threats of other forms of maltreatment (e.g., threats of beating, sexual assault, abandonment).

### Other or Unknown Abuse

This consists of overtly punitive, exploitative, or abusive treatment other than that specified under other forms of abuse or unspecified abusive treatment. This form includes attempted or potential physical or sexual assault, deliberate withholding of food, shelter, sleep, or other necessities as a form of punishment, economic exploitation, and unspecified abusive actions.

Garbarino et al. further define emotional abuse and apply a developmental approach as clarification. They view emotional abuse as a pattern of psychically destructive behavior inflicted by an adult on a child. This pattern may take five forms: rejecting, isolating, terrorizing, ignoring, or corrupting ([Garbarino et al., 1986](#)). Garbarino et al. illustrate these patterns according to stages of child development, as follows:

1. Rejecting
  - a. *Infant*: The parent refuses to accept an infant's primary attachment.
  - b. *Preschooler*: The parent excludes the child from family activities.
  - c. *School-age child*: The parent conveys negative definitions of self to the child.
  - d. *Adolescent*: The parent refuses to acknowledge the changing social roles expected of the child.
2. Terrorizing
  - a. *Infant*: The parent intentionally violates the child's tolerance for change and intense stimuli.
  - b. *Preschooler*: The parent uses extreme gestures to threaten or intimidate the child.
  - c. *School-age child*: The parent places the child in a double bind ("Damned if you do, damned if you don't") ([Garbarino et al., 1986](#)).
  - d. *Adolescent*: The parent threatens to expose and humiliate the child.
3. Ignoring
  - a. *Infant*: The parent fails to respond contingently to the infant's spontaneous behavior.
  - b. *Preschooler*: The parent displays a lack of affect toward and does not engage the child in activities of socialization.
  - c. *School-age child*: The parent does not protect the child or intervene on the child's behalf when made aware of the need for help.
  - d. *Adolescent*: The parent abdicates the parental role and does not have an interest in the child.
4. Isolating
  - a. *Infant*: The parent withholds from the child interactions with parents or other parenting persons.
  - b. *Preschooler*: The parent teaches the child to avoid contact other than with the parent.
  - c. *School-age child*: The parent discourages peer relationships.
  - d. *Adolescent*: The parent attempts to prevent socialization by prohibiting organized and other activities outside the home.
5. Corrupting
  - a. *Infant*: The parent reinforces the development of inappropriate behavior (e.g., sexual) or creates an addiction (exposure to drugs, alcohol).



- b. *Preschooler*: The parent reinforces aggressive or sexualized behaviors.
- c. *School-age child*: The parent reinforces aggressive, sexualized criminal behaviors, or the abuse of substances.
- d. *Adolescent*: The parent promotes in the child socially prohibited forms of sexual, aggressive, criminal, or substance-abusing behaviors.

## **Incidence**

In 1998, 0.7 per 1,000 children or 6% of all children documented as maltreated were found to be psychologically abused ( [U.S. Department of Health and Human Services, 2000](#)).

## **Sequelae**

Emotional maltreatment has been found to be a stronger predictor than physical maltreatment of internalizing and externalizing behavior problems, low self-esteem, suicidal behavior, social impairment, and psychiatric diagnoses and hospitalizations ( [McGee et al., 1997](#); [Vissing et al., 1991](#)).

## **NEGLECT**

### **Definitions**

[Giovannoni \(1988\)](#) generally defined *child neglect* as follows: Whereas abuse is considered an act of “commission,” neglect is considered an act of “omission.” Neglect is perpetrated by caretakers of children who fail to fulfill their caretaker obligations to children. Neglect occurs in the following three situations:

1. Neglect is the result of a parenting problem.
2. Neglect results from social deviance of the caretaker secondary to caretaker problems such as substance abuse, mental retardation, mental illness, criminality, or other problems.
3. Neglect is associated with the physical abuse or sexual abuse of the child.

This definition may be further broken down into the following three categories, discussed at length in the next sections: physical neglect, educational neglect, and emotional neglect ( [U.S. Department of Health and Human Services, 1988](#)).

### *PHYSICAL NEGLECT*

#### *Refusal of Health Care*

This consists of failure to provide or allow needed care in accordance with recommendations of a competent health care professional for a physical injury, illness, medical condition, or impairment.

#### *Delay in Health Care*

This involves failure to seek timely and appropriate medical care for a serious health problem that any reasonable lay person would have recognized as needing professional medical attention.

#### *Abandonment*

This is desertion of a child without arranging for reasonable care and supervision. This category includes cases in which children are not claimed within 2 days and those in which children are left by parents or substitutes who give no (or false) information about their whereabouts.

#### *Expulsion*

This consists of other blatant refusals of custody, such as permanent or indefinite expulsion of a child from the home without adequate arrangement for care by others, or refusal to accept custody of a returned runaway.

#### *Other Custody Issues*

This category involves custody-related forms of inattention to the child's needs other than those covered by abandonment or expulsion, for example, repeated shuttling of a child from one household to another because of an apparent unwillingness to maintain custody or chronically and repeatedly leaving a child with others for days or weeks at a time.

#### *Inadequate Supervision*

In this case, the child is left unsupervised or is inadequately supervised for extended periods or is allowed to remain away from home overnight without the parent or substitute's knowing (or attempting to determine) the child's whereabouts.

#### *Other Physical Neglect*

This involves conspicuous inattention to avoidable hazards in the home, inadequate nutrition, clothing, or hygiene, and other forms of reckless disregard of the child's safety and welfare, such as driving with the child while intoxicated, leaving a young child unattended in a motor vehicle, and so forth.

### *EDUCATIONAL NEGLECT*

#### *Permitted Chronic Truancy*

Habitual truancy averaging at least 5 days a month is classifiable under this form of maltreatment if the parent or guardian has been informed of the problem and has not attempted to intervene.

#### *Failure to Enroll or Other Truancy*

This involves failure to register or enroll a child of mandatory school age in school, causing the child to miss at least 1 month of school, or a pattern of keeping a school-age child home for nonlegitimate reasons (e.g., to work, to care for siblings) an average of at least 3 days a month.

#### *Inattention to Special Educational Need*

This consists of refusal to allow or failure to obtain recommended remedial educational services or neglect in obtaining or following through with treatment for a child's diagnosed learning disorder or other special education need without reasonable cause.

### *EMOTIONAL NEGLECT*

Emotional neglect is defined by the 1986 National Center on Child Abuse study as “a parent providing inadequate nurturance/affection, exposing a child to chronic or extreme spouse abuse, permitting a child to abuse drugs or alcohol, permitting other maladaptive behavior, or refusing a child psychological care” ( [U.S. Department](#)

[of Health and Human Services, 1988](#)).

## Incidence

In 1998, 53.5% (6.8 per 1,000 U.S. children) of documented child maltreatment victims suffered neglect ([U.S. Department of Health and Human Services, 2000](#)).

## Physical Examination Findings

Typical findings are as follows:

1. *Physical neglect*: Malnutrition, repeated pica, constant fatigue, poor hygiene, clothing inappropriate for weather or setting ([Council on Scientific Affairs, American Medical Association, 1985](#))
2. *Medical neglect*: Lack of appropriate medical care for chronic illness, absence of appropriate immunizations or medications, absence of dental care, absence of necessary prostheses such as eyeglasses or hearing aid, discharge from treatment against medical advice
3. *Emotional neglect*: Delays in physical development and failure to thrive ([Council on Scientific Affairs, American Medical Association, 1985](#))

## Psychopathology of the Neglected Child

The effects of neglect have been studied even less frequently than those of abuse. In one of the very few studies on neglect, [Egeland \(1985\)](#) reported that physically neglected children at 12 months were more likely than nonneglected children to have insecure attachments. At 24 months, neglected children were more likely to be noncompliant and easily frustrated, as compared with controls. At 42 months, they were rated as more likely to have low self-esteem and self-assertion, less flexibility, and less self-control and to have a difficult time dealing with frustration. They lacked persistence and enthusiasm on educational tasks. They were more dependent and lacked enthusiasm and interest in the preschool environment. In elementary school, they had attentional problems, low self-assertion, low self-esteem, and greater internalizing behaviors. They were also reported as socially isolated by teachers. Egeland found emotionally neglected children more likely than control children to show declines on cognitive testing, insecure attachment, avoidance of emotional contact, depression, and aggressive behavior. [Bolger et al. \(1998\)](#) found neglected children to have problems with social functioning, including conflict with friends. Neglected children have also been reported to have difficulties in understanding appropriate affective emotional responses to interpersonal situations and to have limited social problem-solving skills ([Haskett, 1990](#); [Rogosch et al., 1995](#)). Greater expressive and receptive language deficits and poorer mathematics and language achievement test scores have been found in neglected than in abused children ([Culp et al., 1991](#); [Eckenrode et al., 1993](#); [Wodarski et al., 1990](#)).

In the United States during 1985, 58 per 1,000 couples experienced severe assault (spouse abuse), which included kicking, biting, punching, hitting with an object, beatings, and threats or harm with guns or knives ([Straus and Gelles, 1986](#)). Children often witness the spouse abuse incidents of their parents. These children are, according to child treatment reporting laws, considered to be victims of emotional neglect by virtue of this exposure. Child witnesses of spouse abuse have been described as having separation anxiety, sleep disturbances, psychosomatic symptoms ([Wolfe, 1987](#)), generalized fearfulness and withdrawal from conflict, impaired social competence, and conduct disorders ([Jaffee et al., 1990](#); [Wolfe et al., 1986](#)). [Pelcovitz et al. \(2000\)](#) found that adolescent exposure to domestic violence, more so than being a victim of physical abuse, increased the risk of the development of posttraumatic stress disorder during adolescence. Witnessing spouse abuse during childhood has also been associated with increased risk for becoming either a perpetrator or a victim of spouse abuse ([Straus et al., 1980](#)).

## Psychopathology of Neglectful Parents

Neglectful mothers are found to be less verbally responsive to their children than nonneglectful mothers ([Aragona and Eyberg, 1981](#)). Neglectful mothers are also found by Aragona and Eyberg to use more direct commands, less verbal praise or acknowledgment, and more critical statements when interacting with their children than are nonneglectful mothers ([Aragona and Eyberg, 1981](#)).

[Zuravin \(1988\)](#) reports that maternal depression is more often associated with physical neglect than with physical abuse of children. [Gaudin et al. \(1993\)](#) find neglectful parents to be more depressed and more socially isolated than are nonneglectful parents. Parents who perpetuate spouse abuse have been frequently found to have substance abuse disorders ([Kaplan, 1989b](#)), whereas their nonviolent spouses frequently had been diagnosed as having depressive disorders ([Kaplan et al., 1988](#)).

## EXPOSURE TO SUBSTANCES IN UTERO

Currently, there is a U.S. epidemic of children being born to mothers who abuse substances. The U.S. [General Accounting Office \(1990\)](#) reports that 325,000 prenatally drug-exposed infants are born annually in the United States, and approximately 100,000 of these infants are exposed to cocaine as well as to other illicit drugs and alcohol used with cocaine.

Reports have noted that infants exposed to substances *in utero* have smaller head circumferences than infants who are not exposed to drugs ([Griffith et al., 1993](#)) and have problems with hyperarousability with prolonged crying, sleeping and feeding difficulties, tonic and attentional problems, low birth weight, and seizures ([Chasnoff et al., 1989](#)). [Rodning et al. \(1989\)](#) report that preschoolers exposed to illicit substances *in utero* when compared with non-substance-exposed preschoolers have lower developmental quotient results on the Bayley Developmental Assessment Test, deficits in spontaneous play and representational play, less secure attachment behaviors, attentional problems with a disorganization of intentionality, and an absence of the impression of delight and pleasure.

Treatment and assessment needs of the *in utero* substance-exposed infant and preschooler include comprehensive programs for parents and children with the following available services: cognitive, language, and mental health assessments of infants and children; dyadic assessment of infant or child-parent attachment; parenting and extended family functioning enhancement strategies; special education, speech and language, and mental health services for children; and substance abuse, mental health, and vocational assessments and programs for parents.

## TREATMENT OF CHILD ABUSE AND NEGLECT

Because parental psychopathology is a high-risk factor for child or adolescent psychopathology in violent families, treatment programs for these families must include treatment for parental mental illness, including substance abuse, as well as treatment for the abused child or adolescent. Specific treatment and prevention strategies for both family violence and adolescent psychopathology and suicidal behavior will be improved, based on further understanding of their association. In fact, the low rate of compliance with treatment in cases of adolescent suicide attempts suggests that adolescents who attempt suicide may come from families that are very similar to families of abused adolescents, and they may therefore require treatment strategies similar to those designed for child abuse. Despite a considerable literature on the diagnosis of child abuse and the initial phases of management, there is a dearth of information regarding long-term psychotherapeutic management. There are virtually no empirical studies evaluating the effectiveness of therapeutic modalities or techniques with this population.

Most child abuse treatment programs have, as their major focus, treatment of the abusing parent, with little treatment of the maltreated child. This model views abuse and neglect as consequences only of parental psychopathology. In contrast, a multidimensional or "ecological" view of child abuse ([Belsky, 1980](#)) defines treatment needs more broadly than the parental psychopathology model. [Main and Goldwyn \(1988\)](#) and [Wolfe \(1987\)](#) emphasize the importance of focusing on the interactive aspect of the cycle of maltreatment and the importance of changing the negative mode of parent-child interactions. In addition to possible emotional disturbances in parents, other variables are seen as contributors to abuse and neglect. These include vulnerability of the child, family dysfunction, and environmental stress factors such as parental unemployment and the degree to which social support systems exist and operate for parents. In this view, child abuse occurs as a function of the degree to which the parents' environment tends to enhance or to undermine good parenting. When support is adequate, parental propensities toward violence are controlled. When there is little social support, violent propensities manifest themselves.

Effective treatment programs recognize the multidimensional nature of the origin of family violence. The multidisciplinary treatment team needs to aim its interventions at variables operating at all levels of the problem: individual psychopathology, family dysfunction, stress, and lack of social supports. In a study done from 1979 to 1981 of 19 child maltreatment clinical demonstration programs funded by the National Center on Child Abuse and Neglect, serving 1,000 families, [Daro \(1988\)](#) found that clinicians rated families who received family therapy and group therapy for 13 to 18 months of treatment as having made the most progress and being the least likely to have a relapse of child maltreatment necessitating rereporting. Parental substance abuse was associated inversely with treatment progress, as were greater



numbers of types of maltreatment exhibited by a family before referral.

In a study of physically abusive families who had a child in out-of-home placement because of abuse, low socioeconomic status, older age of child, greater severity of abuse, or the victim's school behavioral problem predicted poor outcome of social service agency rehabilitative effort and the need for permanent out-of-home care for victims ([Barth et al., 1985–1986](#)).

### Parent Treatment

There is an unusually high dropout rate when abusive parents are referred to traditional community mental health clinics. Given the probability that the parents came from a family environment in which needs for nurturance and dependency were often met with rejection and violence, it is not surprising that these parents often view authority figures with suspicion and mistrust. Initial resistance and missed appointments should be viewed as almost inevitable components of early phases of therapy. To move beyond the parents' lack of basic trust, outreach to these families must be made in a manner not usually needed with nonabusive parents. This includes availability to these parents of the staff on a 24-hour basis and the availability of evening treatment time, to involve both parents. The psychotherapy of the parents is viewed as having two primary components. The first is the provision of intense emotional support and positive models of parenting. The tendency of these parents to make unrealistic demands on their children is dealt with directly by teaching them appropriate developmental expectations and effective, nonpunitive child-rearing techniques. The second component is traditional psychotherapy aimed at insight and conflict resolution. Until a solid, trusting therapeutic relationship is established, this is often done in the context of individual psychotherapy. In later stages, conjoint family sessions deal with the interactional issues, such as the scapegoating of the abused child and marital conflict ([Kaplan et al., 1981](#)).

### Child Treatment

Abused children are at risk of serious behavioral and emotional disorders, developmental disorders, and learning problems. To screen for these problems, complete medical, developmental, and psychiatric evaluations should be performed on all abused and neglected children. School records should be reviewed. Referral for more appropriate educational placement is frequently indicated, as is referral for individual psychotherapy. The goal in psychotherapy is not only to overcome the emotional problems of abuse but also to facilitate emotional development in a manner that will overcome the intergenerational cycle of abuse.

Abuse-focused treatments provided in therapeutic day programs for treatment of young victims have been found to improve social and cognitive skills and to enhance self-esteem ([Culp et al., 1991](#)). Nonabused peers have been used in play therapies that focus on helping socially withdrawn, physically abused, or neglected children ([Fantuzzo et al., 1996](#)). [Urquiza and McNeil \(1996\)](#) and [Borrego et al. \(1999\)](#) find therapy with intensive parent-child interaction therapy (mother-child dyadic therapy) to be effective in families at higher risk of abuse.

### Pharmacotherapy

There have been few studies of the efficacy of medications for abused children. Propranolol was found to lessen hyperarousal and hypervigilance of abused children ([Famularo et al., 1988](#)). [Harmon and Riggs \(1996\)](#) found that clonidine reduced symptoms of aggression, hyperarousal, and sleeping problems of abused preschool children with severe posttraumatic stress disorder. Both these studies used small samples and open medication trial methods and were considered preliminary by their authors.

## LEGAL ASPECTS OF ABUSE AND NEGLECT

All states have reporting laws mandating that professionals in the areas of health care, social service, law enforcement, and education report suspected cases of child abuse or neglect. In approximately 20% of reported cases, children alleged to be abused or neglected become involved in court proceedings ([Besharov, 1971](#)). The purposes of these proceedings are to determine whether abuse or neglect occurred, the services required for family rehabilitation, and the need for custodial disposition.

In most states, abused and neglected children involved in court proceedings receive court-appointed attorneys called *law guardians or guardians ad litem*. The Federal Child Abuse Prevention and Treatment Act of 1974 mandates that states must provide children with representation independent of that of their parents for those children involved in judicial proceedings. The role of these attorneys is to act as advocates for the rights of the child client and the desires of that child. The duty of the law guardian is to permit inclusion of all relevant evidence and argument on behalf of the child client.

## HOSPITAL MANAGEMENT

Child abuse and neglect are frequently diagnosed and reported by hospitals, with children routinely hospitalized after these reports. Many hospitals have developed child protection teams to assess cases of child abuse and neglect. These teams, which often include mental health professionals, cooperate with the state agencies responsible for receiving and investigating child abuse reports and formulating treatment recommendations.

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### CASE ILLUSTRATION

James, a 14-year-old seventh-grade student, was reported by his local police department to child protective services as having been physically abused. Officers in a police car saw him sitting on his front steps with bruises on his face and a swollen left eye. James informed them that his father hit him on the face with a baseball bat after his father saw his report card.

James and his family were referred for evaluation to an interdisciplinary child and adolescent psychiatric outpatient program for families with violence. James, his parents, and his entire family conjointly were psychiatrically evaluated. James' school and pediatrician were contacted after written authorization was obtained from James' mother. James' mother and father were found, respectively, to have major depressive disorder and alcohol abuse. James was found to have attention deficit hyperactivity disorder. James' school indicated that it had been suggested to James' parents that he receive psychotherapy and medication for his attention deficit hyperactivity disorder. However, James' parents had previously been unwilling to obtain mental health services for James, and James did not receive such help. After the evaluation of James and his parents, individual and family psychotherapy and medication were recommended for James. Mr. and Mrs. Smith, James' parents, were given couples therapy for marital problems. James' mother received individual therapy and medication for her depression. James' father was referred to a substance abuse treatment program for his substance abuse problem. James and his parents participated in family therapy, with an emphasis on disciplining James without corporal punishment. James' parents worked in therapy on their relationship and on resolving conflicts over James. Both parents were encouraged to relate to friends outside the family as well as to rekindle relationships with their extended families. They were also encouraged to permit James to be treated for his attention deficit hyperactivity disorder with medication and group psychotherapy. James and his parents complied with therapy, James became less provocative to his parents, and corporal punishment ceased. James' grades improved, and he made his first "best friend." Therapy lasted for 40 sessions. James graduated from high school.

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## PREVENTION

Three types of child maltreatment primary prevention strategies are currently used. The first is competency enhancement, such as parent education programs. The second is preventing the onset of maltreating behaviors through, for instance, media campaigns, crisis hotlines, and community socialization programs for parents. The third is the targeting of high-risk groups, such as single, adolescent, parents of low socioeconomic status and those with complicated pregnancies and deliveries, to attend programs that increase parent-child contact and provide family support, such as visiting nurses or home visiting parent aides ([Britner and Repucci, 1997](#); [Rosenberg and Repucci, 1985](#)). The effectiveness of these programs still remains to be studied because little prevention outcome research has been done using comparison groups.

One methodologically sophisticated study of high-risk mothers indicates that providing visiting nurses reduces the rates of maltreatment, enhances parents' positive perceptions of their infants, improves the health of high-risk mothers, and reduces prematurity rates for their children, as compared with those who do not receive these services ([Rosenberg and Repucci, 1985](#)). [Olds et al. \(1997\)](#) report that high-risk mothers who are part of an intense nurse home visitation program are less likely to be reported for child abuse or neglect than are high-risk mothers who do not receive this service.

## ROLE OF THE PSYCHIATRIST

Psychiatrists can make important contributions to child abuse and neglect case management by consulting with social service agencies, hospitals, and courts, as well as by establishing and participating in treatment programs designed for these families.

Children and parents in child maltreatment cases are often not routinely screened for psychopathology and substance abuse, so the prevalence of psychopathology

and substance abuse has probably been underestimated and untreated. In a survey of hospitals with pediatric beds and of child protective services in the New York City metropolitan area, [Kaplan and Zitrin \(1983a, 1983b\)](#) find that most abused and neglected children and their parents are not assessed by psychiatrists or psychologists, nor are psychiatrists or psychologists members of child protection committees in most hospitals surveyed ([Kaplan and Zitrin, 1983b](#)). In a study of abused children and their parents in cases reaching the Manhattan Family Court, more than half of these children and more than half of their parents have not had psychiatric evaluations before court proceedings, despite having been known to multiple social agencies before court referral. When psychiatric consultation was available to law guardians in family court proceedings, their initial legal custody plans for the children were modified after psychiatric consultation in 21 of 40 cases ([Kaplan, 1981](#)). It is therefore recommended that psychiatrists become involved as members of hospital child protection committees and that routine psychiatric evaluations of parents and children, as well as frequent psychological testing to screen for developmental and learning disabilities and regular review of school records, should be done by agencies involved in planning for these families.

Child maltreatment poses a particular challenge to psychiatry. Child and adolescent mental health treatment compliance depends on parental as well as child and adolescent motivation. Medical neglect reports should be considered by psychiatry when treatment compliance problems exist. This route to overcome resistance to essential mental health treatments for children and adolescents is often overlooked by psychiatrists.

## CONCLUSIONS

Child psychiatric efforts directed at understanding, preventing, and intervening in the area of child maltreatment remain at an early stage. Maltreated children, their parents, and their relatives have been found to have an increased risk of the development of mental disorders, substance abuse, and aggressive behaviors. In addition to the need to advocate for the improvement of environmental stressors, the cessation of exposure of children to family violence, improved parenting, and child development education for parents and children, psychiatry must advocate for an increase in much needed child maltreatment mental health services.

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# 101 CHILD AND ADOLESCENT SEXUAL ABUSE

Sandra J. Kaplan, M.D.

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Child sexual abuse continues to be a major public health problem. During 1998, 1.6 per 1,000 (a total of 103,845) children and adolescents in the United States were sexually abused ([U.S. Department of Health and Human Services, 2000](#)). Research has informed clinical practice by clarifying correlates of sexual abuse and by studying the efficacy of approaches for its assessment and treatment. Professional organizational guidelines pertinent to the mental health care of sexually abused children and adolescents also have been developed.

## DEFINITIONS

The following definition of sexual abuse, used in the most recent National Incidence Study (NIS-3) ([U.S. Department of Health and Human Services, 1996](#)), is used throughout this chapter: Child and adolescent sexual abuse involves a child younger than 18 years of age having experienced one of the following types of sexual acts: intrusion as evidence of oral, anal, or genital penile penetration or anal or genital digital or other penetration; molestation with genital contact, but without evidence of intrusion; or other acts that involved contact with nongenital areas of a child's body (e.g., fondling of breasts or buttocks, exposure) or inadequate or inappropriate supervision of sexual activities when the perpetrator was a parent, parent substitute, or other.

## EPIDEMIOLOGY

There are three major sources of national epidemiologic data on child maltreatment. One is the NIS-3. This study sampled child protective services, law enforcement, juvenile probation, public health, hospital, school, day care, and mental health and social service agencies in counties throughout the United States for a 3-month period during 1993 ([U.S. Department of Health and Human Services, 1996](#)). The incidence of sexual abuse reported by this study was increased compared with that of the two previous incidence studies of 1980 and 1986. Sexual abuse estimates in 1993 increased to 217,700 (3.2/1,000 children) from 119,200 (1.9/1,000 children) in 1986 and 42,900 (0.7/1,000 children) in 1980 ([U.S. Department of Health and Human Services, 1996](#)). Girls were estimated in 1993 to have been targets of sexual abuse more often than boys (4.9/1,000 female children versus 1.6/1,000 male children) ([U.S. Department of Health and Human Services, 1996](#)).

The second major source of national epidemiologic child maltreatment data is represented most recently by *Child Maltreatment 1998: Reports from the States to the National Child Abuse and Neglect Data System* ([U.S. Department of Health and Human Services, 2000](#)). This study, as well as its earlier versions, presents data collected by state child protective service agencies, but not from multiple types of agencies, as did NIS-1 through NIS-3 ([U.S. Department of Health and Human Services, 2000](#)). During 1998, 1.6 per 1,000 (a total of 103,845) children and adolescents in the United States were found to be sexually abused by child protective services. This represented 11.5% of the total 903,000 documented maltreated children and adolescents that year. That year, the rate of sexual abuse was 2.3 female victims per 1,000 female children, compared with 0.6 male victims per 1,000 male children in the population. More than half (55.9%) of the child sexual abuse victims were abused by male parents, male relatives, or other men; 3.8% were sexually abused by female parents only, 12% were sexually abused by both parents, 11.2% were abused by mothers together with others, and 2.2% were abused by female relatives or other women. The average ages of 1998 sexual abuse victims were 10.4 years for girls and 8.6 years for boys ([U.S. Department of Health and Human Services, 2000](#)).

The third major source of national epidemiologic child abuse data consists of population surveys. In a recently published national survey of the child disciplinary practices of 1,000 parents, 5.7% reported that their children had been sexually abused at any time before the survey, and 1.9% reported that their children had been sexually abused in the year before the survey. Sexual abuse was reported as equal in frequency for boys and girls. The sexual abuse cases of this survey were more likely to have been disclosed if parents had been sexually abused themselves, and if families were from lower socioeconomic groups and contained single parents ([Finkelhor et al., 1997](#)).

### Risk Factors for Child Sexual Abuse

Epidemiological studies of the prevalence of child sexual abuse often include a focus on population factors which co-occur with sexual abuse exposure and are therefore considered to increase the risk of sexual abuse. In a survey of women 21 years of age and older, risk factors for child sexual abuse included maternal and paternal alcohol abuse, children's perceptions of mothers and fathers as rejecting and not nurturing, and children not living with both biological parents by 16 years of age ([Vogeltanz et al., 1999](#)). In the 1998 Survey of Child Protective Services' documented sexual abuse cases, risk factors for child sexual abuse included female sex and being approximately 10 years of age if female and approximately 9 years of age if male ([U.S. Department of Health and Human Services, 2000](#)). As mentioned previously, [Finkelhor et al. \(1997\)](#), in their survey of disciplinary practices, found lower socioeconomic status, single parenthood, and parental history of childhood sexual abuse to be risk factors for the sexual abuse of children.

### Recantation of Child Sexual Abuse

After disclosure and documentation of abuse, children may recant. Of 249 state-validated child sexual abuse cases, 4% were found to recant after disclosure of abuse. Half of these occurred in response to pressure from a caretaker ([Bradley and Wood, 1996](#)). [Gonzalez et al. \(1993\)](#) reported that 27% of children who disclosed sexual abuse in therapy later recanted.

## CLINICAL ASPECTS

It is essential that all children and adolescents suspected of being sexually abused be given assessments that include physical examinations by clinicians with training in forensic data collection and interviews by persons with special training in specific methods of interviewing using nonsuggestive methods. If sexual abuse is documented, child and adolescent psychiatric evaluations to define mental health problems and to obtain any needed mental health interventions for the child and the child's caretakers also are essential.

### Physical Examination Findings

Abrasions or bruises of the external genitalia, distortion or attenuation of the hymen, alterations in anorectal tone, sexually transmissible diseases, and pregnancy all

may be findings when sexually abused children are examined ([American Medical Association, 1994](#)).

## Mental Health Correlates of Sexual Abuse

[Kendall-Tackett and colleagues' \(1993\)](#) review of research studies of the effects of child sexual abuse reported that 21% to 36% of child sex abuse victims had no short-term symptoms, and that 64% to 79% of child sex abuse victims had varied patterns of symptomatology. These included, in descending order of frequency, sexualized behaviors, posttraumatic stress disorder (PTSD), poor self-esteem, anxiety, fear, depression, suicidal ideation, somatic complaints, aggressive behavior, running away, and substance abuse.

Posttraumatic stress disorder has been reported as a correlate of sexual abuse during childhood ([Avery et al., 2000](#)). Although sufficient constellations of symptoms frequently are not present to warrant a diagnosis of PTSD using the criteria delineated in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; [American Psychiatric Association, 1994](#)), victims of sexual abuse often present with a constellation of symptoms similar to that seen in victims of other types of interpersonal trauma ([Herman, 1992](#)). *Complex PTSD* ([Herman, 1992](#); [Van der Kolk et al., 1996](#)) often is used to describe this constellation. Complex PTSD symptoms include difficulties with affect regulation, self-concept, and relations with others, dissociative symptoms, and somatization.

Depression also has been reported to be associated with child sexual abuse ([Koverola et al., 1993](#)). Women who were sexually abused as children have been reported also to have increased risk for depression as adults ([Zuravin and Fontanella, 1999](#)). A strong association of suicidal behavior has been reported in survivors of intrafamilial sexual abuse ([Dinwiddie et al., 2000](#), [Wozencraft et al., 1991](#)).

Aggression has been reported frequently in adolescent victims of sexual abuse ([Fergusson and Horwood, 1999](#)) and in adults who were sexually abused as children ([Widom and Ames, 1994](#)). [Lewis \(1992\)](#) hypothesized that child physical or sexual abuse exacerbates preexisting psychobiological vulnerabilities.

Difficulty modulating sexual impulses also has been reported frequently in victims of sexual abuse. Studies of sexually abused children ([Friedrich, 1993](#)) and adolescents ([Trickett and Putnam, 1993](#)) have found these children to differ significantly from their nonabused peers in their displays of unusual sexual behaviors and attitudes.

Risk taking by abused children has been conceptualized to relate to difficulties these children have in modulating their levels of arousal (Cicchetti, 1991). Both sexually and physically abused children have been noted to be more likely than their nonabused counterparts to take part in high-risk behaviors, such as cigarette smoking and alcohol and drug abuse ([Riggs et al., 1990](#)).

Sexually abused children have been found to have fewer close friends and more conflict with parents than nonabused children. They also have been found to have greater numbers of sexual partners, earlier onset of sexual activity, and more frequent unprotected sex than nonabused children ([Johnson, 1996](#)).

Impaired self-concepts in sexually abused children have been reported and have been found to be more evident in older children than in younger children ([Barnett et al., 1996](#); [Cole and Putnam, 1992](#)). Sexually abused children also have been reported to blame themselves for abuse ([Spacarelli, 1994](#)). [Friedrich \(1994\)](#) has hypothesized that increased somatic symptoms in survivors of both physical and sexual abuse in childhood (e.g., [Leserman et al., 1996](#)) are related to the heightened and distorted focus on the physical self that follows abusive experiences.

Dissociation and traumatic amnesia as responses to sexual abuse often have been reported (Putnam et al., 1996; Ross, 1996). Trancelike states, in which a child stares and is inattentive and unresponsive, are the most common dissociation symptoms in children. Dissociation enables the young child to avoid the frightening and overwhelming feelings that accompany abuse. [Ross \(1994\)](#) concluded that over 90% of adults who carried a diagnosis of dissociative identity disorder have a childhood history of physical or sexual abuse. Although dissociation initially may be helpful to the traumatized child, it also may interfere with recovery by interfering with cognitive processing of strong emotions and cognitions associated with the abuse. Dissociation also has been found to interfere with cognitive performance, including school functioning ([Putnam, 1990](#)).

## PSYCHIATRIC DISORDERS

Victims of child sexual abuse also have been found to be at increased risk for a variety of DSM-IV psychiatric diagnoses, most notably major depressive disorders ([Flisher et al., 1997](#)), conduct disorder ([Livingston et al., 1993](#)), and somatization disorder ([Pribor et al., 1993](#)). There is some indication that childhood sexual abuse is associated with disordered eating behavior, particularly bulimia ([Douzinas et al., 1994](#)). PTSD is perhaps the most consistently reported diagnosis in childhood victims of sexual abuse ([Wolfe et al., 1994](#)).

[Friedrich \(1997\)](#) reported that the severity of sexualized behavioral problems associated with sexual abuse is increased if physical as well as sexual abuse occurred. Poor mother–daughter relationship was found by [Hazzard et al. \(1995\)](#) to be the only significant predictor of internalizing and behavioral externalizing problems. Maternal psychiatric status also was found to predict outcome of sexually abused children ([Paradise et al., 1994](#)). PTSD may be predicted by the number of family-related stressors to which the sexually abused child was exposed ([Livingston et al., 1993](#)). Family adaptability and the intensity of parental response to sexual abuse disclosure also were found to predict the intensity of the child victim symptoms ([Mannarino and Cohen, 1996](#)). Adolescent age at the time of sexual abuse also has been found to be associated with increased severity of emotional and behavioral symptoms ([Feiring et al., 1999](#)).

Studies of physically and sexually abused children have documented impaired cognitive and language abilities and school functioning. These problems have included delays in verbal development, impaired academic achievement, and higher rates of school absenteeism, retention, and special class placement ([Eckenrode et al., 1993](#)).

## BIOLOGICAL STUDIES

There have been few studies of the biological correlates of abuse that have included children or adolescents as subjects. Biological studies of traumatized adults have reported that people with PTSD respond to chronic trauma with a state of hyperarousal, including increased heart rate ([Pitman et al., 1990](#)).

[Ito et al. \(1993\)](#) found that when physically or sexually abused children were compared with nonabused children admitted to child and adolescent psychiatric inpatient services, the abused children had more frontotemporal and anterior region brain electrophysiologic abnormalities recorded on electroencephalography.

Dissociative identity disorder, formerly multiple personality disorder, often has been associated with child sexual abuse ([Ross, 1994](#)). Dissociative identity disorder has been found to be associated with optical functioning changes, variances in skin conductance, respiration, and skin temperature, and heart rate with changes in personality alters ([Putnam et al., 1990](#)). "Alters" have been found to be associated with different visual evoked potential and electroencephalographic patterns ([Putnam, 1990](#)).

Changes involving the hormones of the hypothalamic–pituitary–adrenal axis and the hypothalamic–pituitary–gonadal axis also have been associated with sexual abuse. [Putnam et al. \(1990\)](#) found higher morning cortisol levels in sexually abused compared with nonabused girls. In addition, [De Bellis et al. \(1994\)](#) found increased urinary secretion of the catecholamine metabolite homovanillic acid by sexually abused compared with nonabused girls.

Menarche also has been reported to have an earlier onset in sexually abused than in nonabused girls ([Herman-Giddens et al., 1988](#)), as well as an earlier onset in women who reported childhood sexual abuse than in women who did not report such abuse ([Kendall-Tackett and Simon, 1988](#)).

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### CASE ILLUSTRATION

Ten-year-old Nancy cried during her fifth grade teacher's sex abuse prevention educational presentation. After this lesson, when approached by her teacher, Nancy revealed that her father often "touched her private parts." Nancy's teacher made a child abuse report to the state department of social services. Nancy stayed after school with her teacher and was interviewed by a representative from the county child protective service office.

Nancy spoke of her mother working as a nurse during night shifts and of her father caring for her and her 5-year-old brother while her mother worked. Her father worked days as a computer programmer. Each night after he tucked her brother into his bed in his own room, Nancy said that her father came into her bed. She said he took off his underpants, cuddled up to her, touched her private parts, and had her touch his. She said her father then rubbed up against her and that yellow toothpaste-like stuff came out his



privates. Nancy was taken by the child protective service worker to the nearby emergency department, where she was examined by a pediatrician and interviewed by a team that included a pediatric social worker and a child and adolescent psychiatry resident.

Nancy was found to have a dilated and scarred hymen and a vaginal discharge. She was examined using a pediatric colposcope and cultures were obtained of her vaginal discharge for possible venereal disease. During her interview regarding possible sexual abuse, she was able correctly to identify body parts with the aid of male and female anatomically detailed dolls. She was interviewed using nonleading questions and disclosed again that her father had genitally touched her and had orgasms while in bed with her while her mother worked. She further disclosed that her father had told her not to tell her mother because her mother would become upset and might divorce her father and call the police. He said that he would go to jail if the police came and that they would be poor if he could not work, because he would go to jail. Each month, he rewarded Nancy with a secret present to be known only to the two of them. Nancy said that her father began touching her when she was 8 years old and in the third grade. She said that when she began fifth grade, he began putting his private part inside her.

Psychiatric evaluation, at the time of the physical examination, revealed that Nancy was an anxious child who feared retribution by her mother and father because of having disclosed sexual abuse. She expressed fears that her mother would be angry with her and that her father would go to jail. She said it would be her fault if he goes to jail and if her parents get divorced. She said that she was "a bad girl." She spoke of trying to avoid her father, of being afraid of him, of having thoughts all day of him touching her private parts, and of feeling numb and like someone other than herself was being touched by her father. She said that she had recurrent nightmares of being chased by her father and of her mother not wanting to help her from being caught by her father. She said that she had had trouble concentrating in school during the fifth grade and that she was getting only "satisfactories" in reading and mathematics on her report card, even though since first grade, when grades began, she had gotten "excellents" in reading and in mathematics.

There was no evidence of sustained depressive symptoms or of a suicidal plan, attempt, or ideation. There also was no evidence of thought disorder and no history of cigarette or other substance use.

A diagnosis was made of PTSD and Nancy, her mother, and her brother were referred to the child abuse mental health treatment program located in the nearby teaching hospital. Nancy's father was arrested. He was released on bail and told not to return home at this time. He was referred by the child mental health program to a sexual offender treatment program.

Nancy was treated with cognitive-behavioral therapy in a group with other sexually abused girls. Her mother was individually evaluated, diagnosed as having a major depressive disorder, and was provided with individual psychotherapy and antidepressant medication. She also was provided with supportive group therapy together with the mothers of the girls in Nancy's psychotherapy group. Nancy's father was prosecuted, convicted, and released from prison after 1 year. He received cognitive-behavioral and group therapy and was allowed supervised visits with his children. After 2 years of treatment in an outpatient sex offender treatment program, as well as participating in marital therapy with his wife, he was permitted to join in psychotherapy sessions with his children. Three years after his arrest, he was permitted to return to his home. His wife, however, then only worked day shifts and he continued on parole.

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## LEGAL RESPONSIBILITIES

All states have reporting laws mandating that professionals in the areas of health care, social service, law enforcement, and education report suspected cases of child sexual abuse. All child mental health clinicians have reporting responsibilities. It is essential for clinicians to know their particular state's child abuse reporting statute. In addition, child sex abuse may be prosecuted by the district attorney and investigated by the police. Child mental health clinicians may be called on for information by these agencies.

## PROFESSIONAL ORGANIZATIONAL ASSESSMENT GUIDELINES

The following professional organizations relevant to child and adolescent psychiatric practice have issued guidelines for the evaluation of children and adolescents suspected of being physically and sexually abused: the American Academy of Child and Adolescent Psychiatry ( [Bernet, 1997](#)); the [American Professional Society on the Abuse of Children \(1990, 1995\)](#); the [American Psychological Association \(1994\)](#), the [American Academy of Pediatrics \(1991\)](#), and the [American Medical Association \(1994\)](#).

## INTERVIEWING ALLEGEDLY ABUSED CHILDREN: ISSUES

Anatomically detailed dolls have been used to facilitate children's identification of body parts. Interview guidelines for use with these dolls, including those of [Boat and Everson \(1988\)](#) and [White \(1991\)](#), have been developed. A general consensus has emerged that because there is no known behavior with anatomically correct dolls that provides definitive evidence that a child has been abused, these dolls should not be used in isolation as a diagnostic test for child sexual abuse ( [American Professional Society on the Abuse of Children, 1995](#) ). The research of [Bruck et al. \(1995\)](#) and others also has led to the recommendation that these dolls should be used with caution because they have been associated with false reports in preschoolers.

There is no evidence that anatomically correct dolls are superior as interview aids to anatomic drawings or regular dolls. However, some clinicians argue that there may be a place for these dolls in clinical settings, where they may serve as aids to labeling of body parts and demonstration, and as facilitators for children who are better able to "show" rather than "tell" what happened ( [American Professional Society on the Abuse of Children, 1995](#) ).

## MEMORIES OF ABUSE

Since the early 1990s, there has been increasing recognition that, under certain circumstances, false memories of abuse may develop in children ( [Ceci and Bruck, 1993](#) ). Preschoolers, in particular, have been found to be vulnerable to confusion and false memories because of faulty interview techniques ( [Ceci and Bruck, 1993](#) ). Recent studies inform clinical practice by suggesting that children are most likely to be misled during interviews if (a) they feel it is preferable to "guess" or "pretend" during interviews rather than to acknowledge "not knowing" ( [Saywitz and Moan-Hardie, 1994](#) ); (b) their memories are "weak" ( [Saywitz et al., 1991](#) ); (c) they are confused about "adult" language that does not take the child's developmental level into account ( [Saywitz et al., 1991](#) ); (d) the interviewer is seen as unfriendly, intimidating, or authoritarian ( [Goodman et al., 1992](#) ); or (e) the interviewer has a preconceived bias about the event ( [Ceci and Bruck, 1993](#) ).

There also is a general consensus that the use of open-ended questions when interviewing children suspected to have been sexually abused, although associated with lowest risk for confusion, also is associated with the generation of least information. In contrast, leading questions, which pressure a child to agree with the interviewer, can lead to a permanent distortion of memory for what happened. Focused questions, which fall between free recall and leading questions, have been found to be necessary by many clinicians when faced with a child reluctant to discuss abuse ( [Saywitz and Goodman, 1996](#) ). For example, specific nonleading questions, such as "tell me about your dad . . . what do you like about him?" may generate information that the child otherwise would not volunteer. To minimize suggestibility in forensic interviews of allegedly abused children, [Reed \(1996\)](#) has recommended that interviewers systematically clarify to children what is expected of them by specifically teaching children to state when they are "confused" or "do not know" answers to questions.

## TREATMENT

### Psychotherapies

Most clinicians would agree that victims of child abuse should be routinely assessed to determine the need to receive appropriate referrals for mental health treatments. Social service agencies and juvenile courts refer only a minority of victims for treatment ( [Chapman and Smith, 1987](#) ). Multiple reasons exist for this gap in mental health needs assessment and treatment referrals for these children. They include a lack of available child protective services personnel to do other than focus on the protection needs of children, and a lack of available and affordable child mental health services dedicated to serving maltreated children and their families.

Nevertheless, there has been a growing consensus that the most effective therapeutic approaches in dealing with victims of abuse are those that use structured and direct approaches focused on integrating feelings related to the abuse ( [Finkelhor and Berliner, 1995](#); [Friedrich, 1994](#); [James, 1994](#) ). These therapies emphasize helping children to organize and express their memories and feelings regarding trauma in a manner that facilitates their viewing abuse as something bad that happened to them, rather than as indicating stigmatization or destiny.

Most approaches include parallel treatment of the parents involving education regarding abuse effects and methods of helping their children deal with its aftermath. Diagnosis and treatment of psychiatric disorders in abusive parents as components of the treatment of maltreated children also are important because of their high risks for depression, antisocial personality disorder, and substance abuse ( [Kaplan et al., 1990](#) ).

[James \(1994\)](#) makes the important point that therapy of traumatized children requires a developmental approach. Even after termination of an initial course of treatment, it may be necessary to have the child return at later developmental stages to "work through" the meaning of trauma in light of a new stage of development. This is most evident in the case of sexual abuse where the traumatic sexualization, often is seen in victims of incest, may not become fully apparent until the abused child becomes sexually active as an adolescent or a young adult. These considerations need to be balanced with the potential use of long-term therapy with children who have permanent views of themselves as victims.

### Sexual Abuse and Psychotherapy

A major advance in the field of child abuse since the early 1990s has been the systematic evaluation of the effectiveness of a variety of treatments for child targets of sexual abuse ([Becker et al., 1995](#); [Finkelhor and Berliner, 1995](#)). Most of these use either cognitive-behavioral ([Cohen and Mannarino, 1993, 1998](#); [Deblinger et al., 1990, 1996](#)) or structured group psychotherapy approaches ([De Luca et al., 1993](#); [Larzelere, et al., 1993](#)). These studies report that sexually abused children show improvement in some symptoms after even a relatively brief course of treatment. [Finkelhor and Berliner \(1995\)](#), after an extensive review of the empirical literature on treatment of sexually abused children, conclude that although there remains a need for large-scale, randomized trials, research lends support to the effectiveness of abuse-focused directive therapies compared with less trauma-focused treatments.

Because sexually abused children may present with a wide variety of symptom patterns, further research is needed to determine the most effective treatment approaches for particular constellations of symptoms. There is evidence, for example, that aggressive and sexualized behaviors may be more resistant to change than "internalized" symptoms such as depression or anxiety ([Larzelere et al., 1993](#)). Use of specialized treatment strategies for sexualized behaviors therefore may be indicated ([Gil and Johnson, 1993](#); [Ryan, 1991](#)). Optimum therapeutic intervention strategies for the approximately 40% of sexually abused children who have no clearly demonstrable associated symptomatology ([Kendall-Tackett et al., 1993](#)) remain to be determined.

Parenting-focused therapies need to be components of child sexual abuse treatments. However, the high rates of depression and social isolation often seen in abusive parents ([Kaplan et al., 1990](#)) are two of the variables that have been associated with poor prognosis for parenting-focused interventions, unless parental disorders also are treated. In addition, although therapeutic efforts targeting abusive parents may be viewed as important components of treatment processes, interventions that do not include the abused child are, by definition, going to be limited in effectiveness.

### Pharmacotherapies

There have been few studies of the efficacy of psychopharmacologic treatments of PTSD or other mental disorders associated with abuse that have used abused children as subjects. Studies of the effectiveness of medication in adults with PTSD have relied most often on clinical reports or on open trials. These studies suggest that psychopharmacologic interventions may be helpful adjuncts to psychotherapy, but usually do not suffice as isolated treatment. Carbamazepine and the benzodiazepines have been found to be helpful in decreasing affective instability ([Friedman, 1988](#)). However, there is little evidence that either of these medications improves the full spectrum of PTSD symptoms. Two double-blind studies of tricyclic antidepressants ([Davidson et al., 1990](#); [Frank et al., 1988](#)) and one study of monoamine oxidase inhibitors ([Frank et al., 1988](#)) found these medications to be moderately helpful in treating PTSD. [Van der Kolk et al. \(1994\)](#) found that fluoxetine diminished the sense of numbing, the affect dysregulations, the impaired relationships with others, and the loss of sustaining beliefs reported by persons with PTSD. Sertraline has been reported to diminish symptoms of PTSD, depression, and the alcohol cravings of patients with PTSD in substance abuse treatment ([Brady et al., 1995](#)). Several double-blind studies that did not find antidepressants to be helpful in treating adults with PTSD have been criticized for measuring the effectiveness of the medication for only 4-week periods ([Reist et al., 1989](#)).

One of the few studies of the usefulness of psychotropic medication in the treatment of childhood PTSD ([Famularo et al., 1988](#)) found that propranolol, given at a dose of 2.5 mg/kg, was effective in lowering hyperarousal and hypervigilance in victims of physical or sexual abuse diagnosed with PTSD. [Terr \(1989\)](#) has suggested the use of propranolol or other beta-blockers for traumatized children as an adjunct to behaviorally based treatments ([Terr, 1991](#)).

[McCarthy et al. \(1994\)](#) reported that responses of depressive symptoms to fluoxetine were greater in bulimic patients with histories of physical or sexual abuse than in those with no abuse histories. Clonidine has been found to reduce symptoms of aggression, hyperarousal, and sleep problems in preschool children with severe PTSD ([Harmon and Riggs, 1996](#)).

### SEXUAL ABUSE PREVENTION

In contrast to physical abuse, where the focus typically has been on helping parents learn appropriate child-rearing practices, the focus in primary prevention of sexual abuse has been on child education. School-based programs typically aim to teach children to differentiate "good" from "bad" touch, to deal with abusers' efforts to keep abuse a secret, and what actions to take should sexual abuse be attempted. Children also are taught that abuse may be attempted by people they know, and that should abuse occur, the child is never at fault ([Wolfe et al., 1995](#)).

Although research has found that children's knowledge of how to deal with sexual abuse increases after these programs ([Berrick and Gilbert, 1991](#)), there has not been evidence that such knowledge translates into ability of children to take action when exposed to abuse experiences. [Pelcovitz et al. \(1992\)](#) described 22 young school-age children who were sexually abused by a school employee and did not disclose their abuse, despite having received prevention training before and during the abuse. The reasons the children gave for nondisclosure included confusion about what the prevention program told them to do in the case of abuse, fears of retaliation by the abuser, and concerns that they would be blamed by their parents for the sexual activities associated with the abuse. The adequacy of most prevention programs to teach preschoolers even basic concepts has been questioned ([Wolfe et al., 1995](#)). Preschoolers exposed to six sexual abuse prevention curricula often found the material in these programs to be confusing, suggesting their developmental inappropriateness for preschoolers ([Berrick and Gilbert, 1991](#)). However, sexual abuse prevention has been demonstrated to be adequately taught, even to preschoolers, when the curriculum is sensitive to the child's developmental level, is of sufficient length, and includes the active involvement of the child through role play and other participatory techniques ([Kraizer et al., 1989](#)).

There remains little evidence that sexual abuse prevention programs are effective in the primary prevention of child sexual abuse, but there is a general consensus that these programs can be effective in prompting sexually abused children to disclose, for the first time, that they were abused ([Finkelhor and Strapko, 1992](#)). There also now is a need to develop programs that more actively include parents because responsibility for child sexual abuse protection currently rests primarily with children.

### CONCLUSION

Child sexual abuse has been found to involve up to 5.7% of children in the United States ([Finkelhor et al., 1997](#)). It also has been found to be correlated with emotional and behavioral problems, including sexual behavioral problems, of victims. Suicidal behavior, substance abuse, and psychiatric disorders including PTSD also have been demonstrated to be correlates of sexual abuse exposure.

Despite the high prevalence and the mental health consequences of childhood sexual abuse for victims, insufficient efforts are being made to recognize these consequences and there are too few resources available to provide the needed mental health services to these victims and their families. Screening for victim mental health problems does not routinely occur during the abuse legal documentation investigative and case planning period by state child protective services. Victim mental health assessments also do not usually take place when a sexual abuse report is made. Children, as well as their families, are not routinely referred for mental health services after legal documentation of abuse. There also are few specialized treatment programs to which to refer sexually abused children and their families.

Exciting advances have taken place regarding care of sexual abuse victims. Clinical research has demonstrated the effectiveness of psychotherapies and suggested the efficacy of medications in ameliorating the behavioral and emotional consequences of child sexual abuse exposure. Specific clinician interviewing strategies have been developed to avoid suggestion and interference with sexual abuse-associated litigation. Psychiatry and allied mental health professionals have much to contribute in terms of advocacy for sexual abuse victims and an increased understanding of the need for provision of rehabilitation opportunities to these children.

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# 102 MUNCHAUSEN SYNDROME BY PROXY

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## DEFINITION AND HISTORICAL NOTE

### Definition

Munchausen syndrome by proxy is a disorder in which a person persistently fabricates symptoms of illness on behalf of another, thereby causing that person to be regarded as ill ([Meadow, 1977](#)). In instances involving children, it is nearly always their mothers who are fabricating the illness. The severity of the disorder and extent of the fabrication are variable: In the least severe cases mothers only report false symptoms, and the physical harm to the children is only that resulting from the medical investigations carried out in attempting to diagnose the illnesses; at the other end of the spectrum are instances in which mothers have caused severe physical harm to their children or even the death of their children in the continued pursuit of making their children appear ill. Although a form of child abuse, there are characteristics of this syndrome that set it apart from other types of abuse.

### Historical Note

In 1951, Asher first used the eponym Munchausen syndrome to describe adults who consistently fabricate symptoms of illness for themselves, leading to numerous medical investigations and frequently to surgical operations. The syndrome was named after Baron von Munchausen of Hanover, who lived in the 18th century and was renowned for telling greatly embellished stories about his adventures in the wars against the Turks. His tales became even more fantastic when written by a friend, Rudolf Eric Raspe, who published a pamphlet ([1948](#)) retelling some of the stories. In [1976](#), Sneed and Bell used the term "the dauphin of Munchausen" to describe a case in which a 10-year-old boy presented with factitious recurrent urinary calculi and in which the mother was suspected of colluding with the child in fabricating the symptoms. The following year, [Meadow \(1977\)](#) coined the term Munchausen syndrome by proxy in his report of observations of two cases in which mothers repeatedly caused their children to be ill. Prior to this time, there had been reports in the literature of cases referred to as "nonaccidental poisoning" in which children repeatedly presented as diagnostic dilemmas and were found to have been poisoned by a parent ([Lansky and Erickson, 1974](#); [Rogers et al., 1976](#)); such cases are now considered to be variants of Munchausen syndrome by proxy. Subsequent to Meadow's initial report, there were other suggestions for a title for the syndrome; these included the names Meadow's syndrome or Polle syndrome, but these have now given way to the more commonly used Munchausen syndrome by proxy ([Lazoritz, 1987](#); [Meadow and Lennert, 1984](#); [Verity et al., 1979](#)). However, this name may eventually be replaced by the term factitious disorder by proxy, which is listed in Appendix B of *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR) as a category requiring further study ([American Psychiatric Association, 2000](#)).

## PREVALENCE AND EPIDEMIOLOGY

Although the true prevalence remains unknown, Munchausen syndrome by proxy is almost certainly a rare disorder. Active reporting of cases in a prospective study conducted over a 2-year period in the United Kingdom and Republic of Ireland established an annual incidence of 0.5/100,000 children aged under 16 years, and the peak incidence of 2.8/100,000 children in the first year of life ([McClure et al., 1996](#)).

The most comprehensive description of the presentation of cases is found in an article published in 1987 in which Rosenberg conducted a review of the existing literature and summarized all the published reports. These included 117 children in 97 families. In nearly all instances the perpetrator was the mother, although there have now been reports of other persons such as a baby sitter or other relative being involved. Fathers have only been rarely implicated as being the perpetrator or appearing to be complicit in the fabrication of illness ([Makar and Squier, 1990](#); [Meadow, 1984](#)), although recently Meadow has published a series of 15 such cases occurring over a 10-year period (1998).

The diagnosis has been made in children of all ages from the first month of life to 21 years. Rosenberg reported the mean age at time of diagnosis as 40 months, with the mean time interval between the onset of symptoms and time of diagnosis being 15 months. In the more recent epidemiologic study from Britain, the median age of diagnosis was 20 months, suggesting that the diagnosis is now being made earlier ([McClure et al., 1996](#)). There are reports of instances in which the condition started prior to birth with mothers inducing preterm delivery ([Goss and McDougall, 1992](#); [Porter et al., 1994](#)). There is an approximately equal prevalence among male and female children.

## CLINICAL DESCRIPTION

The variety of medical symptoms in children who present with Munchausen syndrome by proxy is extensive and includes practically all organ systems. Generally the illness appears to be multisystem, and the children may appear to have different types of illness at different times. Bleeding is the most common presentation and accounted for 44% of the 117 cases included in Rosenberg's review of the literature. The apparent bleeding may be from many different sites, including the gastrointestinal, genitourinary, and respiratory systems. Seizures accounted for 42% of the cases, central nervous system depression for 19% of the cases, and apnea was present in 15% of the cases. Diarrhea was a presenting symptom for 11%, vomiting for 10%, and fever for 10%. Altogether Rosenberg listed 68 different presentations or pathologic findings. The means by which the perpetrators caused the symptoms or abnormal findings are just as diverse and illustrate the severity and horrifying nature of the syndrome: One mother had put bleach in her child's eye, causing the appearance of a periorbital infection; others had repeatedly suffocated their children so as to simulate recurrent apnea or seizures. Other mothers caused sepsis by putting fecal material into their children's intravenous lines.

In approximately one-quarter of cases in Rosenberg's review, the mother had simulated an illness but had not actually done anything directly to the child to cause harm. These were instances where the mother had done something such as putting drops of her own blood in her child's urine or contaminating the specimen. In these instances, although the mother does not herself physically harm the child, she does continue to collaborate with the physicians as distressing and often painful investigations and procedures are carried out. Meadow also has reported cases in which false allegations of sexual abuse have occurred in the wider context of Munchausen syndrome by proxy with other factitious illnesses ([Meadow, 1993](#)).

[Bools and associates \(1992\)](#) have pointed out that there is a significant amount of comorbidity among cases of Munchausen syndrome by proxy: In a review of 56

cases, 29% had a history of failure to thrive and 25% had a history of either nonaccidental injury or neglect. Siblings also might have a history of such findings or might themselves have been the subjects of fabricated illnesses. This appears to be particularly true among cases that have presented as apnea and which, in fact, are owing to suffocation ([Alexander et al., 1990](#); [Light and Sheridan, 1990](#); [Meadow, 1990](#)). When children present with apnea, Munchausen syndrome by proxy should always be considered if there is a history of death of a sibling or if serious episodes of apnea have occurred only in the presence of one person.

### Description of the Mother

These mothers often have had prior extensive exposure to the health care system. This, in some instances, has been from past training and work experience as a nurse, medical receptionist, or other health care professional. In [Meadow's \(1982\)](#) description of 17 families, nine of the mothers had such a background, and [Rosenberg \(1987\)](#) reported that 27% of 97 mothers had a nursing background and another 3% had worked in medical offices. In other cases, the mother herself has had Munchausen syndrome and therefore has brought to her experience as a mother both her own psychopathology and often a vast knowledge of medicine, hospitals, and medical practice acquired from her experiences prior to her child's birth.

A striking characteristic of the mothers is that they are nearly always considered exemplary in all their interactions with medical staff. This is in contrast to adults with Munchausen syndrome and also parents who provoke sickness behavior in their children and refuse to accept psychological mechanisms ([Kaplan and Sadock, 1988](#); [McKinlay, 1986](#)). Both of these groups are often described as demanding and difficult. In Munchausen syndrome by proxy, the mothers often develop close relationships with the nurses and doctors with whom they come in frequent and continued contact. These relationships sometimes traverse the more usual boundaries between parent and medical staff and may include such things as helping the nurses in their duties, eating meals with the doctors, or maintaining social contact with the medical personnel outside of the hospital. However, these mothers tend to be unavailable for genuine interpersonal interactions, and hospital staff often report subjective feelings of uneasiness or feeling intrusive in the mother's presence ([Zitelli et al., 1987](#)).

The quality of the mother's care for her child is also notable. These mothers are often considered model parents who are extremely attentive to their children. They take over the care of their children to a greater degree than is usual in hospitals. They often live in the hospital and remain with the child constantly. It has been noted, however, that the care given to the child can be of an excessive nature; for example, the child may be dressed in inappropriately lavish clothing, or the hospital room may be stocked with an outrageous number of toys ([Zitelli et al., 1987](#)).

One striking quality of the mother that may be important in recognizing the syndrome is her inappropriate affect when given information about the severity of her child's illness or discussing invasive medical investigations. There is a bland acceptance, rather than obvious distress, and she appears to be relatively at ease with medical uncertainties ([Zitelli et al., 1987](#)). In one report, the mother was even described as appearing euphoric as her child became sicker ([McGuire and Feldman, 1989](#)).

Besides fabrication of symptoms of illness, these mothers often fabricate extensively about other parts of their lives. An example of this is a mother who reported that she had just completed a law degree and was working toward a master's degree in Russian history, both of which were false ([Guandolo, 1985](#)). Certainly an important element of the syndrome is the mother's ability to converse with the medical staff about her child's illness in a very knowledgeable and medically sophisticated manner. The other fabrications often serve to add to the mother's appearance as an intelligent person or as someone who has achieved despite adversity.

### Description of the Father

In contrast to the mother's constant presence, the father may have very little involvement in his child's care and sometimes does not even visit the hospital. This is particularly noteworthy considering the severity of the child's illness. In a review of 37 families, [Gray and Bentovim \(1996\)](#) described 70% of the fathers as peripheral or absent from the family system; often the fathers have jobs that keep them away from the family for prolonged periods of time ([Meadow, 1982](#)). The marital relationship between the parents is often poor, although in some instances the child's apparent illness serves to bring the parents closer together.

In those rare instances where fathers are the perpetrators of Munchausen syndrome by proxy, their interactions with medical personnel appear quite different than is the case with mothers. Like mothers, the fathers often stay with their children in hospital, but are considered by staff to be demanding, overbearing, and unreasonable, and are often quick to make formal complaints and seek legal redress ([Meadow, 1998](#)). It is notable that in Meadow's description of 15 fathers, none were actively employed and 11 had factitious disorders, including five that were considered to be Munchausen syndrome.

## ETIOLOGY AND PATHOGENESIS

Although reports of Munchausen syndrome by proxy are largely found in pediatric journals rather than the psychological or psychiatric literature, there is now an expanding literature that has contributed to a greater understanding of the disorder. Most have been individual case reports ([Chan et al., 1986](#); [Lansky and Erickson, 1974](#); [Nicol and Eccles, 1985](#); [Palmer and Yoshimura, 1984](#); [Waller, 1983](#)), but more recently [Bools and colleagues \(1994\)](#) have reported the systematic evaluation of a series of cases. Thus, there is now some understanding of the underlying psychopathology of the condition and knowledge of features that are common to most cases. However, the full extent to which some of the descriptions may be generalized and an understanding of the limits of the spectrum of the disorder remain undefined.

Three major etiologic factors appear to be important in the pathogenesis of the disorder. These include the mother's experience of abuse or rejection in her own childhood, her pathologic relationship with her child, and the rewarding effect of the medical care system on the mother. In addition, associated psychopathology often contributes to the development of the syndrome.

### Abuse or Rejection of the Mother

Perpetrators of Munchausen syndrome by proxy often have experienced abuse in their own childhoods or have felt rejected by one or both parents ([Krener and Adelman, 1988](#); [McGuire and Feldman, 1989](#); [Palmer and Yoshimura, 1984](#)). In the study conducted by [Bools and coworkers \(1994\)](#) of 47 cases, they were able to interview 19 mothers at variable intervals after the event. Of these 19, 15 (79%) were described as having suffered emotional neglect or abuse in childhood, four had experienced physical abuse, and five had experienced sexual abuse. The experience of rejection continues into adult life, and there is often a poor marital relationship ([Meadow, 1982](#)). The mothers often feel isolated and have a decreased sense of self-worth.

### Pathologic Relationship with Child

These mothers have an extremely close, symbiotic relationship with their children. The child is viewed as very precious but also as somehow damaged and susceptible to illness or harm. [Meadow \(1977\)](#), in his first description of the syndrome, described a child who had been a "long-awaited baby" and who was born after the mother had taken a fertility medication. The child described by [Nicol and Eccles \(1985\)](#) was born after a pregnancy in which there had been a threatened miscarriage at 12 weeks' gestation, an antepartum hemorrhage at 36 weeks, and termination of breast-feeding because of cracked nipples when the child was 5 weeks old. In this regard, cases of Munchausen syndrome by proxy are similar to those seen in the vulnerable child syndrome in which the mother develops an abnormally overprotective relationship with the child following a severe illness early in the child's life ([Green and Solnit, 1964](#)). Obviously, there is the important difference that in the vulnerable child syndrome the mother does not cause her child to be ill, although she views her child as abnormally susceptible to illness. In Munchausen syndrome by proxy there is the complicated and contradictory situation of a mother being excessively concerned about what may be minor complaints while actually causing illness herself.

The contradictory relationship between mother and child is extremely complex and not fully understood. In the case reported by [Nicol and Eccles \(1985\)](#), they describe the mother as experiencing the failure of breast-feeding as "a personal deprivation and attack on herself by the baby." This distorted perception is explained as a protective identification in a stressed person with an abnormal coping mechanism. In the case described by [Palmer and Yoshimura \(1984\)](#), the mother herself had Munchausen syndrome and incorporated her daughter as an integral part of herself. In this way the child experienced both the love and self-hate of the mother.

### Effect of the Medical Care System

Important components in the development of the syndrome are the behavior of the doctors, the hospital environment, and the effect that both have on the mother. In the report by [Nicol and Eccles \(1985\)](#), the mother reported that "she found, in her general practitioner, a source of support and kindness and this reinforced the pattern of very regular attendance at the surgery." [Guandolo \(1985\)](#) has commented that "illness is the ticket of admission to a place where understanding and caring



relieved the feelings of hopelessness and isolation." The interaction, however, is more complex than just the mothers' feelings of being supported. With the increasing severity of illness of their children, these mothers feel a sense of self-worth and importance that is otherwise lacking in their lives. One mother, after confessing to repeatedly suffocating her child, reported that whenever she did it, she experienced a similar feeling to that which she had felt on her graduation day. This mother, during one of the times her child was being resuscitated in the hospital, was overheard by a nurse calmly telling other mothers on the ward that she had already had a child die from a similar episode. This, in fact, was a fabrication but serves to illustrate the mother's need to make the situation even direr so as to gain greater sympathy and appear the more heroic. The hospital environment contributes to these feelings of importance. [Chan and associates \(1986\)](#) described a mother who would visit the intensive care unit just to talk to other mothers, and [Meadow \(1984\)](#) has described another mother who had some nursing training and who would help teach nursing students. There obviously becomes an increasing need for the mother to gain admission to the hospital through her child's illness.

Another factor is the pleasure gained from the contact with the doctors. In the case discussed by [Nicol and Eccles \(1985\)](#), the mother reported that she liked to feel that she "was being considered by intelligent people." There appears to be an additional element in which the mother gains pleasure from outwitting the doctors. It becomes a bizarre game in which the mother matches herself against the specialists, and as one problem is resolved, another one is created.

### Associated Psychopathology

As [Meadow \(1985\)](#) has stated, "Many mothers who have perpetrated Munchausen syndrome by proxy have been referred to psychiatrists, and many have had detailed psychological testing. Usually the tests are normal and no disorder is apparent to the psychiatrist." This likely speaks to the misdirected focus of these evaluations rather than to a true lack of psychopathology. In the follow-up study of 47 mothers conducted by [Bools and coworkers \(1994\)](#), 55% had a history of harming themselves, 21% had a history of abuse of alcohol or drugs (usually prescribed medications), and 72% had a history of somatoform or factitious disorders. For the 19 cases in which there were detailed interviews and completion of the Personality Assessment Schedule, 17 of the 19 were considered to have personality disorders. Many of these had multiple disorders, however; histrionic and borderline personality disorders were predominant. Other disorders included avoidant, dependent, narcissistic, schizotypal, and paranoid categories. However, the cases evaluated in this study likely represent a biased sample with more severe symptomatology, and it is often true that there is sometimes a surprising lack of associated psychiatric symptoms, considering the severity of the nature of the disorder.

Some of the case reports serve to demonstrate a spectrum of illness rather than a universal picture. [Chan and associates \(1986\)](#) described a mother who demonstrated several features consistent with both narcissistic and borderline personality disorders. She was explosive, constantly sought attention, displayed sadomasochistic tendencies, showed marked shifts in attitude and affect, and presented a sense of entitlement. Her primary defenses seemed to be denial and splitting. In the report by [Nicol and Eccles \(1985\)](#) of a mother's progress in psychotherapy, they describe an important part of the pathogenesis of the abuse as being the mother's affect, which included infantile rage as a central component together with devastatingly low self-esteem. In the report by Palmer and Yoshimura of a mother who herself had Munchausen syndrome, psychological testing

revealed a profoundly needy individual whose reality testing was impaired. She perceived the world as a malevolent place and people as attacking. Thought processes were distorted and interpersonal boundaries were blurred. Coping strategies included extreme forms of denial, projection and paranoid vigilance ([Palmer and Yoshimura, 1984](#), p. 506).

The fabrication of illness and often-continued denial do not have the fixed quality of a delusion, although the mother does not appear to be consciously lying ([Waller, 1983](#)). It has been described as "quasidelusional" and Waller has commented that

the disturbance in thought content and behavior may be a dissociative phenomenon or a form of pseudologia phantastica or pathological lying in which the parent comes to believe, at least intermittently, the fantasy that the child has a primary rather than a factitious illness ([Waller, 1983](#), p. 83).

Mothers who have confessed to the perpetration of injury on their children have been able to describe the incident but have little recollection for the details and describe themselves as committing the act in a disassociated-like state.

### PSYCHIATRIC DESCRIPTION OF THE CHILD

There is little in the literature describing the children in this disorder. The one striking comment, however, is that the children, particularly older children, collude with their mothers in the ongoing deception. In the original report of [Sneed and Bell \(1976\)](#), it was the 10-year-old boy who was presenting the pebbles as renal calculi. A 2½-year-old girl at our institution did not cry out when her mother, in what must have been a painful process, produced signs of gastrointestinal bleeding by excoriating her anal canal. Furthermore, these children, like children who have been repeatedly physically abused, quickly learn to passively tolerate medical procedures. Other symptoms that have been described include feeding disorders among infants and toddlers and withdrawn, hyperactive, or oppositional behavior among preschoolers ([McGuire and Feldman, 1989](#)).

### SPECTRUM OF THE DISORDER

[Meadow \(1985\)](#), in reporting on what he terms mild cases, raises questions regarding the limits of the definition of the disorder. He points out that, at times, parents often exaggerate their children's symptoms or may perceive that a problem is present when it is not apparent to the doctor. This is particularly true for some parents who consider their children allergic and limit their exposure to various foods and things in the environment. Certainly some of these parents share a number of the same characteristics as those more severe cases of parents who actually cause their children to be ill ([Warner and Hathaway, 1984](#)).

It is also important to ask the question of when Munchausen syndrome by proxy begins in an individual case, recognizing that some cases start with parental concerns surrounding a real illness and that these concerns, at some stage, overflow into fabricated illness. Also, is it only the lack of the medical knowledge that prevents others from developing Munchausen syndrome by proxy? In fact, the syndrome has been described as only one end of a spectrum of parental behaviors surrounding chronic and factitious illnesses of children ([Eminson and Postlethwaite, 1992](#); [Krener and Adelman, 1988](#); [Libow and Schreier, 1986](#)). It is important, however, that we recognize the significant differences in the psychopathology of the parent who injures her child or causes her child to appear ill and the parent who repeatedly presents as overly concerned about her child's illness. What appears to set Munchausen syndrome by proxy apart is the synergistic effect of the mother's prior experience of abuse or rejection, her pathologic symbiotic relationship with her child, and the powerful rewarding effects of her interaction with the medical environment. [McKinlay \(1986\)](#) describes the parent "who provokes sickness behavior in the child, refuses to accept psychological mechanism, seeks multiple opinions and insists on repeated investigations," and points out that this type of parent is often combative or has a contemptuous style, which is very different from the exemplary, ingratiating parental style seen in Munchausen syndrome by proxy. The former does not appear to experience the elevation of self-worth and feeling of importance that seem so much a part of Munchausen syndrome by proxy. There is, however, a striking similarity between these cases and Munchausen syndrome by proxy in the symbiotic relationship between mother and child and the way in which the child colludes with the mother in continuing the illness ([Woollcott et al., 1982](#)).

In DSM-IV, factitious disorder by proxy has been identified as requiring further study, with the following criteria suggested:

1. There is intentional production or feigning of physical or psychological signs or symptoms in another person who is under the individual's care.
2. The motivation for the perpetrator's behavior is to assume the sick role by proxy.
3. External incentives for the behavior (e.g., economic gain) are absent.
4. The behavior is not accounted for by another mental disorder.

Such criteria would limit the use of the term to cases in which the perpetrator's behavior is motivated by the need to assume the sick role by proxy, which would serve to exclude a number of situations in which the term Munchausen syndrome by proxy is presently inappropriately used, such as with parents who are overanxious or "doctor-shopping" or seek opinions from multiple doctors for other reasons ([Meadow, 1995](#)). However, at present, there continues to be some controversy regarding the spectrum of the disorder and the use of either of the terms Munchausen syndrome by proxy or factitious illness by proxy ([Bools, 1996](#); [Fisher and Mitchell, 1995](#)).

### TREATMENT

The management of Munchausen syndrome by proxy often is extremely difficult. There are a number of reasons why this is true: First, there is the difficulty of making the diagnosis—it often goes unsuspected for a long time, and then, even when suspected, it is often difficult to be sure that one's suspicions are in fact correct.

Second, the disbelief that the diagnosis engenders often serves to sabotage overall management. Last, psychotherapy is extremely difficult when the therapist is reliant on the patient telling the truth, which is something that rarely happens in these cases, at least initially.

## Medical Management

The warning signals that should alert a physician to the possibility of a factitious illness have previously been identified by [Meadow \(1982\)](#) and are shown in [Table 102.1](#). Once it is suspected that an illness may be fabricated, the physician needs to establish the diagnosis with certainty. This is often an arduous and time-consuming task. Obviously, the safety of the child and protection from further harm are of utmost importance during this time.

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Persistent or recurrent illness that cannot be explained  
Discrepancies between the history, clinical findings, and general health of the child  
Wrong diagnosis is a rare disorder, or experienced clinicians have "never seen a case like it before"  
Symptoms and signs occur only in the mother's presence  
A mother who is extremely attentive and always in the hospital  
A child who is frequently intolerant to treatments  
A mother who appears less worried about her child's illness than is the medical staff  
Seizures that do not respond to appropriate therapy  
Families in which sudden unexplained infant death has occurred  
A mother with previous medical or nursing experience or who has an extensive history of illness

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Adapted from Meadow R: Munchausen syndrome by proxy. *Arch Dis Child* 57:92-98, 1982.

**Table 102.1. Warning Signals of Munchausen Syndrome by Proxy**

The physician should review the medical history in detail and distinguish those complaints that may have been fabricated from those that are definitely real; complaints that occurred when only the mother was present need to be separated from those witnessed by others; details of the medical, psychiatric, personal, and social history, as presented by the mother, need to be verified. This may require careful and detailed histories from other family members.

Detailed descriptions of the mother's behavior and incidents that occurred while in the hospital may provide a profile consistent with Munchausen syndrome by proxy. A number of laboratory methods have been used to confirm fabricated symptoms. These include biochemical analyses of blood and urine samples, typing of blood to determine if it is the child's or the mother's, and analyzing recordings from apnea monitors. Continuous observation of the mother by nursing staff is extremely difficult and usually not possible. Also, it is often not possible to exclude the parents from the hospital for a long enough time to establish a temporal association between the symptoms and the presence of the mother. Video surveillance of the mother and child in the hospital has also been carried out. This, like searching a mother's personal belongings, creates ethical and legal problems. However, these need to be weighed against the risks to the child, and if there is no recourse, video surveillance may need to be carried out ([Meadow, 1987](#); [Williams and Bevan, 1988](#)).

Once the diagnosis has been established, the mother needs to be confronted and informed of the doctor's knowledge about what is going on and the course of action that needs to be taken to ensure the safety of the child. Other family members also need to be notified and the diagnosis explained to them. The protective services agency needs to be notified, and legal services need to be involved because in most instances the child has been abused and needs continued protection.

## The Role of the Psychiatrist

Although the early literature seldom commented on the role of the psychiatrist in the management of Munchausen syndrome by proxy, more recent reports of cases being successfully managed with psychiatric approaches suggest that child psychiatrists have a very important role to play in ensuring an optimal outcome for these children and parents. One difficulty is that, although it is a psychiatric disorder, the clinical presentation is medical, and often the apparent lack of psychiatric symptoms precludes a reason for the psychiatrist's involvement. Once the diagnosis is suspected, however, the child psychiatrist's contribution can be very important in a number of different ways.

### HELPING TO ESTABLISH THE DIAGNOSIS

Even though the mother may not herself request psychiatric help, once the diagnosis is suspected, a reason should be sought for the child psychiatrist to meet with the mother. Because care needs to be taken not to sabotage the overall management plan by giving the mother warning that she is under suspicion, the reason might be that the psychiatrist can often be helpful to families of children with chronic illnesses. The history taken at this time should include family, social, and psychiatric information, but the most important focus, and that likely to be most acceptable to the mother, is around the child's illness. In reviewing the details of the child's life and illness, the child psychiatrist may be able to obtain an understanding of the special meaning of this child to the mother and the symbiotic relationship between mother and child. An example of this is a mother's description of how much more worried she had been about her daughter (the child now presenting with illness) than she had been about her older child, a son, although both had been on home monitoring because of concern about apnea (see the [Case Illustration](#)). When the daughter, who was named after the mother, was 8 weeks old, the mother heard of another child who had died of sudden infant death syndrome. Since that time, she had feared that her daughter would die and was compulsively careful about the apnea monitor in a way that she described as being very different from when her son was on the monitor. With her son, she had often turned the monitor around so that she could not see all the flashing digital lights and alarms; with her daughter it was intolerable for her not to see these. It is the ability to elicit this quality of the relationship between the mother and child that might be most helpful in the interview. The child psychiatrist, with his or her training and experience in understanding the relationship between mother and child, is likely to be more helpful in assessing this quality of the relationship than would someone without training in child psychiatry ([Eminson and Postlethwaite, 1992](#); [Meadow, 1985](#)). During the assessment, the child psychiatrist also may be able to elicit other information that provides evidence of fabrication or is indicative of other psychiatric symptoms associated with Munchausen syndrome by proxy. The child psychiatrist also should be able to provide an assessment of the degree of psychological disturbance experienced by the child and should be key in planning how this should be addressed.

### DEVELOPING A THERAPEUTIC RELATIONSHIP

A potentially important part of the early assessment is to provide an opportunity for the mother to start to develop a therapeutic relationship with the psychiatrist. Because in most of the cases reported in the literature the psychiatrist has only been involved once the mother has been confronted, it is unclear whether an earlier meeting, prior to the confrontation, might be beneficial, or whether the mother's later realization that the psychiatrist was complicit in the suspicion of her might be detrimental to ongoing treatment. It seems likely, however, that the mother could accept reassurance that the psychiatrist is intent on helping her and that she could therefore maintain the trust necessary for ongoing therapy. Therefore, it may be important for the psychiatrist doing the early assessment to continue to provide ongoing treatment, although this is not always possible in some centers.

### CONFRONTATION

The aim of the confrontation is to explain to the mother that the clinicians know she is harming her child and the possible consequent effects. She should be informed of the steps that will be taken to protect her child and provide help for her and her family. [Meadow \(1985\)](#) has suggested that the pediatrician alone, without other professionals or family members present, best confronts the mother. Although this may be true for someone like Meadow, who has such a vast experience with this syndrome, it may not necessarily be true in instances where the pediatrician has had no prior experience of Munchausen syndrome by proxy. In these cases, it may be helpful to have a child psychiatrist present to help in the process. It is important, however, that the pediatrician be the one who confidently affirms what has been going on. The child psychiatrist then is able to speak of it as a psychiatric problem and outlines what is likely to happen, at the same time being supportive.

At this stage, only a few mothers will confess to what they have been doing, although at times there are remarks that serve as tacit admission of the activity. The majority of mothers continue to deny their activity, often in a very convincing way, although sometimes in a remarkably calm, flat manner. It is not helpful to try to prove to the mother that you are right and she is wrong, nor is it helpful to counter every explanation she provides.



Some mothers have become extremely agitated, acutely psychotic, or depressed and suicidal following the confrontation ( [Palmer and Yoshimura, 1984](#)). An assessment may need to be made regarding the need for psychiatric hospitalization. Needless to say, it is critically important that the child be protected from the mother at this time, because she may have a heightened need for the child to be truly sick.

#### ASSISTING STAFF IN UNDERSTANDING THE DYNAMICS OF THE CASE

This may be an extremely difficult problem to deal with for medical and social services staff, particularly if they have had no prior experience with or knowledge of the syndrome. The emotional responses of the staff are made more complicated by the fact that some might have known the family for a long time and developed close relationships with the mother and child. For some people, there is complete disbelief; for others, there is anger at the mother and feelings of guilt that they have participated in harming the child or have not been more astute in correctly identifying the problem at an earlier time. Group discussions are helpful in both informing staff about the syndrome and providing them an opportunity to express their feelings. It is also helpful to provide written material about the syndrome for staff to read.

The psychiatrist also may play an important role in providing protective services workers and lawyers with an understanding of the syndrome and may even appear as an expert witness in the courts. In this way, the psychiatrist can help to facilitate an appropriate legal course of action. The psychiatrist's role in this may have important ramifications in later treatment: The assertion of the severity of the disorder and the danger to the child, together with an uncritical view of this as a psychiatric disorder, may have therapeutic implications for the mother.

#### PSYCHOTHERAPY

As noted, there is limited information available in the literature concerning psychotherapy of mothers and children with Munchausen syndrome by proxy. Palmer and Yoshimura have emphasized that the following are important determinants of how a case should be managed:

the degree and duration of abuse; the parent's psychological state; whether the reaction to the confrontation is denial or acknowledgment of behavior; resistance or willingness to engage in treatment; and whether the child is perceived as part of herself or as a separate entity ( [Palmer and Yoshimura, 1984](#), p. 507).

Obviously, each of these factors might be an important indicator of the accessibility of the individual to psychotherapeutic intervention, but an adverse factor should not necessarily be considered a contraindication to therapy.

Nicol and Eccles have provided details of their case in which the mother initially denied the allegations but later confessed when faced with the possibility that her children might be placed in foster care. The mother, in this case, continued in therapy on a weekly basis for 6 months and then biweekly for a further 6 months. From early on it was "clear that the mother had a strong wish to understand herself, that she was intelligent, and that she had a capacity to bring active and painful feelings to therapy sessions." General themes of therapy included the complex reasons for the abuse, where a full realization of the danger she had put her child in gradually emerged. Other important themes were her relationship with her parents and fears about her child's health that first originated during her pregnancy.

In a report describing outcomes for 13 cases receiving psychiatric intervention, Berg and Jones described an inpatient treatment approach provided in the family unit of a children's hospital by a multidisciplinary psychiatric team experienced in management of child maltreatment. "The theoretical orientation of the team is founded on principles of infant-parent attachment theory" and treatment included

psychological interventions targeted at: the parent-child relationship, the quality of the child's attachment to each parent, the abuser's own childhood experiences, and the current social network and family dynamics, together with work with the parental couple ( [Berg and Jones, 1999](#), p. 466).

The length of stay in the family unit varied between 3 days and 4 months with the average duration being 7½ weeks.

#### OUTCOME

The final outcome for children with Munchausen syndrome by proxy is very variable and dependent both on the severity of the disorder and treatment provided. The mother who has other psychiatric disorders in addition to Munchausen syndrome by proxy and the child who has been involved in the fabrication of symptoms for many years are more difficult to treat than those with a simpler presentation. This is likely true for those mothers who have Munchausen syndrome themselves, which is often very difficult to treat successfully ( [Mayo and Haggerty, 1984](#)). The child whose symptom of illness has been caused by a more dangerous activity (e.g., suffocation) is obviously more at risk of dying than the child whose symptom was owing to less dangerous methods (e.g., a mother's putting blood in her child's urine). In [Rosenberg's \(1987\)](#) review of the 117 cases reported in the literature, 9% died and 8% of the survivors had permanent disfigurement or impairment of physical function. The leading causes of death were suffocation and poisoning. Children have died even after the diagnosis was made and their mothers confronted, and younger siblings have been abused after older siblings died.

The psychiatric sequelae have been less well described both for the mother and child. Certainly, some of these children have continued to fabricate illness for themselves, and the child with Munchausen syndrome by proxy has grown up to be the adolescent or adult with Munchausen syndrome ( [Bools et al., 1993](#); [Meadow, 1985](#)). Of the 12 children described by [McGuire and Feldman \(1989\)](#), 11 were described as having adverse effects that included immaturity, abnormal relationships with their mothers, separation problems, and aggressive behavior. Some children have expressed fears of poisoning and death, and at least two children have required psychiatric hospitalization ( [Rogers et al., 1976](#)).

In the most comprehensive follow-up study reported to date, [Bools and colleagues \(1993\)](#) described 54 children for whom they obtained information an average of 5.6 years after the event. Thirty of these children continued to live with their biologic mothers, whereas 24 children were with other family members or in substitute care. For 10 of the 30 children living with their mothers there was evidence of further fabrications, and for another eight there were "other concerns," either about the relationship between the mothers and their children, or about other aspects of the mothers' behaviors. Of these 18 children for whom there were continuing concerns about the family, 12 exhibited psychological symptoms, including somatic complaints, emotional disorders, conduct disorder, and poor functioning at school. Among the 24 children who were no longer living with their biologic mothers, eight had persistent psychological symptoms, and another six children had disorders that had shown signs of gradual improvement. Altogether, the authors of the study concluded that half of the children had outcomes that they considered to be unacceptable, but, because of the variability in cases, it was not possible to comment on whether better outcomes were obtained when children remained with their mothers or were separated. However, the authors did conclude that when children remained with their mothers, the outcome appeared improved when there had been a temporary placement in foster care.

In reporting on the follow-up of cases enrolled in the intensive, inpatient treatment program described above, [Berg and Jones \(1999\)](#) reported greater success. When 16 families were reevaluated an average of 27 months after treatment, there were no ongoing concerns for nine of the families; the mothers had no mental health problems and had insight into their original condition, and the children had no psychological disorders. There continued to be mild concerns for five families in which the mothers continued to have mild to moderate mental health problems, although these did not impact on their relationship with their children or on the children's development. In only two families were there more serious concerns about the mother's mental health, although they were not overtly abusive. However, the authors stressed that these cases had been selected for treatment on their likelihood of achieving success and that cases considered unsuitable for psychiatric treatment were excluded, usually because of persistent parental denial or the severity of the parent's personality disorder.

#### CONCLUSION

In this condition, perhaps more so than any other, there needs to be extensive collaboration between the child psychiatrist and pediatrician, as well as with the other services involved ( [Bentovim, 1985](#); [Eminson and Postlethwaite, 1992](#); [Meadow, 1985](#)). The role of the psychiatrist may be very different from that with other psychiatric diagnoses, particularly when a case is first diagnosed, and although it is recognized as a psychiatric disorder, it needs also to be remembered that it is a serious form of child abuse. It is hoped that with our increased understanding of this syndrome and an appropriate psychiatric approach to management, there will be an improvement in outcome for both the children and their mothers.

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## CASE ILLUSTRATION

A 10-month-old girl was admitted to the hospital for the fourth time over a 3-month period. Each admission was for treatment of unilateral periorbital swelling and inflammation. The presumed diagnosis was recurrent periorbital cellulitis, and on each admission she was treated with intravenous antibiotics. While on treatment, her eye would sometimes improve, and then inexplicably become worse. Numerous consultations and diagnostic studies were carried out. Corneal ulcers were noted on the final admission. Suspicions had already been aroused, and at this time the pH of the conjunctival fluid was tested and found to be more alkaline than that of the other eye. A diagnosis of Munchausen syndrome by proxy was made.

This child was the younger sibling of a 3-year-old boy who had had bacterial meningitis in the neonatal period and had developed apnea when 8 weeks old. He was started on apnea monitoring at home and then, at the age of 11 months, started having seizures. The index child was also reported to have apnea starting at 8 weeks of age. She was monitored at home and had a number of significant apneic episodes. Three of these episodes occurred in the hospital: At the start of each occasion the mother was alone with the child, and when called, the medical staff found the child cyanosed and with gasping respirations. Later, once the correct diagnosis was realized, a recording stored in memory in the apnea monitor was reviewed and showed that prior to the last episode of apnea, the monitor had been switched off for 2 minutes and that when it was switched on again, the child was apneic. The mother had been the only one present during this interval, and there was no reasonable explanation for why the monitor had been switched off.

A child psychiatrist experienced in Munchausen syndrome by proxy was consulted, and the mother was confronted with both the pediatrician and psychiatrist present. She appeared extremely upset and denied all allegations. Her husband and other family members were disbelieving but agreed to the plan of management "in case it were true." The management included referral for ongoing psychotherapy and initiation of plans with the protective service agency to ensure continued protection of the children. Because placement of the children with members of the extended family was a consideration, a family meeting was called to ensure that everyone believed what had happened and understood the seriousness of the disorder. The mother confessed to what she had done prior to this meeting. She sought help from her psychiatrist and, at the meeting, was able to talk about her problem and ask for help from her family. It was decided at the court hearing that the children should be placed in the custody of their father and paternal grandparents, with the mother to have supervised visitation and to continue in psychotherapy.

### Comment

In this instance, the mother shared many characteristics described in cases of Munchausen syndrome by proxy. She had been abused as a child, her parents were divorced, and she had little contact with her father. Her relationship with her husband was described as good, but he had three jobs and was seldom at home. She herself had not had frequent illnesses, nor did she have nursing training, but she had become acquainted with medical care and technology with her first child. She considered her second child to be very special and was particularly concerned that she might die. She was extremely attentive to her child, stayed in the hospital constantly, and was very friendly with the medical staff.

In this case, the mother developed a trusting relationship with the child psychiatrist right from the time when she was most distressed, following the confrontation. The family meeting was extremely helpful in ensuring the family's continued attention to the safety of the children and support for the mother. It should be noted, however, that in some respects this case was less complex than many and less difficult to manage.

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# 103 CHRONIC PEDIATRIC ILLNESS AND MULTIPLE HOSPITALIZATIONS

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More than 3 million children in the United States are estimated to suffer from some type of chronic illness ( [Schoenborn and Marano, 1988](#)). Given the scope and pervasiveness of pediatric problems, the occurrence of a serious physical illness is increasingly understood to represent a major early risk factor for subsequent emotional disturbance ( [Table 103.1](#)). However, it has been difficult to develop a national intervention strategy. One factor that may have impeded progress in this area has been the wide variability in the results of outcome studies designed to demonstrate the association between the occurrence of early illnesses and subsequent problems in emotional development. The range of adaptation to physical illness is broad. Some children seem to thrive heroically despite long periods of difficult symptoms, prolonged and painful treatments, and interruptions in school and peer experiences. This developmental variability in response to early stressors is actually typical of children's response to many risk factors and is well demonstrated by the substantial range of responses that children demonstrate to major life events such as the divorce of their parents ( [Hetherington and Stanley-Hagan, 1999](#)).

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Onset	Particularly difficult developmental periods include early childhood (6 mo-5 yr) and early adolescence.
Etiology	Illness that are the result of some environmental exposure (trauma or infection) or inherited genes provide specific stressors on the family.
Diagnosis	Prolonged delay in diagnosis or misdiagnosis has a negative impact on the family-doctor relationship.
Deformity	Physical deformity and disability negatively influence both the development of self-image and availability of support.
Prognosis	The more negative the prognosis, the greater is the risk posed to adaptive emotional development.

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**Table 103.1. Illness-Related Risk Factors**

Two contrasting approaches can be used to develop a conceptual understanding of the emotional impact of medical illnesses on children. The first strategy is to look for typical responses that occur in children with a range of different types of medical diseases. Using this approach, investigators have described a set of characteristics typical of most children with persistent severe illnesses. Two commonly discussed concerns are problems with self-esteem and the maintenance of supportive peer relationships. However, these difficulties are also present in physically healthy children who grow up in highly stressful environments. Such problems in the formation of identity and relationships are probably better considered to be generic sequelae of early adversity rather than the consequence of a specific risk factor. This approach provides a unifying conceptual model that can be useful politically to advocate for all children at risk. However, it does not provide guidance for the clinician who is faced with the challenge of helping a particular child with a specific chronic disease.

The second approach is to define the unique characteristics of each chronic severe illness. The goal is to identify a specific set of physical symptoms associated with the illness that places children at risk for problems in adaptation. Such a perspective is best organized by considering children within their family and social contexts. This can be conceptualized as defining the mediating variables that modify the child's development. This approach requires an analysis of the interactions of illness-related risk factors, familial risk and protective factors, and other environmental risk and protective factors. This approach also emphasizes strengthening protective influences as an essential aspect of the treatment of the child, to maximize the potential therapeutic impact of the child's family and community. Risk and protective factor analysis has long been used to understand the factors relevant for the development of psychopathology in physically well children ( [Rutter and Garmezy, 1983](#)). This chapter focuses primarily on chronic illness-related risk and chronic illness-related protective factors within the broader context of the family and the community.

## ILLNESS-RELATED RISK FACTORS

Illness-related risk factors are the characteristics of specific chronic diseases that place children at increased risk of emotional disturbance. Physically ill children are not immune to the negative effects of poverty, poor parenting, parental divorce, or overt neglect and abuse. In addition to these and other risk factors, they must cope with hardships imposed on them by their physical symptoms, limitations of function, and disruptive and painful treatments. On a more positive note, they may benefit from illness-related protective factors, such as the support provided by sensitive physicians and nursing staff. These beneficial influences can be further potentiated by protective influences that are not related to illness, such as secure parental attachments and strong family cohesiveness. Many of the more recent innovations in pediatric treatment represent institutionalization of illness-related protective factors. Familiar examples include the systematic preparation of children for difficult medical procedures and the encouragement of parental support and presence throughout the hospitalization of their children.

When a family learns that a child has a serious chronic illness, it does not mean that the child will inevitably become depressed, lonely, or feel socially rejected. Chronic illness should be viewed as a condition characterized by a set of serious challenges that can either be overcome or lead to a sense of emotional defeat. Every pediatrician can provide an anecdote of a particularly competent child who faced dreadful illnesses with great courage. Similarly, pediatricians can describe the sense of accomplishment that some families achieve when they successfully manage the symptoms of their seriously ill child. These stories of victories are retold with pride, because the success of these patients also reflects the skill and support of their committed doctors. It is more difficult to remember the family that falls apart or the child who stops trying. Helping children who cannot overcome the emotional traumas associated with a serious medical illness often becomes the central role of the child psychiatrist in a pediatric setting. The need for psychiatric consultation becomes even more evident when illness-related risk factors begin to contribute to the development of overt psychopathology in either the child or the parents.

### Impact of Time of Onset

The point at which an illness begins has a differential impact on the effect of a chronic illness. A guiding therapeutic principle has been that the painful and frightening symptoms of chronic illnesses are most difficult for young children to manage. This is, in part, because young children lack both the ability to understand the concept



of illness fully and the rationale for required treatments. The exception to this principle is the experience of very young infants. During the first months of life, immature central nervous system organization may contribute to actual amnesia of the traumatic aspects of the illness. A review of the cognitive appraisal of illness supports the general principle by demonstrating a link between cognitive development and the capacity to cope with medical procedures ( [Brewster, 1982](#)).

However, the onset of a serious illness during adolescence can have stressful consequences as a result of quite different developmental considerations. Body image, personal identity formation, and peer acceptance are major issues for the young teenager, and the onset of a chronic illness during these years can dramatically disrupt the normal process of self-development. For example, a child may be able to deal with aspects of an illness quite well in the elementary school years, but with the onset of puberty and a strong desire to establish a sexual relationship, an adolescent may find rejection in early romantic explorations very upsetting. Depressive symptoms clearly increase with the onset of adolescence, and management of suicidal ideation and actual suicide risk must become a more central concern in the care of teenage patients.

### **Nature of the Etiology of the Illness**

It is critical to develop an appreciation of how a particular child understands his or her illness. Similarly, the belief system of the child's parents must be appreciated. Both rational and irrational elements are important to understand. Genetic illnesses provide a special challenge, because there is an element of rational responsibility that must be considered. In many cases, an illness is inherited from one parent. Although passing a problematic polymorphism onto a child may occur with no appreciation of the risk, it is common to feel remorse when a recognized family illness appears in the next generation. As it becomes more common for parents to have a far greater understanding of the negative polymorphisms that they carry, the issue of dealing with parental responsibility will become a more critical aspect of helping families to cope with the consequences of decisions. Such interactions will be particularly challenging when there has not been full disclosure of probable genetic risk between the two parents.

When an illness is caused by an infection, there is an even clearer link between potential parental behavior and outcome. If the illness could have been prevented by recommended immunizations, the parents have clearly made a decision that placed their child at increased risk. The occurrence of traumatic injury is perhaps the most difficult, particularly with young children. If an element of neglect is associated with an injury, particularly in a child who is already compromised by being chronically ill, the negative impact on the parental relationship with the child can be very serious.

In contrast, diseases that are believed to be "an act of God" provide much less direct interpersonal stress for the patient and family. In these cases, there is no one to blame, and the "reason" for the illness is accepted to be beyond the understanding of the family. However, even in the presence of superficial acceptance of the illness, a period of grief and depression often emerges as the family begins to try to understand how such a dreadful outcome as a life-threatening illness could actually be part of a larger plan. Allowing families to work through the loss of their hopes for the future of a healthy child is a critical component of adjusting to the many illness-related stressors that complicate the treatment of severe illnesses.

Regardless of the cause of the illness, the therapeutic task ultimately requires a shift away from a preoccupation with concern about *why* the illness has emerged. It must move forward to a determination of *what* treatment should be initiated and *how* the family can adapt to the changes that the illness will inevitably bring to their lives. A special consideration occurs when the origin of the illness is clearly genetic and there is a quantifiable risk for future children in the family. First the pediatrician or a genetic counselor working as part of a treatment team must explain to the parents the risk for future children. Once the family can appreciate this risk, a child psychiatrist can play an important role in helping families to integrate the meaning of this information realistically and to make well-informed decisions based on the risks involved in having additional children.

### **Certainty of Diagnosis**

Once the diagnosis is established with certainty, the child and family can begin the process of coming to grips with whatever life changes are required to manage the illness. When a long period of uncertainty follows the onset of physical symptoms, various negative processes emerge that can disrupt the doctor-family relationship. Among the most difficult is the situation that arises when the diagnosis is unnecessarily delayed as a result of error or confusion in communication. If it is eventually determined that a specific treatment could have been provided earlier in the course of the illness that would have had a positive impact on the patient's long-term prognosis, the possibility of malpractice suit should not be minimized.

As a general rule, dealing with a defined problem is less difficult than coping with the unknown. This principle often provides guidance to the medical team members, who may be overtly uncomfortable with the painful news of the diagnosis of a chronic illness and may be tempted to delay the actual naming of the disease for months. This avoidance is most common in the case of a terminal illness. In many cases, both the parents and the pediatrician may collude to avoid sharing the grim prognosis with a young child. In such cases, it is not unusual to make the suggestion that the child psychiatrist should be consulted with the explicit expectation that he or she be the one to explain to the child that recovery is unlikely. The consultation is usually prompted by the rationale that the psychiatrist is particularly skillful in speaking directly with children and is trained to understand the nature of their emotional responses. This may ultimately be the only possible way to proceed, and such a strategy is clearly superior to a prolonged pretense during which time the child must try to make sense of the disparity between what he or she is told about the illness and the manner in which the people most important to the child are responding to him or her. Whenever possible, it is more productive for the child psychiatrist to play the role of the facilitator in a session that includes the family and the pediatrician. The goal of this effort is to establish an openness about the prognosis of the child so the child can begin to understand the emotional communications of those around him or her. Ultimately, the objective is to ensure that the parents and pediatrician will be able to be aware of the wishes of the child as they make difficult treatment decisions.

### **Degree of Deformity and Disability**

The presence of physical deformity and functional disability will clearly increase the negative impact of chronic illness. These illnesses are usually associated with some degree of limitation. The first principle of the treatment team is to determine how to help the pediatric patient to address these issues, to minimize negative effects on emotional development. Cumulative evidence suggests that the degree of impairment is directly associated with increased psychopathology.

A different set of considerations is raised when a major deformity in body image is the result of the treatment rather than the disease. Although a major physical deformity is a difficult issue throughout development, the time of greatest vulnerability occurs during early and middle adolescence. The treatment goal should not be to deny the impact of the deformity, but rather to provide critical emotional reassurance that, despite disfigurement, the child is still a valued part of the family and community. In this circumstance, actions are clearly more important than verbal remedies. The child must feel that he or she is included in the life of the family in a substantive way if reassurances are to be at all effective. A particularly useful strategy to normalize the child's experience is to encourage a peer group experience with children with similar deformities who have made an adequate adjustment. One of the therapeutic aspects of these experiences is that peers with similar deformities are highly credible in that they are dealing with similar struggles.

### **Prognosis and Course of the Illness**

A child must learn to live with a chronic illness. When a reasonable hope exists that the illness will either remit completely or eventually provide little disability, an optimistic stance by the medical team and family is highly adaptive. A similar approach is indicated when the illness falls in the category described as the *Damocles syndrome* ([Koocher and O'Malley, 1981](#)). In these cases, a dreadful outcome is possible, but the best strategy is still to assume an optimistic stance, because remission is a realistic possibility. In contrast, terminal illnesses provide a set of specific challenges to both the family and the treatment team. After achieving the first therapeutic challenge, open acknowledgment of the impending death of the child, the treatment team must begin a long process of helping the child and family to come to grips with stages of the illness. Denial is a common defense and must be dealt with sensitively. There are few tragedies more difficult for a family than the impending death of a child. Parents always need time to find ways to mobilize family supports and to reorganize their future hopes and expectations. Staying with them through the process is the central goal of the final stages of support.

## **SEVERE CHRONIC ILLNESSES**

Although many chronic illnesses exist, the most common serious diseases and their estimated prevalence are listed in [Table 103.2](#). Less serious illnesses occur frequently during childhood, but these are for the most part self-limiting problems that are rarely associated with severe emotional sequelae that require child psychiatric intervention. Five quite distinct chronic illnesses are reviewed in this chapter to serve as models of the clinical issues presented by this wide range of pediatric disease. (Further information on some of the illnesses selected may be found in [Chapter 61](#) and [Chapter 95](#).)

Disease	Children Per 1,000
Asthma	62.0
Migraine	15.2
Epilepsy	4.9
Arthritis	1.9
Diabetes	1.2
Cystic fibrosis	0.5
Leukemia	0.15 <sup>a</sup>

<sup>a</sup>Results, except for the prevalence of leukemia, from Adams PE, Hendershot GE, Marano MA: Current Estimates from the National Health Interview Survey: United States, 1996. Vital and Health Statistics, series 10, no. 200, DHEHS pub. no. (PHS) 99-1528. Washington, DC: U.S. Government Printing Office, 1999.  
<sup>b</sup>Leukemia data from Ries LAG, Kosary CL, Hankey BF, et al. (eds): SEER Cancer Statistics Review: 1973-1996. Bethesda, MD, National Cancer Institute, 1999.

**Table 103.2. Prevalence of Serious Chronic Conditions in Children in the United States: 1996.<sup>R</sup>**

## Asthma

Asthma continues to affect more than one in 20 American children and is the most prevalent chronic illness of childhood ( [Myers, 2000](#)). Prevalence rates over a 1-year evaluation in the United States have been reported to be 5.8% of children younger than 5 years of age and 7.4% of children between 5 and 14 years of age ( [Mannino et al., 1998](#)). Incidence estimates in other countries vary widely, reaching a maximum rate of one in 10 in New Zealand. In many ways, asthma is the prototypic psychosomatic illness. Although there is no question that asthma has a demonstrable physical component, both acute and chronic stressors directly affect the management of the illness ( [Mrazek et al., 1999](#)).

*Asthma* is defined as a reversible reactive airway disease triggered by both immunologic and nonimmunologic factors. It is familial, with a significantly increased risk of the disease occurring in first- and second-degree family members. Whereas serum immunoglobulin E may play a role in the expression of symptoms in many patients with asthma ( [Burrows et al., 1989](#)), the disease is heterogeneous in presentation, a finding suggesting that different mechanisms may be prominent in different patients.

The onset of asthma usually occurs quite early in life; approximately 75% of children who ever develop the illness experience it within their first 3 years ( [Falliers, 1970](#)). However, asthma can begin for the first time at any age. This presents an important clinical issue. The panic associated with the rapid onset of intense chest tightness and respiratory distress can be a particularly frightening problem for children younger than 3 years of age. Earlier onset is more difficult to manage, whereas onset during adolescence, relatively unusual, it is easier to adapt to than other chronic illnesses arising during the teenage years. Reasons include the availability of increasingly effective pharmacologic agents to control symptoms and the absence of visible physical symptoms. It has long been recognized that severely asthmatic children have a greater likelihood of anxiety and depressive disorders. In one study of children with persistent asthma who had moderate or severe symptoms, 42% were found to have an Axis I psychiatric illness, with anxiety disorders particularly common ( [Vila et al. 2000](#)).

The origin of asthma is complex and still not completely understood. Despite clear evidence of increased familial risk of developing the illness, twin studies unequivocally demonstrate that environmental factors are necessary for the expression of the disease. Environmental factors that have been considered to be salient include early respiratory infection, difficulties in parenting ( [Mrazek et al., 1995](#)), and excessive exposure to a highly antigenic environment in infants with elevated levels of immunoglobulin E ( [Mrazek et al., 1999](#)). Exposure to cigarette smoke is a nonspecific irritant that may affect the onset of asthma. The wide range of potential factors involved in the expression of the disease complicates the process of attributing causality to specific experiences that have been hypothesized to be associated with its onset. Like all illnesses with a genetic basis, the alleles that a child has at specific gene sites determine the likelihood of disease expression. However, potential therapeutic strategies targeted at familial behavior may well delay or prevent the onset of the symptoms ( [Mrazek, 1999](#)). Evidence examining the links between the quality of parent-child relationships and the occurrence of depressive symptoms in asthmatic children suggests that an ongoing secure relationship may be a protective factor in mediating problems with depressed affect ( [Bleil et al., 2000](#)).

The establishment of the diagnosis can be difficult in the first months of life. In addition, it has been a common practice among some pediatricians consciously to avoid making the diagnosis in the first years of life to avoid "stigmatizing" the child. The rationale for this strategy has included the avoidance of implying that disease may be self-limited. One strategy has been to diagnose wheezing in infancy as "bronchiolitis," even though the attacks may be occurring without a clear concurrent infection. Although it is true that this practice avoids an early confrontation with the potential long-standing nature of the problem, it also may postpone early intensive management of the illness. In short, overly optimistic reassurance that a child may well "grow out of asthma" may inadvertently lead to inadequate treatment of the illness and a worse long-term outcome.

Asthma rarely has any visible stigmata. However, in the 5% to 10% of asthmatic children who become dependent on steroids, striking changes in appearance can occur. These children often suffer from chronic respiratory insufficiency. This may result in the development of a *barrel chest*. In extreme cases, systemic use of steroids can cause cushingoid stigmata and permanent short stature.

The issue of limitation of activity is important for children. A natural parental tendency is to shelter asthmatic children from the rigors of intense exercise. This is clearly inadvisable. [Strunk et al. \(1989\)](#) demonstrate the beneficial impact of improved fitness on the adaptation of asthmatic children. For most children with asthma, an active program of normalization of their activities is the appropriate clinical approach.

The prognosis of asthma is complex. Most children with mild asthma have little risk of a fatal attack and good reason to expect, with proper management, a high probability of remission of symptoms. Consequently, appropriate treatment, which includes pharmacologic, environmental, and psychological interventions, usually results in the control of symptoms. A permanent "cure" or remission is less likely.

The prognosis is less clear for children with steroid-dependent asthma. In these children, the risk of a fatal episode may approach 5%, although more recent evidence suggests that the mortality rates associated with this form of the illness have stabilized ( [Sly and O'Donnell, 1997](#)). These severely asthmatic children must learn to pay close attention to their physiologic state because responding to early warning signs of respiratory distress is often critical, to reach help in time to reverse bronchoconstriction and to avert a respiratory arrest. Prescription of steroids is a complicated issue. Although steroid control is essential in these children, the side effects of prednisone can be disturbing. Negative reactions to steroid treatment are particularly common in young adolescents, who are most self-conscious. Unfortunately, this is the precise developmental period when children seem to be at risk of a fatal episode ( [Strunk et al., 1985](#)). A set of risk factors associated with fatal episodes has been established and essentially creates a profile of children at increased risk. The three most prominent features of this profile are a history of seizures, documented affective disturbance, and demonstrated conflict between the child's family and the medical caregivers.

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### CASE ILLUSTRATION

Sybil was a 12-year-old chronically asthmatic girl with a lifelong history of unremitting asthma. Her symptoms were complicated by serious steroid side effects. She was admitted to an academic medical center to review the management of her symptoms comprehensively. Before this comprehensive evaluation, Sybil experienced little improvement in her symptoms despite more than 40 previous hospital admissions. She had received long-term steroid treatment to control her airway reactivity. This had resulted in severe cushingoid stigmata and retardation of growth, to the extent that her height was that of a 7-year-old child. Her steroid treatment had resulted in the development of scoliosis with respiratory compromise.

Sybil was an extremely intelligent child, with an intelligence quotient of 152. Despite her multiple hospitalizations and many school absences, she was able to score above grade level in basic academic skills. Although her nuclear family was intact, her father had developed chronic pancreatitis secondary to the heavy use of alcohol, and he had become increasingly depressed.

During a 4-month intensive rehabilitation effort, Sybil's steroid dose was dramatically reduced. She received intensive psychotherapy and developed a more realistic perspective on her range of abilities. Concretely, she made a readjustment in personal goals that involved giving up her fantasy of becoming a competitive ice skater. During this time, her depressive affect improved dramatically. Family communication was improved, and her parents were better able to understand her need to individuate from them.

However, Sybil's asthma was acutely sensitive to emotional factors. This was well illustrated after her 4-month rehabilitation, when she developed an episode of acute respiratory distress that was not relieved by oral medication or injections of epinephrine. A plan for an immediate transfer to an intensive care unit for an intravenous aminophylline infusion and the possibility of mechanical ventilation was instituted. While waiting for transfer to the intensive care unit, Sybil requested that her therapist hypnotize her to relieve her intense anxiety. She proved to be a good subject, and hypnosis resulted in a complete remission of her acute respiratory distress within 10 minutes. This remission was persistent, and no intensive care unit admission was required.

On returning to her home, Sybil's course deteriorated dramatically. She became resistant to medical treatment and acutely depressed. Her father's emotional problems became more severe, resulting in intense family conflict. Despite antidepressant medication, Sybil developed intense suicidal ideation. She announced to the psychiatrist at a regular session that she would not see him again because she was going to die before their next meeting. Three days later, she had an acute episode of asthma and died in the back seat of the family car on the way to the emergency room.

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## Epilepsy

Many quite heterogeneous central nervous system disorders can be broadly classified as chronic epilepsy. *Epilepsy* can be defined as a condition characterized by persistent major motor seizures. It affects approximately five children per 1,000. Onset of the seizures can occur throughout childhood, with onset in the first years of life being particularly common. Hereditary transmission is rarely the attributed causality. Unless seizures begin after physical injury, issues of parental responsibility and potential blame are not usually a central consideration. The level of anxiety associated with the diagnosis of epilepsy is clearly linked to how a given family understands the prognosis and cause. Although a single febrile seizure is rarely a cause of persistent concern, focal seizures that suggest central nervous system disease have a major impact on both the child and the family. [Harrison and Taylor \(1976\)](#) examined the long-term outcome of a sample of children with seizures and found a mortality rate of 10.1%. In this sample, 24.2% of the children developed chronic epilepsy. As with many physical illnesses, chronicity and severity of symptoms are among the key factors influencing subsequent deviations in emotional development. Similarly, many different psychosocial stressors have been reported to be associated with the onset or recurrence of seizures. These may include quite discrete events such as the occurrence of sexual abuse ([Greig and Betts, 1992](#)).

The onset of epilepsy in adolescence can be particularly disturbing. A teenager's natural strivings for greater independence can be dramatically interrupted by the uncertainty created by episodic seizures. It has long been demonstrated that children with seizures are at increased risk of emotional disturbance. In the classic Isle of Wight study, nearly 30% of children with uncomplicated epilepsy and almost 60% of children with documented lesions above the brain stem suffered from emotional disturbance ([Rutter et al., 1970](#)). More recent studies report similar rates of psychopathology ([Hoare and Kerley, 1991](#)). These rates have been consistently found to be higher than rates of psychopathology in children with other chronic physical illnesses. The clear inference is that central nervous system damage may often be directly responsible for the onset of behavioral and emotional disturbances ([Howe et al., 1993](#)).

The differential diagnosis of epilepsy can be complex. An initial seizure is often difficult to differentiate from an episode of loss of consciousness that may result from cardiac or functional factors. Confusion in establishing the nature of the episodes can result in both loss of confidence in the medical team and considerable anxiety about the nature of the underlying disorder.

Although epilepsy does not result in persistent physical deformity, the emotional stigmata of an epileptic attack have been well documented. [Taylor \(1987\)](#) suggests that prejudice against persons with epilepsy is in part the result of a publicly perceived association between epilepsy and madness and mental deterioration. There are often real limitations in the ability of an epileptic child to participate in team sports or wilderness activities fully. The subsequent negative impact on self-esteem is frequently compounded by the repeated experience of loss of control. Parents and medical teams need to work together to construct an appropriate strategic plan designed to maximize the opportunities that a child has to develop an appropriate sense of autonomy and independence.

[Hermann et al. \(1990\)](#) adopted a risk factor strategy for predicting the occurrence of psychopathology in persons with epilepsy. More than 100 adults were studied. For most, their disease began during childhood and adolescence. The mean duration of their illness was more than 16 years. Illness-related risk factors included an early onset of symptoms and the perception of being stigmatized. Additionally, experience of embarrassment, disturbances of self-esteem, and the perceived sense of personal rejection were associated with the development of psychiatric symptoms. Other risk factors associated with disturbance were more frequent adverse life events, financial stress, and vocational difficulties. Successful surgical intervention has been reported to be associated with better long-term outcomes ([Seidman-Ripley et al., 1993](#)).

## Cystic Fibrosis

*Cystic fibrosis* is an autosomal recessive illness affecting only about 0.05% of white children and fewer black and Asian children. In white populations, about 5% of adults are carriers of the gene for the disease. The expression of the symptoms of cystic fibrosis occurs almost universally during infancy and involves multiple organ systems. Chronic bronchial airway obstruction often leads to infection. Maldigestion subsequent to pancreatic defects results in unpleasant gastrointestinal symptoms. An alternative name for this illness is mucoviscidosis, which describes the increased viscosity of the secretions that represent the common origin of many of the symptoms. Affected organs include lung, pancreas, liver, intestine, and genitals. The initial presentation of the illness can involve either pulmonary or gastrointestinal symptoms, which can delay the determination of the diagnosis. There has been a dramatic increase in the life span of children with cystic fibrosis. Although previously it had been unusual for children to live to adulthood, the median survival rate has increased beyond 20 years. Sexual development is usually delayed, and 98% of male patients are sterile ([Tausig et al., 1976](#)). Pregnancy is associated with increased risk of deterioration in respiratory function. However, successful pregnancies have been managed within the context of intensive medical support.

Because the diagnosis of cystic fibrosis usually occurs in infancy or early childhood, the child and family must learn early to cope with the related symptoms. Caring for a child with cystic fibrosis requires a major time commitment because of the need for frequent physical therapy to decrease the occurrence of pulmonary complications. [Burke et al. \(1989\)](#) examine the occurrence of obsessive-compulsive symptoms in children with cystic fibrosis and find modestly elevated scores but no overt obsessive-compulsive disorder. Given the demands of cystic fibrosis, obsessive attention to self-care may well be a good coping strategy and may serve a protective function in minimizing disease sequelae.

Adolescents with cystic fibrosis often experience their physical symptoms as intrusive and having a negative impact on peer activities. Problems that typically become more acute during the teenage years include unpleasant flatus, which is associated with maldigestion, anxiety regarding sexual potency, and an increased awareness of coping with a compromised life span. Despite these difficulties, some patients do marry and have reported that their sexual relationships are satisfactory ([Levine and Stern, 1982](#)).

Given that the disease results from a well-described homozygous occurrence of a single gene, genetic counseling is a standard component of a comprehensive plan for supporting these families. Because of the high level of phenotypic expression of the disease in infants, one in four subsequent children born to parents of a child with cystic fibrosis will also develop the illness. Given that the genotype of the fetus can be determined *in utero*, uncertainty concerning future pregnancies is no longer an issue. Both parents must be carriers of the illness for their children to develop the disease. The decision to have a child with cystic fibrosis now includes the awareness that each parent has contributed a dysfunctional gene to the child and both have agreed to proceed with the pregnancy. Because of the need for early intensive treatment, the establishment of the initial diagnosis is an important factor and usually supports the development of a strong and supportive doctor-family relationship.

Studies have suggested variable frequencies of comorbidity of emotional disturbance with cystic fibrosis. Some studies have reported that children with cystic fibrosis have no increased incidence of psychiatric disturbance ([Blair et al., 1994](#)) and that they have normal intellectual and academic abilities ([Thompson et al., 1992](#)). However, a comparison of four chronic pediatric illnesses suggested that methodologic issues of ascertainment of psychopathology in physically ill children may result in lower estimates of psychopathology. Twenty-five percent of the school-age children with cystic fibrosis within this sample were diagnosed with a psychiatric disorder ([Canning, 1994](#)).

Illness-related risk factors have been hypothesized to be minimized by protective factors such as positive familial involvement and high-quality medical support. [Steinhausen et al. \(1983\)](#) examined a set of hypothesized risk factors thought to be associated with psychological disturbance. Only in children with cystic fibrosis was severity of illness an illness-related risk factor. The other risk factors included parental pathology, disturbed parenting, and socioeconomic difficulties. The entire set of risk factors, including severity of illness, was responsible for nearly 37% of the variance in the occurrence of psychiatric disturbance in this sample. Approximately one-third of the children were diagnosed as having severe emotional disturbance. Another 22% of the children had less severe emotional difficulties.

The occurrence of maternal depression has been examined in the mothers of children with cystic fibrosis, based on both the guarded prognosis of their children and the large burden of care that is required as part of the treatment. Mothers of children with cystic fibrosis report higher levels of depression and greater levels of emotional strain than do the fathers of these children ([Quittner et al., 1992](#)). Other studies have linked maternal distress with higher levels of adjustment problems ([Thompson et al., 1994](#)).

[Venters \(1981\)](#) show an association between competent family functioning and the child's adjustment. Two characteristics, seeking out social support during stressful periods and the ability of the family to understand the illness within a broader religious or philosophic frame of reference, are shown to be associated with better outcome. Although a broad range of problems has been reported to occur in families with a child with cystic fibrosis, [Petzel et al. \(1984\)](#) identify concerns about

treatment and premature death as the most salient issues for both the children and their parents.

### Insulin-Dependent Diabetes Mellitus

Juvenile-onset *insulin-dependent diabetes mellitus* (IDDM) is a disease of the islet cells of the pancreas characterized by a deficiency in the production of insulin. It is a rare illness in very young children, but it does occur in about 0.1% of school-age children and adolescents. Reports have suggested that exclusive breast-feeding may be protective and that frequent early infections may be a risk factor for onset ([McKinney et al., 1999](#)). Because of its delay in onset, IDDM presents an interesting contrast to the problems associated with lifelong chronic physical illness. It has been suggested that less evidence of psychopathology has been demonstrated in children with IDDM when compared with children with other serious physical illnesses that begin in infancy. Possible factors that contribute to a better outcome would include the following: (a) the later onset, with less negative impact on early emotional development; (b) a more effective mode of treatment; and (c) minimal early central nervous system involvement.

Questions have arisen related to the role of stress in the initial onset of diabetes. [Barglow et al. \(1986\)](#) report no significant association between psychopathology in mothers with diabetes and the subsequent development of "developmental deficits" in their children when obstetric and perinatal factors other than diabetes are well controlled. [Chase and Jackson \(1981\)](#) report an association between stressor scores and serum glucose and triglyceride concentrations in an adolescent sample. [Gustafsson et al. \(1987\)](#) document an association between disturbed family interaction and diabetic control in adolescents, as measured by elevated glycosylated hemoglobin concentrations 5 years after the initial assessment.

Although associations between poor emotional adjustment and difficulty in control of diabetic symptoms have been repeatedly observed, the nature of the relationship is not clear. It is often suggested that poor emotional regulation subsequently interferes with medical compliance. However, the alternative view, that poor chemical control causes emotional disturbance, remains a viable hypothesis.

Although there is a strong familial propensity for the development of diabetes, the genetic risk is less clear than for illnesses with a better established pattern of inheritance. Diagnostic issues are for the most part less of an issue than the therapeutic issues associated with management of the illness. [Wilkinson \(1987\)](#) identifies two of the characteristics of IDDM that result in later disturbance. The first is that it affects a wide range of aspects of everyday living. The second is that it requires a high degree of personal responsibility to manage the illness medically. In his study of adults with diabetes, as many as 18% of the sample patients were diagnosed with a psychiatric disorder. Depression and anxiety were the most frequently reported conditions.

Little physical deformity is initially associated with IDDM. However, children may experience anxiety related to fears of hypoglycemic or hyperglycemic coma and later vascular complications ([Moran, 1984](#)). Some interference with normal function is a key problem in adolescence, when restrictions in activity and dietary compliance can be particularly problematic. [White et al. \(1984\)](#) studied the association between potential stressors and poor diabetic control in children with very labile IDDM. Ketoacidosis was sometimes associated with "intercurrent illness" (15% to 30%) and insulin mismanagement (5% to 10%). However, most of these children came from families with multiple problems that were believed to be highly associated with their poor medical control.

IDDM has a relatively uncertain outcome. Although most children survive well into adulthood, the fear of diabetic sequelae and the occurrence of severe ketoacidotic episodes can be very upsetting. [Gath et al. \(1980\)](#) demonstrated certain cognitive difficulties in children with IDDM who were between the ages of 9 and 10 years. The duration of the illness was about 3.5 years. A correlation was shown between poor diabetic control and (a) the presence of a psychiatric disorder, (b) problems in reading, and (c) adverse psychosocial factors in the family background. Neuropsychological studies of children who had onset of the disease before 5 years of age have found impairment in the abstract/visual reasoning subscales of the Stanford-Binet Intelligence Scale. These lower scores were noted in some children with only mild hypoglycemia ([Golden et al., 1989](#)). Another study found delays in information processing speed and conceptual reasoning that developed over the course of 2 years after diagnosis in the IDDM cohort ([Northam et al., 1999](#)).

[Kovacs et al. \(1985\)](#) studied a group of school-age children with IDDM and found no evidence of increased life stress or psychiatric disturbance preceding the diagnosis. She described two patterns of coping in these children. Some social withdrawal and sadness were noted in about two-thirds of the children. The remaining one-third met criteria for psychiatric disorder, with depressive syndromes most prominent. Ninety-three percent of the children recovered from these reactions to the diagnosis within the first year of their illness. In a study of the rehospitalization of children with IDDM, externalizing symptoms were shown to be correlated with readmission, a finding prompting the recommendation of appropriate intervention for these problems as a preventive measure ([Kovacs et al., 1994](#)).

[Fonagy et al. \(1987\)](#) conducted a well-controlled study to examine psychological factors that influence the management of children with IDDM. Psychological disturbance in both the children and the parents predicted low glycosylated hemoglobin concentration in the children and accounted for 44% of the variance in blood glucose control. These findings suggest that as many as one-third of the children with IDDM have appreciable emotional and behavioral problems. However, children perceived as having problems were less often responsible for the care of the illness. The authors conclude that children younger than 12 years of age are often unable to manage the self-care regimens prescribed to treat their illness. The importance of parental involvement in maintaining optimal diabetes management has been clearly demonstrated and well accepted ([Weist et al., 1993](#)). A report focusing specifically on adolescent response to both hypoglycemia and hyperglycemia suggests surprisingly inappropriate reactions that were not substantially improved by involving parents ([Johnson et al., 2000](#)).

### Pediatric Malignancies

*Pediatric malignancies* are rare and can begin throughout childhood. The leukemias are the most common, although they still occur in only 15 children per 100,000. They are commonly diagnosed between 2 and 4 years of age. Prior to 1970, childhood malignancies were considered fatal illnesses. Today, because of the dramatic improvements in chemotherapy and radiation treatment, leukemia is now more appropriately considered a chronic illness, given that the current 5-year survival rate is well in excess of 50%.

The causes of most malignancies remain unclear. Evidence of increased genetic risk for some subtypes of leukemia has been determined, but many cases appear to occur sporadically. Although various hypotheses have been developed suggesting that both infectious and immunologic mechanisms are involved in the onset of leukemia, the diagnosis usually comes as a shock, with little sense of parental responsibility.

Malignancies vary in how difficult they are to diagnose. For example, leukemias can be asymptomatic for months. However, once the suspicion of leukemia is raised, the definitive diagnosis can be quickly and reliably confirmed through simple laboratory examination. Given the importance of aggressive early treatment, any delay in establishing the diagnosis presents an important psychological issue in the care of the family.

For the most part, leukemias result in only minimal physical deformity. However, bruising and wasting can be a problem in the later stages of the illness. In contrast, solid tumors that require surgical intervention may result in radical and abrupt changes in the child's body image. In many ways, adolescence is a particularly difficult time for the onset of these illnesses and the often quite extensive treatment that is required. One study examining identity formation in survivors of childhood cancer suggests that these children may have a "foreclosed" or premature formation of identity, which diminishes their exploration of some positive options for the future ([Madan-Swain et al., 2000](#)).

Considerable controversy exists regarding the frequency of emotional disturbance in children with malignancies. Some studies ([Eiser, 1979](#); [Greenberg et al., 1989](#); [Kupst et al., 1984](#)) suggest relatively low levels of either emotional or behavioral disturbance. [Sawyer et al. \(1986\)](#) report significant differences in behavioral and emotional problems between affected children with leukemia and their siblings in the 2-year period after the diagnosis. A longer-term follow-up some 4 years after the initial evaluation ([Sawyer et al., 1989](#)) shows considerable normalization of the functioning of leukemic patients; the only remaining difference between the patients and their siblings is in regard to problems in school performance as noted by both teachers and parents. Three possible explanations for these persistent findings are (a) the direct effect of central nervous system irradiation on neuropsychological functioning, (b) the documented large number of school days missed, and (c) the lasting impact of documented earlier emotional and behavioral problems that had occurred primarily in school-related areas. The treatment of the illness should be considered an illness-related risk factor to the degree that repeated, painful bone marrow biopsies and other invasive procedures are required. This can be particularly difficult for children with early onset of disease.

The cranial irradiation literature suggests that aggressive treatment can result in neuropsychological damage ([Stehbens and Kisker, 1984](#)), particularly if treatment is administered before 8 years of age ([Moehle and Berg, 1985](#)). [Pavlovsky et al. \(1983\)](#) report that children with cranial irradiation and intrathecal methotrexate treatment have the greatest number of psychological sequelae and associated abnormal computed tomography scans. [Brouwers et al. \(1984\)](#) again note associations with computed tomography scans. Specifically, greater neuropsychological deficits and attention deficit problems are noted in patients with evidence of atrophy. In the



relatively few patients who show calcification, the greatest deficits are noted. Long-term follow-up of survivors of acute lymphoblastic leukemia reveals a greater likelihood that the children will be placed in special education programs. However, they generally have reasonably good outcomes. Other studies have suggested that academic problems may be more persistent (Raymond–Speden et al., 2000). Dosage of cranioradiotherapy and earlier age at diagnosis are found to be important education-related risk factors (Eiser, 1991; Haupt et al., 1994; Leung et al., 2000).

The effect of chronic illness should not be considered in isolation from other concurrent stresses. This is illustrated by Kalins et al. (1980), who document the occurrence of non-illness-related stressors in the families of physically ill children. A higher frequency of other serious life events is noted to have occurred in those families with sick children. However, independent studies have noted an association between “family cohesion and adaptation” and better psychological outcome in adolescent cancer survivors across a wide range of stressful experiences (Rait et al., 1992).

Mulhern et al. (1981) describe the differences in the perception of prognosis for recovery in physicians, parents, and children. Physicians have the most negative view of prognosis. Parents hold an intermediate view. Children are the most optimistic about the future. Although it is probably therapeutic to support an optimistic outcome, the authors point out that the somewhat unrealistic expectation may provide an explanation for studies that find few affective and emotional symptoms in children with serious disease. This denial may be an adaptive form of coping with the seriousness of their medical prognosis as long as it does not interfere with maintaining good compliance with treatment recommendations by children and their families.

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#### CASE ILLUSTRATION

An example of an adaptive response to an overwhelming disability was demonstrated by George. At the time of diagnosis, George was 17 years old and was the second of four children in a middle-class, upwardly mobile suburban family. George was not chronically ill as a young child. In fact, throughout high school, he was an outstanding athlete who played three sports and qualified for state finals in track and field. During his senior year in high school, he suddenly developed intense pain in his right leg. The diagnosis of osteosarcoma in his right tibia was made within 2 days of the onset of his first symptom. An immediate amputation was recommended, and the serious nature of his prognosis was openly discussed with George and his family in a sensitive and thorough manner. Surgical intervention was performed without delay.

For George, the impact of the loss of his leg was enormous. His appearance and athletic ability were central components of his identity. Considerable concern that he could become depressed was expressed. Instead, George responded to his surgical treatment in an extremely positive and forward-looking manner. His adaptive and spirited recovery was strongly supported by his family, who could both acknowledge the severity of the illness and at the same time remain emotionally supportive. A very aggressive postoperative rehabilitation program was planned that included visits by teenage amputees to the hospital before George was discharged. By the time he was ready to return home, George had heard from dozens of his classmates, and his courage was widely recognized at his school. This community support was coupled with the unshakable conviction of his family that he would persevere. On follow-up, no depressive symptoms had developed, and George continued to be an outstanding student. The following year, he went onto a prestigious university and established himself as a campus leader.

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## EFFECTS OF MULTIPLE HOSPITALIZATIONS

A pediatric hospitalization should be considered as an experience during which a child will be exposed to a set of illness-related risk factors that are likely to have a negative impact on subsequent adaptation (Mrzcek, 1986). Substantial evidence suggests that a single hospitalization, particularly if managed sensitively, will have relatively minimal impact on the long-term development of the child (Quinton and Rutter, 1976). However, as is true with many risk factors, multiple exposures increase the potentially negative impact of the experiences. At least five sets of variables influence the impact of hospitalizations. These include (a) the nature of the disease, (b) the nature of the hospital experience, (c) the sociocultural context of the hospitalization, (d) the adaptive abilities of the parents, and (e) the child's capacity to cope with the illness. Developmental considerations are particularly important. Early hospitalization, occurring within the first 6 months of life, has not been associated with later difficulty. However, some early studies show that children between 1 and 4 years of age may be particularly sensitive to prolonged hospitalizations (Mrzcek et al., 1984; Prugh et al., 1953). During this period, the parent is perceived as the primary attachment figure. Unfortunately, parents may be unable to modulate the experience of a preschool child during difficult procedures that are part of the hospitalization. This results in the parent's being unable reliably to provide a secure haven for the child and in more persistent perturbations in the quality of the primary caregiving relationship. Preschool children would normally be developing a sense of independence and autonomy, which is essentially incompatible with the passive, dependent behaviors that are required during the hospitalization. Yet another issue is that preschool children have preoperational cognitive abilities. As a consequence, it is not possible to rely on concrete operational thinking. This means that therapeutic strategies that require the understanding that specific causes will have predictable effects cannot be used to help these children anticipate likely positive or negative outcomes that will result from necessary but uncomfortable aspects of their treatment.

Ensuring that parents become a part of the treatment team during hospitalizations is critical. The basic objective during acute hospitalizations is to provide a sense of emotional security for the child. To the degree that the parents are unable to cope with their own anxieties about the illness of their child, they will have difficulties in modulating stressful experiences. It is critical for parents to take an active role in providing emotional support. This includes the realization that their physical presence is important. However, they can also be involved in helping their child make sense of what can sometimes be a series of bewildering experiences. As stressed earlier, this is particularly true for the preschool child. It is usually best to assume a proactive stance. Even in a maximally sensitive hospital, the emotional needs of a child in a medical crisis cease to be a primary focus. At such moments, the pediatric team is appropriately exclusively concerned with preserving the life of the child. In this climate, the parent is often the critical link between the pediatric treatment team and the child. If the parents are able to assume this role, it is often advisable for the medical team to encourage parents to become a partner in the provision of their child's care. The child psychiatrist can play a role of supporting the parents in this difficult position and then can help them to understand retrospectively what has happened after the crisis has passed. Although this is demanding for all involved, the successful accomplishment of helping a child through a critical episode provides a strong basis for all subsequent consultative efforts.

It is essential to help children proactively to cope with the most difficult aspects of hospitalization. The evolution of Child Life programs is a good example of the development of many proactive strategies. The alliance between the medical team and the family is of central importance. A trusting and communicative relationship with the pediatrician can be extremely important in managing the anxiety level of the parents and the child. This primary relationship can be either enhanced or threatened by the nursing staff. Nurses have become increasingly sophisticated in the identification of parents who are having difficulties with coping. They are now able to organize nursing care to maximize potential parental emotional support.

The child psychiatrist who provides pediatric liaison is a critical part of the treatment team. In addition to the classic role of diagnostician and therapist able to intervene at moments of family crisis, child psychiatrists have increasingly taken proactive roles in the development of systems designed to help families to deal with the experience of hospitalization. One specific aspect of this role is taking the responsibility to ensure that both cognitive and emotional understandings of the implications of medical procedures are considered. All too often, misunderstandings about medical care can arise that lead to major disruptions in the emotional well-being of the child. Ensuring that both the family and the child have a good understanding of the need for medical procedures and of the implications of proceeding in a particular clinical direction is a cornerstone of appropriate care for ill children.

In analyzing the impact of hospitalization on an individual child, two variables should always be considered. The first is an assessment of the severity of illness. The second is the duration of the discomfort associated with required therapeutic procedures. Minde et al. (1982, 1983) developed a morbidity scale to monitor these variables in infants. This approach highlights the specific needs of some families, who may cope well during early phases of an illness but do less well when the stressors associated with hospitalization continue for prolonged periods.

During the prolonged hospitalization of young children, the single most important aspect of treatment is to ensure that the primary attachment figures are available for the children. For older children who must remain in the hospital for prolonged periods, other issues are important. First, it is helpful to provide opportunities for educational and tutorial experiences to ensure that these children do not fall behind in their learning objectives. Furthermore, the maintenance of close contact between friends and classmates can support the coping strategies of most teenagers. However, it is helpful to consider a hospitalization as a new opportunity during which new peers can be identified. In some cases, this can lead to the creation of a new peer support group consisting of children with similar physical challenges. Such groups can be particularly helpful during very extended hospitalizations.

## SUMMARY

In conceptualizing the impact of chronic medical conditions on child development, it is helpful to integrate the stressful aspects of an illness into a broader systems perspective that considers the entire spectrum of early childhood adaptation. Both illness-related and more generally appreciated protective factors should also be considered in developing a treatment plan for a particular child. This approach represents an appreciation that whereas all chronic illnesses have some similar negative impacts on development, illness-related stressors are associated with each individual disease. Illnesses that have a long, unremitting course share characteristics, as do illnesses with uncertain prognoses, with early onset, and with similar causes. To the degree that the illness-related characteristics of various diseases are similar, their emotional sequelae will also be similar (Table 103.2).

## RESEARCH DIRECTIONS

The need to have ongoing prospective longitudinal studies is overwhelmingly apparent in a review of the multiple cross-sectional studies of physically ill children. When one is considering problems with a genetic origin, the optimal plan is to recruit a sample of children before the onset of illness and to follow them longitudinally. When illnesses have more spontaneous onsets, the best approach is a study that is designed to follow-up children after the initial diagnosis. Any prospective longitudinal study should include the documentation of not only the key characteristics of the illness itself but also of the familial risk and protective factors that simultaneously influence development. Through the judicious examination of the interaction of these factors, primary prevention strategies can probably be developed to minimize the emotional sequelae of physical illness. Given the prevalent nature of these problems, the implications of early intervention for physically ill children are of substantial public health importance.

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# 104 DYING AND DEATH IN CHILDHOOD AND ADOLESCENCE

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Dying is a transitional state during which the child and the family may look to the physician for understanding, support, and direction. However, a physician may experience anxiety in the presence of a dying child because of (a) the feelings of impotence, failure, and anxiety that are aroused in a physician when confronted with his or her own limitations and mortality; (b) the regressive pull the child's loneliness, abandonment, neediness, and insecurity evokes in the physician; and (c) the difficulty of dealing with the parents' anxiety, depression, anger, resentment, and denial.

Yet there is probably almost nothing that the child or parent fears, imagines, feels, or experiences that cannot be discussed with the child in an honest way. The basis for the discussion is a trusting relationship. Children in particular soon learn whom they can trust and hence to whom they can open up. Indeed, the child often senses more accurately what the adult can tolerate than the adult senses how much the child can assimilate, and the child acts accordingly.

## CHILDREN'S CONCEPTS OF DEATH

The child's reaction to his or her own dying or the death of others is related in part to his or her concept of death ( [Schonfeld, 1993](#)), which in turn is related to the child's developmental stage ( [Table 104.1](#)).

Age	Concepts of Death	Implications for Adjustment to Loss
0-2	Death is seen as a permanent phenomenon from which there is no return.	Failure to comprehend this concept prevents the child from engaging in the process of mourning from the deceased, a necessary step toward successful mourning.
2-4	Death is seen as a state in which all the functions cease completely.	Examples of incomplete understanding: The child worries about a fourth relative being cast or in pain; the child wishes to bury both with the deceased.
4-7	Death is seen as a mixture phenomenon that no living being can escape eventually.	Examples of incomplete understanding: The child views significant individuals (i.e., self, parents) as immortal; if the child does not view death as inevitable, he or she is likely to view death as a punishment rather than a choice or thought of the cause of the deceased leading to excessive guilt and shame.
7-10	The child develops a realistic understanding of the causes of death.	Examples of incomplete understanding: The child who relies on magical thinking is apt to assume responsibility for deaths of a loved one by desiring food thoughts or unrealistic actions toward cause of death.
10-12	Death is seen as a punishment rather than a choice or thought of the cause of the deceased leading to excessive guilt and shame.	Implication: Tends to lead to excessive guilt that is difficult for the child to resolve.

Table 104.1. Death and Childhood

The stages in the child's understanding of the concepts of death have been well studied ( [Hostler, 1978](#); [Kastenbaum, 1967](#); [Smilansky, 1987](#); [Speece and Brent, 1984](#); [Wass, 1984](#)). Four major concepts related to death have been noted consistently: irreversibility, finality (also termed *nonfunctionality*), causality, and inevitability (also termed *universality*). Failure to comprehend each of these concepts impairs the child's ability to mourn successfully ( [Table 104.2](#)). [Speece and Brent \(1984\)](#), in a review of the literature, concluded that most studies found the age of acquisition of the three concepts they reviewed (irreversibility, nonfunctionality, and universality) to occur between 5 and 7 years; earlier studies citing an older age of acquisition typically were noted to have significant procedural flaws.

Concept	Example of Incomplete Understanding	Implication	Adjustment to Loss
1. Irreversibility	Death is seen as a permanent phenomenon from which there is no return.	Failure to comprehend this concept prevents the child from engaging in the process of mourning from the deceased, a necessary step toward successful mourning.	
2. Finality (nonfunctionality)	Death is seen as a state in which all the functions cease completely.	Examples of incomplete understanding: The child worries about a fourth relative being cast or in pain; the child wishes to bury both with the deceased.	
3. Inevitability (universality)	Death is seen as a mixture phenomenon that no living being can escape eventually.	Examples of incomplete understanding: The child views significant individuals (i.e., self, parents) as immortal; if the child does not view death as inevitable, he or she is likely to view death as a punishment rather than a choice or thought of the cause of the deceased leading to excessive guilt and shame.	
4. Causality	The child develops a realistic understanding of the causes of death.	Examples of incomplete understanding: The child who relies on magical thinking is apt to assume responsibility for deaths of a loved one by desiring food thoughts or unrealistic actions toward cause of death.	Implication: Tends to lead to excessive guilt that is difficult for the child to resolve.

Table 104.2. Concepts of Death and Implications of Incomplete Understanding for Adjustment to Loss

During the first few years, the child ordinarily has virtually no concept of death other than that of a disappearance. However, when faced with traumatic events, such as the death of a parent, children younger than 5 years of age may develop what seems to be a precocious understanding of death. In general, the role of personal exposure to death of others has been controversial; although several studies have shown that such personal experience may promote the acquisition of the concepts of death ( [Kane, 1979](#); [Reilly et al., 1983](#)), other studies have failed to support this conclusion ( [Jenkins and Cavanaugh, 1986](#); [Townley and Thornburg, 1980](#)). Cross-cultural comparisons ( [Florian and Kravetz, 1985](#); [Schonfeld and Smilansky, 1989](#); [Wass et al., 1979](#)) also have illustrated significant cultural variations and important cross-cultural consistencies, suggesting that although the underlying developmental framework is likely to be robust across cultures, various sociocultural variables may have a significant and profound impact on the rate of acquisition of important individual concepts.

Children between 5 and 10 years of age (approximately) continue to clarify their concepts of death but still are confused at times. For example, a child may say, "When I die, my heart stops, I can't see, and I can't hear. But if I'm buried, how will I breathe?" Some of the child's difficulty in thinking clearly about death is



developmental, but some of the difficulty is emotional. If the child of this age has a heightened concern about a part of his or her body and its functioning, the child may tend to think of death in terms of the harm to that part of his or her body and its functioning, especially because the child also tends to think in concrete terms at this stage. Children in this age group with a terminal illness have been shown to have a marked awareness of the seriousness of their illness, even if never told that their illness is fatal, and a precocious understanding of personal mortality ( [Clunies-Ross and Lansdown, 1988](#); [Spinetta, 1974](#)).

Somewhere between 10 and 15 years of age, the average child acquires a grasp of the meaning of mortality ( [Kastenbaum, 1959](#)). The child's reaction to death at this time is influenced more by his or her emotional struggles than by the child's intellectual capacities. Thus, a young adolescent who is concerned with, among other things, sexual performance, control of impulses, physical intactness, and separation from parents may react with anxiety if any one of these sensitive conflict areas is involved in the fatal illness.

## THE CHILD'S REACTION TO HIS OR HER OWN DYING

The very young child mostly is preoccupied with the discomfort of the illness, whether acute or chronic, and the separation and withdrawal that occur when hospitalization is necessary. A somewhat older child, although also troubled by pain and separation, interprets his or her illness according to his or her level of cognitive development and emotional conflicts. Thus he or she may interpret the illness as an act of "immanent justice" for the guilt he or she feels about some real or imagined misdeed. Usually the child shows regressive behavior in the face of the illness, hospitalization, and its treatment, and a fear of mutilation. Occasionally the child shows a denial of discomfort or dread ( [Solnit, 1965](#); [Solnit and Green, 1959](#)). An older child who is aware of the irreversibility of death may deny his or her own anxiety but may exhibit a depression, occasionally mixed with outbursts of anger and anxiety. This reaction is especially common in adolescents. On the other hand, some children are astoundingly courageous and steadfast in the face of death.

The range of reactions is great. In a sense, all that has gone before contributes to the child's understanding of death and his or her reaction to it. Each child is an individual, and a myriad of variables influence the behavior of the child, the family, and the helping persons around the child.

## REACTIONS OF OTHERS TO THE DEATH OF A CHILD

An important determinant of children's reaction to death is the reaction of those around them to death. Those others may be parents, siblings, or hospital staff members.

### Reaction of Parents

#### GENERAL REACTIONS

The general reactions of parents have a chronologic sequence, starting before and continuing during and after the moment of death. Initial shock and denial of the diagnosis may last from a few seconds to a few months. This stage may be followed by anger ("Why my child?") or guilt ("If only I had..."). Sooner or later, the parent starts to bargain ("If he could only live to..."). This stage is followed by normal grieving and mourning over the impending loss and by the beginning of separation. Finally, a stage of resignation or acceptance can be reached.

#### SPECIFIC REACTIONS

##### *Reactions to a Rapid Death*

When the death has occurred relatively rapidly (e.g., perhaps as a consequence of a brief illness), the period before death is filled with anxiety and concern. (For a discussion of sudden infant death syndrome, see [Chapter 95](#).) The parents may be desperately hopeful, but they also may have feelings of guilt and a need to deny the possibility of death as an eventual outcome. After a rapid death of their child, the parents may again feel some diffuse anger, which may be displaced onto the physician. This reaction may occur regardless of whether the physician has been diligent, but it is more likely to fester and be prolonged if the physician fails (out of his or her own discomfort) to show consideration at the time and to provide the opportunity for a follow-up interview. Over time, the parents will then go through their own characteristic mourning process. Their mourning may include some identification with the lost person and, occasionally, an overidealizing of the lost person (particularly in cases in which the parent also experiences the loss of what they had expected for the lost child). Further possible normal reactions (within limits) may include (a) a displacement of attitudes toward the dead child onto one or more of the surviving children, (b) attempts to fill the loss by another pregnancy, or (c) withdrawal for a time. These and other normal reactions, in general, should be respected and left alone. Extreme forms of defenses may be dysfunctional and are an indication for therapy ( [Krell and Rabkin, 1979](#)).

##### *Reactions to Prolonged Dying*

Occasionally, premature mourning may occur, with anticipatory grief and withdrawal of interest in the dying child, perhaps accompanied by the displacement of warm feelings onto an infant child in the family. Often, unacceptable thoughts arise. For example, a parent may find himself or herself wishing that the child would finally die and relieve everyone of the emotional and financial burden and suffering. Such a wish may horrify a parent and lead to the immediate mobilization of certain defense mechanisms. A common defense mechanism is that of reaction formation: The parent becomes extra protective in caring for the dying child. The parent also may feel guilty and express his or her guilt (and anxiety) by asking repetitive questions that require tactful answers.

As a chronically ill child nears death, the parents may be filled with remorse and may experience a resurgence of love. Rarely, a denial that death is imminent may remain in force. After the death of a chronically ill child, parents may feel a mixture of relief and guilt, perhaps with feelings of remorse being uppermost.

### Reactions of Siblings

Siblings who are very young, especially those younger than 5 years of age, feel the withdrawal of the parent intensely and consequently feel a loss of love. Young siblings may view the death as an abandonment, as punishment, as the realization of unacceptable wishes, or as all three. Children between approximately 5 and 10 years of age in general are somewhat more concerned for the dying child and also may be fearful for themselves. Although it is expected that older children usually can muster a supportive attitude and temporarily assume parental roles for the younger siblings at home, even teenagers feel and react to parental withdrawal and may "act up" during such a trying time. They too require special attention ( [Adams and Deveau, 1987](#)). Children as well as adults may experience survivor guilt after the death of a child ( [Lifton, 1967](#)). Some surviving children suffer serious symptoms and subsequent distortions of character structure ( [Cain et al., 1964](#)). Through identification with the deceased, thought to be more common and dramatic in children than in adults, surviving children also may manifest conversion symptoms. In one series of conversion disorders in children, 58% were associated with unresolved grief reactions ( [Maloney, 1980](#)).

### Reactions of the Hospital Staff

Hospital staff members also experience anxiety in the presence of a dying child or a grieving parent ( [Solnit and Green, 1959](#)), and they tend sometimes to deal with that anxiety by withdrawal and a conspiracy of silence. These reactions may hamper them from giving the dying child and his or her family the best care possible and may prevent the staff members from carrying out certain essential psychological tasks. Besides comforting the parents, such tasks include helping the child feel as active as possible in his or her attempts to cope with anxiety and allowing the child some hope. Furthermore, the privacy and dignity of the child require protection. Last, certain distortions require correction. The child, for example, may show his or her concern by asking such questions as "Am I safe?" "Will someone be with me when I need them?" "Will I be helped to feel better?" The need for tact carries through into the period of care for the survivors.

Survivors outside of the family also will need assistance in dealing with their grief. Psychiatrists not infrequently are consulted to provide crisis intervention for bereavement support in settings outside of the hospital, such as in the schools. Different techniques often are required to provide this type of intervention service and are described elsewhere ( [Newgass and Schonfeld, 2000](#); [Schonfeld, 1989](#); [Schonfeld et al., 1994](#)).

## CLINICAL CARE OF THE DYING CHILD

## Talking with the Parents About Fatal Illness

The hardest task for the physician is to tell the parents that their child is fatally ill. The physician should take the parents into a private office and allow at the very least half an hour, uninterrupted by telephone calls or other tasks. He or she can begin by telling the parents the diagnosis and the nature of the illness. He or she might then go on to describe the treatment that is available to offer some relief for the child's symptoms. At some point, the physician will have to tell the parents that there is no treatment that can cure the child of the illness. Throughout this interview, the physician should pause and give the parents every opportunity to express their feelings and ask questions. The physician must resist an understandable impulse to "shut off" the parents' grief. If the parents ask whether the child will die from the illness, the physician will have to say that the child will. If they do not ask, the physician should at some point attempt to clarify that the illness is progressive and that the child will die. At the same time, the physician must remember that the parents will not necessarily understand or accept what they have been told.

The physician should not end the interview then but should stay with the parents as they experience their shock (and perhaps anger) and grief. The physician might then tell the parents how he or she plans to treat the child and help the parents feel some measure of participation in and control of the treatment.

The physician is obliged to tell the parents what to expect as the illness progresses. This information need not be given in full in the first interview; rather, it should be given in stages over an extended period. The goal is to give the parents information that will enable them to anticipate the child's needs at each stage. The parents usually will indicate by their questions what they need to know.

Regular contact with the parents should then be planned. The contact should take the form not of comments made in passing but of time set aside to talk, review, and listen in the privacy of an office. The physician should resist the natural impulse to avoid the parents or avoid the subject. To do this, the physician must recognize any impotence and anger he or she may feel in the face of death. The parents will come to trust the physician and feel safe expressing, if they so wish, some of their less acceptable feelings if they are sure that the physician is available and ready to listen. During these planned interviews, the physician can discuss with the parents their child's behavior, their management of the child, what to tell the child's siblings, and whether and in what way the parents would like their minister, priest, or rabbi involved. The parents should be reassured about their own handling of the situation, and the physician should feel free to share admiration for how well they are meeting the child's needs.

The physician may be asked for advice about religious rites for the sick. The practice of offering prayer with the child or administering the Sacrament of the Sick, although intended to comfort, may be anxiety arousing. Although it sometimes happens that the Sacrament of the Sick is given without the parents' consent, or even knowledge, most priests prefer to involve the parents and family first, and to have the family present in the room. However, administering the Sacrament of the Sick may cause as much upset in the family as in the child. The 1972 modification of the Sacrament of the Sick (["Ordo Unctionis," 1972](#)) does not alter very much the way in which the child might experience the ritual. Indeed, there are no specific modifications for children other than those based on the judgment of the particular priest. The physician should discuss with the parents and the priest the child's needs and how the child might experience the praying or the Sacrament before any step is undertaken.

## Talking with the Child

In regard to talking about death with the sick child, especially the young sick child, the parents' feelings and wishes must be respected. Some parents, for example, do not wish the child to be told that he or she is going to die, whereas others do. Some families need to use denial as a protective device.

There is no simple answer to the question of whether the particular child should be told. One useful approach is to discuss with the parents how they think they would respond if their dying child asked them whether he or she was going to die. There are several stages of response to such a question that might be suggested.

First, the child's reason for asking the question must be clarified. The child may be responding to the parents' or the hospital staff's anxious behavior, or the child may be concerned about such things as pain, mutilation, loneliness, and the needs of others. The child then can be given repeated opportunities to talk about what he or she is worried about.

Second, if the parents decide that they want the child to know that his or her illness is fatal, the process of telling the child about the illness and impending death should have the characteristics of a dialogue rather than of an announcement. Some children simply cannot understand and do not want to hear the truth; they should not be told. Others have to arrive slowly at the realization of the significance of their illness; it is too much for them to understand and grasp at one time.

Third, the child must be given hope. Even when the child is told that the illness is one that causes death, the child can and should be told that the physicians will do everything they can to fight the illness.

The adults must agree not only on how the child should be told but on *who* should be the first to tell him or her. Sometimes the physician is not the right person to disclose this information. A parent or a member of the clergy who is close to the child may have a more sensitive understanding of the child's needs.

A discussion of this kind with parents often helps them to express some of their own concerns. It also promotes in them a feeling of trust and of being understood, as well as a feeling that they have some control over the care of their child. Nothing is so painful to the parents as their feeling of helplessness as their child's life ebbs.

Most important, the physician who has had a dialogue with the parents is prepared to have a dialogue with the child, always keeping in close touch with the parents. The physician, for example, can convey assurance while imparting to the child as much of the truth as the parents want and the child seems ready to know. There is no blueprint answer.

The ward staff members should be clear about who has the primary responsibility for talking with the parents and child about the seriousness of the illness. If it has been agreed on by the parents that the physician should talk with the child, the physician should first establish a relationship of trust with the child. Before the physician talks with the child, he or she should make sure that someone is available to be with the child after the physician has left the room, if the child so desires. The involved staff members should be informed about how much the child knows about the illness so that the child does not receive conflicting and therefore puzzling information. Ward staff members also experience anxiety when in the presence of a dying child or such a child's family. Their natural inclination to avoid these feelings may cause them to stay away from both the child and the grieving family. Such inclinations may be better controlled if the ward staff members have the opportunity to explore and share their feelings in meetings ([Berman and Villarreal, 1983](#); [Lewis, 1962](#)).

## Specific Management of the Child Who Is Dying

[Kübler-Ross \(1972\)](#) gives a beautiful description of a dying boy's expression of his thoughts and feelings:

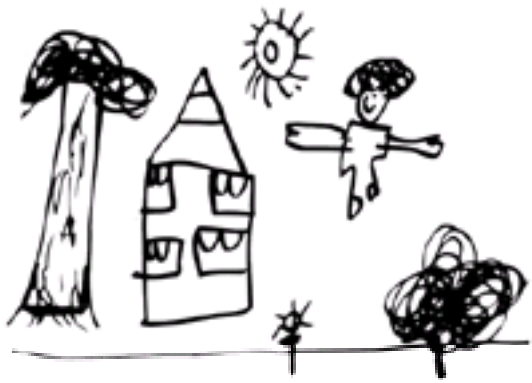
[The dying] boy tried to paint what he felt like. He drew a huge tank and in front of the barrel was a tiny little figure with a stop sign in his hand. This to me represents the fear of death, the fear of the catastrophic, destructive force that comes upon you and you cannot do anything about it. If you can respond to him by saying it must be terrible to feel so tiny and this thing is so big, he may be able to verbally express a sense of smallness or impotence or rage. The next picture he drew was a beautiful bird flying up in the sky. A little bit of its upper wing was painted gold. When he was asked what this was, the boy said it was the peace bird flying up into the sky with a little bit of sunshine on its wing. It was the last picture he painted before he died. I think these are picture expressions of a stage of anger and the final stage of acceptance.

This description underscores the importance of one aspect of the care of the dying child: With the parents' permission and in the privacy of his or her own room, the young child should be given the opportunity on different occasions to express his or her concerns through drawings or through play with toys and dolls ([Fig. 104.1](#) and [Fig. 104.2](#)).





**Figure 104.1.** Self-portraits done by a 4-year-old girl hospitalized with osteosarcoma. **A:** Self-portrait drawn within the first week of diagnosis. The body is intact; it is a very positive body image (i.e., profuse facial and body details, including eyelashes and hair). **B:** This portrait was drawn approximately 1 month after diagnosis. The patient was still hospitalized. Note the disintegration: The body is no longer intact, and the Broviac is highlighted, as is her stomach (she was experiencing nausea, and her Broviac became a prominent part of her experience of her body). **C:** This last drawing was done a week before death. Note the tumor looming largely in the picture. She died 3 months after diagnosis.



**Figure 104.2.** Self-portrait drawn by a school-age hospitalized patient dying of acquired immunodeficiency syndrome. The drawing expresses the child's sadness, isolation, and fears of death associated with this illness. The picture was drawn in black, and noteworthy are the major objects that appear to be floating in space with no roots, no foundation, and no sense of security. The house without a door offers no entrance or exit, implying perhaps little family support and isolation. The child is flying to heaven. He stated, "This is me flying to home in heaven in the sky."

If needed, and if the parents agree, the collaboration of a child psychiatrist here can be very helpful. The opportunity for expressive play may enable the child to exercise some control over his or her anxiety. If the child expresses concern about such problems as pain, loneliness, and fear through this play, this fact should be noted mentally by the pediatrician or child psychiatrist. At another time, the child could be reassured, without reference to the play session, that the physicians will make sure that he or she does not have pain, that there will always be someone available to help him or her, and that everything will be done to help him or her feel better.

Patients—adults and children alike—feel threatened by the passivity imposed on them by illness. Every effort must be made to give the child a feeling of active participation in treatment. The child should be informed at each stage what is being done, why it is being done, and what he or she can expect. Some feeling of hope needs to be provided. Last, the dignity of the child requires protection, and the child's privacy should be ensured.

Denial of death in a child, as in an adult, is a defense against anxiety, and it should be respected. Each person must be allowed his or her own way of dealing with the dread of death. At the same time, certain distortions should be corrected. For example, the child may require reassurance that the illness was not brought about by anything the child did (or thinks he or she did). Reactions that generate further anxiety, such as regression, should be gently but firmly controlled by the parents as well as by the hospital staff; excessive regressive behavior is uncomfortable for a child as well as for those caring for him or her.

Children vary in their capacity to deal with the inevitability of their impending death or with the diagnosis that implies impending death ( [Greenham and Lohmann, 1982](#)). Some children, particularly older children, want to know, whereas others do not want to know or cannot comprehend. In some cases, older children and adolescents possess the cognitive and emotional maturity to render them competent to take part in even the most difficult decisions in their medical care, including the decision to forego life-sustaining treatment for terminal illness ( [Leikin, 1989](#)).

### The Moribund Child

In a situation in which a child has no brain activity and is being kept "alive" by artificial methods, and there is no hope of spontaneous respiration or recovery of brain function, the physician must proceed with tact. First, before any decision is made to stop artificial life supports, the parents must be fully informed and prepared. The physician first might tell them that the child is being sustained by machines but that there is no possibility that the child will breathe on his or her own or will recover brain function; that is, the child in essence is dead. The physician should explain why this is so. In some instances, the parents have already considered the issue and will have decided to discontinue artificial life supports. Such parents also may have decided whether they want to be in the room at the time of death. Other parents may experience great anguish at the burden of deciding when to discontinue artificial respiration and also may prefer not to know exactly when it will be stopped. In a tactful way, the physician can say to such parents that there is nothing more that can be done. Then, when the parents do decide to discontinue artificial respiration, the physician should ask the parents where they want to be when it is discontinued.

The parents' wishes must be respected at all times; the child is theirs. The parents should not be rushed. It usually is their decision to make, and they need all the help they can get.

It is essential also that the parents feel a sense of unanimity with and security in the entire ward staff. Therefore, before the plan is carried out, the staff physician should discuss with all the ward staff members the steps just outlined and encourage them to express their thoughts and feelings. Parents tend to seek different answers from different staff members, and it is essential that all the staff members be aware of the way in which an individual case is being handled so that their responses do not conflict.

If the child dies suddenly without the parents being present, the parents should be informed of the death immediately, no matter what the time of day. The parents' sense of guilt at not being present when their child dies is an enormous one.

### Management of Parents During Prolonged Illness in the Child

The many different reactions that parents may have when the period of dying is one of prolonged suffering necessitate sensitive management. Some parents may request other opinions regarding prognosis or treatment. Often, they should be given this opportunity. Sometimes, however, a futile search for a magical cure may devastate a family emotionally and financially. The physician then should gently attempt to steer the parents toward a more realistic and helpful way of coping with their feelings of impotence. The physician can help parents by giving them opportunities to talk about their feelings in an accepting, nonjudgmental way. For example, parents often feel relieved when the physician reassures them that they are doing everything they can and that the physician knows how hard it is for them. The physician also can say, "Many parents have told me how at times they had wished it would all finally end, and then felt bad about thinking that. But it's a natural thought to occur. We all have many kinds of thoughts. What is important is that you have done everything that could possibly be done."

Questions may arise about child-rearing during the long period of time during which remissions occur and treatment is administered. Although the child's condition is far from normal, there often is a wish on the part of the child to feel normal. Perhaps this represents in part the child's wish that he or she no longer had the disease, that there was no longer a need for painful treatments, and that he or she could talk freely with others about feelings of frustration, anger, and resentment.

At the same time, parents may be in a quandary about how to rear the child, siblings about how to relate to the child and deal with their guilt, and teachers about how to educate and deal with the child and the other children in the classroom. Once again, there is no blueprint answer; indeed, blanket recommendations (e.g., "Treat the child normally") may only burden the parents with more conflicts and guilt. Each situation must be thought out and managed individually, taking into account the many needs of the child, the parents, and the siblings.

### Management of Siblings

Good medical management takes into account how the child's dying and death affect the child's siblings. Siblings of all ages need support and explanations, and the physician can help the parents provide them. The physician may suggest that the parents gather the family together and then give a simple explanation to all the children. The explanation should include the facts that Johnny is very ill, that everyone is doing his or her best to make the child as comfortable as possible, that the illness Johnny has could not be prevented, that it is no one's fault, and that it is necessary to figure out together how everyone can help. Later, individual children in the family may be given more information as they give evidence that they require it. If the siblings are told that death is near, they also will need help on how to conduct themselves in the presence of the dying child. The dying child needs their support, and they can give it by doing such things as making drawings for the child, bringing messages from others, and getting things he or she may need. If the dying child asks them whether he or she is going to die, they can say, "I don't know. I know it's a serious illness. Would you like me to ask Mommy and Daddy, or do you want to ask them yourself?"

When the child dies, to avoid hurt feelings, all the siblings should be told of the death at the same time if possible. A simple account of the death can be given if the children ask about it. It is better to avoid such statements as "He died in his sleep," especially when young children are present, because of the danger of engendering in them a fear of sleep.

A visit to the home by the physician is nearly always deeply appreciated by the parents and siblings of the dead child. If the physician has had a long-standing relationship with the family, he or she should ask about the funeral or memorial service and should attend the service. A physician who has provided extended care for a dying child may be remembered only for failing to attend the funeral service or convey his or her condolences.

Sometimes parents will ask whether a young sibling should attend the funeral. The physician first should decide whether the parents will probably be in control of themselves and who else will be present at the funeral who could support the child. For children younger than 5 years of age, the funeral can be a puzzling experience unless it is explained and unless a great deal of support is given by a familiar and caring adult. Attendance also depends on cultural practices. Children older than 5 years often can use the funeral rite in the same way adults do, especially if they have adults in attendance who also can explain to them their feelings and describe what is taking place. A child who does not wish to attend the funeral should not be made to feel guilty. Rather, arrangements should be made for the child to be in the company of an understanding adult during the time of the funeral. Older children should be encouraged to attend the rites and observe the rituals the adults are attending and observing because, again, these practices usually help the person to deal with the reality of death. If the older child chooses not to attend the funeral, the reason for the choice should be explored, but if the child continues to feel that he or she does not want to attend, this wish should be respected. Each person mourns in his or her own way. In no circumstance should the subject of the dead person be closed off. A wall of silence hampers the child as he or she struggles with the reality of the death and his or her feelings about death. Some of the specific ways of helping children understand the many facets of death have been described by [Wolf \(1958\)](#).

Other questions involving siblings may arise later. A younger sibling may ask to have some of the dead child's toys. The transfer can be done in a helpful way by suggesting that the dead child would have wanted his or her younger brother or sister to have his or her toys. Other decisions, such as rearranging the dead child's room or giving a sibling the dead child's room, might be deferred until most of the work of mourning has been done. Such decisions probably can be made on a rational basis then, when the mourners are less affected by their emotions.

### Requesting Autopsy Permission or Organ Donation

A difficult task for the physician is requesting autopsy permission or organ donation. Because of the difficulties, the request frequently is made in a hasty, tactless manner. The physician must be aware that many families have strong feelings against such procedures. For example, Orthodox Judaism prohibits the permanent removal of organs. Despite the physician's medical curiosity and zeal to learn, he or she must resist pressuring a family into agreeing to a procedure to which it objects. On the other hand, the physician may legitimately describe an autopsy to the parents as a postmortem internal examination that determines the cause of death and the effects of the treatment given. He or she can honestly present to them the potential benefits of an autopsy or organ donation to others. If parents ask whether the child will be cut open, the physician must answer honestly, even if the physician knows that the autopsy or donation request may then be refused. Families who refuse to agree to such procedures should not be made to feel guilty about their refusal.

## WHEN A PARENT DIES

### The Children

When a parent dies, the physician should help the surviving parent anticipate the reactions to be expected from the children. Children, particularly young children, are unable to tolerate—and therefore complete—the painful task of mourning the death of a parent. Sad feelings often are curtailed, and often the child quickly returns to everyday activities as if nothing had changed. Although the immediate manifestations of mourning in extremely young children usually are brief, it is difficult to assess the impact of a parent's death on the child's future personality development. Occasionally, a child may express hostile feelings toward the surviving parent. The child actually may be angry at and feel abandoned by the parent who died. Because such feelings usually are experienced as unacceptable, the child displaces them onto the surviving parent. The expression of hostile feelings toward the surviving parent unfortunately invites punishment when it is misunderstood. Frequently, a child, by virtue of his or her still somewhat primitive way of viewing the world, is convinced that he or she caused the parent's death, either by not being a good child or by having at one time or another wished the parent dead. When the child provokes the surviving parent, he or she may in part be seeking punishment to assuage feelings of guilt. Therefore, it is necessary to prepare a parent for these reactions as well as to attempt to correct the child's fantasies. Some families will benefit from participation in support groups for grieving children or families; recommendations for written resources for further information often are appreciated ([Emswiler and Emswiler, 2000](#)).

The child who has lost a parent is a child at psychiatric risk. After a review of the literature on bereavement in childhood, the Institute of Medicine ([Osterweis et al., 1984](#)) summarized the factors that are associated with an increased risk of psychological morbidity for children after the death of a parent or sibling:

1. Loss in a child younger than 5 years of age or during early adolescence
2. Loss of a mother for girls younger than 11 years of age and loss of a father for adolescent boys
3. Premorbid psychological difficulties in the child or lack of prior knowledge about death
4. When the relationship with the deceased had been conflictual or when the parent remarries and there is a poor relationship between the child and the stepparent
5. When the surviving parent is psychologically vulnerable and excessively dependent on the child, or the environment is unstable and inconsistent
6. When there is a lack of adequate family or community supports, or when the surviving parent is unable to access available supports
7. When the death was unanticipated or the result of suicide or homicide

Impairments in the child's capacity to form new, lasting relationships may show up later. Shame at being different may be experienced. Impaired sexual identity and conscience formation also may occur ([Bonnard, 1962](#); [Neubauer, 1960](#)). In addition, the loss of a parent during childhood may predispose a child to attempt suicide during adolescence.

The family disruption and the reactions of the surviving parent may lead to a depression in the child. The presenting symptom may be a school learning problem or a behavior difficulty. Another hazard that sometimes occurs is a morbid attachment of the surviving parent to a child of either sex, particularly an adolescent. The



adolescent in question may have great difficulty in separating from the parent or may develop along homosexual lines.

On the other hand, as development proceeds, the child may be able to continue the work of mourning on a piecemeal basis. As his or her cognitive capacity matures and reality testing is strengthened, the child may at some later date be able to express some of the feelings that he or she had earlier repressed. These feelings may include yearning and sadness, which may occur at times of special significance in the form of anniversary reactions ( Fox, 1985), as well as anger and resentment.

The physician can help most if he or she can enable the parent, who also is in a state of mourning and withdrawal, to recognize the needs of the child. The child needs to know that there is someone he or she can depend on to meet his or her needs, and to whom feelings can be expressed. In some instances, the physician may appropriately support the parent in this role by making himself or herself available to the child, if the parent agrees. The pediatrician should suggest a consultation with a child psychiatrist if there is concern about the child's behavior.

### The Surviving Parent

The death of a parent almost always disrupts a family. As much as possible, the physician should help the family to maintain its stability and to avoid making hasty decisions while the family is in a state of acute grief. The services of a relative or a homemaker may be helpful during this acute period. Some tact is required as the physician tries to steer a course that will not be experienced by the parent either as intrusive or as an abandonment.

The disruption in the family caused by the death of a parent is not confined to the period of mourning. Loss of income, reduction in the amount of time that can be spent with the children, changed roles for the surviving spouse, caretaking responsibilities for the older children in the family, and altered social relationships are some of the repercussions that continue to affect the family. The physician should remain accessible to members of the family. Sleep difficulties, psychosomatic disturbances, and school learning difficulties are some of the common signs of continuing distress that may require further psychiatric evaluation.

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# 105 EARLY CHILDHOOD EDUCATION

David Elkind, Ph.D.

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The theory and practice of early childhood education, like that of so many other disciplines, has a long past and short history. When education is taken in its broad sense, including everything a child is taught and learns during the first years of life, then early childhood education has existed for as long as humankind has been rearing children. However, in the narrow sense of education—nonparental instruction in knowledge, values, and skills—the education of young children is a relatively modern innovation.

We are concerned only with early childhood education in the narrow sense in this chapter. In the first section we look at some of the factors that have contributed to the rapid growth of early childhood education in modern times. In the second section we briefly review the contributions of some of the major figures in early childhood education as we know it today. In the third section we highlight the basic parameters of early childhood education. In the closing section, we look at some of the major mental health issues raised by early childhood education in the United States today.

## THE GROWTH OF EARLY CHILDHOOD EDUCATION

The growth of early childhood education in the modern era has many different roots. Darwin's theory of evolution, for example, transformed our ideas about the insane, the retarded, and children. Just as insanity and retardation came to be seen as natural phenomena, rather than as the work of the devil, children's behavior came to be viewed as reflecting their immaturity rather than their original sin. Humanitarian practices progressively replaced restriction and punishment for each of these groups.

Another factor that contributed to the growth of early childhood education was the establishment of socialist societies in countries such as China, Russia, and Israel. In these countries, the society rather than the family was to be the prime identity and allegiance of the individual. Accordingly, massive early childhood care programs were instituted, in part to enable women to join the work force, and in part to weaken the strength of family bonds. In many ways, these state-supported programs were large-scale experiments in early childhood education. Unfortunately, they were uncontrolled experiments, and we have no good way of assessing their effects.

In this country, a different type of social change eventually contributed to the growth of early childhood education, namely, the movement of farm families to the cities and factory work, as well as the waves of immigration around the turn of the century. Initially, in the United States, early childhood programs were introduced for the children of poor, two-parent working and often immigrant families. Many day schools, such as the "Home for Little Wanderers" in Boston, Massachusetts, were founded in major cities in the first two decades of this century.

Although programs for young children began as a service for working mothers, after World War I many of these programs were institutionalized and redirected to serve the children of single mothers or those of troubled families. Day care programs were thus associated with social pathology. As a consequence, day care centers acquired a social stigma. Children who were in out-of-home programs for a full day were looked on as unfortunate and underprivileged. The social stigma associated with day care is gradually being overcome, but some traces of that stigma may account, at least in part, for the continued resistance to early childhood education in some quarters.

Another contribution to the growth of early childhood education in this country was the nursery school movement. In contrast to the evolution of day care, the nursery school movement in America was directed toward children of middle-income parents. Between the two world wars, the nursery school movement flourished, and training schools for nursery school teachers, such as the Bank Street College of Education in New York, the Merrill Palmer School in Detroit, Michigan, and the Nursery Training School in Boston, Massachusetts, prepared young people to work with young children in nursery school settings.

In contrast to the day care programs, the nursery school program was a half-day affair and often involved the parents in one or another form of "guided observation." The contemporary growth of early childhood education has its roots in the nursery school movement, not day care programs. In fact, however, there is now so much overlapping that it would be hard to tell the difference between a good day care program and a good traditional nursery school program.

## MAJOR CONTRIBUTORS TO CONTEMPORARY EARLY CHILDHOOD EDUCATION

Many writers, both inside and outside the discipline of education, have contributed to the theory and practice of early childhood education. Among the most prominent of these are the contributors described in the following.

### Heinrich Pestalozzi (1746–1826)

Heinrich Pestalozzi was a Swiss educator who extended some of the ideas of the Enlightenment to early childhood education ( [Green, 1914](#)). The spirit of the Enlightenment was to look to experience, rather than authority, for true knowledge. Pestalozzi began several schools for children whom today we would call disadvantaged. None of these schools was very successful or lasted very long. This was probably more a result of Pestalozzi's limitations as an administrator than of the programs he initiated.

Pestalozzi was nonetheless a prolific writer and gave what is perhaps the first systematic approach to the education of infants and young children in his many books. In his most famous book, *Gertrude Teaches Her Children*, which takes the form of a series of letters to mothers, Pestalozzi described many exercises the mother could engage in with her child. These exercises were aimed at helping children develop their intellectual and physical abilities. These exercises were revolutionary in the sense that they took account of child development and catered to it. Up until that time young children were taught what adults thought they ought to know, namely, the catechism.

Although he was personally not successful in maintaining a school run according to his own principles, Pestalozzi did influence many parents and other educators,



who also began to look at what children needed to learn rather than at what adults wanted them to know.

### **Frederich Wilhelm Froebel (1782–1852)**

Frederich Wilhelm [Froebel \(1893\)](#) is generally regarded as the originator of early childhood education as a distinct field of pedagogy. Froebel believed that children could and should be taught outside the home as well as within it and that trained teachers should provide such education. He developed not only a curriculum for young children, but also a training school to prepare teachers to work with them.

Froebel is perhaps best known for his creation, kindergarten, (“children's garden”) where children could learn through play. Froebel recognized the educational value of certain toys and advocated toys and play as the appropriate way of instructing young children. His kindergarten included blocks, pets, and simple games. The toys Froebel introduced for educational purposes, such as a ball, he termed “gifts.” He believed that children could not only learn simple ideas such as “roundness” from playing with the ball, but also could begin to comprehend the shape of the earth and the concept of unity also implicit in the sphere.

### **Maria Montessori (1870–1952)**

The first woman in Italy to gain admittance to and graduate from medical school, Maria Montessori ( [1912/1964](#)), laid the foundations for early childhood education as we know it today. After graduating from medical school, Montessori worked with retarded children, for whom she designed some innovative learning materials. She was then invited to set up an educational program for young children in a low-income housing project. Using untrained teachers and modifications of the materials she had devised for retarded children, she created what is known today as Montessori education.

Like Pestalozzi and Froebel, Montessori began with observations of how children engage in learning on their own. From these observations she not only was able to choose materials that were uniquely suited to the intellectual needs of young children, but she was also able to describe some general principles of early childhood education. For example, Montessori believed that young children should always be exposed to the object, property, or quality before they are given the name for that object, property, or quality. Put differently, she believed that sensory learning should precede symbolic learning.

Many of Montessori's innovations have become part of accepted early childhood teaching practice. For example, like Froebel, she believed that early childhood teachers had to be specially trained, and she worked out an elaborate training program of teacher preparation. Today, Montessori-oriented teacher training is one of the more rigorous early childhood teacher training programs in this country and abroad. Montessori teachers are particularly well schooled in the use of manipulative materials with young children.

In addition to rigorous teacher training, Montessori also introduced what are now staples of most early childhood classrooms—child-sized tables and chairs, dishes, and utensils. Other curriculum innovations were form boards, sandpaper letters, button and shoe lacing boards, and much more. Montessori believed that if the environment were properly prepared with the right learning materials, then children would spontaneously move to these materials and extract the most educational benefit from them. Montessori programs have multiplied rapidly since the 1960s and are now in all of the states and many public schools.

### **John Dewey (1858–1952)**

Although trained as a philosopher, John Dewey became America's first original educator. Like Pestalozzi, Froebel, and Montessori, Dewey believed that childhood was a unique stage of life that should not be rushed. In addition, he believed that education should be practical—his brand of educational philosophy has been called *functionalism*—and should prepare children for the life they will experience outside of school. Although Dewey was not opposed to classical education at some levels, he believed that children also should learn more practical skills ( [Dewey, 1916](#)).

Dewey contributed to educational philosophy and introduced instructional innovations. Perhaps the most well known is what has come to be called the *project method*. Rather than teach subjects as separate and apart from one another, Dewey believed they could be taught in an integrated way if children were engaged in particular projects. In putting on a play, children would have to learn the lines (reading and vocabulary), build the sets (measurement and carpentry), and design and sew the costumes (history, measurement, and sewing), as well as follow instructions and work cooperatively (social skills).

Dewey's ideas and methods became a major movement in the United States that lasted until the mid-1950s and was called *progressive education*. It disappeared from the scene for many different reasons, not the least of which is because, in distorted form, it had become associated with “permissiveness” ( [Cremin, 1961](#)).

### **Sigmund Freud (1856–1939)**

Sigmund [Freud's \(1938\)](#) discoveries of infant sexuality, the stages of psychosexual development, and the Oedipus complex have had a significant impact on early education. Although most of what Freud had to say about early childhood dealt with parent–child relationships, there were some implications for out-of-home care as well. This is particularly true with respect to children's play.

Whereas writers such as Froebel and Montessori saw play as an all-important mode of learning for young children, Freud saw play as comparable to a dream. Like a dream, play has both a *manifest* and a *latent* content. Children's play can reflect inner complexes and conflicts and also be a way of working them out. A child who has just returned from the doctor, for example, may play at being doctor as a way of relieving some of the anxiety aroused by that visit. The technique of play therapy has grown out of this recognition that children's play can have symbolic meaning. It is now generally recognized that the play of young children has both *cognitive and affective* value.

### **Erik H. Erikson (1902–1994)**

Erik H. Erikson was trained as a child analyst but later departed significantly from analytic theory ( [Erikson, 1950](#)). In his writings, Erikson focused on the social dimensions of development and the evolution of such social orientations as trust and identity. Four of Erikson's eight stages of development occur in early childhood.

We are born with a number of social potentials in Erikson's theory. How these potentials are realized depends very much on the sort of experiences to which we are exposed, as well as our own unique characteristics. Each potential exists as a kind of polarity and has a particular time when the balance between the two opposing tendencies will be determined (although changes can occur at later ages as well).

To illustrate the relevance of the Eriksonian stages of early childhood education, we can look at the stage of initiative versus guilt, which has its critical time around the ages of 4 and 5. If the child is given the opportunity to explore the world and undertake projects on his or her own, the child will develop a sense of initiative that is stronger than the sense of guilt. In contrast, if the child is constantly told what to do and corrected if he or she makes mistakes, then the child will acquire a sense of guilt that is greater than his or her sense of initiative.

Erikson's theory, then, has rather direct implications for the practice of early education, as well as child rearing.

### **Jean Piaget (1896–1980)**

Although he was trained as a philosopher and biologist, Jean Piaget's greatest contributions were to developmental psychology. Piaget elaborated a theory of intellectual development that has influenced many different disciplines, including early childhood education. Although [Piaget \(1970\)](#) himself wrote about education in general terms, his work has had a major impact on the pedagogy of young children.

From a theoretical point of view, Piaget's work has emphasized the importance of development and the fact that learning cannot accelerate growth. Piaget's theoretical work has been a major factor in the current effort by early childhood educators to get schools to provide “developmentally appropriate” practice for young children ([Bredenkamp, 1987](#)). In addition, Piaget's work on the development of children's understanding conceptions, such as numbers, has led to major curriculum innovations ([Kamii, 1982](#)).

## Lev Vygotsky (1896–1934)

Although Lev Vygotsky ([Kozulin, 1990](#)) was a contemporary of Jean Piaget, his work is becoming generally known in America only today. Thanks to changes in Russian policy, his works are being made available and translated at an increasing pace. Although he worked in many different areas of psychology, he is perhaps best known for his contributions to our understanding of the relationships between language and thought.

Vygotsky argued that there were two levels of development: biological, which we share with animals and sociological, which is unique to humans. Both animals and humans, to illustrate, employ tools or implements to attain desired goals—a process Vygotsky described as mediation. However, only humans use language as a mediator; language is acquired socially and not as a biological given. Vygotsky believed that thought and language initially were a single undifferentiated activity and that they became separate only later in development. Initially language is entirely social, derived from hearing adults speak. As children develop, language becomes increasingly internalized (whispering is a step along the way) and individual rather than social.

Vygotsky, like Piaget, believed that children construct and reconstruct their ideas until they approximate those of the adult world. Nonetheless, he also contended that when adults mediate the child's learning, the child will arrive at the socialized concept earlier than if he or she has no adult model. Observing a parent use a spoon, for example, enables the child to use a spoon earlier than if he or she had never been witness to a model. The difference in time between the child's spontaneous attainment of a concept, and the time when the concept is attained with adult mediation is the "zone of proximal development." Vygotsky, like Montessori, believed that children could progress more effectively with a prepared environment and adult modeling than if left to their own spontaneous devices.

It is important to emphasize that Vygotsky was not arguing that development could be quickened with adult intervention. Perhaps his position is best understood in contrast to that of Piaget. In general, Piaget believed that children spontaneously maximize their abilities given an average family environment. Vygotsky, in contrast, argued that children without adult mediation do not maximize their abilities but can quicken their development with the aid of adult mediation. There is a zone of undeveloped capacity that can be achieved with adult intervention, but this zone is limited by the child's native capacity.

## MAJOR PARAMETERS OF EARLY CHILDHOOD EDUCATION

Every system of education presupposes a set of assumptions regarding the learner, learning process, knowledge to be acquired, and aims of education. Contemporary early childhood education in America can be described within these parameters. It must be said that this description represents the views of mainstream early childhood education and does not purport to convey the many varieties of early childhood education currently available.

The discussion of the parameters of early childhood education has implications for dynamic theories and therapy with young children. For example, any theory or therapy applied to young children must take the child's developing mental powers into account. The therapist must understand the child's level of mental ability, what the child can and cannot do cognitively. A case in point is the writings of Melanie Klein. Although her books have raised important issues and concepts, she nonetheless seems to attribute too much to the young infant in the way of cognitive processes. For example, [Klein \(1957\)](#) suggests that young infants have aggressive fantasies. Yet [Piaget's \(1951\)](#) work demonstrates that children do not really have images before the second year of life. Inasmuch as fantasy is comprised of images, the child could hardly engage in fantasy activity before he or she was capable of constructing images. From a developmental point of view, Klein is attributing too much mental ability to the young infant.

### The Learner

Young children are growing individuals first and foremost. From this point of view, all children are seen as having the same basic learning abilities, which they may nonetheless attain at different ages. For example, we expect that all children (with the exception perhaps of the extremely retarded) will eventually attain the mental abilities that [Piaget \(1950\)](#) calls *concrete operations*, which enable young children to reason in a syllogistic way and follow rules. Although most children acquire these operations at about the age of 6 or 7, bright children may acquire them at the age of 4, whereas slow children may attain them at the age of 8 or 9.

The view of children as endowed with developing abilities has many educational implications. It is important to match the child's developing mental abilities with a curriculum that is nicely suited to those potentials. Curricula have to be studied to determine their "developmental difficulty," the level of mental ability required for their comprehension. For example, instruction in phonics requires children to have attained the concrete operations described earlier. This is true because children require these abilities to grasp that one and the same letter can represent different sounds and that different letters can represent the same sound. Teaching a child phonics before he or she can grasp that one and the same thing can be two things can be frustrating and demoralizing.

### The Learning Process

Learning is always a creative activity from a developmental point of view. The child never simply copies information from the external world but always transforms it in significant ways. The Connecticut youngster who heard the Lord's Prayer as, "Our Father who art in New Haven, Harold be thy Name" is not the exception but the rule. In effect this means that we cannot really separate the learning process from the content to be learned. Every different content engages us in a unique way. There are no disembodied learning principles that operate entirely apart from the content to be learned. Learning is always content oriented.

Here again this view of the learning process differs from the view of learning held by those who educate at the primary and secondary levels. At those levels there is a strong belief in the separation of learning and content. Currently, for example, there is a great deal of interest in teaching "thinking skills" ([Baron and Sternberg, 1987](#)) and "learning strategies" ([Weinstein and Mayer, 1986](#)). Such efforts reflect the belief that the learning or thinking process can be taught and learned apart from specific contents.

The issue here is what has been called transfer of training. To be sure, the developmental approach recognizes that such transfer occurs. However, those who wish to teach thinking or learning skills assume that transfer is automatic and hence unconscious. A developmental approach, which argues for the necessity of content for the operation of learning and thinking, insists that transfer can only occur when the learner is consciously aware of content and is making the effort to generalize.

On the other hand, there is a growing recognition of the importance of individual learning styles; for example, some children learn best through the visual modality, others through the auditory ([Elkind, 1994](#)). Learning styles, in contrast to thinking skills, are individual differences that transfer from one type of learning task to another. Here again, however, the identification of learning styles means that we have to adapt our materials to the child's learning mode, not decide on our own what skills they should learn.

From a therapeutic point of view, this conception of the creative nature of learning means, for example, that the child's language always contains individual symbolic significance. In this respect, the developmental approach to learning would be in keeping with the creative role attributed to language by Jacques [Lacan \(1968\)](#).

### The Nature of Knowledge

Knowledge is always a construction from an early childhood education perspective. That is to say, reality as we know it is never simply a copy of the external world but rather is a construction or creation that bears something of ourselves and something of the external world and that cannot be reduced to either one. To be sure, we all have the same sensory apparatus, and there are constants in the external world; therefore, our individual realities are never entirely egocentric. Nevertheless, we never see the world exactly the same way everyone else does.

To this individual difference dimension we must add the developmental one. As the child matures, so too do his or her mental abilities. As a consequence, the child must construct and reconstruct reality during the process of growing up. The same holds true for our memory of past reality. The child reconstructs the past each time he or she attains a new, higher level of mental ability. Thus, memory is never a simple recall of previously stored images, but rather is an active, constructive process that transforms the information derived from earlier experience.

This way of viewing knowledge and memory implies an approach to curriculum different from that which is currently taken in our schools. The idea that knowledge is tied up with thought and action and should not be separated, as was first put forward by John [Dewey \(1916\)](#) and reiterated by Vygotsky. As mentioned, in the project method, children engage in putting on a play, building a house, and so on. In the process they learn language, math, science, social cooperation, and much more.



The project method speaks to the conviction that content is an integral part of the learning and knowing process and should not be separated from it.

From a therapeutic point of view, the idea of reality as a construction again has important implications. It suggests that realities can be different and not just right or wrong. The young child who believes that adults are all-powerful and all-knowing has a different reality from the adult, but it is not wrong from a developmental point of view. Indeed, that conception is as developmentally appropriate as the more negative perception of adults held by adolescents.

The rule that there can be different realities without these being necessarily right or wrong is as important in therapy as it is in education. A child's fear of a particular animal is very real for him or her, even though it appears baseless to us. Rather than try and disabuse the child of this fear, we need to accept it as a reality for the child and reassure the child that we will not allow the animal to do him or her any harm. On the other hand, there are certainly some beliefs that are right or wrong (e.g., the Bastille was stormed in 1789 and not in 1960), and there are some evil realities. The difficult thing is always to make the right discriminations as to when we should bring in the value judgments of right or wrong, good and evil.

## THE AIMS OF EDUCATION

The aims of early childhood education have been clearly enunciated by Jean Piaget:

The principal goal of education is to create men who are capable of doing new things . . . men who are creative, inventive and discoverers. The second goal of education is to form minds which can be critical, can verify, and not accept everything that is offered ( [Ripple and Rockcastle, 1964](#), p. 18).

According to Piaget, the legitimate aim of early childhood education is to produce self-motivated learners and critical thinkers. From a therapeutic point of view, this aim suggests that therapy be directed not solely at removing this or that problem but also at helping the child acquire an orientation and strategies that will enable him or her to deal with the later stresses of life more effectively and productively.

## ISSUES IN EARLY CHILDHOOD EDUCATION

There are many different issues currently being debated regarding the education of young children. Only a few of these can be dealt with here.

### The Effect of Early Childhood Education on the Infant

Today 57% of mothers with children under the age of 6 have jobs. In addition, more than half of the mothers with infants of less than 1 year of age are employed. This means that a large percentage of infants and young children are being cared for by relatives within the home or by paid caregivers outside the home. What effect is being cared for by nonparental figures going to have on these children? There has been a longstanding debate on this issue. For example, John Bowlby wrote:

The absolute need of infants and toddlers for the continuous care of their mothers will be borne on all those who read this book. . . . We must recognize that leaving any child under three years of age is a major operation to be undertaken with good and sufficient reasons ( [Bowlby, 1950](#), p. 16).

Many contemporary researchers do not agree, however. They find that the critical issue is not the parent as caregiver but rather the *quality* of the care the child receives. There is no evidence of lasting untoward effects when an experienced, warm adult cares for a child; one adult cares for not more than three infants; and the environment is safe, pleasant, and interesting. According to one investigator, with quality child care of the sort described in the preceding, "the major effect of day care seems to be a speeding up some of the child's social and intellectual skills during the preschool period" ( [Clarke-Stewart, 1982](#), p. 75). Likewise, the authors of a leading child development textbook conclude their review of the literature with the following statement:

There is little clear evidence that day-care is likely to cause disruption of the infant-parent attachment relationship. In fact, day-care may have positive effects for some infants, particularly when it is of high quality ( [Hetherington and Park, 1993](#), p. 43).

The real issue for contemporary working parents is to find quality, affordable childcare. With such care, there is no evidence of any immediate or lasting harm to young children. However, there is one important caveat. It does appear that children in child care settings, particularly large day care settings, are more subject to communicable diseases than children cared for at home or in the home of the caregiver.

### Does Early Childhood Education Increase a Child's Intelligence or Give Him or Her an Academic Head Start?

One of the questions usually raised in relation to early childhood education is whether it will raise the child's IQ. This question is interesting because it is rarely, if ever, raised with respect to elementary or secondary education. If neither elementary nor secondary education is expected to raise a child's IQ, why should it be expected of early childhood education? Indeed, there is no evidence that early education raises a child's IQ on any permanent basis.

It is still possible, however, that early education can give children who have it an edge over those who do not. Does this in fact occur? Certainly some high-quality age-appropriate programs for disadvantaged children seem to have demonstrated long-term positive benefits ( [Schweinhart et al., 1985](#)).

However, these results cannot be generalized to all programs and to all children. A simple analogy may help to make the dangers of such generalizations concrete. Assume a group of children who are badly undernourished and who are well below their age norms for height and weight. If these children are now placed on a full-calorie, nutritionally well-balanced diet, they will make quite noticeable progress in height and weight in a reasonably short period of time. If, however, you take a group of children who are already well nourished and at or above the norms for height and weight for their age group, a comparable regimen will have little or no effect.

Does this mean that advantaged children receive no benefits from early childhood education because it merely duplicates what they have at home? Not at all. The issue here is comparable to the one raised about home schooling. Yes, parents can and do successfully school their children at home, even through high school. This does not signify that early childhood programs are unimportant. It does mean that parents who have the time, energy, and commitment to home school can do so successfully. However, parents who do not have the time, energy, and commitment to home school do need the public education to fill that role.

Exactly the same holds true for early childhood education. If parents have the time, energy, and commitment to provide an intellectually challenging environment at home; spend regular periods of time with the child reading, talking, and engaging in other activities; and have other children in the neighborhood over on occasion, there is really no need for the child to attend a preschool. But if the parent is not ready or able to devote the time and energy to providing that kind of experience, then a good preschool experience would be enriching and rewarding for the child.

### Is Earlier Better?

The idea that earlier is better when it comes to early childhood education is widespread. It stems from the 1960s, when many writers hoped that early childhood education might be the way to break the cycle of poverty. It was hoped that if disadvantaged children could be given a head start at the preschool level, then they would be on a level playing field with advantaged children when they entered school.

The promise of early childhood education seemed unlimited at that time. Jerome Bruner wrote that "we begin with the hypothesis that any subject can be taught effectively in some intellectually honest way to any child at any stage of development" ( [Bruner, 1962](#), p. 22). It was hoped that if children were taught the basics of reading, writing, and arithmetic during the early years, then this would not only ensure them a head start but also insure them against failure later.

The idea that earlier is better was reinforced by the writing of Benjamin Bloom, who contended that "general intelligence appears to develop as much from conception to age four as it does during the fourteen years from age four to eighteen" ( [Bloom, 1964](#), pp. 207-208). Bloom went on to argue that because intelligence grows so rapidly in early childhood, it is the time to engage in serious instruction rather than play. The idea that little children are sponges ready and eager to soak up all the information we pour over them became a widespread and accepted conviction. It encouraged many parents to try to teach their infants and young children everything from reading to violin to karate.

However, is early childhood a critical period for learning basic skills? It is certainly true that infants and young children are eager and avid learners. Yet what young children need to learn is not academic subjects and skills but rather the basics of the world in which they live. Young children need to learn sights, colors, sounds, shapes, textures, and tastes. They need to discover *up* and *down*, *behind* and *on top of*. They need to discover what floats and what sinks, and encounter the many forms, sounds, and smells of different animals and plants.

Yes, infants and young children are avid learners, but they are eager to learn from their own explorations and discoveries and not from formal instruction. That is what all of the workers in early childhood education have emphasized, from Pestalozzi to Piaget. The child needs to acquire a conception of the immediate fundamental world before he or she begins to learn the derived, symbolic world of formal instruction.

#### *PROS AND CONS OF UNIVERSAL PRESCHOOL*

There is currently much discussion about making preschool education available, if not mandatory, for all young children. In some states, such as Georgia and New York, such programs are already in process. Universal preschool has long been a national policy in many countries. In France, for example, state-supported preschools for all children age 3 and older have been in operation for decades. Likewise in China, state-supported preschools are provided for children as young as 1 year of age. In both of these countries the initial impetus for supporting universal preschool was the need for quality childcare, as large numbers of young mothers entered the work force. This has changed, however. French preschools are heavily academic and early education is now regarded as part of the education system. China has not moved in this direction; their preschools are still primarily dedicated to childcare.

There is certainly a need for universal preschool in our country. Currently more than 60% of mothers with infants younger than 1 year of age are in the workforce either full or part time. Unhappily, we do not have the quality, affordable, and accessible childcare needed by these mothers and those of older preschoolers. Much of what is available is substandard because preschool teachers are poorly paid and often relatively untrained. The need for quality early childhood care is becoming even more pressing now that so many welfare mothers are joining the work force. These mothers can hardly afford to pay for high-quality childcare because they are in low-level, or entry-level jobs.

Universal preschool supported by the state makes sense for all of these reasons—particularly so, if teacher training and teacher pay are placed on a par with that for elementary and secondary schoolteachers.

The risk, with universal preschools, is what has happened in France. That is, what was originally a childcare initiative has become an educational initiative. Young French children are taught reading and math in the manner of grade school children. Many early childhood experts fear that what happened in France will happen in the United States. That is to say, developmentally appropriate early childhood programs will be transformed into academic programs. Quality early childhood education programs are academic, but in a hands-on rather than a symbolic way. Much damage can be done if the all-important distinction between hands-on learning and symbolic learning is lost, and symbolic academic learning is pushed down to the preschool level. To illustrate, children have many more reading problems in France than they do in the Scandinavian countries, where reading is introduced at a later age level.

Accordingly, universal, state-supported early childhood education is a good idea and would fill a very dire need. The real danger is that if it is not done right, if it becomes a size smaller first grade, many children will be put at risk for learning disabilities.

#### *COMPUTERS AND YOUNG CHILDREN*

Computers are here to stay, unlike many other educational innovations, such as overhead projectors and teaching machines. They are becoming an increasing part of our environment and impact all facets of our lives. Nonetheless, the personal computer is still a very complex machine that does not always work the way you want it to. There is considerable debate about whether or not young children should be exposed to computers. Some argue that such exposure takes children away from the hands-on learning so critical for young children. Others claim that computers are part of the world and that young children have a natural affinity for them, and should not be kept from using them.

This argument is academic in some ways. Many parents I have talked to tell me their children naturally gravitate to the computer when they themselves are working on them. These are the parents who are likely to buy computer programs for their young children and even give them a secondhand computer when the parents move up to a more powerful model. So, regardless of what the experts think, many young children are being exposed to computers. It is my impression that this is happening in numerous early childhood centers as well. Many early childhood programs now have one or more computers available for young children to use. That is certainly true for the Children's School and Educational Day Care Center at Tufts. Teachers have chosen what they regard as quality programs and the children are allowed to spend some time on them each day. Some children seem to be more drawn to computers than others.

In general, it seems that after the age of 3 or 4 there is little harm to exposing young children to computers so long as they do not spend too much time on them. It is important to recognize that we still do not know a lot about children and computers and that it is important to watch how they use them. I am often impressed that young children like to work at the computer in groups helping one another along with the program. It would be a mistake for a teacher to interfere and insist that the children take turns. We can learn much about computers and young children if we allow them the freedom to experiment and explore on their own.

Accordingly, computers do have a permanent place in early childhood education if we are careful about the programs we choose for young children and make sure they do not spend too much of their time at them.

#### **SUMMARY AND CONCLUSIONS**

Early childhood education is a relatively modern innovation. It had its origins in the industrial revolution, humanitarian thrust of the theory of evolution, and emergence of socialist societies and communal child rearing. In the contemporary United States, early childhood education has grown to accommodate the needs of two- and single-parent working families, as well as to provide a head start for disadvantaged children.

There are four basic principles of developmental early childhood education. The first is that the learner is an individual with growing mental abilities. A second is that learning is a creative process and necessarily involves both the subject and object or material to be learned. The third is that knowledge is always a construction and always contains something of the subject as well as the material to be known. Finally, the aim of developmental early childhood education is to produce active, self-motivated learners and critical thinkers.

There are many unresolved issues surrounding early childhood education. Among these are the questions of whether early childhood education can harm the child, improve the child's IQ, and give the child a head start.

Early childhood education is coming into its own in the United States as a part of the educational system. It is increasingly recognized that early childhood education is not a "size smaller" first grade; that young children learn differently from older children and adults; and that they require their own curriculum, teaching modes, and method of evaluation. Early childhood education is coming to be accepted on its own terms.

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## 106 IMPROVING PSYCHOEDUCATIONAL OUTCOMES FOR AFRICAN-AMERICAN CHILDREN

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[Relationship Dynamics of Children Developing in Families, Social Networks, and the School](#)  
[Economic and Social History: European, Asian, and African-American Experiences Compared](#)  
[Yale School Development Program](#)  
[Role of the Child and Adolescent Psychiatrist](#)  
[Conclusion](#)  
[Chapter References](#)

There has been a dramatic increase in problems facing families and children in the inner cities of the United States during recent years. These social difficulties are having a significant negative impact on the school readiness and school success of African-American children and are resulting in an urgent need to improve psychoeducational outcomes for these children. Indeed, providing low-income African-American children with a successful school experience is one of the most important tasks facing the United States today. If these children remain undereducated in disproportionate numbers in this age of science and technology, all our most troublesome social problems will be exacerbated—low productivity, dependency, substance abuse, crime, children having children, and others. Across the country, in areas where such children are located in disproportionate numbers, they are often up to 2 years behind children from better-educated families. In large urban centers, school dropout rates are as high as 50% ([Children's Defense Fund, 1989](#)). Evidence indicates that these youngsters can achieve at a much higher academic level ([Comer, 1980](#); [Comer, 1988](#); [Comer, 2001](#)).

Although it is more difficult to document, some social and behavioral scientists and educators believe that many African-American young people from better-educated, middle-income families may not be achieving at the levels of their ability because of negative peer pressure, as well as insensitive schooling practices ([Ogbu, 1986](#)). If this is the case, the leadership group needed to improve opportunities for all African-Americans, which, in turn, would benefit all Americans, will not grow sufficiently rapidly or large enough.

Several causes are commonly suggested for the educational underachievement of low-income black children: school segregation, inadequate schools, disparity in instructional materials, inadequate attention to language and cultural considerations, low teacher expectations and bias, too few educational role models such as teachers, continuing insensitive schooling practices, and unemployment and its detrimental effects on families ([Boykin, 1983](#); [Clark, 1983](#); [Haynes, 1993](#); [U.S. Department of Health and Human Services, 2000](#)).

Some African-American children achieve despite these obstacles. In 1939, one of us (Comer), then a student, entered an elementary school in East Chicago, Indiana, with three other black youngsters from a low-income community. The school was racially integrated, served the highest socioeconomic group in town, and was considered one of the best. All four students were from two-parent families. All four fathers made a living wage as laborers in the local steel mills. Despite similar intellectual potential, all three of Comer's friends had difficult educational and life outcomes—one died prematurely of alcoholism, a second spent a significant part of his life in jail, and the third has been in and out of mental institutions.

The reason for the more favorable outcome for Comer was largely that his parents provided him with a preschool experience that gave him the confidence and kinds of skills necessary to elicit support in school. His parents interacted with school people in a way that encouraged support for him. Beyond home and school, a constructive social network of friends and kin, religious and other institutions, helped to make academic success possible for him.

Although another of them (Haynes) was raised in another culture, Trinidad, where racism and segregation were not part of his educational experience, but class distinctions were, he also found that his mother's understanding of the importance of positive social relationships and the existence of a supportive network of caring adults and good friends were critical in his educational and social experiences. The notion that *it takes a village to raise a child* was very much true.

A similar experience is usually found among African-Americans who have had academic success. Many, perhaps most, of today's educated, middle- and upper-income African-Americans are from low-income backgrounds ([Jaynes and Williams, 1989](#)). Most educated blacks who are more than 45 years of age, and a large percentage of the leadership group, attended racially segregated primary and secondary schools, even postsecondary schools ([Jaynes and Williams, 1989](#)). Yet many black children have not done well in racially integrated schools, even schools considered very good and serving middle- and upper-income families ([Ogbu, 1986](#)). At the same time, although unemployment is a serious problem and has probably served to increase the number of single-parent families, some black children of unemployed parents and low-income families do reasonably well in school ([Clark, 1983](#)).

How are we to explain the underachievement of American-born black youngsters from middle- and upper-income families ([Fordham, 1988](#))? This is especially curious in view of the finding that West Indian children do as well as Asian and white children. Our usual explanations for school underachievement among African-American youngsters are simplistic, adynamic, and not very useful.

In the 1960s, in part because of his own experience, Comer began to speculate that the relationship experiences of students before school and in school, and relationships between parents and school staff, affect the child's development and, in turn, the child's ability to achieve in school. This gave rise to the hypothesis that the almost exclusive focus of schools on instruction and curriculum and the comparatively little attention given to issues of relationships and development are probably at the root of the academic underachievement of many students.

Moreover, because of his own experience and his psychotherapeutic work with middle- and upper-income black children, Comer became aware of the troublesome racial identity problem that such young people face. Often without conscious intent and because of historical situations and conditions, U.S. society attributes characteristics to African-Americans or blacks such as bad, unsuccessful, having limited interest in academic learning, and the like.

Being white or a member of the majority and more powerful group in the society—even when not in a majority in a particular situation, such as school—is characterized by almost exactly opposite traits: characterized as being good, successful, highly motivated to learn, and the like. Identity as a black person, then, can be problematic ([Comer, 1972](#); [Oyserman, 1999](#)).

Slavery in a society committed to Judeo-Christian principles and democracy had to be rationalized. The argument was that blacks were inferior; thus, slavery was justified. This notion has been transmitted across time. As a result, many mainstream organizations and people are carriers of negative and stereotypical ideas and attitudes about blacks. The mass media transmits the message ([Luthar et al., 1997](#); [Watson, 2000](#)). In addition, too many teachers, health care workers, other service providers, and the general public are carriers. African-American children are aware of these stereotypes and are adversely affected by them, especially in terms of their self-evaluations and educational aspirations. Young African-American male students appear to be especially at risk.

Young people develop best when they experience positive, self-affirming relationships. The skills they can then develop provide them with a chance to function well. This, in turn, creates a sense of adequacy and belonging: These are the critical dynamics, regardless of age, sex, race, or class. We hypothesize that the effectiveness of our social institutions, especially our schools, is better based on the degree to which these self-affirmative relationships are fostered and encouraged.

Why has society largely ignored relationship issues in its efforts to improve education, particularly that of minority children? In part, it is because educational theory is influenced more by industrialization than by child development and the social and behavioral sciences. Society tends to understand teaching and learning as a mechanical process. Thus, academic learning seems to have been thought of as an entirely cognitive (mechanical) process, willfully engaged in or rejected by the individual and facilitated or limited only by the individual's ability level.

In addition, social scientists usually analyze the behavior of societies, groups, and individuals in the here and now, as if it were unrelated to their histories. The impact of powerful structural forces created by political, economic, social, geographic, and other conditions is often ignored. The effects of these forces, positive and negative, are transmitted from generation to generation—parent to child—greatly influencing child development, subsequent social and academic performance in



school, and opportunities in life ([Clark, 1983](#); [Fiscella et al., 2000](#)).

Finally, society's neglect of these effects results in part from societal guilt relative to the experience of Native Americans, Hispanics, and African-Americans in particular ([Helms and Carter, 1990](#); [Steele, 1991](#)). Even the European and Asian immigrant experience and the experience of groups isolated from the positive effects of scientific and technologic changes (and not all were positive) have not been adequately explored to help us understand individual, group, and societal functioning, teaching, and learning in school. It is necessary to consider the effects of the black experience on the functioning of the community and individuals to understand the obstacles and opportunities involved in the education of African-American children ([Anyon, 1997](#)).

We need an understanding of learning and a method of teaching that look beyond the child in school and that consider the complex interactions of children developing in families and family interactions in social networks within a larger society. This perspective must include an understanding of the force of past social and economic conditions on the present. Thus, in this chapter, we (a) discuss the relationship dynamics of children developing in families, social networks, and the school; (b) explore the relevant economic and social history of the United States and compare the differences among European and Asian immigrants and African-Americans and the corresponding psychosocial consequences; (c) present a discussion of a specialized child development program in schools as a basis for addressing the complex problems involved; and finally, (d) consider the role child and adolescent psychiatrists can play in influencing practices and policies relevant to the education of African-American young people.

## RELATIONSHIP DYNAMICS OF CHILDREN DEVELOPING IN FAMILIES, SOCIAL NETWORKS, AND THE SCHOOL

Children are born totally dependent, with biological potentials that must be developed and aggressive drives that must be channeled and sublimated into the energy of learning, work, and play. They are also born with a capacity to form relationships. When competent caretakers (usually parents) provide for them, a strong emotional attachment and bond develop between the child and the caretakers. This bond enables parents and other caretakers to help children to grow along many developmental pathways ([Bowlby, 1952](#); [Stern, 1977](#)). At least six pathways are critical for future academic learning: physical, social–interactive, psychoemotional, moral–ethical, speech–language, and intellectual–cognitive. Whereas development along the pathways takes place simultaneously and each facilitates the other, the growth of the brain is critically important. Brain growth is facilitated by child interactions with people and things in emotionally powerful environments ([Greenspan, 1997](#)).

Families are enmeshed in a social network of friends, kin, and religious and social institutions ([Erikson, 1963](#)). Social networks or groups have a history that influences their attitudes, values, and ways. However, in large, modern societies, families live in primary reference groups or subcultures as well as the larger mainstream culture. Generation after generation, mainstream parents interact with their children from birth forward and transmit attitudes, values, and ways that make it possible to participate successfully in the mainstream as children and as adults. Marginalized or nonmainstream parents often transmit attitudes, values, and ways that enable their children to function in the subculture or primary reference group but do not adequately prepare them to participate in the mainstream culture.

The school is a mainstream institution. When children's growth is adequate along critical developmental pathways in their family and social network, and the culture and expectations of the network are similar to those of the school, most children are adequately prepared to achieve in school at the level of their ability.

Development along critical pathways takes place through incidental experiences that children have with their caretakers, as well as through systematic teaching efforts on the part of the adults around them. For example, a 2-year-old child who wants to play with the ball of another child does not know that he or she cannot just take the ball. He or she may attempt to do so, and a struggle or fight may ensue. A caretaker intervenes and mediates. In the process, the caretaker spells out several options for the aggressor—do something else until Johnny is through playing with the ball, play with Johnny if there is an interest on his part, or just go away.

In this single incident, the child learns appropriate social interaction. The impulse to hit or take is controlled, and sometimes the caretaker must help the child to handle emotions or feelings related to being denied. What is right and wrong is learned. Language is involved, and thinking is involved. In the process, the caretaker is helping the child to grow along the critical developmental pathways.

The interaction most closely related to academic learning is reading. Parents who are part of the mainstream of the society usually read to their very young children. They often do so at the end of a busy day, and the child has the parent's total attention. Reading, then, is a special time and becomes a positively charged emotional experience. Children's stories often deal with the fears and anxieties they experience while attempting to cope, with few skills, in a threatening world. Thus, children want to hear the stories repeatedly. Eventually, they memorize the words and associate them with the pictures on a page. Delighted parents, and other important adults in their lives, often excitedly express their approval.

Children are programmed to seek the approval of adults. With approval, they want to read more and to master other aspects of their environment to win the recognition of important adults around them. The motivation for learning and mastery, then, grows out of early relationships with important adults. The attitude about learning and the positive learning experiences with parents and other primary caretakers at a very early age become the basis for a comparable reaction with other adults, particularly school people, later in the development of the child ([Elias et al., 1997](#)).

Children who have been living in average, expected mainstream environments approach the school experience, and the adults involved, with attitudes, values, and ways similar to those expected in school and with development at a level necessary to meet school tasks. Such children are able to interact appropriately with other students. They are able to sit still and to listen or attend to a task when it is appropriate to do so. They are able to be spontaneous and curious when it is appropriate.

Such behavior elicits a positive response from adults in the school environment and promotes positive transference between the child and school people. This permits a positive attachment and bond to take place between the child and school people, similar to, but not as strong as, the attachment and bond that took place at home between the child and the primary caretaker. The child then has the same need to please school people as to please parents at home. This enables school people to help students continue to grow along the critical developmental pathways mentioned earlier. Growth facilitates academic learning of increasing complexity.

The utility and benefits of academic learning accrue. Eventually, the child develops an inner motivation to learn and to achieve, and it is as powerful, if not more so, than the outside approval of teachers, other adults, and other external rewards such as money or gifts. Learning to read is the critical academic challenge because all other academic work requires this skill. In addition, reading success leads to the sense of confidence and competence that children need to be motivated to interact with other students and staff appropriately. Reading difficulty or failure often promotes troublesome feelings and behaviors among children, such as acting-up and acting-out behaviors that make matters worse.

The preschool and school developmental experience is often different for children growing up in families that are marginal to and outside of the mainstream of the society and often under economic, racial, or other stresses. The relationship processes in such families less often lead to school success.

Income that is not adequate to enable heads of households to meet basic family needs is a source of excessive stress. Negative societal attitudes about minorities and poor people are stressors. They contribute to the increased frequency of divorce and to never-married single-parent families, which are, in turn, a major source of poverty. Children from poor families more often grow up in neighborhoods with a disproportionate number of social problems—delinquency and crime, violence, teenage pregnancy, welfare dependency, undesirable housing, and inadequate health care ([Children's Defense Fund, 1989](#); [Zigler and Styfco, 1996](#)). Although successful families and social networks exist within such neighborhoods, extraordinary pressures interfere with the development of children at all times. Thus, all such families are under stress.

Parents often lack education themselves or bear social and psychological scars related to their social status and conditions. They often sense exclusion and feel alienated from the mainstream of society ([Mead, 1996](#)). They often carry some attitudes, values, and ways that are different from those of the mainstream. They often simply do not know mainstream expectations and ways ([Anderson, 1998](#); [Lightfoot, 1978](#)). Thus, even when they want to prepare their children to succeed in school—a mainstream institution—they are not able to do so adequately.

For example, learning to negotiate and to work things out rather than fight is expected in school. However, in many nonmainstream communities, children are expected to fight rather than to negotiate ([Anderson, 1998](#)). Many such families do not read to their children, take them on recreational and learning excursions, or otherwise give them the informal preschool learning and social skills needed to grow along the critical developmental pathways discussed earlier. By school age, such children are underdeveloped, or differently developed, along the developmental pathways most related to academic learning. They often have skills that enable them to function adequately on the playground, the halls of the housing project, and a variety of other customary places, but such skills elicit a negative staff response in

school.

The work of the Yale School Development Program suggests that children from low-income or marginalized backgrounds have the potential to achieve at high levels. The Samuel Gompers Elementary School in Detroit, Michigan has a 98% student poverty level. Yet they made the highest test scores in the state on the fourth grade Michigan Educational Progress Test in 2001. This was possible because of the collaboration in school and between the school and families that promoted the development of the children to the point that they could make significant academic gains.

However, the preparation of most school people does not help them to understand the behavior and apparent ability level of such children as underdevelopment or different development. It is viewed as “bad” and “dumb” or as evidence of inadequate learning potential. Many believe the students are willfully not trying. Most school staff members attempt to change the behavior of the children through punishment. This often leads to even more troublesome behavior and accompanying low expectations on the part of students and, in turn, to increased control efforts and continued low expectations by school staff. Some students eventually respond with severe acting-out behavior or withdrawal and apathy. This situation leads to hopelessness and despair among all in school settings ( [Edmonds, 1979](#)).

Parents who had ambivalent feelings about the school in the first place—who hoped that it would give their children a chance for greater success than they experienced, but feared that it would not—are often brought into conflict with school people, or they withdraw and avoid the school. The attachment and bonding that should occur between school staff and students do not occur, and the staff is not able to help the students invest strongly in the academic learning task. When parent–child bonding is not strong because of difficult social circumstances, inadequate bonding in school is doubly troublesome.

Before to 8 or 9 years of age, most students are able to establish the level of attachment and bonding that enables adults to influence their behavior ( [Bowlby, 1952](#)). However, around this period, the academic performance demands of the school (more abstract learning) begin to outstrip their performance preparation. Second, a child's cognitive capacity now enables him or her to understand his or her social status—at home and in the community, and in the classroom. Third, the thrust for independence is greater at this age ( [Flavell, 1985](#)). These developments limit or facilitate mainstream aspirations and reduce the ability of adults to influence positively the behavior of young people. If constructive attitudes toward learning have not been internalized by this point, it can be difficult for adults to introduce them.

Students with a weak attachment and bond to the people and the program of the school, or with extraordinary pressures to belong to troublesome social networks in and outside of school, begin to pull away ( [Anderson, 1998](#)). At this point, around third grade, one sees the academic performance gain of children from families and communities under stress begin to reach a plateau. Future brain research may well show organic changes underlying this process.

As young people confront the developmental issues of preadolescence and early adolescence, they “place” themselves in the social scheme of things. They learn to develop an understanding—correct or not—of their exclusion from, or their limited opportunities in, the mainstream of the society. Their source of self-affirmation is from their own families and social networks. In fact, when schools ask young people to achieve at a high level, they are often asking them to be different from their own parents, the people with whom they have the most important and powerful social ties.

Around 12 to 13 years of age, children develop the cognitive capacity to consider hypothetical situations—not only what is, but what could be ( [Piaget, 1970](#)). This ability, in addition to widening their world, also makes it more complex. Simultaneously, numerous confusing physiologic and physical body changes take place. Succeeding and belonging in groups—with attendant group pressures—become very important tasks at this point. In addition, in seventh grade, in many places, young people are required to change schools and to make numerous social adjustments associated with doing so.

Most young people who are developing well and who are from the societal mainstream are able to manage these challenges with the average expected difficulty. However, many capable young people go on a sharp psychosocial downhill course at this point, leading to academic underachievement, school dropout, teenage pregnancy, delinquency and crime, and other social problems ( [Luthar et al., 1997](#); [Watson, 2000](#)). Disproportionate numbers of such youngsters—for reasons we discuss later—are African-American ( [Anyon, 1997](#); [Children's Defense Fund, 1989](#)).

By midadolescence, at around ninth grade for most, social class-related and race-related expectations and occupational and career possibilities begin to influence student performance greatly. Many high-ability African-American students from low-income backgrounds begin to do less well in school, often less well than the low-ability students from middle- and upper-class backgrounds ( [Fordham, 1988](#)). In addition, by this point, many students have serious academic deficiencies and negative attitudes and beliefs about themselves that make it difficult for them to perform adequately even when they desire to do so ( [Oyserman, 1999](#)).

The situation for middle-class African-American youngsters is different but still problematic. With adequate income, such families are under less stress and are better able to prepare their children to meet the expectations of the school. Many such parents believe that their economic well-being will protect their children from negative societal attitudes and behaviors related to race. In fact, many such parents avoid any discussion of race. At the other extreme, parents express angry and hostile feelings about racial injustice in a way that conveys powerlessness. They often encourage their children to be individuals, as if raceless, and as if they will not be forced to confront and manage racial issues in their lives ( [Fordham, 1988](#)). These youngsters often receive the same message in school.

The developing young person has no way to understand the disproportionate number of problems he or she observes among African-Americans. Such persons are not prepared to manage racial antagonisms, deliberate and unintentional, in ways that minimize the negative effects on their self-esteem. With little understanding of the experience, strength, and successes of their racial group, they cannot experience the level of positive identification with the group that is needed to manage the racial antagonism in the larger society more successfully. In adolescence, some young people from better-educated, middle-income black families who are struggling to establish their identity are drawn into behaviors defined by the society as black and internalized by some as “true” black behavior—low academic achievement, troublesome social performance, and the like ( [Anderson, 1998](#); [Fordham and Ogbu, 1986](#); [Oyserman, 1999](#)).

Pop culture entertainment has made this matter worse. Many nonmainstream young people are vibrant and intelligent and have developed powerful self-affirming artistic expressions of one kind or another. Some of it has become mainstream art and entertainment and is transmitted back as such to people from all socioeconomic groups. However, these expressions and the accompanying lifestyles lead to mainstream success for only a few. Many African-American young people embrace it as “their culture” without mainstream skills, thus limiting their ability to participate in the mainstream.

Although this chapter focuses on psychoeducational outcomes for African-American children, and although the historical political and economic issues are different, the same principles of child and adolescent development and behavior hold for mostly white schools and, indeed, for all children. Whenever parenting styles are ineffective and even harmful, for whatever reason, and whenever attention to students' emotional development is insufficient compared with cognitive growth in school, there is a higher probability of problem behavior on the parts of students. Our current concern about violence in schools is directly related to inadequate attention to students' emotional development at home and at school. Schools serving African-American children are disproportionately affected because of their difficult historical, political, and economic circumstances.

## **ECONOMIC AND SOCIAL HISTORY: EUROPEAN, ASIAN, AND AFRICAN-AMERICAN EXPERIENCES COMPARED**

Since the mid-19th century, the United States has moved through four economic stages: agricultural, before 1860; late agricultural and early industrial, from 1860 to 1900; midindustrial or heavily industrial, from 1900 to 1945; late industrial and early scientific and technologic, from 1945 to 1980; and postindustrial, highly scientific and technologic, after 1980. European and Asian immigrants arrived in the United States in large numbers before 1915. Most were able to maintain a reasonable degree of cultural continuity—religion, language, and other aspects of their culture—because they lived in ethnic enclaves, resulting in a reasonable degree of social cohesion ( [Lieberson, 1980](#)).

Some immigrants had ties to financial resources in the old country. Many created local and administrative structures in their attempts to gain power and control not available to them through state and national electoral representation ( [Anyon, 1997](#)). They obtained the vote in one generation. Cultural cohesion, the vote, and economic resources, together, permitted most groups to gain political, economic, and social power in one generation at a level that permitted their families to undergo four generations of change and development that paralleled economic change and development in the country ( [Lieberson, 1980](#)).

Before 1900, heads of households could be uneducated and unskilled and could earn a living in the economy, provide for themselves and their families, and give their children the kind of experiences that enabled them to gain the moderate level of education and skill needed to succeed in the midindustrial period between 1900 and 1945 ( [Wilson, 1987](#)). Successful heads of households in this economy were able to give their children the kind of experiences that enabled them to gain high levels of education and skill needed for the last stage of the industrial era, 1945 to 1980, and, of course, a similar process permitted most heads of households to prepare their



children for the postindustrial era.

European and Asian immigrant groups experienced numerous hardships, and as a consequence many unsuccessful families from these groups are living under stressful conditions in various areas of the United States today. However, most did not experience overwhelming abuse and denial of access to political, economic, and social structures; therefore, they did not undergo significant group and family deterioration. The experience and outcome for a significant number of African-Americans, Hispanics, and Native Americans were different.

The African-American experience was characterized by cultural discontinuity ( [Franklin, 1947](#); [Frazier, 1957](#)). The sense of adequacy, worth, and direction given by culture was lost in the separation from Africa. Because the central organizing force of West African culture was tight-knit kinship and lineage groups with communal traditions, a slave culture—and ultimately, release into a society that highly valued independence and competition—was extremely traumatic ( [Hildreth et al., 2000](#)). Slavery was a system of forced dependency, inherent inferiority, and no opportunity for a better future. These are three of the most problematic conditions that can be imposed on people who are then expected to function in a competitive economic and social system ( [Berry and Blassingame, 1982](#)).

Again, because the United States is based on Judeo-Christian principles and a commitment to democracy, there was a great need to rationalize the enslavement of blacks and their exclusion from the political, economic, and social systems after the abolition of slavery. First a non-Christian religion and later racial inferiority were used. After the Bacon Rebellion in Maryland in 1676, and perhaps others, the potential of a coalition of indentured white servants, black freeman and slaves, and poor white farmers represented a threat to the white population of significant wealth. To reduce the threat, white indentured servitude was eliminated. The notion of the superiority of whites and of the inferiority of blacks was gradually written into law in surrounding areas, eventually nationwide. These doctrines flourished because of the economic, psychological, and social benefit to whites across the socioeconomic spectrum.

These conditions created negative psychosocial consequences among many, and they were transmitted from generation to generation, from parents to children, even down to the present day ( [Elkins, 1963](#)). However, many African-Americans were protected to some degree by the "Black Church," fashioned from aspects of African culture and Protestant religions in the South, and by living under better conditions of slavery. Moreover, southern rural culture tended to reinforce the church culture and to facilitate effective family functioning ( [Comer, 1972](#); [Frazier, 1962](#)).

After the abolition of slavery, blacks in large numbers were denied the vote and, in fact, denied in significant numbers right up until the 1960s, the middle of the last stage of the industrial era. In addition, blacks had no ties to capital ( [Jaynes and Williams, 1989](#)). Without the vote and capital, it was not possible for the black population to limit the level of racial antagonism or racism. Extreme violence, terrorism, and pervasive subterfuge were used to bring about these conditions. Whites often walked off better paying jobs when white employers attempted to introduce black workers, and white workers were often victimized if they agreed to work with blacks. As a result, blacks were closed out of the political, economic, and social mainstream. Most black heads of households worked at the lowest levels of the economy—sharecroppers and tenant farmers, domestics, and industrial laborers.

Without political and economic power, blacks were closed out of educational opportunities. In the eight states that had 80% of the black population, four to eight times as much money was spent on the education of a white child as on the education of a black child ( [Bose and Caliber, 1936](#)). Where the black population was disproportionate in numbers, the disparity was as great as 25 times and more ( [Bose and Caliber, 1936](#)). This situation continued into the 1940s, perhaps beyond. As late as 1964 to 1965, the combined endowment of two prestigious white women's colleges was half the endowment of Harvard. One-half of Harvard's endowment was greater than that of all black colleges put together (totaling more than 100) ( [Council for Financial Aid to Education, 1967](#)).

Racial antagonism followed the black population from the South to the North, although in a less overt form. Thus, blacks could not experience the benefits of mainstream political, economic, and social participation in the North anymore than was possible in the South. Educated blacks were limited to professional areas—teachers, physicians, nurses, and the like. As a result, the kind of information, contacts, and connections needed to make the social system work for its members was not available. Even the growing political power since the 1980s has been in the absence of economic power; thus, it is less useful in making the social system work for the entire black community ( [Anyon, 1997](#)). All of this had a negative impact on black community and family functioning. Despite these difficult conditions, the black population did reasonably well right into the 1950s ( [Wilson, 1987](#)).

As late as 1950, only 22% of all black families were headed by single parents; now the number is more than 60% ( [Lugaila, 1998](#)). Moreover, black communities were reasonably safe. Today's serious conditions among a disproportionate segment of the black population began with (a) the mass migration of undereducated people from rural to urban areas in the 1940s and (b) education's increasingly becoming the ticket of admission to living wage jobs after 1945 ( [Wilson, 1987](#)). Blacks were disproportionately undereducated, compared with other groups, and they lacked the power to gain well-paying, low-skill jobs. In addition, previous organizing cultural ties were lost with mass migration. Many families that once functioned well in the traditional church-based, extended family, rural culture of the South began to function less well under urban conditions.

In addition, families functioning well in urban areas began to reduce family size, except for religious reasons. Families not functioning well did so less often. As a result, the growth in population from 1961 to 2001—among all groups—has been among low-income families operating under the greatest stress ( [U.S. Bureau of the Census, 2000](#)). For the reasons cited, African-Americans have a disproportionate number of families operating under excessive stress.

Most industrial countries developed social and educational policies that facilitated family functioning in the 1940s and 1950s. The United States did not do so to the same extent ( [Kamerman and Kahn, 1981](#)). As a result, many minority group families—more than among groups able to undergo several generations of development and stabilization—are not able to give their children the kind of preschool experiences that will enable them to meet the expectations of the school. For the reasons described earlier, some mainstream middle-class minority youngsters are affected. Thus, too many minority group children are undereducated.

## YALE SCHOOL DEVELOPMENT PROGRAM

In 1968, a Yale Child Study Center team (directed by Comer and made up of a social worker, a psychologist, and a special education teacher) began a collaborative project with the New Haven, Connecticut school system. This team worked in two schools in the inner city of New Haven as a subsystem of the school system. The plan was to develop a successful change process within these two schools, in collaboration with the parents and school staff, in a way that could be transmitted to all New Haven schools and beyond, eventually influencing national school policies and practices ( [Comer, 1980](#)).

Work began in the Martin Luther King Elementary School, from kindergarten to fourth grade, with approximately 300 students, and in the Simeon Baldwin school, from kindergarten to sixth grade, with approximately 350 students. After 5 years, the team left Baldwin, already vastly improved but resistant to additional changes, and began to work in the Katharine Brennan School, a school with students from kindergarten to fifth grade and with a profile similar to that of the original schools. These schools were 99% black, and almost all the students were from poor families. The students ranked 32nd and 33rd of 33 city schools on standardized achievement tests. They were 19 and 18 months behind in achievement in language arts and mathematics by the fourth grade. Their attendance was among the lowest in the city, and there were serious behavior problems in both schools. Teacher turnover was common, and parent dissatisfaction with the schools was widespread.

By 1979, without a change in the socioeconomic makeup of the school communities, the children were at grade level on standardized achievement tests. The improvement process was institutionalized, and it allowed the team to leave these two schools in 1980. In 1984, the two schools, again with no change in the socioeconomic makeup, tied for the third and fourth highest levels of achievement in the city on standardized achievement tests, a year above grade level at King School and 7 months above grade level at Brennan School. King School's attendance had been first or second in the city for 5 of the previous 6 years, and Brennan School's attendance was improved and ranked first once during that period. Behavior problems were greatly decreased in both schools; teacher turnover was extremely rare, and there were no serious behavior problems ( [Comer, 1976](#); [Comer, 1980](#)).

What the team found initially in these school communities served to provide insight for the formulations described earlier. The parents and the students were largely the descendants of rural, southern African-Americans, undereducated and working at the bottom of the job market, or not working and on public assistance. Many were experiencing significant economic and social stress. The underdevelopment and different development of their children made it difficult for them to meet the expectations of the school.

The school staff was not prepared to adjust successfully. The resultant difficult interactions between home and school, among school staff and between staff and students, led to alienation, distrust, disrespect, inadequate student-staff attachment and bonding, difficult teaching conditions, and limited academic learning.

The team from the Yale Child Study Center took an ecologic and child development perspective in identifying the problems and opportunities. They realized the need

to (a) change the difficult interactions among the adults into cooperative and collaborative interactions so that they would be able to support the level of child development that would make successful academic learning possible and (b) make teaching a gratifying experience. However, the team could not mandate a change in the culture of the school—its beliefs, attitudes, values, and ways. Efforts to teach school people about child development and behavior, in the hope that they would apply their knowledge in the classroom, were unsuccessful. The team instead gradually had to develop mechanisms that permitted the staff and parents to carry out a process that allowed them to function in a way that changed the culture and climate of the school and promoted child development, teaching, and learning, as needed.

The team members used their clinical skills to help the staff work more effectively with children displaying behavior problems. With each success, the team gained staff interest and appreciation for clinical methods. Eventually, the team was able to share child development and child-rearing knowledge, skills, and sensitivity with the staff and help them to appreciate how school attitudes, beliefs, values, and practices either facilitate or limit student growth and development, teaching, and learning.

With school staff and parents, the team eventually developed a change process called the *Yale School Development Program*, which has three mechanisms, three operations, and three guidelines designed to address the problems and opportunities found in all schools, but especially in schools serving low-income minority students. The three mechanisms are the Governance and Management Team, the Mental Health Team, and the Parents' Team.

The Governance and Management Team is the most important program element. It is representative of all the adult stakeholders in the schools—parents, teachers, administrators, Mental Health Team, the nonprofessional support staff, and so forth. It carries out the three critical operations: development of a comprehensive school plan, staff development based on the plan, and systematic assessment and modification of the program. This mechanism and these operations bring the key adults together in a way that enables them to establish agreement and to coordinate and carry out a program that reduces duplication and conflict, facilitates communication, and establishes mutual trust and respect. The comprehensive school plan focuses on carrying out activities that systematically serve to improve the climate of relationships and the academic program.

The Mental Health Team brings together personnel usually found working separately in schools—social worker, psychologist, special education teacher, nurse, and the like. (As this team took on more functions, the name was changed to the School Staff Support Team.) The team works with individual children, but the focus is on prevention. Team members often gain clues from individual student problems about the kind of building-level changes that can be made that will reduce them. For example, difficult adjustments to transferring into the schools led to serious behavior problems among some students. Transfer problems were reduced by orientation sessions for new students at the classroom and building level, thus preventing immediate, sustained, and more serious problems. Problems of discontinuity in the lives of children, interfering with their ability to establish the kinds of attachments needed to trust and take chances, led to a program of keeping children with the same teacher for 2 years, with great success. A program for helping children in crisis greatly reduced disruptive acting-out behaviors. A “Discovery Room” enabled many withdrawn children, doubtful of their ability to manage in the school setting, to function adequately.

The Mental Health Team person serving on the Governance and Management Team helped that group to establish policies and practices that prevented student behavioral problems or reduced their adverse consequences. In the process, this person provided all adults in the school with knowledge about how children grow and develop and how that is related to teaching and learning. The Mental Health Team also helped staff members to interact well together and to develop a school climate that facilitated the work of all involved. Adults functioning well in a good climate, who are knowledgeable about child growth and development, can facilitate attachment and bonding, child growth, and development, leading to adequate teaching and learning.

The third mechanism was the Parents' Team. Parents were involved at three levels: as members of the Governance and Management Team, selected by other parents; as members of the parents group that put on activities with school staff in support of the school program; and as visitors and participants in school activities. A school social worker or teacher served as a liaison between the staff and the parent group and facilitated their work in the school. This approach reduced the likelihood of the development of an adversarial relationship.

During one period, parents worked as assistants in the classroom, receiving a minimum wage for 10 hours and often volunteering for 20 to 30 hours more. The presence of parents in the school working closely with school staff reduced the alienation and distrust between home and school. It facilitated the attachment and bonding between students and school staff and the program of the school. Many parents working in this way were motivated to go back to school themselves; at least seven went on to college and became professional people. The presence of a core of parents in school increased the social comfort of all parents in the school setting. Because of this and other things done to improve school operations and climate, parent turnout for an important school activity, the Christmas Program, went from 15, in a school serving a little more than 300 students, to 400 in the same building 3 years later. Similar increases in parent participation have occurred in almost every school district in which the team has worked.

However, school restructuring alone was not responsible for these outcomes. It was necessary to change the culture of the school from being punitive with low expectations to having a focus on supporting growth and development, cooperation, collaboration, and high expectations for all. The team's three working guidelines were designed to change the culture. The mechanisms and operations of the program generated and carried the new beliefs, attitudes, values, and ways, or culture.

The three guidelines were (a) a “no-fault” policy, (b) consensus decision making, and (c) an agreement that the members of the Governance and Management Team could not paralyze the principal, but also that the principal could not ignore the input of team members. In this way, time was not wasted placing blame but was used to solve problems, decision making through consensus rather than voting kept groups focused on child development principles and avoided winner–loser behavior, and leadership was based on multiple perspectives. Although the Governance and Management Team generated the desired culture, its style permeated the school because all involved carried out the work set forth in the comprehensive school plan.

When the three mechanisms, the three operations, and the three guidelines were all working appropriately, everyone experienced ownership and responsibility for the school program, and a kind of synergism developed that gave the school hope and forward momentum. It enabled student growth and development to take place; adequate teaching and learning were then possible.

The team eventually developed a program that very systematically taught low-income minority group children the same skills that mainstream children from better-educated families gain simply by growing up with their parents. The team developed units in politics and government, business and economics, health and nutrition, and spiritual and leisure time in which there was an integrated teaching of basic academic skills, social skills, and appreciation of the arts. The artistic programs enabled children to channel aggressive energy that would otherwise be harmful to themselves and others, or at least disruptive, into the energy of recreation and learning. The social skills students gained through these activities enabled them to elicit a positive response from teachers and other people in the mainstream of the society. This facilitated the needed attachment and bonding to mainstream people and programs and motivated young people to achieve. Because of the beneficial outcomes of this approach, the New Haven School System has established a social development program from elementary school through high school in which students are taught appropriate problem-solving and decision-making skills.

Initially, it was expected that the Yale School Development Program would create conditions in schools that would motivate students to learn. It was expected that the curriculum and instruction would be provided by the district and school building. Rather than a curriculum focus, there was to be an academic focus through the comprehensive school plan. The team urged the use of age-appropriate academic programs and activities, a recognition of cultural diversity, and accurate information about the nature of the African-American experience. This gave schools the opportunity to use their own creativity in implementing district required curriculum and instruction activities.

Although many schools thrived under this approach, achieving remarkable gains, some did not. The Yale School Development Program team then undertook a more active role in promoting academic development in those schools that showed only incremental or no achievement gains. A Balanced Curriculum program was developed, as well as a Teachers Helping Teachers program and an Essentials of Literacy program. All these approaches improved instruction and achievement where they were implemented appropriately. The Balanced Curriculum program integrates the many demands on schools in a way that makes it manageable for the classroom teacher. The Teachers Helping Teachers program enables the staff to share and deepen knowledge and skills. The Essentials of Literacy program has helped nonreaders in the third grade learn to read in a remarkably short time. All these approaches are being studied and integrated into systemwide school change efforts.

The Yale School Development Program expanded nationally to approximately 700 schools in 68 districts. The team soon learned that to sustain these efforts it was necessary to work systemically or on a district-wide basis. Thus, the team reduced Yale School Development Program involvement to 500 schools, and with the expectation of eventually working back to a nucleus of 600 schools, all supported by district level school officials. Although most of the students in the schools serviced by the program are African-American or from other minority ethnic backgrounds, increasing numbers of schools with majority white populations are opting to



implement the program because of its demonstrated effects in improving the psychoeducational outcomes for large numbers of African-American and culturally diverse groups of students.

The research of many investigators ([Becker and Hedges, 1992](#); [Cook et al., 1999](#); [Haynes and Comer, 1993](#); [Millsap et al., 1997](#); [Noblit et al., 1997](#); [Noblit et al., 2001](#)) has documented the program's effectiveness in improving educational and psychosocial climate of schools. The research indicates enhanced student self-esteem, motivation, learning, and achievement. The work of the Mental Health Team in focusing attention on child development principles, proactive and preventive interventions, and child and family issues from a systems perspective has served to increase parents' involvement, their sense of efficacy in helping to raise and educate their children, and, in turn resultant and significant reductions in school absenteeism and behavior problems.

The Yale School Development Program has generated a wealth of knowledge and professional expertise regarding how to help people change and improve schools. The program leaders also learned that reforming educational services is an ongoing process, one that must be nurtured and constantly reshaped to respond to larger political issues, to new service developments, and, above all, to changing child and family needs. With this in mind, the Yale School Development Program has brought together and supported activities and programs that engage scholars from within and beyond the disciplines of child psychiatry around issues that intersect with the fields of child development and public education. One such program is the Comer–Zigler Initiative. This program is a structure that combines the existing School Development Program network and organizational plan with a school-linked program model that transforms a school into a year round, multiservice center: School of the 21st Century (21C). 21C was conceptualized by Yale University professor Edward Zigler, one of the principal architects of the Head Start Program. In designing the 21C model, Zigler recognized that changes in patterns of work and family life in recent decades have meant new concerns for parents, especially a pressing need for affordable, high-quality child care.

The Comer–Zigler Initiative is a unique collaboration because it capitalizes on the natural synergy of family and children's services connected to schools. It provides a process to bring all the adults in the community to the same table, where they can plan and share information in a comprehensive way. The guiding principles of the Yale School Development Program and 21C encourage the forging of a common mission, which keeps the child's development at the heart of all planning and decision making. Just as the three Comer mechanisms, operations, and guiding principles fostered collaboration that improved a school's capacity to affect student outcomes, so may they support and facilitate these kinds of practices and collaborative connections, as well as open doors for powerful new ideas and ways of doing business.

## **ROLE OF THE CHILD AND ADOLESCENT PSYCHIATRIST**

General psychiatrists focus more on the individual person than on his or her environment. However, because children are in a state of significant dynamic development and are dependent, the people and institutions around them (their critical environment) greatly affect their behavior. With greater appreciation of the power of people and institutions, child and adolescent psychiatrists can help to promote policies and practices in organizations and institutions that promote growth and development. Child and adolescent psychiatrists have this opportunity in their work with families, the juvenile justice system, social welfare agencies, schools, and others. Schools probably offer the best opportunity to affect the most people under the best conditions.

Going to school is a normative function with no stigma attached to it. It is the final common pathway for almost all children today. It is strategically placed in the developmental pathway, early enough to compensate for potentially troublesome preschool experiences. School is the work of young people and is of long enough duration on a daily basis and over the years to influence their development significantly. Thus, the child and adolescent psychiatrist should be involved in helping people engaged in every aspect of schooling—those providing preventive and direct services, policy, and research.

Although most child and adolescent psychiatrists do not have full-time involvement in the way the Yale Child Study Center team has had, many are engaged in regular consultation, accept school referrals, and interact with school people regarding patients referred to them. Child and adolescent psychiatrists doing school consultation usually work with staff or individual students and small groups. The work is usually child and family centered. However, as the psychiatrist enables teachers and administrators to help students, the child's respect and trust develop and allow him or her to begin to help the staff look at school practices and policies that often interfere with student growth and development or aggravate existing psychological problems. Sharing such insights often leads to the child psychiatrist's involvement in building-level staff development sessions. Such involvement provides even greater opportunities.

Through such sessions, the child psychiatrist can help school staff to understand how the fears and anxieties of students, the threat that school presents to many (and the related behaviors that arise), and the staff's own tendency toward control and punishment make matters worse. The psychiatrist can help staff to understand that academic learning is more than a mechanical process in which students willfully participate or refuse—that the affective component is important and the motivation for learning grows out of relationships at home and school and between home and school. The psychiatrist can help staff members to learn how their own behaviors promote either desirable or undesirable student responses. As the child psychiatrist and his or her team work with individual children, they can share their knowledge of child development and ways to promote growth and development with teachers, administrators, and parents.

Many schools lack orientation programs for new students, conflict-resolution approaches, activities designed to promote cooperation and collaboration, and a focus on helping students to develop inner discipline, self-motivation, and personal responsibility. Social problem solving and social skills are not taught systematically in most schools. Social activities are thought of as frills rather than as opportunities to help young people who are underdeveloped or differently developed grow and develop in ways that enable them to meet the expectations of the school. The psychiatrist is in a position to help school people understand how these skills and the resultant successes, confidence, and growth in self-esteem facilitate (a) bonding and attachment to school people and the program of the school and (b) teaching and learning.

Few school people understand the real or potential conflict that exists because of the income, education, racial, and other differences between home and school. Even fewer understand how, when, and why these differences influence academic learning ([Stipek, 1997](#)). Child psychiatrists can help teachers to understand the kinds of experiences that children from poor families need to be prepared for the level of abstract learning that is more required after 8 or 9 years of age, as well as the need to appreciate the effects of the thrust for independence among young people at this age and the later effects of social and racial status "placement" or identification with their respective groups. With such understanding, practices and programs can be devised at the building level to facilitate an appreciation for learning while enabling students to make a positive identification with their own group. In some cases, there will be opportunities to influence preservice programs and schools training future teachers and others. This will help them to be better prepared to support the development of children in school once they are in practice (Knitzer, 1997).

Knowledge of how and why children learn and behave is also needed among policy makers. Decisions about the employment and use of social workers, psychologists, special education teachers, and the like depend on their understanding of child development and behavior or lack of understanding. Likewise, school size and architecture, compensatory education programs, busing, school desegregation, the management of trauma, racial identification issues, class and race relations in general, gender, and various other issues require knowledge of the behavioral and social sciences held by child and adolescent psychiatrists. When child and adolescent psychiatrists are helpful with social issues at the building level, school people at the central office level are more likely to use them to help school board members, legislators, and the media to understand school district needs, opportunities, and limitations better.

The surge in violence in recent years has resulted in significant demand for child psychiatric services in schools. Children now, more than ever before, attend schools with serious psychological problems such as posttraumatic stress disorders, severe depression, behavior disorders, and attention deficit problems, many of which result from exposure to the chronic violence in their lives. The Yale School Development Program school-involved clinical consultants and Yale Child Study Center crisis teams provide significant and valuable interventions in New Haven schools to prevent and treat violence-related psychological problems among children. The result of these vital services is a special collaborative relationship between the New Haven schools and the Yale Child Study Center in which child psychiatrists, social workers, and psychologists participate in strategic planning and program development sessions with school officials. Notable also is the police fellowship program directed by Dr. Steven Marans, in which New Haven police officers are trained in child and adolescent development and psychology to improve their skills and increase their sensitivity ([Marans, 1994](#)) (Chapter 127).

Child and adolescent psychiatrists, because of their clinical focus, are probably better prepared than are other researchers to do the kind of research that is helpful in schools. The experimental research designs favored by nonclinicians take behavior out of context in a way that limits the value of their findings. The clinician looks at dynamic interactions in context, just as school people must do. In fact, school change leaders are suggesting that teachers be trained in a way that they can engage in the clinical practice of education. The participant–observer, ethnographer, case-study approach child psychiatrists are inclined to use is much more helpful. Child psychiatrists are in a good position to understand the insights and limitations of experimental design research findings and to help school people to use them appropriately.

Any effort to understand child developmental trajectories must focus not just on the child, but also on the child's ecosystem (Zigler and Styfco, 1996). Since the 1970s, there have in fact been many innovative and sophisticated studies of child developmental pathways specific to youth outside the sociocultural mainstream (Luthar et al., 1997). Richters and Cicchetti (1993), for example, argue the need to distinguish between two broad pathways to conduct disturbances: dysfunctional pathways, which are associated with neuropsychological or social cognition deficits; and normal pathways, in which conduct problems are essentially the outcome of extrinsic, environmental factors rather than internal dysfunction. These investigators conclude that antisocial behaviors may often occur among essentially normally functioning persons, as a reflection of their adaptation to prevailing mores and norms of a counterculture subculture. Collectively, the data emphasize that conceptions of "normal" developmental trajectories can vary substantially among children and families living in poverty as opposed to those who live in more affluent, mainstream families. These empirically derived findings are supported by other researchers (Luthar et al., 1997). Yet insights from these efforts are rarely integrated into the academic literature on child development. Many textbooks continue to be written as though these new research theories and findings did not exist (Luthar et al., 1997). The analysis of these variables often emerges as either an afterthought or as nuisance variables. Many researchers may be reluctant to enter into discussions about the impact of race and socioeconomic status because of the intricacies (both practical and political) of defining and isolating the variables.

In future theoretical conceptualizations of psychosocial development among inner-city youth of ethnic minority groups, it will be useful to acknowledge from the outset not only those developmental tasks that confront all children and adolescents, but also those that are unique, significant, and inevitable in the lives of the disadvantaged (Luthar et al., 1997). Developmental psychological research must aim to (a) shed light on the factors within and across economic and ethnic groups that influence development and (b) broaden our understanding of the range of variation that is possible over the course of development. A more complete understanding of human behavior will require us to consider, in a conscious, critical way, the impact of race, gender, and other important social categories not only on study participants and clients, but also on ourselves as researchers and clinicians (Luthar et al., 1997). As we broaden and augment our theoretical conceptualizations, it will also be crucial to redress the training of mental health professionals across disciplines (Knitzer, 1996). Child psychiatrists, for example, nowadays appreciate the impact of the social, cultural, economic, and political history on child development. Indeed, knowledge and understanding of the impact of family and social network experiences on children and an appreciation of historical, sociocultural effects are easily gained. The psychoeducational outcome of African-American students and of all other students in homogeneous and complex societies will be improved as we further our understanding of how societal, institutional, family, and individual developments amalgamate to promote or limit student readiness for academic learning and teaching.

## CONCLUSION

The work of the Yale School Development Program suggests that socioeconomic conditions, race, ethnicity, and other factors are less important than the quality of the developmental experience that children receive. Schools that provide children with good developmental, teaching, and learning experiences can promote greatly improved academic and psychosocial outcomes despite significant risk factors. There is a need to demonstrate the saliency of a focus on child and youth development in school and to create large numbers of educators capable of supporting it. It is also important for institutions preparing teachers and administrators for service and leaders who craft education policies to understand and to use sound child and youth development principles. The child and adolescent psychiatrist can provide knowledge and understanding that can be useful in all these efforts. A focus on child and youth development can go a long way toward overcoming the ill effects of traumatic historical conditions.

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# 107 SCHOOL AVOIDANCE, SCHOOL PHOBIA, AND TRUANCY

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## SCHOOL AVOIDANCE

In developed countries, education is provided for all children, and there is an expectation that they should attend school regularly to take advantage of the opportunities for learning that are offered. Penalties may be imposed if children fail to attend school without an adequate excuse ([Berg and Nursten, 1996](#)).

In the United States, attendance laws were passed early in the 19th century requiring 12 weeks at school in a year, but enforcement came only much later ([Hersov, 1990](#)). In Britain, the current system of compulsory schooling, backed by legal procedures that can be used if attendance is unsatisfactory, was introduced nearly 100 years ago. Throughout most of the 19th century, children were affected to a considerable extent by extreme poverty, crippling diseases, early death, and employment in mines, agriculture, and factories, all of which made it difficult for any sort of national system of education to be organized. There were then few schools, and those that did exist often had poor facilities and inadequate teaching. However, there were some schools that attempted to encourage regular attendance. Inducements sometimes were offered in the form of a distribution of welfare benefits. Occasionally, lists of enrolled pupils who failed to attend regularly were displayed publicly as a means of deterring absence. A century ago, laws began to be enacted that enabled schools to be set up throughout the country, that compelled all children of specified ages to attend, and that also stated how regular attendance should be enforced. Courts were empowered to act when there was lack of compliance ([Berg et al., 1988](#)).

Despite this legal framework of mandatory education, many children have always had some time off school without adequate excuse. On average, at any one time in the United Kingdom, 10% of all children are out of school. Most are away with an acceptable reason, namely, a physical illness or a family holiday, but possibly as many as a third of them are off without adequate explanation. Community surveys have indicated what social, educational, and individual factors are associated with failure to go to school ([Farrington, 1980](#); [Fogelman and Richardson, 1974](#)). Epidemiologic studies also have shown that there is a small proportion of children who remain away from school for more than half the time they should be there ([Berg et al., 1993](#); [Galloway, 1980](#)).

In general, there is no strong relationship between the sex of the child and absence from school. In this respect, there is a big difference between this problem and delinquency, which is so much more common in boys. However, there is a clear connection between absence from school and poor social circumstances, as well as lack of parental interest in the child's education ([Fogelman et al., 1980](#)). Factors in the school also have been found to be related to children's staying off ([Reynolds et al., 1980](#); [Rutter et al., 1979](#)). It also has been found that a sizable proportion of children at school show considerable dislike of it and show some reluctance to attend ([Mitchell and Shepherd, 1980](#); [Moore, 1966](#)). The simple fact of failure to attend school also has been considered to be an important indicator of antisocial conduct occurring in later life ([Robins, 1978](#)). It is important to view the problems of school phobia and truancy against the general background of school attendance.

## SCHOOL PHOBIA (SCHOOL REFUSAL)

Difficulties in going to school may be due to emotional upset. This was probably first described by Jung in 1913 ([Jung, 1961](#)). Soon afterward, Melanie Klein and Anna Freud also reported the psychotherapeutic treatment of children who had fear of going to school ([Sayers, 1991](#)). [Broadwin \(1932\)](#) reported several children who appeared afraid to go to school and stayed at home. [Partridge \(1939\)](#) also provided descriptions of children who did not go to school and who preferred to remain close to their mothers. They were generally well behaved. The term *school phobia* was later introduced ([Johnson et al., 1941](#)) essentially for neurotic difficulties in leaving mother that were manifested as fear of going to school. In Britain, an alternative term, *school refusal*, was used more generally for similar problems ([Hersov, 1960b](#)). In this chapter, to avoid confusion, the descriptive label *school phobia* is used throughout because the view is taken that *school phobia* and *school refusal* can be used interchangeably. There have been many papers and review articles on school phobia, and some of the best accounts of childhood anxiety and depression have appeared in them. It would seem that school phobia often is the presenting problem that brings disabling emotional disturbances to professional attention ([Hersov and Berg, 1980](#)).

### Definition

School phobia is easier to describe than to define. Many children are referred to clinics for treatment who stay home, apparently unable to face the prospect of having to attend school, despite normal parental efforts to help bring about their attendance and despite the absence of any evidence that the child is antisocially inclined and is deliberately attempting to avoid going there. Such children tend to show emotional upset in various ways.

Although any clear set of defining criteria would appear to exclude some children who in all other respects would be considered to be suffering from school phobia, it probably is helpful to have in mind the following features and also to be aware of the pitfalls of applying them too rigidly ([Berg et al., 1969](#)):

1. *The child remains at home with the knowledge of the parents.* This is in marked contrast with truancy, which often is considered to be characterized by staying away from home as well as from school and attempts to conceal the absence from parents ([Hersov, 1960a](#)).
2. *There is an absence of severe antisocial behavior.* This also is in marked contrast to truancy, which often is found to be associated with antisocial conduct ([Hersov, 1960a](#)).
3. *Parents make reasonable attempts to secure their child's attendance at school.* This distinguishes the problem from condoned absence ([Kahn and Nursten, 1962](#)), in which irresponsible parents allow the child to stay away from school.
4. *There is emotional upset at the prospect of having to go to school.* This may be confined to the situation of leaving home or going to school, or may be part of a more general disorder characterized by anxiety and depression ([Atkinson et al., 1985](#); [DeAldaz et al., 1987](#)).

Problems associated with use of these criteria should be mentioned ([Bools et al., 1990](#)). Staying home during school hours, with parents knowing about it, often occurs when other features of school phobia are not evident ([Belsen, 1975](#)). Parents who are at work when the child should be at school obviously are less able to influence the situation. Sometimes parents, concerned about their child's getting into trouble wandering about, reluctantly accept that they are better off staying home. Mothers weighed down with family commitments in poor social circumstances may appear too easily prepared to accept the child's complaints of illness, and this can lead to unwarranted absence, with the child remaining at home. Occasionally, there is the situation of a child who is manifestly afraid to go to school, who hides in the



vicinity of home or takes refuge with a friend or relative. However, in general, staying home in the knowledge of parents is a necessary, if not a sufficient, criterion for considering that school phobia exists. To this extent, the term *home-bound school absence* (Waller and Eisenberg, 1980) is appropriate. Considering the second criterion of antisocial behavior, confusion may arise because some apparently school phobic children manifest aggressiveness and resistiveness at home toward members of the family. However, they do not show other antisocial tendencies, such as lying, stealing, destructiveness, or wandering from home, and characteristically appear rather timid with other children away from home. Regarding the third criterion, concerning parental attitudes, it can be difficult to distinguish the irresponsible permissiveness of the parent who encourages or condones absence from school and the oversolicitous attitudes of the typical school phobic child's parents who give way to their offspring's demands to avoid having to go to school because of fears of putting too much pressure on the child or because of their own difficulties in separation engendered by overclose emotional attachments (Eisenberg, 1958). The fourth criterion of emotional upset in the child also presents some difficulties when the reaction is confined almost entirely to the situation of having to attend school, is expressed as refusal and determined resistance without obvious fearfulness, or is mainly in the form of vague physical symptoms without any discoverable cause. This last situation has been referred to as the *masquerade syndrome* (Waller and Eisenberg, 1980).

It is possible that the syndrome of school phobia is an artifact created by the obvious need to treat children who are not fulfilling their legal obligations to go to school and at the same time have an emotional or affective psychiatric disorder. Severe separation anxiety can occur without refusal to go to school (Last et al., 1987), and school phobia can occur without separation anxiety, or indeed much evidence of anxiety or depression other than in the immediate situation of school attendance (Bools et al., 1990; Smith, 1970). It will require further epidemiologic surveys to clarify this issue.

### Clinical Features

Many cases of school phobia manifest the interplay of two tendencies: avoidance behavior in relation to school and active seeking of situations providing comfort and security (Perugi et al., 1988). Unlike children displaying the common fears of childhood, such as those of insects, loud noises, or the dark, whose apprehension increases when the phobic stimulus is present, once in school, children with school phobia quite often appear to behave normally. The fear in their case appears to dissipate very rapidly, only to recur the next day at the prospect of having to go to school. So many reviews of school phobia are available (Hersov, 1990) that it is unnecessary to give yet another account of the protean manifestations of this condition; just some of the main features are outlined.

### Background Characteristics

Boys and girls are approximately equally affected. The condition is most frequent in the early teenage years, although it can occur anytime a child is enrolled in school. No social class bias is apparent. Intelligence and educational achievement are in no way different from what would otherwise be expected. Children without brothers and sisters do not suffer from school phobia more than children with siblings, but there may be a tendency for younger children in the family to be affected (Berg et al., 1972). Correspondingly, parents tend to be older than otherwise would be the case.

### Onset and Course

There usually is a gradual onset, with increasing upset on school mornings, although a sudden occurrence of the problem is by no means infrequent. Absence from school because of illness or holidays may precipitate school phobia, and there seems to be a relationship with change of school, particularly to the more demanding educational settings a child moves to with increasing age. Stressful events at home, at school, or in the peer group outside school may be related to onset. Often, however, the problem comes on out of the blue (Gittelman-Klein and Klein, 1980). Some school days may be particularly avoided because of lessons or activities the child finds stressful, but in general it is most difficult for a child to get to school after the weekend at home. The distinction between problems of leaving home and of getting to school is not an easy one to make, so often both tendencies appear to be of equal importance and inextricably intermingled. Nevertheless, some workers in the field do make this distinction (Last, 1993). Efforts on the part of the child to overcome the problem vary enormously. At one extreme, the child gets ready for school and may even leave home, but cannot progress any further. At the other, there is total unwillingness even to contemplate return to school, refusal to get ready, staying in bed, and threats of self-harm or of running away. Emotional upset is manifested by obvious fearfulness, as well as by physical complaints that appear to be due to anxiety, such as anorexia, pallor, headaches, abdominal pain, diarrhea, and frequency of micturition. Depressive features, such as misery, tearfulness, and lack of enjoyment, often are found but are less persistent than anxiety (Berney et al., 1981). Manifestations of anxiety and depression overlap in many instances (Bernstein and Garfinkel, 1986). Although some school phobic children who show a very circumscribed difficulty in getting to school behave normally at home and with their friends, most affected youngsters are severely socially impaired by the condition. They appear concerned about being seen to be off school and often go out little or not at all; they cut themselves off from social contacts and become increasingly anxious and depressed. The problem can continue indefinitely if little or nothing is done about it. However, if efforts are made to restore regular school attendance by a combined effort of family, school, and professional workers involved with the problem, they often are successful, particularly in children younger than 11 years of age (Rodriguez et al., 1959) and in the more circumscribed school attendance difficulties (Kennedy, 1965). Otherwise, return to school and improvement in more general neurotic and affective disturbances are less likely to be satisfactorily achieved (Berg, 1970; Berg and Fielding, 1978).

### Associated Psychiatric Disorders

School phobia has been characterized by symptoms of emotional disturbance, including physical complaints that can be seen as manifestations of anxiety, for a long time (Berg et al., 1969). With more recent developments in categorizing psychiatric disorders affecting children using the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) and *International Classification of Diseases* (ICD) systems of classification, importance has been given to the kinds of disturbance that are part of the problem of school phobia, while playing down the particular disabilities that are associated, namely the difficulties in relation to school attendance. Also, repeated revisions of these classifications have made it difficult to compare studies that use different versions, including ICD-9, ICD-10, DSM-III, DSM-III-R, and DSM-IV. Nevertheless, some kinds of disorder have been consistently present throughout these changes (Werry, 1996). School phobia is commonly associated with separation anxiety disorder, but other anxiety and mood disorders also are found, alone or in various combinations, including specific phobias, generalized anxiety disorder, depression, adjustment reactions, and, rarely, the more severe psychiatric disorders, such as schizophrenia and bipolar manic-depressive disorder. In a study of children who met the DSM-III criteria for separation anxiety disorder (Last et al., 1987), approximately three-fourths were having difficulty going to school. Additional diagnoses were made in 90% of the entire sample: Half of them had overanxious disorder, and a third, major depressive disorder. An investigation of approximately 50 school phobic children 9 to 14 years of age showed that approximately one-half had depression as assessed on well defined criteria (Kolvin et al., 1984). Separation anxiety, more or less corresponding to DSM-III separation anxiety disorder, was found in only approximately one-seventh of a group of Japanese school phobic children (Hoshino et al., 1987), a third of a sample of British school phobic students (Smith, 1970), and four-fifths of cases studied in New York (Gittelman-Klein and Klein, 1980). It is difficult to imagine that cultural factors are entirely responsible for discrepancies of these magnitudes. Referral practices determining the characteristics of samples of cases studied, differences in diagnostic criteria, and possibly unusually severe educational pressures, in the case of Japan, may have played a part (Chiland and Young, 1990).

In ICD-10, (World Health Organization, 1992) Separation Anxiety Disorder of Childhood (F93.0) is often used. Phobic Anxiety Disorder of Childhood (F93.1) is another category that is used. Social Anxiety disorder of Childhood (F93.2) also will be appropriate sometimes. Categories not limited to children are useful in some instances: Agoraphobia Without Panic Disorder (F40.00), some other Anxiety Disorders (F41), Reactions to Severe Stress and Adjustment Disorders (F43), Somatoform Disorders (F45), and Mild Depressive Episodes (F32.0) are likely to be found in some school phobic children. In DSM-IV (American Psychiatric Association, 1994), the category in the section of Other Disorders of Infancy, Childhood or Adolescence: Separation Anxiety Disorder (309.21), is particularly relevant. Of the DSM-IV disorders not confined to young people, Specific Phobia (300.29), Social Phobia (300.23), Agoraphobia Without History of Panic Disorder (300.22), Generalized Anxiety Disorder (300.02), Avoidant Personality Disorder (301.82), Posttraumatic Stress Disorder (309.81), Somatoform Disorders (300.81, 300.11), and Depressive Disorders (296 etc.) all have a place. Other categories of disorders also occasionally may be appropriate. School phobia has also been found to occur in the general population when no associated classifiable psychiatric disorder exists (Bools et al., 1990).

### Occurrence

The incidence of school phobia among referrals to child and adolescent psychiatric clinics has been reported as 11% (Chazan, 1962), 8% (Kahn and Nursten, 1962), and 4% (Smith, 1970). It thus forms a small, but not inconsiderable, part of the work of these services. In the cross-sectional Isle of Wight epidemiologic survey of children 10 to 11 years of age in the general population, the prevalence of school phobia was less than 3% of all psychiatric disorders (Rutter et al., 1970). A follow-up study at age 14 years produced substantially more cases of school phobia in the Isle of Wight, but no precise prevalence figures were given (Rutter et al., 1976). The severity of the manifestations of anxiety and depression that characterize school phobia, coupled with the severe social impairment that also is typical of the condition and the urgent need to do something about it that the legal requirement to attend school engenders, would provide obvious reasons for most cases coming to professional attention. However, there is some evidence that a considerable number of school phobic children are not identified as such and are dealt with more generally as school attendance problems (Galloway, 1985). A nonclinical sample of 100 severe school attendance problems (Bools et al., 1990) contained a

considerable proportion of children with school phobia, but only some of them had a classifiable ICD-9 emotional disorder. A study of 80 13- to 15-year-old children who failed to attend school for more than 40% of the time over several months ([Berg et al., 1993](#)) identified school phobia without an identifiable psychiatric disorder as well as school phobia accompanied by a DSM-III-R anxiety/mood disorder; 15 children in the sample had one of the latter disturbances. Few had had any contact with psychiatric services.

## Dependency

A child's attachment to and reliance on parental figures is referred to as *dependency* ([Bandura and Walters, 1963](#)). School phobic children often have been described as overdependent on their parents and as being overprotected by them ([Kahn and Nursten, 1962](#)). A self-administered questionnaire, completed by mothers, was developed to measure dependency: the Self-Administered Dependency Questionnaire (SADQ) ([Berg, 1974](#)). Compared with children in the general population, young adolescents with school phobia were found to be more likely to require assistance in washing, dressing, and carrying out other domestic tasks. There therefore was some evidence for undue reliance on parents. In the case of school phobic girls, they were less willing than control subjects to travel around unaccompanied. The SADQ includes a preference rating, and it appeared that mothers of school phobic children were more likely to encourage dependency in their children than would be expected ([Berg and McGuire, 1974](#)). However, an investigation of family life variables failed to show that families containing a school phobic child are deviant in the way they live their lives, looking at contact with relatives and friends, leisure and work activities, and how household tasks are apportioned ([Berg et al., 1981](#)). Some deficiency in self-reliance in the child's makeup therefore would appear to be a feature of children who get school phobia.

## Agoraphobia

The essentially adult disorder of agoraphobia is characterized by difficulties going out, traveling, and entering places from which escape is difficult, such as trains, buses, elevators, and crowded shops. There is a tendency to rely on the company of friends and relatives in going around because their presence seems to reduce anxiety. Agoraphobia has been thought of as a complication of panic disorder, which consists of severe and regular anxiety attacks. However, because agoraphobia often occurs without panic disorder, the suggestion has been made that some predisposition exists as shown by previous school phobia or a dependent personality ([Gelder, 1989](#)). A small proportion of adults who have agoraphobia have had the condition from before the end of the period of compulsory education ([Marks, 1969](#)). Transposing the features of adult agoraphobia to childhood and imagining how it would affect children, the result is very much what is observed with some children with school phobia who will not travel alone, who cannot go into school assembly, who are fearful of crowded playgrounds, and who rely excessively on their parents for comfort and security. Follow-up studies, in which adult psychiatric patients were asked if they had suffered from school phobia when they were young, showed an increased incidence compared with what would be expected in the case of affective and anxiety disorders in general, not just agoraphobia ([Berg et al., 1974](#); [Tyrer and Tyrer, 1974](#)). It also was found that children of agoraphobic women were more likely to have school phobia ([Berg, 1976](#)).

## Chronic Fatigue Syndrome

The so-called chronic fatigue syndrome, as it affects children, appears to have much in common with the masquerade syndrome described by [Waller and Eisenberg \(1980\)](#), where there is "home-based school absence" and physical symptoms without any apparent organic cause. In itself, staying home with little or no physical exercise can lead to a state of persistent fatigue and debility ([Wright and Beverley, 1998](#)). This suggests that those who advocate active rehabilitation and prompt return to school, in essence treating the problem as school phobia, have the correct approach ([Marcovitch, 1997](#)).

## Outcome

A number of follow-up studies of school phobic children have been carried out ([Gittelman-Klein and Klein, 1980](#)). In one of them ([Berg and Jackson, 1985](#)), 160 young adolescents with school phobia were reviewed on average 10 years after treatment in an inpatient psychiatric unit, when they were a mean 24 years of age. It was found that a third of them had required further treatment for psychiatric illness, mostly anxiety and depression. Five percent of the group had been readmitted to hospital because of severe affective disorders. Half were well on discharge after their treatment for school phobia, and they remained essentially free from further problems. The remainder had persistent but fluctuating symptoms and social impairment. More psychiatric illness had occurred than would have been expected. Similar findings were reported from a follow-up of 35 younger Swedish school phobic children with separation anxiety disorder (DSM-III), after 30 years of age, compared with other psychiatric patients and general population control subjects. Outpatient psychiatric treatment had been given in more of them and more of them had lived longer with their parents, as well as having produced less children themselves ([Flakierska-Praquin et al., 1997](#)).

## Management

So much has been written about the treatment of school phobia ([Berg, 1980, 1985b](#); [Hersov, 1990](#)) that it is necessary to include only a brief outline here. The principal aim is to bring about an early return to school ([Hersov, 1980](#)) because many of the symptoms of anxiety and depression that continue while the child is at home rapidly resolve once regular school attendance is restored. Management of associated psychiatric disorders is dealt with elsewhere in the book, so is not given much attention in this chapter.

### *Parental Counseling*

Regular contact with parents is important, on their own or with the affected child and other members of the family, when appropriate. It may be difficult to persuade parents of the need to bring about an early return to school. The usual reasons are either that they have been told by teachers, doctors, or others that the child is too ill to go and must be treated first, or they believe there is some physical illness as yet undiagnosed that should be identified and dealt with before the child can be expected to return. Less commonly, parents have a fixed view that something is wrong at school and that the child should not go back until that is put right. The process of counseling is to help disabuse parents and secure their cooperation. When there is the question of undiagnosed physical illness, the condition is called the *masquerade syndrome* ([Waller and Eisenberg, 1980](#)); persuading parents to accept that the problem is school phobia can take a little time. By rearranging hours of work, it may be that both parents can be there when it is time to go to school, so as to provide the child with more firm support. It may be necessary to accompany the child to school. It sometimes is only when the child realizes that the parents are at last determined to restore normal school attendance that resistance begins to crumble and progress is made. Counseling techniques along behavior modification lines can be used with parents to reduce their overprotectiveness and bolster their resolve, so that the child gets consistent cues and parent responses that help restore normal attendance ([Yule et al., 1980](#)).

### *Assisting the Child*

It is important to try to get the child's cooperation in going back to school. Regular individual counseling sessions may be helpful in achieving this. Initially there may be frank hostility, with the child refusing to cooperate in any way, but the situation usually improves with time, and it may help for the child to have some say in when and how return to school is brought about.

### *Behavioral Methods*

There is some evidence that a behavioral approach to ensure return to school and help parents and teachers respond appropriately to the problem of school phobia can be successful ([Blagg and Yule, 1984](#); [Yule et al., 1980](#)). A cognitive-behavioral approach used in a randomly assigned study indicated that school attendance improved significantly and emotional upset was reduced ([King et al., 1998](#)). However, an equally well designed and even more substantial investigation of cognitive-behavioral treatment failed to show that it was any more effective than traditional educational and supportive methods of management ([Last et al., 1998](#)).

### *Medication*

Some anxiolytic medication may be helpful in the initial stages of reintroducing a child to school ([Elliott, 1999](#)). There is no evidence that tricyclic antidepressant medication adds anything therapeutically to the clinical and psychological methods of management mentioned previously ([Berney et al., 1981](#); [Klein et al., 1992](#)). In their review of the use of antidepressant medication in school phobic children, [Murphy and Wolkind \(1996\)](#) take the view that it is not to be recommended because safer psychological treatment is available.

### *Placements*

Change of school often is suggested as a means of overcoming school phobia. It rarely is successful because the same problems arise at the new school, and neither



teachers nor other children have known the affected child under more normal circumstances. Sometimes special educational centers are available with transport provided, which can admit the child for a few weeks or months on a daily basis. The use of such units is recommended only when all attempts to get the child to normal school have failed. Special units normally have few children and a high staff-to-student ratio. Children with school phobia often find such centers acceptable and attend regularly without too much difficulty. From time to time, outpatient treatment, either alone or combined with a day placement, has no success, and admission to a residential center is required. Such centers often are part of psychiatric services and may be run on a weekly basis so that the child can be at home on weekends. There often are difficulties in getting the child to accept admission, but once this is accomplished, symptoms of emotional upset soon improve, and a planned return to school from home is arranged. Unit staff can be helpful in bringing this about. One or 2 days a week at the child's own school while he or she is still in the unit can be a useful stepping stone. The hospital admission has the added advantage of enabling associated psychiatric difficulties to be treated ( [Berg and Fielding, 1978](#)) by such methods as family therapy, social skills training, psychotherapy, and behavior modification, as appropriate. Bringing about separation from the family by admission does appear to have a beneficial effect in itself and starts the process of rehabilitation because the child usually attends the unit school and mixes with other children. Only rarely is admission to a boarding school, as a definitive solution to the problem, necessary. However, this may be the only answer when home circumstances warrant it (e.g., when there is mental or physical illness affecting a parent).

## TRUANCY

### Definition

Staying away from school without good reason with attempts to conceal the absence from parents is probably the most satisfactory definition of truancy ( [Fogelman et al., 1980](#)), although the term often is used more loosely to cover any unwarranted failure to go to school ( [Robins, 1978](#)).

### Prevalence and Etiology

Teachers' estimates of truancy often are preferred to those of parents or children, for obvious reasons. In one survey carried out in Glasgow, teachers judged that 3% of boys and girls were truanting at any one time ( [Institute for the Study and Treatment of Delinquency, 1974](#)). In the British National Child Development Study, teachers estimated that 8% of children had truanted in the previous year ( [Fogelman et al., 1980](#)). There probably are more boys than girls among truants ( [Fogelman et al., 1980](#)). Truancy predominantly affects poorer homes, where adverse social factors are evident, including large families, overcrowding, and multiple family problems ( [Farrington, 1980](#); [Hersov, 1960](#)). Some schools seem to foster truancy more than others ( [Reynolds et al., 1980](#); [Rutter et al., 1979](#)), possibly because of their more authoritarian regimes. There is a well established relationship between truancy and poor educational achievement ( [Douglas and Ross, 1965](#); [Fogelman and Richardson, 1974](#); [Gittelman-Klein and Klein, 1980](#)). There is a definite association between truancy and delinquency, even in girls ( [Berg et al., 1985](#)). The British Cambridge Study of Delinquent Development found truancy and delinquency to have similar backgrounds characterized by social adversity ( [Farrington, 1980](#)).

### Conduct Disorder

Psychiatric disorders characterized by antisocial behavior are common ( [Quay, 1986](#)) but tend to be confused with hyperactivity and attention deficit disorders, which frequently coexist ( [Taylor et al., 1986](#); [Trites and LaPrade, 1983](#)). In ICD-10 ( [World Health Organization, 1988](#)), truancy is given as one of the antisocial behaviors that occur in conduct disorders, but a recommendation is made that it should have persisted for 6 months before a child is considered as having a conduct disorder on the strength of it. Two main varieties are described: unsocialized conduct disorder, in which there is a pervasive impairment of relationships with other children, and socialized conduct disorder, in which there is not. Conduct disorders long have been thought of as the basis of truancy ( [Hersov, 1960a](#)). Suggestions that children who truant from school often fail to show the features of conduct disorders emerged from a general population survey of persistent absentees from school in Sheffield, England ( [Galloway, 1985](#)), and in a clinical study of 100 children who persistently failed to attend school without adequate reason ( [Bools et al., 1990](#)) who had been dealt with as disciplinary problems. It was found that approximately one-half of the group were typical truants, with a sex ratio of 3 boys to 1 girl. A cluster analysis indicated a group of 11 children nearly all of whom had both truancy and a severe conduct disorder, but there also was another group of 68, 44 of whom were categorized as truants but who usually had no conduct disorder.

### Clinical Features

Severity and persistence of truancy vary enormously. On the one hand, a child may take odd days off school because of some transient problem, and on the other, there may be persistent absence extending over weeks or months. Absence from school may be a solitary activity, or it may be in the company of others. The day may be spent wandering around, or the child may come home while the parents are at work ( [Belsen, 1975](#)). Efforts are made to pretend that normal school attendance is continuing, and accusations of staying off school frequently are met with denials. As would be expected based on previous discussion in this chapter, educational backwardness usually is associated with truancy, and when there is an associated conduct disorder, the situation is complicated by aggressiveness, destructiveness, poor personal relationships, wandering from home, dishonesty, and other antisocial activities, with or without delinquency. The family situation is, likewise, usually unsatisfactory in various ways, with severe social adversity creating stress in many instances. The child and adolescent psychiatrist is likely to encounter young teenagers who truant and who come into hospital having taken an overdose ( [Hawton, 1982](#)).

In Britain, disruptive pupils, many of whom truant, sometimes are permanently excluded from school when their conduct is particularly unacceptable. A recent survey by the Youth Justice Board found that the level and frequency of criminal behavior substantially increased after such pupils were expelled from school.

### Outcome

Severe and persistent truancy is an important forerunner of antisocial conduct occurring in adult life ( [Robins, 1978](#)). This may take the form of criminal behavior with convictions in the courts ( [Farrington et al., 1989](#); [Huissy and Howell, 1985](#); [Loeber and Dishion, 1983](#)). The importance of truancy as a predictor of antisocial tendencies in adult life is well established, but it should not be forgotten that many truants develop normally without manifestations of deviant behavior in later life.

### Management

Truancy normally is dealt with as a social, legal, or educational problem by social work, law enforcement, and school agencies. The severity and duration of the problem and its associated difficulties, particularly the occurrence of delinquent acts, often determine the ways in which it is handled ( [Turner, 1974](#)). Similar types of problems may go to juvenile court or be referred for psychiatric treatment ( [Gath et al., 1972](#)), so there appears to be an element of chance about which agency is left to manage the problem.

Some schools are more assiduous than others about checking up on and finding ways of preventing school attendance problems, by keeping in close touch with parents, providing education in keeping with the needs of their pupils, and motivating children to keep coming to school ( [Boyson, 1974](#); [Jones, 1980](#)). Special day units sometimes are available, which have a high teacher-to-pupil ratio and offer a limited education, which truants sometimes find more acceptable than normal school ( [Galloway, 1980](#); [Sproule, 1974](#)).

In Britain, there are education welfare officers, employed by local education authorities, who have the task of enforcing school attendance ( [Clark, 1976](#)). They keep an eye on school registers and visit homes where they suspect a child may be away from school without good reason. Parents or children may be taken to court if unwarranted absence from school continues. A series of randomly controlled trials carried out by juvenile court magistrates showed the value of courts supervising children who were persistently out of school without a satisfactory excuse in reducing unwarranted absence ( [Berg et al., 1988](#)). Occasionally, children who continue to truant are taken into care or sent to residential schools when efforts to provide a solution to truancy have failed.

Confronted with the problem of truancy, the clinician often must deal with associated conduct problems, educational backwardness, and family disadvantage. Close liaison between home, school, and the professional agencies involved is essential in restoring and maintaining normal school attendance. Particular difficulties may be helped by clinical procedures. Thus, poor peer group relationships may respond to social skills training. Social work counseling along behavioral lines may be helpful in motivating parents to keep in regular touch with school and take the child to school when appropriate. Very little has been written about the efficacy of routine clinical methods in truancy ( [Berg, 1985a](#)). If the analogous problem of absconding from residential institutions is anything to judge by ( [Clarke, 1980](#)), then close supervision, surveillance, and preventative activity by home and school will be the most useful approaches. Occasionally, children who come to clinics with problems of truancy have satisfactory homes, no educational backwardness, and few associated conduct problems. Clinical techniques such as counseling and behavior modification procedures, including the use of reinforcement, may be worth trying. Further work on the evaluation of different treatment approaches in truancy

is urgently needed.

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# 108 ADOPTION

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Adoption involves the child psychiatrist primarily in terms of clinical consultation and treatment of the child–adoptive parent family unit. This chapter focuses on extrafamilial adoption, which currently comprises close to one-half of adoptions. The other half of adoptions, intrafamilial adoption, is comprised of adoption by step-parents after divorce, adoption by fathers of their out-of-wedlock children, and adoption of family members by uncles, aunts, and grandparents. The major psychodynamic issues affecting children who experience intrafamilial adoption are quite different from those affecting children who are adopted extrafamilially and are not treated in this chapter.

## ADOPTION AND SOCIAL CHANGE

Spanish immigrants brought to this country adoption modeled on the Roman tradition, in which adoption primarily benefitted the adopter ( [Huard, 1956](#); [Presser, 1971](#)). The purpose of adoption soon changed to that of providing homes for children in need of parents. Adoption, like divorce, was an occasional event until well into the 1800s. If one wished to adopt or divorce, a petition was made to one's state legislature ( [Witmer et al., 1963](#)). In 1851, Massachusetts was the first of the states to provide a general adoption law ( [Whitmore, 1876](#)).

Until the middle of the 20th century, adoption by deed without involvement of public agencies or the courts was the rule. In Texas until 1931, a child could be adopted by filing a written statement of adoption with the county clerk ( [Jacobs and Goebel, 1952](#); [Texas Revised Civil Statutes, 1925](#)). If the natural parents were living, a written transfer of parental authority accompanied the statement of adoption. After World War II, as adoption became part of the child welfare system, the system of agency adoption with screening and preparation of parents and counseling with birth parents was established ( [Kadushin, 1976](#)).

The number of children that young single women have given up for adoption has diminished because of increased use of contraception and wider availability of abortion, but probably most of all because these mothers choose to raise their children. Adoption of healthy infants appears to be increasingly the domain of independent adoption arranged by individuals and their attorneys ( [Wells and Reshotko, 1986](#)). Nonrelatives adopt probably 50,000 children yearly. Roughly 40% involve public agencies, and about 30% involve private agencies and independent adoptions ( [Berry et al., 1996](#)). International adoptions have stayed at about 10,000 per year for a number of years ( [Altstein et al., 1994](#)). Transracial adoptions within the United States of Native American children by white couples have been very few since the passage in 1978 of the Indian Child Welfare Act. Adoption of African-American children by white couples has varied according to changing federal legislation ( [Grotevant and Kohler, 1999](#)). Children who come from the foster care system and those with special physical and emotional needs are generally placed by public agencies. More than 50% of all adoptions via public agencies involve children designated as having special needs ( [Rosenthal and Groze, 1994](#)), which includes children who: (a) are over 10 years of age; (b) have serious physical, intellectual, or emotional problems; or (c) are members of a large sibling group. The most commonly occurring special needs are emotional disturbance and mental retardation ( [Rosenthal, 1993](#)). Recent developments in this area are presented in some detail because of the importance of the child welfare system to adoption and because consultation is often requested of child psychiatrists by public agencies.

## The Child Welfare System and Adoption

Some changes have taken place regarding the stability of foster placements, and foster parents being allowed to adopt children in their care. The changes have resulted in large part because of increased public awareness of the problems facing children in care. [Maas and Engler \(1959\)](#) published their study of over 4,000 children in foster care in 1959. Their data indicated that more than half of the children would remain in foster care for most of their childhood. A 1973 study of children in foster care in Massachusetts indicated that 60% had been in foster care between 4 and 8 years, with the average being over 5 years ( [Gruber, 1973](#)). The Columbia University longitudinal study of children in foster care in New York City showed that two-thirds of the 36% of children who remained in foster care had lost contact with their biological parents at the end of 4 years ( [Fanshell, 1976](#)). In 1983 a class action suit was brought against the entire foster care system of Jackson County, Missouri, which encompasses Kansas City. Twenty-nine percent of the children had been placed in four or more homes within a span of less than 5 years. The longer a child remained in care, the more likely he or she was to experience movement from home to home ( [Mushlin et al., 1986](#)). A federal court ordered specific improvements in foster care policies and practices ( [G.L. v. Zumwalt, 1983](#)). That decision relied on a doctrine that children in public care have a "right to freedom from harm" ( [New York State Association for Retarded Children v. Rockefeller, 1973](#)).

Various schemes have been devised for stimulating permanency planning and activity in order either to return the foster child to the parental home or make the child available for adoption ( [Bush and Goldman, 1982](#); [Festinger, 1975](#); [Seltzer and Bloksberg, 1987](#); [Ten Broeck and Barth, 1986](#); [Wiltse, 1985](#)). The 1980 Adoption Assistance and Child Welfare Act (PL 96-272) stipulated federal requirements for a reporting and tracking system for children in foster care ( [Hartley, 1984](#)).

## The Foster Parent: More Rights and the Ability to Adopt

Traditionally foster parents have not been considered to have a legally recognizable interest in their foster child. When foster parents have attempted to contest removal of the child by the welfare agency or have attempted to adopt the child against agency wishes, courts have almost uniformly upheld the sanctity of the placement contract between foster parents and the agency along with the rights of the biological parents ( [Katz, 1971](#)). In the 1977 case of *Smith v. Organization of Foster Families*, the Supreme Court recognized that for a child



who has been placed in foster care as an infant, has never known his natural parents, and has remained continuously for several years in the care of the same foster parents, it is natural that the foster family should hold the same place in the emotional life of a foster child, and fulfill the same socializing functions as a natural family. For this reason, we cannot dismiss the foster family as a mere collection of unrelated individuals (pp. 844–845).

The Court, however, judged the procedures utilized by the State of New York to be constitutionally adequate for the protection of the foster care parent–foster child relationship. Any rights that foster parents accrue can benefit the foster children in their care because of the enhanced stability of the foster placement ( [Derdeyn, 1986](#); [Derdeyn et al., 1978](#)).

Studies of children's experience in foster care and adverse publicity regarding various aspects of the public child care system have led to some significant changes with regard to foster parents' becoming adoptive parents ( [Coyne and Brown, 1986](#)). In the past child welfare agencies selected adoptive and foster parents according to quite different standards. Now the requirements for foster and adoptive parents vary little, and adoption by foster parents has become commonplace ( [Derdeyn, 1990](#)).

### Openness Versus Privacy in Adoption

Adoption policy until the last three decades has required that the biological parents, whether terminating rights voluntarily or by legal force, disappear completely from the child's life ( [Wrobel et al., 1998](#)). In recent years, because of pressure from some adopted persons and advocacy on the part of some professionals, there is interest in and more adoptions taking place under conditions of "open" adoption ( [Avery, 1998](#); [Baran and Pannor 1990](#); [Belbas, 1987](#); [Berry, 1993](#); [Chapman et al., 1987](#); [Demick and Wapner, 1988](#); [Sachdev, 1991](#); [Triseliotis, 1987](#); [Watson, 1988](#)). This means that the birth parent meets the adoptive family and has some arrangement regarding sharing information or visiting after adoption. In a related phenomenon, an English study ( [Ryburn, 1996](#)) discovered postadoption contact after termination of parental rights between children and their birth relatives to be over 50%.

Some states have passed laws to open the sealed adoption record for adoptees wishing to make contact with birth parents. The birth parent is notified and is given the option to consent or to refuse the adoptee's request. If one birth parent has died or a birth parent does not consent, the adopted person may petition the court to open the birth record. The court then acts on the basis of weighing the interest of all the parties ( [Auth and Zaret, 1986](#); [Weidell, 1984](#)).

In the areas of joint custody and grandparent visitation, the courts and society are increasingly accepting of diminished family boundaries. In these instances, however, it is adults and not the children of divorce who have been motivated for change and effected that change by political means ( [Derdeyn, 1985](#); [Derdeyn and Scott, 1984](#)). The desire for change in adoption is different from that in divorce, in that it is the adoptees themselves who are feeling the need to make changes in the structure of adoption to diminish the secrecy and mystery, and to provide knowledge of and contact with biological parents. Some years ago, [Triseliotis \(1973\)](#) suggested that people searching for biological parents might have unsatisfactory relationships with adoptive parents and poor self-images, whereas [Sorosky and associates \(1975\)](#) determined that their interview study established that was not the case. Today the desire to make some sort of contact with biological parents is widely accepted as a normal phenomenon devoid of implications of psychopathology or maladjustment ( [Anderson, 1988](#); [Aumend and Barrett, 1984](#); [Campbell et al., 1991](#)). Clinical experience as well as some reports in the literature ( [Moran, 1994](#); [Sorosky et al., 1974](#)) indicate that making contact with birth parents is generally found to be arduous but ultimately relieving emotionally for the adoptee and birth mother, and challenging for adoptive parents ( [Silverman et al., 1994](#)). It is becoming apparent that many women who relinquished a child have a desire to establish contact and gain some emotional relief from doing so ( [Field, 1992](#)). A group of clinicians have determined a need for and have developed adoption triad—adoptee, adoptive parents, and birth parent(s)-support groups ( [Valley et al., 1999](#)). A suggestion has been made of a relationship linking dissimilarity between adoptee and adoptive parents and the drive to search for birth parents. The drive to search would arise out of the more problematic identification with dissimilar parents ( [Hollingsworth, 1998](#)).

A survey regarding 1,396 newly adopted children in California indicated that most of the adoptions were open, and that children in open adoption had better behavior scores than children without access to birth parents ( [Berry, 1991](#)). One investigator found that adoptive mothers who had prenatal contact with the child's mother reported a significant positive effect on their attitudes toward both the biological mother and adopted child ( [Lee and Twaite, 1997](#)). Two-thirds of these adoptive parents had met one or both of the birth parents. A study of 12 high-risk adopted children and their families determined that cooperation between the children's adoptive and birth family members appeared to positively affect social and emotional outcomes for these children ( [Grotevant et al., 1999](#)). Agencies, typically protective of confidentiality, had 40% of their adoptions open, whereas 80% of the independent adoptions were open ( [Berry, 1991](#)). Regardless of whether adoption was open or closed, families adopting through private agencies have been found to feel best prepared and satisfied ( [Berry et al., 1996](#)). A 4-year follow-up study of a sample of 764 adoptions showed a marked drop in contacts in open adoptions over that span of time, and indicated no effect of openness on child adjustment ( [Berry et al., 1998](#)).

### ADOPTION OUTCOME

Reports of the incidence of adoption in children referred for outpatient treatment have established that adopted children are overrepresented compared to their proportion in the population, which is usually estimated to be 3.5% ( [Schwam and Tuskan, 1979](#)), and that they tend to be referred for externalizing, acting-out behaviors ( [Finley, 1999](#); [Goodman et al., 1963](#); [Humphrey and Ounsted, 1963, 1964](#); [Menlove, 1965](#); [Offord et al., 1969](#); [Schechter, 1960](#); [Schechter et al., 1964](#); [Simon and Senturia, 1966](#)). A study of 2,148 international adoptees found that early neglect, abuse, and the number of changes of care taking environment increased the risk of later maladjustment ( [Verhulst et al., 1992](#)). Recent studies have continued to find an overrepresentation of adopted children and adolescents in outpatient clinics ( [Kotsopoulos et al., 1988](#)) and on inpatient wards. A study of 3,698 adolescents, 145 of whom were adopted, corroborates the overrepresentation of young adoptees in clinical settings. The adoptees did have more problems than the controls, but were also referred more readily for lesser problems ( [Warren, 1992](#)).

Adopted children are often classified for investigative purposes according to the following factors: adoption at birth; adoption at an older age after satisfactory early-life care; and adoption at an older age after adverse early-life care. Problems in childhood and adolescence tend to increase with age at adoption and having experienced adverse care ( [Howe, 1997](#)). Some children adopted from orphanages in foreign countries have experienced the deprivations of limited caretaker contact, yet adapted surprisingly well, and their parents were satisfied with the adoption ( [Groze and Ileana, 1996](#)). Developmentally vulnerable is the term often applied to children with multiple developmental challenges: having been born to drug abusing mothers, having histories of low birth weight, failure to thrive, neurological problems, and so on. The drug-exposed children tend to become indistinguishable from non-drug-exposed children ( [Barth and Needell, 1996](#)), and children with other developmental vulnerabilities tend to do well and have satisfied adoptive parents ( [Hoopes et al., 1987](#)).

A 1985 study of inpatient adolescents found a great overrepresentation (over 21%) of adopted children, but no difference in diagnosis from the control group ( [Senior and Himadi, 1985](#)). Senior and Himadi cite a number of studies of adopted inpatients. Although one of the studies found a slight underrepresentation ( [Humphrey and Ounsted, 1963](#)), the rest showed a significant overrepresentation: 9.4% of girls and 9.3% of boys ( [Work and Anderson, 1971](#)), 7% of boys and 11% of girls ( [Kellman-Pringle, 1961](#)), and 4.3% of both boys and girls ( [Kirk et al., 1966](#)). A review of over 5,000 patients registering for their first psychiatric hospitalization found a moderate overrepresentation of adoptees on the children's service and an underrepresentation on the adult service ( [Brinich and Brinich, 1982](#)). [Rogeness and colleagues \(1988\)](#) found 8.7% of 763 consecutive admissions to a children's psychiatric hospital to be adopted. [Dickson and colleagues \(1990\)](#) found 11.7% of their inpatient population to be adopted.

[Kim and associates \(1988\)](#) found an overrepresentation of adoptees in their hospitalized children and an underrepresentation of adopted children referred to the regional juvenile court. These authors postulated that the relatively high socioeconomic status of adoptive parents was instrumental in their adoptive children's entering the psychiatric rather than the criminal justice network. A 1975 study of adopted children before the juvenile court found the children's parents to be extremely rejecting of them and the courts to be particularly harsh with them, compared to a group of neglected children also referred by the court to the psychiatric clinic. The authors ascribed the difference in the court's dispositions in the two groups to the parents of neglected children contending for their custody and the parents of the adopted children failing to defend them or even requesting placement in correctional facilities ( [Lewis et al., 1975](#)). A more recent study of 3,280 juvenile delinquents did not find significant differences between adopted and nonadopted children in terms of their dispositions ( [Kim et al., 1992](#)).

Studies carried out with nonclinical school populations also found some differences for the adopted group. [Brodzinsky and colleagues \(1984\)](#) studied 260 adopted and nonadopted children by means of maternal and teacher ratings. Adopted children were found to be higher in psychological and school-related problems and lower in social competence and school achievement than were nonadopted children. [Lindholm and Toulaitos \(1980\)](#) compared 41 adopted and 2,991 nonadopted children of a suburban school district by means of teacher ratings on a behavior problem checklist. Adopted children evidenced more conduct and personality problems. The Ontario Child Health Study, a community survey of 3,294 children, 104 of whom were adopted, found that adopted boys had a significantly increased risk of psychiatric disorder versus nonadopted boys ( [Lipman et al., 1992, 1993](#)). A recent comprehensive review of adoption as a risk factor for development pointed out that

the increased involvement of adopted children in mental health treatment may be because of their parents' and other adults' predisposition to seek treatment, and because of disturbance related to early-life maltreatment ([Haugaard, 1998](#)). The issue of adoption and psychopathology might best be stated: Adopted children exhibit more psychopathology than nonadopted ones but not as much as their adoptive parents think they do ([Sullivan et al., 1995](#)).

### Transracial Adoption

The literature indicates that the adjustment of these children is indistinguishable from that of white children ([Feigleman and Silverman, 1984](#); [McRoy et al., 1982](#); [Shireman and Johnson, 1986](#); [Silverman and Feigleman, 1981](#); [Tizard, 1991](#)). An 18-year follow-up study of Asian, black, and white children adopted by white couples showed better adjustment of Asian and black children than of white ones ([Brooks and Barth, 1999](#)). A comparison of early adopted Korean-American adoptees with biological children of their adoptive parents showed lower social competence in the adopted children and higher internalization in biological children, but very little difference in adjustment overall ([Kim et al., 1999](#)).

Although adopted children and the adoptive family receive a disproportionate share of clinicians' attention, it is also evident from the literature that most adoptions go very well indeed. [Kadushin \(1967\)](#) reviewed a number of follow-up studies regarding children adopted in infancy ([Brenner, 1951](#); [Davis and Douck, 1955](#); [Edwards, 1954](#); [Fairweather, 1952](#); [Fradkin and Krugman, 1956](#); [Morrison, 1950](#); [Nieden, 1951](#); [Theis, 1924](#); [Witmer et al., 1963](#)) and found "success" rates of 78% to 86%. The younger the age of adoptive placement, the better the outcome ([Humphrey and Ounsted, 1963](#); [Kotsopoulos et al., 1993](#); [Offord et al., 1969](#)).

### BENEFITS OF ADOPTION

Adoption is very desirable and constructive for those children who are in need of it. Although the fact and the mystery of being relinquished by parents is an important issue for every adopted person, it is the early life experiences that are surely the most problematic for children who are later adopted. Studies of children who moved from foster to adoptive families have found that immediate and long-term problems were greater when the move was made between 6 and 12 months than when it was made before 6 months ([Yarrow and Goodwin, 1973](#); [Yarrow and Klein, 1980](#)). The deleterious effect of early experience was mitigated by high quality of care in the adoptive home.

The value of adoption to children is made particularly evident by studies that include children who have fates other than adoption. A group of children who spent their first 2 to 7 years in an institution was followed for a number of years. The children who were adopted were noted to be overly friendly with strangers and attention seeking, and to cause moderate to severe problems in the school situation. The children who were in foster care or who returned to their families, however, fared much worse ([Tizard and Rees, 1975](#)). [Bohman and Sigvardsson \(1980\)](#) identified 624 Swedish children who were candidates for adoption as infants. At 11 years of age they evidenced greater maladjustment than classmates, whether they had been adopted, placed in foster care, or remained with their biological mother. At age 15, adjustment of the adopted children differed only slightly from that of their classmates, whereas children living in foster homes or with their biological mothers had a high degree of social maladjustment and/or scholastic underachievement. The military enlistment process at age 18 confirmed the findings at age 15. The adopted boys were indistinguishable from age-matched controls. Boys who remained with their mothers or were raised in foster homes scored lower in intelligence and psychological tests and were more frequently exempted from military service for social or psychiatric reasons than the adoptees ([Bohman and Sigvardsson, 1990](#)). [Dumaret \(1985\)](#) followed the progeny of 28 French mothers: 35 children were abandoned and adopted early in life by well-to-do families, 46 were raised by their biological mothers and/or her relatives, and 21 were raised in institutions or foster homes. In primary and secondary schools the percentage of school failure for the adopted group was 17%; for the biological family-raised group, 67%; and for the institutional or foster care group, 100%. (All the institutional or fostered children had been excluded from the normal secondary school curriculum.) Teachers' ratings of behavior showed both the adopted and family-raised groups of children to be more problematic than their classmates.

### HEREDITY AND ENVIRONMENT

The developing field of behavioral genetics has shed much light on issues of nature and nurture through studies comparing siblings, partially related siblings, twins raised together and apart, and biologic parents and their adopted-away offspring. Although quantifying the role of genetics, these studies are also quite remarkable for quantifying the effects of environmental influences. Two general forms of environmental influence, shared and unshared environment, have been shown to contribute to developmental processes and outcomes in the context of genetic influence. Shared environment refers to the environment common to siblings raised together. Nonshared environment refers to influences and experiences unique to an individual within a family of siblings. It may include perinatal factors, traumatic experiences, extrafamilial experiences, illnesses, different parental expectations and reinforcement, or even common factors experienced differently by different children ([Pike and Plomin, 1996](#); [Rowe, 1987](#)). Behavioral genetics gives us a unique window into the interaction of genes and environment in adoption.

### Intelligence and Cognitive Abilities

An early study by [Skodak and Skeels \(1949\)](#) suggested a substantial influence of early environment in raising the average IQ of a group of adoptive children. The brighter children had brighter biological mothers, however, indicating that heredity played a role in individual differences, although the environment raised the IQs of the group of adopted children as a whole.

Subsequent studies have indicated that individual differences in IQ are substantially influenced by genetic differences among individuals, but that family environment also has a significant impact ([Scarr and Weinberg, 1983](#)). A Texas study involved 300 families who were adopted immediately after birth from a home for unwed mothers ([Horn, 1983](#)). Psychometric testing was carried out with the biological mother, the child, and both adoptive parents. The adopted children were more influenced by their biological mothers than by the intellectual milieu of the adoptive family. Other research suggests the environmental influence of middle- to upper-class families on the IQ of adopted children may be a temporary one limited to childhood ([Rowe, 1997](#)).

Environmental factors play an important role in childhood, but by late adolescence genetic influences best predict intelligence ([Lynn, 1994](#); [Scarr and Weinberg, 1983](#)). [Alarcon and colleagues \(1998\)](#) assessed data from the Colorado Adoption Project and found that heritability accounts for around half of the correlations among cognitive abilities at age 12. Plomin also has suggested that genetic influences account for approximately half of the variance in general cognitive ability ([Plomin, 1999](#)). Reviewing the environmental influence on intelligence, Kaufman has compared it to the environmental influence on weight, where lifestyle plays an important role in the context of a genetic predisposition ([Kaufman, 1999](#)).

### Behavior Disorders

Studies in this area are notable for findings of both significant hereditary predisposition and significant environmental effects of the adoptive home. [Cadoret and Crowe \(1983\)](#) found a marked increase in antisocial behaviors in adolescence when the adoptee had both a genetic factor and an adverse environmental factor such as psychiatric problems, antisocial behaviors, or drinking problems in the adoptive home. Biological parents who were alcoholic or antisocial tended to have adopted-away offspring who were antisocial in adulthood. Two environmental factors significantly increased adoptee antisocial personality: placement in a home where there was an alcohol problem or antisocial behavior, and placement in a lower socioeconomic home for a child whose biological parents had exhibited criminality or delinquency ([Cadoret et al., 1990](#)). [Roth and Finley \(1998\)](#) reviewed the literature on the development of antisocial behavior and concluded that biological parent antisocial behavior, adoptive parent antisocial behavior, and low adoptive family socioeconomic status all predict antisocial behavior in adoptive children, although the association appears to be strongest with the biological parent status. [O'Conner and coworkers \(1998\)](#) investigated antisocial behavior and depressive symptoms over a 3-year period in a population of siblings and twins, finding a strong role for both genetic and nonshared environmental influences on the stability of these symptoms. Shared environment influenced antisocial behavior but not depressive symptoms. Additionally, genetic factors appeared to vary over the 3-year period, suggesting that the nature and impact of genetic factors on behavior may change through the developmental process. [Gjone and Stevenson \(1997\)](#) showed a genetic influence on the covariance between emotionality and attention problems and emotionality and aggressive behavior. The importance of nonshared influences on antisocial behavior and depressive symptoms is underscored by findings that suggest almost 60% of the variance in antisocial behavior and almost 40% of the variance in depressive symptoms is because of conflictual and negative parenting behavior directed toward an individual ([Reiss, 1995](#)).

### Depression

Most investigations into the development of depressive symptoms show that heredity causes between 33% and 50% of the variance between individuals, with nonshared environment being the dominant environmental factor ([Eley et al., 1998](#)). Eley found that in childhood, nonshared environmental factors have the greatest impact on the development of depressive symptoms, followed by shared environment, and finally genetic factors. There is evidence to suggest that the effects of heredity may increase with age, but favorable environmental factors may delay or alter the manifestation of hereditary influences ([Muhs and Schepank, 1995](#)). [Pike](#)



[and Plomin identified maternal negativity to be particularly salient in the development of depressive symptoms \(1996\)](#).

## **Adoption and the Interaction of Heredity and Environment**

Genes and environment have an interrelated impact on the development of adopted children. The parenting practices of adoptive parents (adoptive environment) are affected by the behavior of their adopted children, which itself may be significantly modulated by genetic factors. Thus, genetic influences on the child may also act to modify the environment in which the child lives. The impact of a child's genes on the environment has been demonstrated to begin in infancy through both cognitive and temperamental factors ([Saudino and Plomin, 1997](#)), and there is also evidence for genetic influence on the environment through antisocial and hostile behavior ([Ge et al., 1996](#); [Roth and Finley, 1998](#)). The relationship between negative parenting, generally seen as an environmental influence, and antisocial or depressive symptoms may be primarily because of genetic factors (Niederhiser et al., 1999).

Behavioral genetics has shown that genetic factors play a significant role in child and adolescent development, and that these factors influence the cognitive and personality traits of adopted children. As significant as these influences are, environmental influences, especially nonshared environment, play a vital role as well. Genetic influences, with the possible exception of some cognitive abilities, do not lock individuals into a particular developmental trajectory. Rather, there is a complicated and diverse interaction between genetic and environmental factors affecting development.

## **ADOPTIVE CHILDHOOD: CHALLENGES AND DIFFERENCES**

It is a testament to the complexities of the human mind that the fact of adoption makes the difference that it does. One could surmise that on the face of it, adoption should not make much difference to a child adopted early in life, for the child can have no perspective on the identity of the care taking adults. Later, however, when the child is told of adoption, he or she must deal with the incomprehensible questions of who the other parents are and why they left him or her. Although the practice of telling children early of their adoption is almost universal, the benefits and liabilities remain unclear ([MacIntyre and Donovan, 1990](#)). [Brodzinsky and colleagues \(1981, 1984, 1995\)](#) have established that "being told" about adoption leads to a process of understanding on the basis of outside information provided and inside cognitive development. As in most areas of significant communications between parent and child, the parent's comfort regarding adoption is of utmost importance. The parent who can talk easily of adoption is likely to be able to observe the child's response and gauge what is communicated by the child's response at a given time.

### **Adoptees' Images of Biological Parents and Self**

Children tend to "solve" the narcissistic injury and unfathomable mystery of having been given up for adoption by assuming that they were unlovable, dirty, too angry, or otherwise bad or unrewarding to their biological parents.

One adopted boy talked about how at the orphanage the children were not cared for and were fed slop. When his parents brought him home at 3 months, he vomited white stuff that he had been fed at the orphanage. At the orphanage they only cared for themselves and were just trying to get rid of all the little kids, which is what his real mother wanted to do also.

Nickman points out the complex of losses adoption entails: the knowledge of having been relinquished, with its deleterious effect on self-esteem; lack of knowledge regarding one's birth parents; and the multitude of stigmatizing experiences society visits on adopted children.

The core issue for the child is "not having been kept by one's parents . . . the fact that the child did not come into the world as a wanted person, into the arms of a man and woman who together could eagerly and lovingly assume his care." ([Nickman, 1985](#), p. 376). Nickman advocates a continuing dialog regarding adoption between the child and adoptive parents to grieve the various losses and counter the tendency of adoptees to experience themselves as devalued.

Some children deal with the injury and mystery by attributing the cause or blame to the biological parents. They assume their parents were bad, alcoholic, or mentally ill; their fathers were criminal and their mothers were prostitutes. This process of blaming their parents still does not leave the child unscathed, because the identification with "bad" parents can be quite strong.

### **Anxious Attachment**

Many adopted children readily assume that abandonment or other loss could well happen again. This is probably enhanced by unconscious rage at having been abandoned previously, in that the parents may retaliate against the anger by "again" abandoning the child. For example, an adopted 13-year-old boy reported that his parents had gone for a few days to Israel. He immediately commented in what appeared to be a casual fashion that if something happened to them, he would have to be adopted again, and he sure would hate that.

### **Identity Issues in Adolescence**

Knowledge about and experiences with one's parents and extended family contribute greatly to the establishment of a sense of identity. [Erikson's \(1959\)](#) definition of the sense of ego identity as "the accrued confidence that one's ability to maintain inner sameness and continuity is matched by the sameness and continuity of one's meaning for others" (p. 89) suggests the liabilities adoption might bring with it. [Wellisch \(1952\)](#) and [Sants \(1964\)](#) has written of the adolescent experiencing "genealogical bewilderment."

### **Searching**

Adolescents are curious about their own and their family's history. Adoptive children have a double handicap in this regard, in that they find it difficult to locate their own personal history within that of their family, and they are likely to experience their curiosity about their origins and early life as conflictual within themselves and difficult for their adoptive parents.

Many adult adoptees continually search faces for resemblance and wonder if a person talked with or observed might be a relative. [Sants \(1964\)](#) described adoptees developing a fear of committing incest, and this also has been an issue in the author's clinical experience with adoptees.

[Schechter and Bertocci \(1990\)](#) reject the frequently mentioned "curiosity," and explain its use as relegating the search to a simple cognitive issue and as an attempt to avoid any impression of pathology. Underlying the relatively conscious and commonplace motivations—dissatisfaction with the adoptive family, finding someone who looks and is like the adoptee, seeking genealogic or medical information, finding an way to feel "connected"—is a powerful need to be the driver instead of the passenger in life's drama.

All studies of reunions indicate positive experiences by the adoptees. Relatively few develop intense connections with their biological parent, in spite of that being an important initial motivation for their search. The sense of relief, completeness, and renewed readiness for life that people with a completed search almost universally experience is an internal event of great developmental importance. The wrong is righted; the lost parent (child) is found; and control is gained over the chaos, confusion, and helplessness with which life began.

## **ADOPTIVE PARENTHOOD: CHALLENGES AND DIFFERENCES**

### **A Reciprocal Relationship between Parent and Child**

Under the best of circumstances, there are challenges to the development of a reciprocal relationship between parent and child. There are differences of "fit" between parent and child in terms of responsiveness, activity level, and other characteristics. Because of marked genetic differences, the fit of parent and child may be especially problematic in adoption. At the least, the child is days old on arrival at the adoptive home, or the child may be years old. The child's relationships may have been disrupted several times before; therefore, the child brings ambivalent attitudes to the relationship with the new parents. Differences in intelligence between the adoptive parents and adopted child may create problems. The legal course of adoption, where the adoption is not finalized for many months, with the parents being essentially on probation for that period of time, also can impede the relationship's development. The rights of biological parents continue to be a threat, in that courts

may belatedly remove a child from adoptive parents ([Kermani and Weiss, 1995](#)). Other issues of importance that can cause attenuation of the parents' attachment are societal ambivalence regarding adoption and grandparents' disapproval of the adoption ([Blum, 1983](#)).

### **Infertility and Situations Where Pregnancy Is Medically or Genetically Contraindicated**

Infertility has not usually been suspected by either of the adults, requiring a painful alteration of self-concept of the prospective adoptive parent. Medical problems of the woman and genetic liabilities of either spouse may lead to a choice not to conceive.

### **The Fate of the Biological Child**

Adoptive parents must acknowledge emotionally that the adopted child is a replacement of the child that they cannot have ([Solnit and Stark, 1961](#)). If the parents are not able to adequately grieve the wished-for biological child, the extent and nature of their attachment to the adopted child may be affected. In addition, there may be a parental analogy to the child's confusion about having two sets of parents: The parent may maintain a fantasy about a dual identity of the child—the natural and adopted child. Insufficient emotional acceptance for the adoption alternative may be accompanied by an idealized image of the natural child who might have been. In addition, the adopted child may be the target of some anger because of the child's not affirming the parental biological role and reminding the parent of what is not possible.

### **The Absence of Pregnancy**

Most couples experience many months of preparation for parenthood throughout the pregnancy. The myriad manifestations of pregnancy serve as a focus to stimulate both parents to come to grips with some of the changes occurring in the marital relationship. The birth of the new child is increasingly anticipated, and the pending loss of freedom and of being able to attend to one's own and each other's needs unavoidably will be faced because of various aspects of the pregnancy process. In adoption, these processes tend to be attenuated because there is no pregnancy, and often because it cannot be predicted when a child will become available for adoption.

### **The Adoptive Parents as Rescuer**

There is a particular danger to the parent-child relationship if the parent views adoption as a rescue. When there is frustration of rescue fantasies in the course of the conflict inherent in child raising, a very angry response from the adoptive parents often results. Problems in this area may be closely connected to the issue of failure to work through the loss of the child one cannot have. When the child has had significant deprivation in the past and exhibits the characteristic sequelae of emotional withdrawal, indiscriminate friendliness, or provocative behavior, however, even the most capable adoptive parent is likely to feel rejected.

### **Entitlement or Ownership of the Child**

Adoptive parents sometimes feel that their right to be the parents of the child is tenuous. This attitude may, in major part, be the result of insufficient resolution of the losses and issues inherent in the developmental process of adoptive parenthood. As a result, the adoptive parents may fail to take on the full role of parenthood in terms of confidently providing the guidance and control that enhance feelings of security and self-confidence in younger children. A recent study suggests that adoptive parenthood is associated with enhanced paternal and diminished maternal interaction with the adopted child ([Holditch-Davis et al., 1999](#)).

### **The Adoptee as Alien Child**

For the biological mother, her child is, or was, part of her. For the adoptive mother, the child was part of someone else. This may relate at the mildest level to difficulties of attachment and of the parents' feeling entitled to the child. But there may also be implications for strongly negative parental attitudes. When there are difficulties in adoption, one quite often observes that the parent has trouble accepting the adoptive child's instinctual expressions in a variety of areas—soiling, sexual curiosity, aggression, and, very frequently, eating ([Brinich, 1980](#)). The child's instinctual side is the target of strong and quick prohibitions and expressions of negative expectations. There may be a rather disconnected pair of internal representations, one acceptable and one not. Referral tends to revolve about behaviors in the instinctual sphere.

## **CLINICAL ISSUES**

Parents who bring their adopted children to psychiatric facilities often have a high degree of concern regarding the child's behavior, typically sexual behavior in girls and aggressive behavior in boys. The degree of parental concern is often notably disproportional to the actual behavior the child is exhibiting. An example of such over concern is a 2-year-old referred for "stealing." A source of this concern relates to the parents' fantasies of the behavior and personalities of the people whose union produced the child and whose imagined weakness, immorality, or instability also led to the child's adoption. And again, in the adoptive parents' minds there may be a too-readily available transition from "our good child" to "their bad child" ([Brinich, 1980](#), p. 126), although parental ambivalence is certainly not unique to adoption ([Brinich, 1995](#)). Another aspect of importance is guilt for having someone else's child. The adoptive parents' fantasies regarding the biological parents plus their guilt and fear that they might not deserve the child may predispose them to be overly concerned about the child's behavior. These phenomena may predispose adoptive parents to develop an inaccurate parental portrait of the child ([Ferholt, 1996](#)) because of the adoptive parents attributing known or imagined aspects of the biologic parent(s) to their adopted child. They may tend to look to the child for excessive reassurance to assuage their own anxiety about the child becoming antisocial and their guilt about having someone else's child. Open adoption may mitigate this situation through the adoptive parents' enhanced knowledge of or acquaintance with the biological parents. Alternatively, in some instances such knowledge may elicit more negative parental expectations than the adoptive parents' negative fantasies ([Riggins-Caspers, 1999](#)).

[Johnson and Szurek \(1952\)](#) described how parental concerns might lead to their acting in ways that may influence the child to engage in the behavior that the parents consciously least desire. Because of the parents' anxieties regarding such matters, there is a tendency to express unnecessary prohibitions about sexual and aggressive behavior. Parents may express unfounded but vocal allegations of drug use, ask their adolescent daughter whether she kissed her date or "went further," or otherwise express what the child eventually views as an expectation that he or she is going to do some of these things. As Johnson and Szurek state it, "Such constant checking, or such warning, means to the child that there is an alternate image of him in the parent's mind." The transmission to the child of an alternate, antisocial image on the part of the adoptive parents may have a peculiarly powerful effect on the adopted child. The child's having two sets of parents makes it more difficult to fuse the intrapsychic good and bad parents' images of infantile object relations into a workable, realistic identification. To use [Erikson's \(1968\)](#) terminology, the child may be particularly vulnerable to the development of a negative identity because of the unhappy combination of the child's fantasy that he or she could be "someone else" and living in another home and the adoptive parents' unfortunate expression of their negative expectations to the child.

Sources of the adoptive child's hostility to the adoptive parents have the usual developmental determinants as well as one unique to the adoptive situation: rage at having been abandoned. This can readily be displaced from the biological parents to the adoptive parents. The child's rejection of the adoptive parents may serve as an illusory reversal of the painful actuality of history, by way of identifying with the rejecting biological parents. Often all that is immediately evident is provocative behavior. Generally this antagonistic, aggressive behavior serves as a defense against feeling vulnerable and is one of the major reasons that adopted and foster children are referred for therapy in the first place. One author describes such an adopted child's desire that someone see through her aggressive shield and reach out to her "scared and longing core" ([Cohen, 1996](#), p. 292). In the treatment situation, the therapist and any treating institution may become the objects of the adolescent's rejection. At breaks occasioned by the therapist's vacation or other activities, children often play out compensatory displays of strength ([Sherrick, 1983](#)), and adolescents tend to leave therapy or run away from the institution.

Another illusory reversal related to repair of narcissistic injury that is often exhibited by adopted children is the telling of incredible stories. [Sherrick \(1983\)](#) suggests this is a form of "fantasy lying" as described by [Fenichel \(1945\)](#), p. 529). Such stories relate to such things as possessing superior wealth and expensive automobiles and having great mental or physical attributes. These types of fantasies are surprisingly powerful and persistent. The children may be of normal physical and intellectual ability, manifest minimal signs of emotional disturbance, and be the object of considerable teasing by peers because of what is perceived as bragging. Yet the behavior continues unabated. This phenomenon is also quite resistant to psychotherapy, where interpretations of the meaning are often drowned out by a continuation or elaboration of the theme. [Wilkinsen and Hough \(1996\)](#) call attention to the therapist's impulse to dismiss these productions as untrue as a reenactment of the role of the denying, neglectful parent, and emphasize the difficulty in formulating interventions regarding this behavior. Responses on the part of the therapist that are restricted to reflecting the wish to be wealthy, powerful, or famous are much more likely to be heard than are interpretations of what these stories are geared to accomplish in terms of repair of self-esteem. It is the impression of the writer that in many instances these fantasies are most responsive to improvements of



self-esteem related to the development of an attachment to the therapist and improvement in other relationships that result from the therapist's work with parents or residential school staff. The ideal situation for resolving these or other problems, of course, is when such children are in good adoptive homes and the therapist can work with the child and adoptive parents (Samuels, 1995). One might look on these stories as adoption self-narratives constructed in isolation by the adopted child (Lichtenstein et al., 1996), in contrast to the adoptive self-narratives developed in conjunction with helpful adults; these overshoot their mark.

Brinich suggests that the core problem for some adopted children is to create a self-representation as a "wanted" child. He also points out some practical limitations of therapy:

All the psychoanalytic investigation and assistance in the world cannot undo the fact that the adopted child has been rejected or abandoned. This is one reason why adopted children present a particularly difficult therapeutic challenge ( Brinich, 1980, p. 126).

When things do not go well in adoption, there is often a combination of some of the issues discussed in the preceding. Parents may have insufficiently worked through giving up the biological child and accepting adoption and the adopted child, and feel insufficient entitlement to and ownership of the child. Their over concern about problematic behavior has had a paradoxical effect whereby the child has started to identify with the parents' apparent expectations and started to act accordingly. The parents' lack of feelings of entitlement undermines their confidence; therefore, their efforts to direct and set limits are ineffective. Parental frustration leads to increasing hostility on their part, which triggers the child's separation anxiety. The child typically deals with the separation anxiety in a counterphobic manner in terms of provocative behavior ( Stein and Derdeyn, 1980), which is, of course likely to make things worse.

In one study of a hospital population, it was found that adopted adolescents had significantly more runaway episodes and terminated hospitalization via a runaway prior to the planned discharge more often than did the nonadopted adolescent population ( Fullerton et al., 1986). The authors postulated that increasing anxiety about attachment might play a role, as well as "taking charge" of leaving the hospital in lieu of being discharged. These authors suggested that particular attention should be paid to the adolescent's dependency conflicts and defensive needs for inappropriate pseudoautonomous actions. In other studies of inpatient adolescents, a trend was noted for these children to be discharged to placements out of the parental home ( Senior and Himadi, 1985), and it was concluded that the parent-child relationships of hospitalized adopted adolescents were more problematic than those of hospitalized nonadopted adolescents ( Weiss, 1984).

### Adoption Disruption

Adoption disruption refers to adoptions that end with the return of the child to the adoption agency either before or after formal legalization. A number of issues have been identified as contributing to the likelihood of disruption: limited ability of the child to attach to the new family or let go of the birth family; the parents' expectations of a less difficult child; unresolved infertility issues; gaps in information and child history; and inadequate support for the adoptive parents ( Schmidt et al., 1988). Previous adoption, older age at adoption, adoption by a nonfoster parent, the number of child problems, and the higher level of education of the adoptive mother predict a significant portion of disruptions ( Barth et al., 1988; Berry and Barth, 1990). Of the issues identified, the population with the greatest number of adoption disruptions is children with special needs ( Barth et al., 1988; Groze and Ileana, 1996; Rosenthal et al., 1988). When adoption disruption does occur, the emotional aftermath is often intensely negative for both the child and adoptive parents. Not only the child but also the adoptive families are in great need of some form of postdisruption support ( Deykin, 1984; Elbow and Knight, 1987; Valentine et al., 1987).

### COMMENT

There is a consensus among therapists that there are unique challenges to being an adoptive parent and an adopted child. It is important for a therapist to be familiar with the challenges and differences in adoptive families, so as to make the family feel accepted and empowered to take a helpful parental role regarding their child (Nickman and Lewis, 1994).

Many interacting forces shape the unique experience of adoption. The adoptive parents' fantasies, fears, and perceived lack of entitlement to the child can detrimentally influence their parenting abilities. The adopted child's often powerful need to master early abandonment may lead to maladaptive behaviors that feed the adoptive parents' fears and fantasies regarding the biologic parents' past and the child's future. The therapist familiar with the challenges of adoption can take an empathic stance with the adoptive parents and the adoptive child that can contain their anxieties and conflicts and pave the way toward a better resolution of their individual and shared experience.

Adoption continues to change, and will continue to offer children who need homes the opportunity to be raised by people who desire to have a child and who have generally made difficult conscious decisions and gone to efforts that most biological parents do not even approach on their route to parenthood. This chapter of necessity focuses on differences and difficulties pertaining to adoption; however, the great majority of adoptions go well.

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# 109 THE CHILD AND THE VICISSITUDES OF DIVORCE

Judith S. Wallerstein, Ph.D., and Shauna B. Corbin, Ph.D

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Changes within the contemporary family are reshaping the experience of growing up in the United States. The steep rise in recent decades of the incidence of divorce is foremost among the changes that are profoundly influencing the lives of children and their parents. The number of children from divorced families more than doubled between 1960 and 1980 ([Spanier and Glick, 1981](#)). There have been a million children from divorced families each year since 1972 (A.J. Norton, U.S. Bureau of the Census, personal communication, 1998). By the 1990s, demographers informed us that one-fourth of all adults between the ages of 18 and 44 years had experienced their parents' divorce (Statistical Abstract of the U.S. Bureau of the Census, 1997). A newer study reveals that Americans are spending more of their adult lives outside the institutions of marriage and parenthood, and children are experiencing three or more different family structures and living arrangements while growing up ([Teachman et al., 2000](#)).

Although many children weather the stress of marital discord and family rupture without psychopathologic sequelae, many falter along the way. As a result, the high divorce rate has also had a notable effect on the makeup of clinical populations. Children of divorce are greatly overrepresented in outpatient psychiatric, family agency, and private practice populations relative to their presence within the general population ( [Furstenberg et al., 1983](#); [Gardner, 1976](#); [Kalter, 1977](#)). Parental divorce and parental loss significantly predict mental health referrals for school-age children ( [Felner et al., 1975](#)). A national survey of adolescents whose parents had separated and divorced by the time the children were 7 years old found that 30% of these children had received psychotherapy by the time they reached adolescence, compared with 10% of adolescents in intact families ([Zill, 1983](#)); by young adulthood, 40% had received psychological help. The representation of children from divorced families is even higher among inpatient populations. Although national figures are unavailable, many inpatient psychiatric facilities for adolescents report informally that 75% to 100% of their patients are from nonintact families. Overall, national data show that young people from single-parent families or stepfamilies have a two to three times greater likelihood of experiencing emotional or behavioral problems, and a higher incidence of learning problems, than children and adolescents living with both biological parents ( [Zill and Schoenborn, 1990](#)).

The divorce literature, which scarcely existed before the 1970s, has proliferated as growing numbers of investigators in psychiatry, psychology, and sociology have examined the processes involved in family separation and marital dissolution. As a result, we have begun to acquire knowledge in many critical areas: the nature of the divorce process, the responses of children and adolescents by age and gender, the impact of divorce and parental conflict on parent–child relationships, factors in good and poor outcome in the short- and long-term perspectives, patterns of custody and visitation, the role of the father, the roots and dimensions of interparental conflict, and some of the issues that children and adults confront in remarriage. More recently, as findings from longitudinal studies have become available, we have been able to shed light on divorce-specific anxieties that emerge belatedly in the lives of children of divorce, when they enter young adulthood and well into their 30s and 40s ([Wallerstein et al., 2000](#)).

By and large, however, professional interest has not been directed toward issues of treatment. With the exception of mediation programs and court- and school-based groups that have been developed in scattered areas of the United States, and a few pilot and demonstration projects (some clinical, others primarily educational), there has been a paucity of preventive or clinical services specifically designed to respond to the stressful changes in divorcing families or to the special needs of children and adolescents whose family structure has been temporarily or more lastingly weakened by marital distress and breakdown. Most families struggling with divorce have relied perforce on the traditional mental health services available in the community.

In the 1980s, an increasing number of courts offered lectures on parenting to divorcing, and some also offered groups for children that customarily meet for several sessions to discuss the children's feelings. By and large, the effectiveness of the parent education lectures and of the children's groups has not been evaluated in terms of psychological change in adults or children or changes in the parent–child relationships. Not surprisingly, the interest of the court has been first and foremost in diminishing the amount of litigation over custody and visitation. It seems evident, however, that the groups for children can offset the child's loneliness at the time of the breakup, and it may relieve the child's suffering to be with peers who are undergoing the same difficult changes in their lives.

Additionally, much of the interest in exploring the theoretical issues inherent in the family forms that are being created (e.g., joint-custody families; single-parent families in which one parent, usually the father, continues to visit; remarried or redivorced families) has centered around reassessing the role of the father in child development ([Cath et al., 1982](#); [Hanson and Bozett, 1985](#); [Lamb et al., 1997](#)). Although it is widely acknowledged that the psychological theory that underlies established clinical interventions with children (whether in psychoanalysis, family systems theory, or child development) was developed primarily within the paradigm of the intact two-parent family, there has been little theoretical exploration of how changes in family structure can or should modify the goals or approach of the clinician, either in work with parents, who find themselves cast in unfamiliar roles, or with the child, whose primary identification figures are often shifting and unclear.

## NATURE OF DIVORCE: DIVORCE AS A PROCESS

Divorce is a process of social and psychological change in the individual and in family relationships that can extend over many years. It has no true counterpart in other crises of adult life. Although it was initially considered analogous to bereavement in the central significance of loss as the critical component of the adult experience, we have come to recognize that, in divorce, grief is only one of many powerful affects; rage, sexual jealousy, and unrequited love share equal power and significance ([Wallerstein and Kelly, 1980](#)). Divorce is not a time-limited event for the adults or the children involved, in part because a complex undulation of changes including remarriages and redivorces and love affairs leads up to and, in turn, is set into motion by the marital rupture. These changes often occupy a significant portion of the adult's postdivorce life. They typically occupy a significant portion of the youngster's childhood and adolescence and, as we are learning, of his or her own young adulthood.

Divorce can be broadly conceptualized as progressing through three successive phases ( [Wallerstein and Blakeslee, 1989](#) ). The ambiance of the first, acute phase of dramatic and highly emotional responses is established by the fact that divorce in a family with children is rarely a mutual decision. Separation often occurs amid escalating spousal conflict, which can include physical violence between the parents. During this acute phase, in extreme cases, one or both parents may experience depression with suicidal ideation and may regress considerably in behavior. Similarly, one or both parents may experience ego-syntonic rage, which can reach paranoid dimensions. There is, in many of these people, a temporary weakening of ego control over aggressive and sexual impulses, accompanied by lapses in customary judgment. This acute phase may be relatively brief, or it may extend over several years. Sometimes, the divorcing couple remains fixated in this acute phase for years, reenacting the separation drama again and again, in the vain hope of modifying the events or the ending but never obtaining relief from the narcissistic injury that was initially sustained. These reenactments may take place in the courts, or they may be played out in the many other arenas available when



there are children from the broken marriage. In most instances, the acute phase is followed by a transitional phase in which the parents begin to disengage from each other's lives and move into new relationships and new work and home settings. The physical, social, and emotional environments of the family during this time may be in continual flux. This intermediate period may be relatively brief, or it, too, may last for several years. Finally, with the onset of the third phase comes the establishment of the relatively stable postdivorce single-parent or remarried-parent household, each of which has its own associated strains and gratifications.

Ongoing stresses confront many postdivorce families. Some of these stresses are rooted in convergent economic and social issues that communities have been reluctant to address, particularly those that affect the serious economic disadvantaging of women and children after divorce ( [Weitzman, 1985](#)). Other reports describe the many complex psychological issues that adults face in reconstructing their postdivorce lives and how their tasks are affected by the number of years invested in the marriage, the age of the adult at the divorce, and the discrepant opportunities that are available to divorced men and women in the sexual, social, and economic marketplaces ( [Wallerstein and Blakeslee, 1989](#)). Moreover, observations on remarriage have called attention to the many psychological differences between first and subsequent marriages and the complex challenges that a second marriage, with children, poses to all the participants ( [Pasley and Ihinger-Tallman, 1987](#); [Wallerstein and Blakeslee, 1989](#)). These concerns have been reinforced by reports from demographers that the incidence of divorce in remarried families with children from a previous marriage is even higher than the divorce rate in first marriages ( [Pasley and Ihinger-Tallman, 1987](#)).

## EFFECTS OF DIVORCE ON THE PARENT-CHILD RELATIONSHIP

### Diminished Parenting

[Hess and Camara \(1979\)](#) point out that "for children, the threat of divorce lies in the disruption of relationships with the parents." This applies both to the availability of the parent and to the quality of the postdivorce relationship itself. Hetherington and her associates observe disorganization, deterioration of discipline, rising angers, and lowered expectations for appropriate social behavior by their children on the part of custodial mothers ( [Hetherington et al., 1978](#); [Hetherington et al., 1982](#)). Others ( [Santrock et al., 1982](#)) note the more conflicted postdivorce relationship of children with an opposite-sex custodial parent. The impairment of relationships between parent and child may appear early in the separation and divorce process, or may remain latent, emerging only in young adulthood. Mother-child relationships that have previously weathered the storms of adolescence have been noted to deteriorate in young adulthood. This finding parallels Wallerstein's observed "sleeper effect" in young women from divorced families ( [Wallerstein, 1991](#)).

[Wallerstein \(1985a\)](#) suggests that a diminished capacity to be an effective parent is an expectable short-term consequence of divorce, and it has the dangerous potential of becoming chronic when the custodial parent or joint custody parents fail to reconstitute or becomes involved in new relationships that overshadow or replace the relationship with the child. This diminished capacity is most evident in the parent's decreased ability to separate the child's needs and reactions from those of the adult. Wallerstein proposes that the fantasy underlying the sometimes astonishing changes in the parent's relationship with the child is a conscious or unconscious wish to abandon the child coincident with the breakup of the marriage. As a fantasy, this impulse to leave the child behind may remain unacted on, or it may unconsciously spur sudden flight or unexpected rejection by a parent of a child who had previously been cared for properly. Whatever the conscious or unconscious roots of the disrupted parent-child relationship may be, the consequence is that the child's fears of abandonment at the time of the breakup are powerfully reinforced by a parent's changed attitudes and behavior. Often, these fears lead to a hypervigilant tracking of the parent's responses and an intense anxiety that can dominate the child's inner life and can intrude on his or her capacity to accomplish normative developmental tasks.

[Wallerstein \(1985a\)](#) also proposes that a contrapuntal theme to the temporary rejection of the child at the time of the marital breakup is an intensified need of the parent for the child. This dependence on the child by one or both parents is often at the core of parental conflict and prolonged litigation over custody and visitation. In extreme cases, the dismantling of customary supports within the marriage, combined with the humiliation of the narcissistic injury inflicted by the divorce and the painful persistence of attachment to the divorcing partner, may result in severe ego regressions in parents whose previous functioning, separately or together, may have been at least adequate. Feeling suddenly bereft and in need of help, such parents turn to their children for help in warding off the threatened depression.

In many divorcing families, the temporary dependence of the adult on the child is a transient phenomenon that has no lasting deleterious effects and may indeed be of benefit to a child suddenly elevated to unaccustomed importance. However, as [Wallerstein \(1985b\)](#) points out, turning to a young son or daughter as a peer, or worse, as to a parent, may overwhelm the child with responsibility and may place the child at serious risk if the intense dependence continues. These overburdened children—out of worry, guilt, compassion, or, indeed, out of their conviction that it is their assigned task to keep the parent alive—may devote themselves entirely to maintaining the psychic functioning and physical needs of the ailing parent and may relinquish their responsiveness to their own needs over many years.

There is by now convincing evidence that witnessing violence between the parents has very serious lasting effects on the psychological development of children, on their conscience formation, and especially on their future relationships with the opposite sex. The violence, which may arouse erotic excitement, especially for the daughters, may lead them in adulthood to seek out men who abuse them. As reported in [Wallerstein et al. \(2000\)](#), the young women are often governed by the fantasy that they can rescue the loving man who is hidden inside the abuser. This can, of course, have tragic consequences for the young woman.

### Children in High-Conflict Divorce

The struggle for and through the children embodies the intense conflicts that often accompany the failing marriage. Although competition for a child's affection may occur in an intact, even well-functioning family, disruption of the family system and its resulting angers bring the parents' competitiveness into sharpened focus. It is not uncommon for angry, sometimes distraught parents to cast their children in a great many roles during postmarital battles, ranging from that of audience, whose presence appears to be a necessary backdrop for the parental fighting, to that of fully positioned battle allies. The children often range in their participation from astonished, frightened observers to denunciatory Greek chorus or, in some instances, ardent champions of one parent against the other.

Many of the anger-driven parent-child relationships that emerge at the time of the marital breakup are new alliances that diverge from the pattern that existed within the intact marriage. The child's conscious behavior may be powered by loyalty to the disrupted marriage or by the quixotic impulse to defend or rescue the parent who has been identified by the child (sometimes erroneously) as the victim. Not atypically, a child may take up the cause of the absent parent, representing his or her interests in the events of the custodial household. A child's active involvement in the marital battle is additionally fueled by the rise in physical violence between the parents that often erupts at the separation, even though it had never been a feature of the marriage ( [Wallerstein and Kelly, 1980](#)).

Such alignments occur most often between a late latency or early adolescent child and the parent who has vehemently opposed the divorce. The adult's participation is almost always rooted in an entrenched sense of moral outrage at having been betrayed and cruelly exploited during the marriage. The avowed agenda of these alignments is likely to be the restoration of the failed marriage; the unspoken agenda is almost always revenge.

These anger-driven alliances serve a range of psychological purposes for both parent and child. The loneliness of the divorce period is reduced significantly by the new partnership. The child's own gnawing fear of being abandoned is alleviated by becoming the needy parent's trusted companion. Directing their anger outward against the absent parent serves as a powerful antidote to the intolerable pain of rejection and helplessness that the allied child and parent experience, nor is it accidental that many children join cause with a parent with whom their relationship during the marriage had been tenuous or emotionally impoverished and take up an angry campaign against the parent they once had cherished. Additionally, for the child, the new alignment provides an opportunity to resolve any existing ambivalence of feeling toward the parents by creating a clear repository of virtue in one and villainy in the other. These alignments have triggered court fights and have resulted at times in the removal of the child from the mother's custody. These court cases have been rooted in the writing of a psychiatrist Richard A. Gardner, who has characterized them as evidence of *parent alienation syndrome*, alleging that the mother is the primary initiator of these alignments, and removal of the child in severe cases is the treatment of choice. Gardner's influence has been challenged by the absence of supporting research for the diagnosis of parental alienation or its recommended treatment and the absence of this condition from accepted classification of psychiatric illnesses ( [Gardner, 1992](#)).

Evidence indicates that such active alignments generally do not survive the adolescence of the young ally. Nevertheless, many young people come to adulthood complaining that their responses to the "villainous" parent had been governed by perceptions that they did not truly share. They regret deeply that they were not given the opportunity to form their own judgments, and their anger at having been cheated out of a true relationship with both parents is often intense. Some also express profound and pervasive guilt at having participated in lying or even more serious misbehavior directed at hurting their parent ( [Wallerstein and Blakeslee, 1989](#)).

One of the most significant findings in family research concerns the serious hazard posed to the psychological health and development of children by continued exposure to high conflict between the parents, whether in the context of an intact marriage or as part of ongoing postdivorce antagonism ( [Emery, 1982](#)). In one California study of 1,124 divorcing families, one-fourth were characterized by high levels of conflict at an average of 3½ years after separation ( [Maccoby and Mnookin, 1992](#)). There is increasing interest in the plight and fate of children in such families and the impact of witnessing abuse and violence between the parents,

especially if it is prolonged. The psychological effects and disturbances in parent–child attachments observed are not unlike those reported in families where the child has been the target of abuse directly. As yet, it is not clear whether this detrimental impact is mediated by the influence of the conflict on critical aspects of the parent–child relationship or whether it largely represents the intense reactions of the children who directly witness or overhear the fighting. Nor is it known whether children's reactions are primarily governed by the stimulation of seeing the parents fight, by the anxiety engendered from parents who appear to be out of control, by the fear that one or both parents—and perhaps the children as well—may be severely harmed, by complex issues of identification, or by some combination of all these factors. The persistence of high conflict for many years, even after the parental divorce, may be particularly psychotoxic to children in that it appears to lack any remedy.

The link between parental conflict and the subsequent psychological adjustment of children represents one of the most fertile areas for increased theoretical understanding and new models of psychotherapeutic intervention. [Johnston and Campbell \(1988\)](#) and [Johnston and Roseby \(1997\)](#) address the serious effects these lasting impasses create in some families after divorce and report severe reactions among the children, including impairment in reality testing. Adding to this concern is the report from a longitudinal study that, at 10 and 15 years after the divorce, many young people remember with great clarity incidents of physical conflict between their parents and describe having been continually haunted by those memories during their waking and dream lives throughout the postdivorce decade and well into their young adulthood. Many of these children from homes where there was physical and verbal abuse between parents during the intact marriage became involved, at entry into young adulthood, in abusive relationships of their own, although they had been separated by at least a decade from the trauma of witnessing their parents' quarrels ([Wallerstein and Blakeslee, 1989](#)).

At the cutting edge of research in this area is the work of Johnston and Roseby on the developmental impact of high-conflict divorce and of separations in the context of violent relationships. Children caught in such circumstances show problems in psychological separation and individuation and disturbances of gender and sexual identity ([Johnston and Roseby, 1997](#); Roseby and Wallerstein, in press).

## EFFECTS OF DIVORCE ON CHILDREN

### Initial Reactions

Children and adolescents experience parental separation and its immediate aftermath as an immensely stressful period in their lives. Indeed, for middle-class children in the United States, their parents' divorce is likely to be the central stress of their growing-up years. The family rupture evokes an acute sense of shock, intense anxiety, and profound sorrow. Many children can feel relatively content and even well parented within families in which one or both parents are unhappy. A surprising number (one-third in one study) did not know that their parents' marriage was troubled ([Wallerstein and Kelly, 1980](#)). Few youngsters experience relief with the divorce decision; those who do are usually older and have witnessed open conflict between their parents. Children's initial responses are typically governed neither by an understanding of the issues leading to the divorce nor by the fact that divorce has a high incidence in their community. To children, their own parents' divorce signifies the collapse of the structure that was responsible for providing them basic nurturance and protection, even when the family was performing poorly in this role.

The initial pain experienced by children and adolescents in response to a marital separation is compounded by their fantasies of the catastrophes they fear the divorce will bring in its wake. Children suffer with a pervasive sense of vulnerability as they experience the family's breaking apart. They grieve over the loss of the intact family, including the hopes and dreams attached to it, and over the absence of the noncustodial parent. Often, they must also confront the additional losses of familiar friends, neighborhood, and school. Children worry about their distressed parents. They are concerned about who will take care of the parent who has left and whether the custodial parent will be able to manage alone. They experience intense anger toward one or both parents for disrupting the family. Some of their anger is reactive and defends them against their own feelings of powerlessness, their concern about being lost in the shuffle, and the fear that their needs will be disregarded as the parents give priority to their own wishes and needs. They often feel a painfully divided loyalty, as if they were being forced to choose between their parents, even when this question has not been put before them. They may suffer with imagined guilt over having caused the divorce. This is especially likely among young children, or when the parents have fought over child-related issues. Young children sometimes decide heroically that it is up to them to mend the broken marriage.

The responses of children, particularly their sense of loneliness and social isolation, are also strongly influenced by the social context of the divorce. Children all too often must face the tensions and sorrows of divorce with little outside help. Fewer than 10% of children in one widely reported study ([Wallerstein and Kelly, 1980](#)) had any support at the time of the crisis from adults, other than relatives, who could have helped them, such as teachers, pediatricians, clergy, or family friends. Only 25% felt that grandparents came to their aid emotionally. Many children are poorly prepared by their parents for the impending upheaval, some not at all. Thus, it is a striking feature of divorce that when it occurs—unlike in bereavement or other stressful events that can occur in childhood—customary support systems, either through adult ignorance or diffidence, fall away.

### Developmental Factors

Developmental factors are critical in the responses of children and adolescents at the time of the marital rupture. Despite significant individual differences in children, in their families, and in parent–child relationships, it appears that children's dominant concerns, their capacity to perceive and understand family events, their central psychological preoccupations and conflicts, their available repertoire of defenses and coping strategies, and the dominant patterning of their relationships and expectations, all primarily reflect their age and developmental stage at the time of the parental separation.

A major finding in divorce research has been these common patterns of response within age-related groups ([Wallerstein and Kelly, 1980](#)). The groups reported to share significant perceptions, responses, underlying fantasies, and behaviors are as follows: (a) preschool age, 3 to 5 years; (b) young school or early latency age, 6½ to 8 years; (c) later latency age, 8 to 11 years; and (d) adolescent age, 12 to 18 years. The responses of young adolescents have also been differentiated from those in middle and late adolescence ([Springer and Wallerstein, 1983](#)). It may be that the similarities observed within children's age groups represent their commonly shared responses to acute stress generally, not simply to marital rupture. Until recently, no systematic research has addressed the short- or long-term effects of divorce on infants and toddlers less than 3 years of age. The newest work in this area is a longitudinal study in progress by [Solomon \(1992\)](#), who examines the effects of overnight visitation on the parent–child attachments of 100 children ages 12 to 18 months at the time of the family rupture. This is an important subgroup to understand, because many divorces occur within the first 2 years of marriage.

Observations about preschool children derived from longitudinal studies in two widely divergent regions, northern California and Virginia, are remarkably similar ([Hetherington et al., 1982](#); [Wallerstein and Kelly, 1980](#)). Preschool children are likely to regress after one parent's departure from the home. The regression usually occurs in the most recent developmental achievement of the child. Intensified fears are frequent and are evoked by routine separations from the custodial parent during the day and at bedtime. Sleep disturbances are common. The preoccupying fantasy of many of these youngsters is fear of abandonment by both parents. Yearning for the departed parent is intense. Preschool children are likely to become irritable and demanding and to behave aggressively with parents, younger siblings, and peers.

Children 5 to 8 years old grieve openly for the departed parent. Many share the terrifying fantasy of replacement: "Will my daddy get a new dog, a new mommy, a new little boy?" Little girls weave elaborate Madame Butterfly fantasies, asserting that the departed father will someday return to them, that he loves them "the best." Many very young children cannot believe that the divorce is permanent. In their preoccupation with their fantasies, there is often a precipitous decline in their schoolwork ([Wallerstein and Kelly, 1980](#)).

For those 8½ to 12 years old, the central response often seems to be fierce anger at one or both parents. These children grieve over the loss of their intact world and suffer anxiety, loneliness, and a humiliating sense of their own powerlessness. They often see one parent as "good" and the other as "bad," and in this latency age, they appear especially vulnerable to the blandishments of a parent to participate in the marital battles. They also have a high potential for assuming an empathic and engrossing role in the care of a needy parent. School performance and peer relationships may consequently suffer ([Wallerstein and Kelly, 1980](#)).

Adolescents are also vulnerable to the impact of their parents' divorce. The incidence of acute depression, accompanied by suicidal preoccupation and acting out, is frequent enough to be alarming. Anger can be intense. Several instances have been reported of direct violent attacks on custodial parents by young adolescents who had not previously shown such behaviors ([Springer and Wallerstein, 1983](#)). Preoccupied with issues of morality, adolescents may sit in judgment on their parents' conduct around the divorce, and they may identify with one parent and do battle against the other. They are often anxious about their own future entry into adulthood, fearful that they may experience marital failure like their parents. Nevertheless, as researchers have pointed out, many adolescents show an impressive capacity to grow in maturity and independence as they respond to the family crisis and their parents' need for help ([Weiss, 1979](#)). The presence or absence of perceived fairness and integrity in their parents' postdivorce dealings with each other is not lost on them; yet they are capable of considerable compassion for their parents' weaknesses



and struggles, even as they continue to grapple with their own.

## Gender Differences

Although it had been widely accepted by researchers that boys are more vulnerable than girls in both initial and long-term responses to divorce, this finding has been called into question by a critical analysis of the methods employed in a range of studies ( [Zaslow, 1988](#); [Zaslow, 1989](#)). The picture is confusing, in part because the comparative developmental course of boys and girls in intact families, from infancy to young adulthood, is far from being clearly understood. The current state of our knowledge of divorce populations links gender differences to the different developmental stages. Thus, major differences between preschool boys and girls at approximately 4 years after the separation have been observed on a wide range of cognitive, social, and developmental measures ( [Hetherington et al., 1982](#)). Although traditional sex-role typing in girls does not appear to be disrupted by divorce, boys score lower on male preference and higher on female preference on the sex-role preference tests at this same time. The boys also spend more time playing with girls and with younger children. They show affective narrowness and a constriction in fantasy and play and are more socially isolated than their female peers.

Gender differences were observed as well in the California Children of Divorce Study ( [Wallerstein and Kelly, 1980](#)). Although boys and girls did not differ in their overall psychological adjustment at the time of the marital breakup, 18 months later, the boys' psychological adjustment had deteriorated, whereas that of the girls had improved, making for a significant gap between the two groups ( [Springer and Wallerstein, 1983](#)). [Guidubaldi and Perry \(1985\)](#), in a national survey of elementary school-age children 6 years after their parents' divorce, found that boys, but not girls, tested significantly below a matched control group from intact families in academic achievement and social relationships. Other evidence suggests that, in general, marital turmoil has a greater impact on boys than on girls, both in divorced families and in intact, discordant families ( [Block et al., 1981](#); [Emery, 1982](#); [Rutter, 1970](#)). In fact, a report of two national, longitudinal studies of divorce effects on children in Great Britain and the United States suggests that, at least for boys, negative symptoms that are usually considered divorce sequelae are actually apparent before the marital split ( [Cherlin et al., 1991](#)).

A critical question is how much of the reported differential response between the sexes, if it does exist, is mediated by being in custody of the mother. One small study from the late 1970s found that children of latency age who were in the custody of the same-sex parent showed greater sociability and independence than did those boys and girls in the custody of the opposite-sex parent ( [Santrock and Warshak, 1979](#)).

Finally, increasing evidence indicates that adolescent girls in divorced and remarried families confront particular difficulties. Kalter describes special problems that girls from divorced families face in their relationships with their mothers, especially the difficulties of separating at adolescence ( [Kalter et al., 1985](#)). The 10-year study of Wallerstein and Blakeslee ( [Wallerstein and Blakeslee, 1989](#) ) also reported that young women from divorced families often have a turbulent adolescence and a conflict-ridden entry into young adulthood. Many young women at the 10-year mark were caught up in a web of short-lived sexual relationships, some with much older men. They described themselves as fearful of commitment, anticipating infidelity and betrayal. Many of the young women who encountered difficulties in late adolescence had done well during the early years after the divorce, when they were preschool and latency children. It may be that boys, especially oedipal boys and those of and latency age who are in the custody of their mothers, have a more difficult time immediately after the divorce, whereas girls in the custody of their mothers find adolescence and entry into young adulthood particularly hazardous. Clearly, gender differences need to be explored further for the various age groups and within different family structures.

## Long-Term Outcomes in Children

Mounting evidence indicates that the effects of divorce in a general population extend well beyond the previous expectation of a several-year, but limited, aftermath to the marital rupture. Wallerstein's research over a 25-year period ( [Wallerstein et al., 2000](#) ) represents the most extended longitudinal study of children and adults after divorce. Their findings show ongoing as well as delayed effects that come to the fore during the years from midadolescence to young adulthood, as relationships with the opposite sex move to center stage. The ongoing effects associated with good or poor adjustment are likely to reflect the quality of life and the parent-child relationships within the postdivorce or remarried family. The delayed (often long-delayed) effects are more likely to reflect concerns associated with the possibility of repeating the failure of relationship between a man and a woman that the child observed during the marriage, as well as with the parents' subsequent failures in coparenting or new love relationships that the child may have observed. When the postdivorce relationship that the parents develop with each other is more satisfactory, and when the parent individually is able to reconstruct his or her life successfully, the child's memories of the first poor marital relationship are less likely to be disturbing but remain of central significance in their search for lasting love and commitment.

Observations over the 25-year postdivorce period have led Wallerstein to propose that the inner developmental course of children of divorce is significantly altered by the parents' failed marriage and its frequently troubled, long-lasting aftermath ( [Wallerstein and Blakeslee, 1989](#) ; [Wallerstein et al., 2000](#) ). There is no question that children of divorce face more difficulties in their relationships. They not only fear failure but also feel poorly prepared for the complex interactions of adult intimacy. Many state that they have never seen a man and a woman "on the same beam." Amato also finds that the greatest impact of divorce occurs in adulthood in troubled relationships between the sexes ( [Amato, 1996](#) ; [Amato, 2000](#) ). He has called attention to the lack of social skills of these persons. However, there is reason to assume that the problem is much deeper than the educational issue of needing social skills. Wallerstein suggests that they lack the requisite internal images to construct a lasting relationship, and their internalized images include those of failure to resolve conflict and flight when frustrations within the relationship begin to mount.

Men, particularly in adulthood, feel helpless about their capacity to influence the woman's behavior or feelings. Additionally, many fear that disaster will strike at any time and will take away what they have. Their fears of loss increase when things are going well in their lives. They live with chronic anxieties that they attribute to their parents' divorce when they were children and their remaining sense that the family rupture occurred when they did not expect it ( [Wallerstein et al., 2000](#) ). This is true whether the child falls into a more resilient group with better outcomes or into a more vulnerable group with poorer outcomes. The reports from the children over the many years of the follow-up suggest that the internal developmental tasks of establishing intimacy and trust in their own relationships with the opposite sex are felt to be persistently burdened to a greater or a lesser degree by the template of a failed man-woman relationship that these children carry within them. Additionally, the fears of disappointment, betrayal, and abandonment that are legacies of the failed parental marriage are often reinforced by extended periods of diminished parenting during the postdivorce period. When many find as well that the bond with the father does not survive transplantation into the alien, rocky soil of arranged visitation, this adds to their sense of the unreliability of relationships.

Other delayed effects of divorce may not emerge until many years later. There is evidence that, at adolescence, the need for the father increases in both boys and girls, and feeling rejected by the father at this critical development time may pose special hazards. For the young adult, relationships with both parents appear burdened by divorce-specific issues surrounding the normative events of separation from home and family, which are emotionally much more complicated if the custodial parent will be left alone. In brief, children of divorce face many tasks in addition to the usual ones of growing up. These tasks are formidable and may require more help than children in intact families in our society typically receive ( [Wallerstein, 1983](#) ). What seems very clear is that, over the years of their growing up, children of divorce find it necessary to work hard and consciously on the mastery of their experiences. It may be for this as well as other reasons that they continue to think of themselves as "children of divorce," as if that were their fixed identity ( [Wallerstein and Blakeslee, 1989](#) ).

## Factors in Outcome

The initial responses of children do not predict long-term consequences for psychosocial adjustment, either for those who did well at the time of the divorce or for those who fared poorly. Nor do preliminary findings at the 15-year mark of the California Children of Divorce Study indicate that even 10-year outcomes have remained stable ( [Wallerstein and Blakeslee, 1989](#) ). There appears to be considerable shifting in individual adjustment as the young people, now in their third decade of life, either seek psychotherapy for themselves after several relationship failures or succeed in building gratifying heterosexual relationships and marriages. No single theme appeared among the children in this study who were functioning well immediately after the separation and divorce, or over the years that followed, nor was there a single thread associated with poor outcome. Many of the children who seemed to be functioning appropriately at the 10-year mark were well parented or had had considerable help along the way from a parent or grandparent. Only a few were helped by both parents. Visiting frequency or patterns of visiting were unrelated to outcome, but whether or not the child felt rejected by the father remained a critical factor. Some were fortunate enough to find adult mentors, and those who did showed particular promise in scholarship or athletics. Many had taken a great deal of responsibility for their own upbringing.

Although in remarried families the stepparent can, on occasion, play a critical role in the child's development, the extent to which this occurs is doubtful. In the cited 10-year study, few stepparents took on a central role in the child's life. Moreover, in many remarried families, the children felt excluded from the orbit of the remarriage. The latest national figures provide no clear support for the protective or mitigating influence of remarriage for children of divorce, although when divorce had occurred early in the child's life, parental remarriage seemed to offer some benefit to the child ( [Zill et al., 1993](#) ).

In the Wallerstein study, the amount of stress noted in the postdivorce family was considerable. One of two children experienced a second parental divorce. One of two continued to live with intense anger between their parents that did not subside over the years. Three of five felt rejected by one or both parents. There were additional economic stresses, and one-fourth of the youngsters experienced a significant drop in their standard of living, which they did not recoup during the postdivorce decade.

In effect, in investigating the long-term adjustment of the child of divorce, we confront a rich mix of individual issues in the resiliency and vulnerability of child and parent, the individual talents and staying power of the child, the nature of the relationship between the child and each parent, the extent to which the postdivorce coparenting relationship is relatively free of continued conflict that involves the child, and the encouragement and support available to the child from whatever other sources are available within or outside the family.

## DISPUTED CUSTODY AND VISITATION

The most tragic children of divorce are those aptly described as “the children of Armageddon” ( [Watson, 1969](#)), who are caught in the entrenched legal battles of their parents. No national figures are available on the numbers of families involved in full-scale legal battles over custody and visitation, but based on an early estimate, it is likely that 10% to 15% of families with children struggle in the courts over many years, and that one-third of divorcing families return to court for modification of the initial orders ( [Freed and Foster, 1974](#)). In the study by Maccoby and Mnookin, researchers estimated that 10% of families encountered “substantial” legal conflict over custody and visitation, whereas 15% experienced “intense” legal conflict ( [Maccoby and Mnookin, 1992](#)). Thus, although most families make custody and visitation arrangements without recourse to the courts, many relying on the advice of their attorneys, even private arrangements are very much influenced by court decisions, which cast a long shadow over all postdivorce arrangements, including those that were never in dispute ( [Mnookin and Kornhauser, 1979](#)).

The causes of continued legal contest between divorcing spouses are complex and multidetermined. Fortunately, studies have begun to shed light on the interlocking issues that maintain the conflict at high intensity over many years. [Johnston and Campbell \(1988\)](#) suggest a triad of factors that contribute to the impasse in the family: those that reflect psychopathology in the individual, those that derive from the marital relationship and the changed interaction between the partners, and those factors in the social surround that support the continued litigation and contribute to the increasing stress of the parties involved. In the individual, a history of repeated un mourned losses is not uncommon and may go hand in hand with a pathologic dependence on the constant presence of the child. The severe narcissistic injury of the divorce may trigger a rage against the divorcing spouse that continues to bind the partners to each other through conflict over the children. The same psychodynamics may underlie child stealing.

There is growing recognition among mental health professionals that the adversarial system of the courts is not only poorly suited to resolving family conflict but also may intensify it by further dividing the hapless parents and adding to the stress on the family. Concern with “family values” has become almost a code word to reflect the widespread worry about the high incidence of divorce. Political and religious groups in several states have tried to pass legislation that would narrow the exit from marriage with a range of restrictions but mostly by a return to “fault divorce” in which one person is held culpable for the deterioration of the marriage and is punished by withholding the children or by imposing greater financial burdens. These efforts have largely been unsuccessful. As [Gardner \(1982\)](#) notes, “the adversarial system is ill suited to deal optimally with custody conflicts, is psychologically detrimental to children, and is therefore antithetical to good psychiatric practice.” The [Group for the Advancement of Psychiatry \(1980\)](#) recognizes the impact of the entire family's interrelationships during the postdivorce years and strongly advocates that all family members should be examined before a court decision on custody or visitation is made. Practitioners have increasingly refused to appear as an expert witness for one side in a custody or visitation dispute and have insisted on meeting with both parents before rendering a recommendation to the court.

Much progress has been made in understanding the tormented and driven behavior of parents whose fight for the child is mostly determined by the unbearable narcissistic injury of the divorce and the dependence on the child to offset their sense of abandonment and to keep severe depression at bay. The work of Janet Johnston, Vivien Roseby, and their colleague has been at the forefront of the progress that has been made ( [Johnston, 1994](#); Johnston, in press; Johnston and Campbell, 1998; [Johnston and Roseby, 1997](#); [Roseby, 1993](#); [Roseby and Johnston, 1995](#)). In two seminal books, Johnston and Campbell and Johnston and Roseby propose a complex model for understanding the impasses that baffle judges and attorneys that shows how the internal dynamics of one or both parents are soon reinforced by the ways that the couple continue to hurt each other and by the presence of the Greek chorus of attorneys and mental health professionals who are caught up in mischievous countertransferences and, of course, family members on each side ( [Johnston and Campbell, 1988](#); [Johnston and Roseby, 1997](#)). Conflicts that are reinforced in this way can go on seemingly forever, with little perceptible dimming of the anger that led to the dispute. Children caught in these battles are tragic casualties whose interest are soon lost sight of by everyone, including the courts, which are caught up in adjudicating the rights of the parents.

## Mediation

Mediation has attracted considerable interest in recent years as the intervention of choice for disputing families ( [Coogler, 1978](#); [Haynes, 1981](#)). Reports from a study of mediation in four court systems (Hartford, Denver, Minneapolis, and Los Angeles) indicate that families agreeing to make use of the mediation services in the courts are generally pleased with the process and outcome. Many families, however, reject mediation even when it is made available without charge. Many issues continue to hinder the full realization of mediation's early promise. Research shows that severely conflicted parents are unable to make use of the mediation process successfully ( [Kressal et al., 1989](#)), and long-term studies do not distinguish outcome in children whose parents mediated their differences from those whose parents relied on traditional negotiation between the respective attorneys ( [Emery and Dillon, 1996](#)). Still unresolved is the issue of whether mediation falls within the domain of the attorney or the mental health professional or whether they should work in concert with the disputing family. A critical issue is that the mediator's role may leave the child's interests without adequate protection, because mediators, by and large, lack training in child development or psychopathology and are unable, except from a common sense vantage point, to assess how the mediated agreement will influence the child. Furthermore, the mediation process makes the assumption that the child's interests will be protected by the parents, an assumption often unwarranted at divorce, especially in the case of intensely conflicted parents. Finally, whereas the judge is charged with protecting the best interests of the child, the mediator in most settings does not share this responsibility.

In 1981, California enacted mandatory mediation for divorcing families who are disputing custody or visitation. Reports from different jurisdictions within that state show a high settlement rate, ranging from 55% to 85% of the disputing families in the different counties, who then refrain from taking their case to court. There is still much to be learned about the nature of these agreements and their impact on the psychological adjustment of the children or their parents. Overall, however, the courts have considered mediation services to be of great benefit, and the availability of mediation services has increased rapidly within courts throughout the United States.

## Custody

The changing roles of men and women are mirrored in the courts and in legislation regarding custody and visitation. Early in the 1980s, the courts relied extensively on the concept of “the psychological parent,” assuming that, except in unusual circumstances or for older children, the mother would fulfill this role. Our society has now moved away from the expectation that single-parent custody, combined with reasonable visitation with the noncustodial parent, is the legacy of divorce. Attention has increasingly focused on the contribution of the father as parent and as a potential primary parent ( [Cath et al., 1982](#); [Jacobs, 1982](#); [Ricci 1980](#)). Custodial arrangements have changed since the mid-1980s ( [Maccoby and Mnookin, 1992](#)). We still lack sufficient information about the extent of this change in the direction of joint custody. It is important, however, that during the 1980s more than one-half of the states enacted legislation that permits joint custody. In several instances, the public policy has leaned toward a presumptive preference for joint custody. In California, in 1988, legislation went against the presumption of joint custody; while acknowledging the importance of both parents for the child, it emphasized the necessity of matching the custody arrangement to the needs of the individual family. Clearly, community attitudes and social policy are in flux.

Joint custody remains a variously defined arrangement, differing not only among states but also even among local jurisdictions. Joint *legal* custody typically refers to an equally shared responsibility between parents for major decisions regarding their children's lives and well-being. Joint *physical* custody indicates that the child actually resides for substantial periods in each parent's home, although the proportion of time spent and the schedule of transitions between households may vary widely.

Joint physical custody can be properly regarded as a new family form. The motivation for its choice varies widely. Some parents select joint custody out of commitment to the child's continuing relationship with both parents; others, however, select this custody form out of the demands of the workplace; and still others select joint custody because neither parent truly wishes to take responsibility for the child. Obviously, the experience of the child will vary with the parents' motivation and emotional investment.



Researchers have raised the question of how important the custody arrangement by itself is to the psychological adjustment of the child. [Kline et al. \(1989\)](#), in a sample of 93 white middle- and upper-class divorcing families, compared the psychological adjustment of those 38% of the children who were living in joint custody with that of the remaining group who were in sole custody. She and her colleagues found that neither the custody arrangement itself nor the frequency of access and visitation with the father influenced the child's psychological adjustment. The factors affecting the child's psychological and social adjustment, regardless of custody arrangement, were the prior psychological functioning of the parents and the degree of postdivorce hostility and conflict between the parents.

One study of intensely conflicted families, in which the court had ordered joint custody over the considerable reluctance of one or even both parents, showed that children in involuntary custody situations seemed seriously deteriorated in their psychological and social adjustment, school performance, and peer involvement, as observed over a several-year period. Both boys and girls seemed to suffer when frequent access to both parents was imposed on families locked in ongoing disputes ([Johnston et al., 1989](#)). This work addressed the very serious issue that has been raised in many jurisdictions regarding whether the courts should award joint custody in the face of one parent's strong opposition. Findings from this study are very much in accord with the clinical opinions that practitioners have held over many years.

Evidence from numerous studies indicates that many children prefer joint custody to sole custody, and many children benefit from this arrangement ( [McKinnon and Wallerstein, 1986](#); [Steinmann, 1981](#)). Our knowledge at present indicates that, when it is entered into voluntarily by both parents with dedication and conviction, joint physical custody can be regarded as a viable family form. Under appropriate circumstances, it serves well, especially in the transition from divorce to remarriage. Joint custody does demand special effort and commitment from the parents, the ability of the formerly married partners to remain in close touch with each other's lives, and considerable flexibility from both child and parents ( [Wallerstein and Blakeslee, 1989](#)). There is an insufficiency of research in this entire domain, especially on the long-term effects of joint custody. We especially await findings from the study of infants and young children to shed light on how the frequency of going back and forth from one home to another affects bonding and development.

Several studies, most notably that of [Maccoby and Mnookin \(1992\)](#), show that whatever the court order, many children change their residence from one person to another during the postdivorce years. Joint physical custody is particularly likely to become a situation in which the mother is the primary parent who tends to medical and dental appointment, clothing, and other important aspects of the child's life. Dornbush find that adolescents who are in trouble are more likely to gravitate or be sent to live with their father during the postdivorce years.

## **PUBLIC POLICY AND DIVORCE RESEARCH**

Although policy makers, legislators, and judges have increasingly sought support from the findings of behavioral science and guidance from the mental health professions, the accumulation of psychological knowledge has not kept up with the rapid evolution of family law. Knowledge about children and parent-child relationships in the postdivorce family is still fragmentary and is insufficient to support many of the legislative changes in family policy that have found powerful adherents. The subtleties of psychological thinking and shadings of individual difference that are so critical to the perspective of the behavioral scientist translate poorly into the arenas of court and legislature. The several years of follow-up required to assess the impact of changed circumstances on altered family structure are ill-suited to the pressured agendas of the political and judicial process. Despite the widespread acknowledgment given to the important interface between family law and mental health, the major task of building cooperation and mutual understanding on a firm basis of empirical knowledge and shared values still lies ahead.

## **INTERVENTIONS**

It is evident that many families need professional advice and guidance in negotiating their way through the complex and tangled pathway of divorce and the postdivorce years. Moreover, it is important not only to provide the services that they need but also to find ways to reach both adults and children at the appropriate times, namely, at the marital rupture and at critical turning points along the arduous road that lies ahead. Essentially, divorcing families confront two sets of divorce-related issues that fall within the domain of the clinician: those associated with the acute crisis engendered by the marital breakup and those associated with rebuilding the family, and subsequent families, that will provide a "holding environment" for children and adults during the postdivorce years. These two sets of issues translate into a series of immediate and long-term psychological and social tasks for adults and children ( [Wallerstein, 1983](#); [Wallerstein and Blakeslee, 1989](#)). They translate as well into two separate preventive and clinical agendas: one addressed primarily to the amelioration of the psychological disequilibria of the separation crisis and its immediate aftermath and a second addressed to building or restoring family structure and parent-child relationships within the postdivorce or remarried family ([Wallerstein, 1990a](#)).

A third, clinical agenda is addressed to children who show a relatively consolidated psychological disturbance. Although children in this group present clinical issues that are relatively familiar, the therapist's relationships with the parents and other significant adults will differ sharply from those with most intact families. Thus, for example, in the divorced family the issue of who bears primary financial responsibility for the child's therapy is often in dispute. The treatment itself, whatever its course, has a high potential for being caught within the continuing angers between the parents, and the therapist is likely to be identified by one or both as allied with "the other side." Furthermore, there is the thorny issue, which needs to be resolved on a case-by-case basis, of which adults should be included in the therapist's relationship with the family. When and under what circumstances should a biological father who has had little contact with the child be seen? When should a stepparent be dealt with as the primary parent? When and how should live-in lovers be included? These multiple relationships need to be carefully assessed by the clinician not only for their bearing on the child but also because of their importance in constructing a network that will support the child's treatment.

Another critical issue that will influence treatment process and goals is that the development of many children is hindered by the continuing failure of their families to provide sufficient nurturance and protection to sustain the youngsters' developmental progress. As a consequence, in addition to addressing issues of neurotic conflict, the clinician may need to take a more supportive role. These supportive parameters, which so many of the children need, have implications not only for process but also for the duration of treatment.

### **Tasks of Divorce**

The reorganization and readjustments required of the child of divorce, namely, the psychological tasks that need to be addressed, represent a major addition to the expectable tasks of childhood and adolescence in our society. In effect, the child of divorce faces a special set of challenges and carries an added burden, which may indeed require professional help at different points along the developmental course. The individual child's resolution of these tasks is profoundly influenced by the family ambiance and by the extent to which the parents have made progress in resolving the many issues to which divorce gives rise. Nevertheless, it is the child who must carry the burden of mastery and resolution on the way to successfully achieved adulthood; there is no necessarily determining relationship between the resolution and adjustment achieved by either of the parents and the outcome for any particular child in the family. There is, in fact, a widening difference in outcome among siblings as they approach young adulthood ( [Corbin, 1988](#)).

These readjustments are likely to stretch over the growing-up years and through adolescence. They are the coping tasks that are shaped by psychological threats to the child's psychic integrity and development. These tasks have been conceptualized as a hierarchical series, which follows a particular time sequence beginning with the critical events of parental separation and culminating at late adolescence and young adulthood. They represent the agenda for the child as well as for the therapy.

Six psychological tasks have been formulated by [Wallerstein \(1983\)](#). They fall into an unfolding sequence with varying time spans attached for the accomplishment of each. Task 1, *acknowledging the reality of the marital rupture*, and task 2, *disengaging from parental conflict and distress and resuming customary pursuits*, need to be addressed immediately at the time of the decisive separation, and they are optimally resolved within the first year. The child's successful mastery of these two immediate tasks is tied to the maintenance of his or her appropriate academic pace and overall developmental agenda, after the initial dip at the time of crisis. However, the child's successful mastery of divorce-engendered stress is only partially related to the early period after the marital rupture. Task 3, *the resolution of losses* (including the loss of the presence of one parent in the home), task 4, *resolving anger and self-blame*, and task 5, *accepting the permanence of the divorce*, will be worked and reworked by the child over many years. They, along with the final task 6, *achieving realistic hope regarding relationships*, will become salient at adolescence and entry into adulthood.

### **Some Treatment Considerations**

Various treatment modalities have been employed with this population to facilitate adjustment, including family therapy, individual psychoanalytically oriented therapy for the child, parent guidance, supportive group therapy for parents, and supportive group therapy for children. Cognitive-behavioral approaches have been used, primarily in groups for children within school settings. In working with all these modalities, it is important to keep in mind that treatment of the adult *qua* adult does not necessarily trickle down to help the child. Our own work of many years shows that parent-child relationships need to be addressed directly within the treatment and

cannot be counted on to extend from the treatment of the parent as the primary patient to the child ( [Wallerstein and Blakeslee, 1989](#) ). The conjoint treatment of divorced parents and their children may place children in jeopardy. It is not uncommon for the child in a divorced family to be punished severely by disputing parents for statements that may have been acceptable within the framework of an intact family. The child's greater vulnerability in these families needs to be understood by the clinician.

The psychological assessment of both child and parent takes on special importance in work with a divorce population. A significant measure of responsibility for the selection of the treatment goals falls on the clinician. In the ordinary course of child psychotherapy, parents present their concerns, which provide the starting point for the therapeutic alliance. By contrast, divorcing families, especially those coming to a preventive program, bring multiple and conflicting overt and covert agendas, each of which may cancel out the other. These agendas range from the wish to reconcile the marriage to fantasies of Medea-like revenge, from the need to hear that the children are untroubled to well-founded serious concerns about their welfare. Nowhere is the difference between an intact family, however conflict ridden, and a disrupted family more evident than in the difficulty in arriving at a common treatment agenda.

An additional consideration in the clinician's greater responsibility for the agenda of the intervention is that often parents are unaware of what may be the grave psychological condition of the children. Time and again, we have observed children in serious trouble, even in dire emergencies, whose normally attentive parents seem oblivious to the danger, caught up as they are in their own concerns. This is true not only of threats to psychological health of the child but also of a decline in the physical health of the child as well. Both need to come under the special scrutiny of the psychiatrist.

A considerable likelihood exists that children in these families will have witnessed severe verbal abuse or physical violence between the parents. Often, the effects of such experiences will not show up initially in the child's symptoms. They may well be associated, however, with serious superego lacunae, deficits in object relations and identity, and skewed expectations regarding relationships between men and women, which, although they may not become manifest during childhood, may, if left untreated, come to the fore at subsequent developmental stages. The therapist is well advised to make appropriate inquiries about parental conflict in the child's history that may not be readily forthcoming and to attempt to engage the child in an exploration of his or her reactions to what may have been searing incidents.

Finally, treatment of children and parents in the separating and divorced family often depletes the therapist's emotional resources. The final scenes of the failed marriage are played out in the consulting room not only in the painful context of the verbal interactions but also, just as importantly, in the transference reactions of the client caught up in the crisis and in the subsequent countertransference responses of the clinician. There is an extraordinary lack of psychological distance between therapist and client as identifications move back and forth across the therapeutic interface. The most common countertransference is for the clinician to experience anxiety about the stability of his or her own intimate relationships ( [Wallerstein, 1990b](#) ).

### **Future Directions in Interventions**

There are indications of mounting community concern about the changes in the family in our society and their impact on children, particularly the initial and long-term effects of divorce. Whether this concern will translate into adequately funded preventive and clinical programs, we cannot predict. Thus far, mediation and court-based programs have traditionally excluded children, whereas school-based programs have typically excluded parents. Nevertheless, the possibility is greater that the next decade will see a proliferation of new and integrated educational and preventive programs that will address families at the time of the separation crisis. Such programs should ideally reach out to parents within a general divorcing population and provide them with guidance in regard to the many decisions that they face and with specific advice about how to restore parenting and how to help their children during the crisis and its often extended aftermath.

One such demonstration project has been developed in northern California at the Judith Wallerstein Center for the Family in Transition, funded by private foundations. The outreach includes a letter sent to every family filing for divorce within the county. Children and adolescents are assessed individually and receive intensive counseling over an initial 3-month period, returning for brief follow-ups at the 1- and 2-year marks ( [Wallerstein, 1990a](#) ). This model has been copied in several clinics within the United States and Canada. There has also been some recent interest in modifying the model to meet the demands of the private sector. The work of Johnston and her associates offers another important template for working with families in high-conflict situations.

Precisely because divorce draws a large segment of the general population, it provides an unprecedented opportunity for developing and testing models of prevention in mental health. Because the subgroups at greater risk can often be identified at the time of the separation, among both parents and children, the clinician is provided with the opportunity to offer a range of intervention programs early in the process. One such subgroup that may well receive increased attention consists of those high-conflict families who fall through the net of the mediation services provided by the court. It is possible that the courts will recognize the grave psychopathology among these children and their parents and will develop referral networks within the private sector. The high incidence of allegations of physical and sexual abuse in divorcing families may also encourage the development of clinical services through a referral network attached to the courts.

Among the hopeful signs is the new willingness of public, private, and parochial schools to recognize the link between the learning and behavioral problems of many children and the weakening in the family structure. School systems are increasingly willing to permit or even welcome groups for children within the school setting, especially in the elementary schools.

Finally, there is burgeoning recognition of the needs of children of divorce that emerge with new intensity at young adulthood. Group programs are being developed that primarily attract adults in their 20s and 30s who find their shared experience a useful supplement to individual psychotherapy.

Overall, however, our society has been reluctant to undertake measures related to helping families deal with change. The mental health professions have not taken a leadership role, and children continue to lack a powerful voice raised on their behalf. Still critically needed are research that will address the large lacunae in our knowledge and programs in prevention and intensive clinical interventions that can push our knowledge far beyond its current compass. Both the research agenda and the intervention agenda appear to lengthen and to unfold before us as we come closer to understanding the psychodynamics of these new family forms.

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# 110 LEGAL CONSIDERATIONS IN THE PSYCHIATRIC HOSPITALIZATION OF CHILDREN AND ADOLESCENTS

Stephen Wizner, J.D.

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[The Parham Decision](#)  
[State Legislation](#)  
[The Responsibility of the Psychiatrist](#)  
[The Medical Decision to Hospitalize a Child or Adolescent](#)  
[Chapter References](#)

The mode and procedure of medical diagnostic procedures is not the business of judges. What is best for a child is an individual medical decision that must be left to the judgment of physicians in each case (1).

No area of medical practice affords a physician as broad and far-reaching discretion in the exercise of professional judgment as does psychiatric hospitalization of children and adolescents. Unlike the circumstances under which adults are admitted to psychiatric hospitals or wards—as voluntary patients, under short-term emergency procedures, or after a judicial hearing replete with due process protections and specific commitment criteria—children and adolescents, as a rule, may be confined at the request of a parent or guardian. In most states, only the admitting physician (and, in many cases, a managed care organization) stands between a parent's desire to hospitalize a child, for whatever reason, and the child's interest in not being improperly or unnecessarily institutionalized. Even in those states that afford objecting minors some judicial review of their involuntary confinement, review procedures and substantive commitment standards tend to be less formal and precise than those applicable to adults.

In addition to the lack of procedural formality and precise commitment criteria, the “mental illness” that brings most minors to psychiatric hospitals in the United States is manifested by behavioral problems rather than by cognitive or affective disorders. The typical minor whose psychiatric hospitalization is sought by parent or guardian is given a diagnosis of conduct disorder, personality disorder, adjustment reaction, or transient or situational disturbance. Only a relatively small proportion of hospitalized youngsters, less than one-third, are hospitalized for severe or acute mental illness, such as psychotic, organic, or serious affective disorders (2).

The reasons that some seriously disturbed minors who are hospitalized are diagnosed as having conduct disorders or a situational disturbance may reflect a reluctance to label a child as having a serious mental illness prematurely and the difficulty in diagnosing a child or adolescent without a significant history of psychiatric disturbance. Nevertheless, the fact is that most minors who are hospitalized are hospitalized because of conduct or personality disorders and do not have a serious mental illness.

In her 1988 study of psychiatric hospital admission rates of juveniles, psychologist Lois Weithorn reported that in 1980 there were 81,532 psychiatric hospital admissions of persons younger than 18 years of age, of which 49,910, or 61%, were to private hospitals (3). The fact that large numbers of minors without serious psychiatric disorders are confined in mental hospitals is attributable to several factors, not least of which is the absence of clear procedural protections and precise commitment standards. Since managed care has become more prevalent, fewer children and adolescents receive inpatient treatment (4), but the medical and profit-maximizing judgment of the managed care industry still is not an adequate substitute for legal rights.

During the 1970s, the practice of parental volunteering of minors into psychiatric hospitals was the subject of scholarly criticism (5) and judicial disapproval (6) as a violation of the constitutional right of juveniles not to be confined in institutions without due process of law. These academic critics and lower court judges concluded that parental discretion, subject only to psychiatric ratification or acquiescence, was insufficient to protect the liberty interests of minors.

## THE PARHAM DECISION

In 1979, in the case of *Parham v. J.R. and J.L.*, the United States Supreme Court upheld the practice of parental volunteering of minors into mental institutions, stipulating only that a physician at the admitting facility be “free to evaluate independently the child's mental and emotional condition and need for treatment” and that this physician “has the authority to refuse to admit any child who does not satisfy the medical standards for admission” (7).

The Court used the opportunity presented by the *Parham* case to constitutionalize a presumption that parents act in their children's best interests and therefore ought to be able to decide, free of state interference, that their children require psychiatric hospitalization. The Court sought to justify the state's failure to institute procedural safeguards by hypothesizing benign, and even beneficent, reasons the state might have for refusing to provide procedural protections for institutionalized children. The Justices speculated that a cumbersome or intrusive procedure might dissuade parents from taking advantage of state-provided health opportunities, to the detriment of their children and the public. Also, the Court inferred that the state could have an interest in ensuring that psychiatrists and other staff spend their time treating patients, rather than preparing for and appearing in court. Nevertheless, balancing the interests of the parents and the state against the “risk of error” from the child's perspective, the Court concluded that “some kind of inquiry should be made by a ‘neutral factfinder’ to determine whether the statutory requirements for admission are satisfied.” That “neutral factfinder” is the physician who approves the parent's decision to hospitalize the child.

The Justices were unwilling to accept the notion that children's rights might be violated by apparently well-meaning parents and predicted that the focus of an evidentiary hearing was likely to be an examination of the parents' motives rather than evaluation of the child's need for hospitalization.

The Court expressed concern about the possible antitherapeutic consequences of such a hearing, theorizing that it would exacerbate tension between parents and children and make the child's subsequent return home more difficult. Finally, the Court observed that the adversary system is not the best mechanism for discovering truth when psychiatric decisions are at stake.

Under the Supreme Court's ruling, the kind of inquiry that must occur is to be conducted by a staff physician at the admitting facility. The review of the admission decision should focus on “what is best for the child.” “Informal, traditional medical investigative techniques” are satisfactory. There must be an interview with the child; mere review of records is not sufficient. Finally, periodic review is required, although the Court did not specify how frequently review must occur, nor whether it may be less formal than the preadmission screening.

On only one remedial point did the Supreme Court agree with the trial court. The danger to children who are state wards of becoming “lost in the shuffle” after being admitted to the hospital might necessitate some formal postcommitment review procedures.

The facts of the *Parham* case, which the lower court had found to be “representative” of the class of children volunteered into state mental hospitals, would appear to cast doubt on the presumption that parents (and guardians) always act in the best interests of their children. At the request of his mother, J.L. had been admitted to a state mental hospital in 1970 at the age of 6 years. His diagnosis was “hyperkinetic reaction of childhood.” He was still there 5 years later when the case was filed because his parents would not take him home and, in fact, had voluntarily terminated their parental rights the year before. Hospital staff had recommended that J.L. be placed in a foster home, but that recommendation had not been carried out.

J.R., the other named plaintiff, had been declared a neglected child and removed from his parents' custody at the age of 3 months. He had been placed by state authorities in a succession of seven different foster homes and was eventually volunteered by social workers into a state mental hospital at the age of 7 years. His diagnosis was “unsocialized, aggressive reaction of childhood.” More than 5 years later, when the lawsuit was filed, he remained in the hospital, solely because state authorities had failed to implement a hospital recommendation that he be placed in a foster home.

In the words of Justice Brennan in a dissenting opinion, “In these circumstances, I respectfully suggest, it ignores reality to assume blindly that parents act in their children's best interests when making commitment decisions and when waiving their children's due process rights” (8).

## STATE LEGISLATION

Although the Supreme Court in *Parham* expressly rejected the views of academic critics, civil rights lawyers, and lower court judges, it left the door open for states to provide additional protections for minors beyond the minimum that it found to be constitutionally sufficient. Virtually every state currently has legislation that applies to the admission to (and discharge from) psychiatric facilities. Because of the significant variation in standards and procedures contained in the various state statutes, it is essential for the physician contemplating psychiatric hospitalization of a child or adolescent to be familiar with the law in his or her own state ( 9). Statutes change frequently, so although the following discussion provides guidelines for understanding the law in a given state, it cannot substitute for thorough research into the current applicable statutory scheme for a physician's own state.

In many states, a minor's admission to a psychiatric hospital is governed by statutes different from those used in an adult's admission to a psychiatric hospital. The statute relating to the admission of minors may be in a different part of a state's code altogether and may not even be referred to in the code relating to admission of adults (10). This is not true in all states; Iowa, for example, addresses the admission of minors and adults in the same statute ( 11). In general, however, a physician should be careful to ensure that he or she has identified the applicable statutes.

Once the proper set of statutes is identified, the physician should determine what kind of psychiatric admission is required. There are two primary types of admission to psychiatric hospitals: voluntary and involuntary. In cases involving minors, however, these terms may be misleading. Often, if a parent consents to the minor's admission, even if the minor is opposed to the admission, the admission is considered voluntary and governed by voluntary admission statutes ( 12). Even if the terminology of "voluntary" admissions is not used, separate statutes may govern parental admission of minors and involuntary admission of minors ( 13).

A psychiatrist's approach to admission also may depend on the age of the minor in question because the weight given to the minor's opinion about admission sometimes depends on the minor's age. For example, Virginia requires no hearings or court reviews for admission of a minor younger than 14 years of age if the minor's parent consents to admission, regardless of whether the minor consents ( 14), but does require judicial approval for admission of an objecting minor 14 years of age or older, even if the parent consents to the minor's admission ( 15). [The minor's age may continue to be relevant even after admission: For example, Illinois (16) permits minors 12 years of age or older to request postadmission judicial review, whereas Louisiana grants the right to all minors, regardless of age, to request a postadmission judicial hearing ( 17), although only those minors 16 years of age and older may institute certain formal objection procedures ( 18). And, of course, a person's age determines whether he or she is considered a minor at all. For example, in Connecticut ( 19), a person 16 years of age or older is covered by the adult voluntary and commitment statutes, whereas in New Mexico (20), a child is anyone younger than 18 years.]

After determining whether the admission is legally voluntary or legally involuntary, the psychiatrist should determine the standards for admission as outlined by the relevant statute. The specificity of these standards varies. Some state statutes merely enact into law the vague admonition of the Supreme Court that "some kind of inquiry" be made at the time of admission, and periodically thereafter, by a hospital physician to ensure that hospitalization is appropriate. For example, the Arizona statute permits hospitalization of a minor on application of a parent, guardian, or custodian, after a determination by the medical director of the admitting facility that the "child needs an inpatient evaluation or will benefit from care and treatment of a mental disorder or other personality disorder or emotional condition in the agency" and that "the evaluation or treatment goals can[not] be accomplished in a less restrictive setting" ( 21). Some states, like Idaho (22), do not require any type of review before the voluntary admission of a minor, beyond that conducted by the admitting physician. In contrast, some states make such hearings mandatory. Florida ( 23), for example, mandates that a person 17 years of age or younger may be admitted to a psychiatric facility only after a hearing to verify the voluntariness of his or her consent.

State laws also differ in the form of judicial review provided and the procedural protections afforded to minors in the case of involuntary admissions. For example, Connecticut (24) provides for an evidentiary hearing, with representation by court-appointed counsel, an independent psychiatric evaluation, and the right to present evidence and cross-examine witnesses. New Mexico (25) also gives the child the right to be represented by counsel, present evidence, and cross-examine witnesses, as well as giving the child the right to a complete record of the proceedings and the right to "an expeditious appeal of an adverse ruling."

States that provide judicial hearings to minors also differ with respect to the substantive criteria to be applied at such hearings. Connecticut ( 26), for example, requires a judicial finding that "the child suffers from a mental disorder, is in need of hospitalization for treatment, and such treatment is available, and such hospitalization is the least restrictive available alternative." For involuntary commitment of minors, New Mexico ( 27) requires a judicial finding

- (1) that as a result of mental disorder or developmental disability the minor needs the treatment or habilitation services proposed;
- (2) that as a result of mental disorder or developmental disability the child is likely to benefit from the treatment or habilitation services proposed;
- (3) that the proposed involuntary placement is consistent with the treatment or habilitation needs of the minor; and
- (4) that the proposed involuntary placement is consistent with the least drastic means principle.

Iowa (28) takes a somewhat different approach; it classifies an admission of a minor on the application of a parent or guardian as voluntary, but requires that "[t]he juvenile court shall order hospitalization of a minor, over the minor's objections, only after a hearing in which it is shown...that: (1) The minor needs and will substantially benefit from treatment. (2) No other setting which involves less restriction of the minor's liberties is feasible for the purposes of treatment," without further definition or specificity.

In contrast, although Virginia allows any minor younger than 14 years of age to be admitted as a voluntary patient if the minor's parent has requested the minor's admission, Virginia has extensive requirements for involuntary commitment of a minor. Specifically, it requires that for a minor to be involuntarily committed, the court must find that

1. Because of mental illness, the minor (i) presents a serious danger to himself or others to the extent that severe or irremediable injury is likely to result, as evidenced by recent acts or threats, or (ii) is experiencing a serious deterioration of his ability to care for himself in a developmentally age-appropriate manner, as evidenced by delusory thinking or by a significant impairment of functioning in hydration, nutrition, self-protection, or self-control;
2. The minor is in need of compulsory treatment for a mental illness and is reasonably likely to benefit from the proposed treatment; and
3. If inpatient treatment is ordered, such treatment is the least restrictive alternative that meets the minor's needs.

If the parents or guardians do not agree to the child's hospitalization, the requirements are even stricter: In that case, "the court shall order inpatient treatment only if it finds, in addition to the criteria specified in this section, that such treatment is necessary to protect the minor's life, health, or normal development and that issuance of a removal order or protective order is authorized ..." ( 29).

As this brief overview shows, statutory schemes for the admission of minors to psychiatric facilities vary widely from state to state, and may change over time even within a state. Psychiatrists should determine the current relevant statute in their respective states, paying attention to issues such as the applicability of the statute to minors; the distinctions drawn between involuntary and voluntary commitments; the age distinctions drawn by the statute; and the procedural and substantive protections provided to minors by the statute.

## THE RESPONSIBILITY OF THE PSYCHIATRIST

It is unlikely that either the Supreme Court decision in *Parham* or the wide variety of state legislation governing the admission of children and adolescents to psychiatric institutions will make a difference in most cases of parental hospitalization of children for psychiatric treatment. In many, if not most cases, the decision to hospitalize a child is made by responsible parents and competent psychiatrists, and only when there is no other way to provide necessary treatment. In such cases, whether a minor has a right to a hearing or exercises his or her right to a hearing, and regardless of the standards for commitment, the decision to hospitalize is likely to be the same.

However, there will remain a significant number of cases in which children and adolescents face unnecessary, or unnecessarily prolonged, confinement in mental institutions. State statutes will protect some of them. But there will continue to be young people, particularly in those states that have chosen not to enact detailed child commitment statutes, whose right to liberty must be protected, if at all, not by lawyers or courts but by the medical profession.

The psychiatrist called on to make the decision whether to institutionalize must not allow himself or herself to be party to using the process of commitment to remove unruly or unwanted children from unpleasant situations. Particularly in light of the long stays of some children for lack of a home to which to return or a more



appropriate placement, the admitting psychiatrist should see that discharge planning begins at the time of admission rather than at the time the child is ready for release.

Finally, the psychiatrist should realistically view the dangers inherent in forced hospitalization by taking the long view of treating a child's problems. More than determining whether the child can benefit from a stay in a hospital, the psychiatrist should ask how this period of institutionalization will likely affect the child's life over a period of years. What kind and quality of treatment will the child actually get? Will the family or state agency use this as a way to abdicate responsibility for the child? Will hospitalization improve the child's lot, taking into account the entire living situation, including the home to which the child may return? If a seemingly therapeutic short stay in a hospital stretches into an unnecessarily long period of time, will the benefits of the originally prescribed treatment be undone?

Although it may be true, as Chief Justice Burger asserted in *Parham*, that "in the typical case" the decision to hospitalize a child is made cautiously, by competent, caring parents in consultation with well trained and qualified psychiatrists, even the Chief Justice would have to concede that there are instances, such as those presented in the *Parham* case, involving uninformed and expedient decision making by parents and other caretakers; conflicts of interest between children and their parents; abandonment of troublesome or intellectually limited children; use of mental institutions as asylums or as a form of incarceration; confinement of children and adolescents in understaffed, overly restrictive, depressing state institutions; and hospitalization of youngsters who could receive appropriate treatment in nonhospital, community-based programs.

In 1969, the Joint Commission on the Mental Health of Children reported that most state mental hospitals for children and adolescents provided little or inappropriate treatment because of shortages of professional staff, untrained aides, lack of educational and recreational programs, and outmoded facilities, and it concluded that, for these thousands of hospitalized youngsters, "instead of being helped, the vast majority are the worse for the experience" ( 30). More than a decade later, the Children's Defense Fund surveyed mental health facilities for children and adolescents and reported disturbingly similar findings ( 31).

Shortly after the Supreme Court's decision in the *Parham* case, Edward Futterman, a child psychiatrist, drafted a partial "dissenting opinion":

While children do need protection from their own rebelliousness, they also need protection from arbitrary and capricious decision-making by parents and caretakers. While children do need protection from decision-making beyond their developmental capabilities, they also need protection from abandonment and from the use of the hospital as a form of incarceration. While parental responsibility, family autonomy and cooperation by families in the care of their children need to be respected, children need protection when their own interests are in conflict with parental wishes. While children need to be protected in their access to necessary treatment, they need protection from the imposition of overly restrictive treatment approaches when less restrictive alternatives are available (32).

Parents' decisions to hospitalize their children may bear little relation to the severity of the child's mental condition or to the optimal therapeutic approach. Parents simply may not want to or not be able to continue living with a difficult child. Parental unwillingness to live with a difficult child may result in placement in a mental hospital because parents know of no other alternative, either because of a failure to devote sufficient effort to looking for appropriate services or inability to afford whatever appears to be available, or a combination of both (33).

Even if less restrictive alternatives exist, a youngster may be accepted for inpatient treatment by a psychiatric hospital because hospital staff are not required to investigate the availability of alternatives, may lack the time or competence to assist parents in identifying and making arrangements for nonhospital treatment, or may have a financial interest in accepting a child into their program.

Parents who have volunteered their child into a mental hospital may undercut the child's treatment by failing to cooperate in family therapy. Parents may be inconsistent or hostile in their attitudes toward their child's treatment and improvement and reluctant or unwilling to have their child return home, even when hospital staff conclude that the child should be discharged ( 34).

In addition to parent-child conflicts of interest and parental ignorance of less restrictive treatment alternatives, there are other causes of inappropriate hospitalization of children and adolescents. Admission usually is based on a short interview with the child, together with additional information supplied by the parent, guardian, or referring agency. Diagnosis is difficult, and diagnostic labels are vague. "Adjustment reaction" and "transient situational disturbance" are ambiguous and encompass a broad range of "normal" as well as "sick" behavior. Youngsters can spend extended periods of time in mental hospitals despite early findings of "no mental illness."

Often there is third-party pressure on the child, the parents, and the hospital. Judges, police, probation officers, social workers, and school personnel may recommend or insist on psychiatric hospitalization, either for evaluation or treatment. All of these individuals may be acting from motives that, however well-intentioned, do not provide adequate protection against erroneous and unnecessary hospitalization.

Children and young adolescents may experience painful and traumatic psychological effects from being "locked up" in mental hospitals. Disruption in the continuity of relationships with adults is harmful for both young children and older adolescents. The committed child lives in a restrictive environment characterized by frequent staff turnovers and three different "shifts" of staff each day, making it difficult, if not impossible to develop new, stable relationships. The committed child is denied the opportunity to explore the world. Instead, he or she confronts an environment that places a premium on institutional efficiency and encourages compliant and passive behavior (35).

Because of the adverse effects of institutionalization on a child's cognitive, emotional, and social development, mental health professionals caution against hospitalization of a child, even in a superior facility, except when clearly necessary: "[N]o matter how good a treatment program for children in the state hospital is, hospitalization of an emotionally disturbed child is not the best answer . . . [The] basic experiences which a child needs in order to grow into an emotionally healthy, happy and productive adult . . . cannot be found in a hospital" ( 36).

Confinement in a mental hospital creates a special stigma not present in the placement of children in general hospitals or residential schools. The personal and social consequences of being labeled "mentally ill" are well documented in the professional literature ( 37). The label becomes "a double-edged blade . . . caus[ing] [the individual] to demean himself and to magnify social ostracism" ( 38).

The problem of stigmatization is magnified for children, who are more impressionable than adults, who are more likely to accept as true society's judgment that they are "crazy," and whose peer group is less tolerant of those who are "different."

For the child who requires psychiatric hospitalization, stigmatization is an unfortunate "side effect." But when a child is confined unnecessarily, the profound and long-term harm that results cannot be justified. A system that provides few procedural protections at the time of admission and no assurance that discharge will occur when therapeutically appropriate cannot protect children from the severe deprivation of liberty and other suffering caused by unnecessary, or unnecessarily prolonged, confinement in mental hospitals.

## THE MEDICAL DECISION TO HOSPITALIZE A CHILD OR ADOLESCENT

There exist differing professional standards, and differences in professional practice, regarding the conditions and circumstances that warrant psychiatric hospitalization of children and adolescents. Other than in the relatively uncommon, clear cases involving suicidally depressed or dangerously psychotic juveniles, most minors whose hospitalization is sought by parents or other legal guardians manifest symptoms of conduct or personality disorders, adjustment reactions, or situational disturbances. Adults carrying such diagnoses would not be considered appropriate for involuntary commitment because, as a rule, such persons do not pose an imminent risk of physical harm to themselves or others and are not wholly incapable of caring for themselves as a result of mental illness.

The National Association of Private Psychiatric Hospitals, the American Psychiatric Association (APA), and the American Academy of Child and Adolescent Psychiatry each have issued standards for psychiatric hospitalization of children and adolescents ( 39). Notwithstanding their clinical language, the National Association guidelines are sufficiently general and behaviorally based that they could be used to justify the involuntary hospitalization of a wide range of acting-out and nonconforming, as well as mentally ill and emotionally disturbed, youths. These guidelines include "self-defeating" or "self-destructive" behavior, inability "to attend to age-appropriate responsibilities," and "pronounced affective and/or behavioral disturbance" as reasons for psychiatric hospitalizations. An example of self-defeating or self-destructive behavior that might require "immediate acute-care hospitalization [as] the only reasonable intervention" is "sexual promiscuity," although the standards provide no definition of the type of sexual behavior to which that term ought objectively to apply ( 40). Behavior demonstrating "inability to function" that might justify hospitalization under these guidelines could be refusal to communicate with parents, angry outbursts, or participation in disapproved social activities, rather than

doing homework, having a job, participating in socially approved extracurricular activities, and spending time with one's family. Not only do the National Association guidelines emphasize behavioral disturbances, they lack a clear definition of the "mental disorders" that justify hospitalization.

The APA standards provide a more "medical" definition of the type and severity of "mental disorder" that would justify the emergency or long-term psychiatric hospitalization of a minor: "'Mental disorder' means a substantial disorder of the child's cognitive, volitional, or emotional processes that grossly impairs judgment or capacity to recognize reality or to control behavior" (41). Under the APA guidelines, involuntary civil commitment of a minor would require a judicial finding "by clear and convincing evidence, that (a) the child has a mental disorder and that (b) the child is in need of treatment or care available at the institution for which certification is sought and that no less structured means are likely to be as effective in providing such treatment or care" (42).

The American Academy of Child and Adolescent Psychiatry standards are even more precise and medically based and assume that inpatient treatment should be used only in situations where outpatient psychotherapy or other community-based treatment programs have been shown to be inadequate (43), and the youth is suffering from a severe problem that is primarily attributable to a "psychiatric disease" (44). Specific criteria justifying hospitalization under the American Academy standards include (a) "acute disabling symptoms of mental illness such as impaired reality testing, disordered or bizarre behavior, psychotic organic brain symptoms"; (b) "acute danger to property or self or others ...attributable to primary psychiatric disease"; and (c) "severely impaired social or family or educational or vocational or developmental functioning" (45).

Whatever standards or guidelines are followed, the admitting physician who is called on to make the decision whether to hospitalize a child or adolescent must exercise clinical judgment. He or she must determine whether the youngster suffers from a severe or acute disabling mental disorder that requires psychiatric treatment, whether he or she requires hospitalization to receive the treatment, whether such treatment is actually available in the hospital, whether he or she could obtain the treatment in a less restrictive setting or on an outpatient basis, and whether the potential benefits of involuntary psychiatric hospitalization outweigh the restriction of liberty, separation from family and friends, stigmatization, and other harms associated with institutionalization. In addition, the physician must carefully monitor the course of the treatment to ensure that the hospitalized juvenile does not spend any more time in confinement than is necessary to carry out the treatment plan, which should, in any event, be designed to help him or her return and adjust to life in the community as quickly as possible.

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11. See Iowa Code Ann. § 229.2 (West Supp. 2000).
12. See, e.g., Iowa Code Ann. § 229.2 (West Supp. 2000); N.C. Gen. Stat. § 122C-221 (1999).
13. E.g., compare Va. Code Ann. § 16.1-338 (Michie 1999) (parental admission of minors younger than fourteen and nonobjecting minors fourteen or older), Conn. Gen. Stat. § 17a-79(a) (1999) (parental admission of minors), with Va. Code Ann. § 16.1-345 (Michie Supp. 1999) (criteria for involuntary commitment of minors), and Conn. Gen. Stat. § 17a-76 (1999) (commitment of mentally ill minor).
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31. Knitzer, J., *op. cit.*
32. Memorandum from Edward H. Futterman, M.D., Chairperson, American Psychiatric Association Council on Children, Adolescents and Their Families, to APA Commission on Judicial Action, et al., dated June 29, 1979.
33. See Ellis, *op. cit.*, 851–852.
34. In general, children are more likely to stay longer in mental hospitals than are adults. *Crisis in Child Mental Health*, supra, p. 271. See also Rieger-Norbert I: Changing concepts in treating children in a state mental hospital. *Int J Child Psychother* 89:104, 1972: "[M]ost mentally ill children who are treated in a state hospital overstay, by months and even years, the optimum period required for their partial or total social restoration." A nationwide study reported that 40% to 50% of children and adolescents confined in mental hospitals are admitted inappropriately or remain hospitalized longer than necessary; Knitzer, *op. cit.* A recent national survey found that the average length of stay of children and adolescents in private psychiatric hospitals had decreased between 1990 and 1992, for children from 36.4 days to 27.6 days, and for adolescents from 33 days to 23.6 days. For adults during this same 2-year period, the average length of stay in private psychiatric hospitals declined from 19.6 days to 15.8 days. National Association of Psychiatric Health Systems, *1992 Annual Survey: Final Report*, summarized in *Hospital and Community Psychiatry*, 44(9):898–899, 1993. The survey did not include public psychiatric hospitals, where nearly half of all hospitalized children and adolescents are confined and where the lack of private insurance constraints often results in longer periods of confinement than typically occur in private hospitals.
35. "[H]ospitalization in a mental institution is a serious and possibly injurious event in the life of an individual, particularly a child or adolescent. Institutionalized individuals are cut off from significant interpersonal relationships with family and friends, and from their usual life activities. Institutionalization creates a major discontinuity in a person's life. It also places him or her within an artificial, regimented, and restrictive environment that can seriously harm those not in need of such placement." Affidavit of Eli C. Messinger, M.D., filed on August 28, 1975, in the case of *Poe v. Weinberger*, Civ. No. 74-1800, U.S. District Court, District of Columbia.
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45. *Id.*, Specific Guidelines I.A. 1, 2,8 (emphasis added).



# 111 THE MENTAL HEALTH PROFESSIONAL IN THE JUVENILE JUSTICE SYSTEM

Stephen Wizner, J.D.

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Mental health professionals occupy a special place in the juvenile justice system. Because of the rehabilitative and therapeutic goals of the juvenile court and its expressed commitment to individualized, nonpunitive dispositions for children and adolescents who are brought before it, mental health professionals are frequently called on to evaluate juveniles, recommend treatment or other rehabilitative programs, and provide consultation and expert testimony.

Juvenile court judges, probation officers, and attorneys defer to and rely on the opinions and recommendations of mental health professionals. Therefore, it is important for the clinician who would provide services in the juvenile court to be familiar with its history, jurisdiction, and procedure, as well as its limitations.

## HISTORY OF THE JUVENILE COURT

Jane Addams and other turn-of-the-century social reformers brought the juvenile court into being in order to rescue misbehaving children from the harshness and rigidity of the adult criminal justice system (1). In contrast to the criminal justice system, designed to apprehend, prosecute, and punish offenders for the protection of society, the goals of the child-saving movement were the identification, evaluation, and treatment of maladjusted youths for their own benefit, and ultimately society's.

Beginning in Chicago in 1899 and eventually spreading throughout the country, the juvenile court (in some states called the family court) was envisioned as a humane, informal, treatment-oriented social agency that would "save" delinquent children from what the reformers saw as the failure of many immigrant and other impoverished parents to cope with the adverse effects on their children of an increasingly urban and industrial environment.

Initially, the jurisdiction of the juvenile court was broad, encompassing both criminal and noncriminal misbehavior. Children who had committed no crimes but who were alleged to be truant, disobedient, engaging in undesirable behavior, associating with unacceptable companions, or otherwise beyond the control of their parents ("ungovernable") were brought before the juvenile court, together with those who were charged with criminal offenses, in order that they might receive the treatment, discipline, and care they required. These so-called "status offenders" were treated the same as youths who were charged with criminal offenses, although in many jurisdictions they were given a different label, such as "persons in need of supervision."

The juvenile justice reformers believed that juvenile misbehavior, criminal or otherwise, was not a law enforcement problem but a social-psychological problem of children and their families, requiring state interference with and assumption of the parental function of child rearing. It was possible, they believed, and therefore proper, to discipline, educate, and reform the errant and neglected children of the urban poor through the intervention of kindly judges, supportive probation officers, and well-meaning social workers and volunteers. If necessary, children were to be removed from their families and placed in rural reform or "training" schools where they would be subject to long hours of physical labor and strict discipline.

There were theoretical flaws and practical problems in the child-saver's program for curing juvenile delinquency. The coercive nature of involuntary court-imposed "therapy," however benevolently intended, must have seemed punitive from the child's perspective, because it was imposed as a consequence of the child's having engaged in disapproved conduct. The "treatment" provided—judicial admonition, probation supervision, counseling, and confinement in reform schools—was frequently both excessively intrusive and ineffective. Moreover, the theory was based on a simplistic and sentimental view of the nature of children and their psychological development and a naive and romantic faith in the extent to which youthful rebelliousness and antisocial behavior were amenable to treatment of the type offered by the juvenile court.

From the outset, juvenile courts lacked the resources even to attempt to carry out their program. Insufficient funds were appropriated by legislatures to enable juvenile courts to provide individualized justice or implement costly treatment plans for the majority of juveniles who appeared before them. There were not enough qualified mental health professionals available to the court to perform the evaluation, referral, and treatment services that were required. As with many social reform movements, rhetoric outpaced reality.

In addition, the court's treatment-oriented approach was thought to provide insufficient protection for the community from certain juveniles who committed serious crimes or were repeat offenders. Thus, as early as 1903, only 4 years after the first juvenile court was established in Chicago, that court transferred 14 children to the adult criminal system (2). By the 1970s every state, the District of Columbia, and the federal government had laws authorizing or requiring the criminal prosecution in adult courts of certain minors found to be "not amenable to treatment" (3).

Critics have pointed to the unbridled discretion, rehabilitative pretensions, and punitive realities of the juvenile court. They have called for restriction of juvenile court jurisdiction, limitation of the discretion of juvenile court judges, diversion of petty (or first) offenders to nonjudicial alternatives, procedural safeguards, and improved facilities, resources, and treatment (4).

The rising tide of criticism of the juvenile court culminated in the 1960s and thereafter in legislative reforms and judicial decisions that mandated procedural protection for juveniles and narrowed the court's jurisdiction. The New York Family Court Act of 1962 (4a) introduced due process into that state's family courts. A major innovation of the act was its provision for "law guardians," independent lawyers who were appointed to represent juveniles who came before the court. In 1966, the United States Supreme Court decided the landmark case of *Kent v. United States*, holding that the transfer, or "waiver," of a juvenile to an adult criminal court was "an invitation to procedural arbitrariness," and a decision "of such tremendous consequences" that it ought not be made without a hearing that provided "the essentials of due process and fair treatment" (5).

In 1967, in *In re Gault*, the Supreme Court expanded on its decision in *Kent* and ordered the provision of due process protection to all juveniles in delinquency proceedings, characterizing the existing informal juvenile court procedure as "a kangaroo court" (6).

Finally, in 1977 the United States Congress enacted legislation requiring states seeking federal financial assistance for juvenile delinquency programs to cease confining status offenders—children charged with noncriminal misbehavior—in secure detention or correctional facilities intended for juvenile delinquents (7).

The combined effect of the introduction of due process protections into juvenile delinquency proceedings, due process restrictions on the transfer of serious offenders to adult courts, and the effective removal of status offenders from the court's delinquency jurisdiction, together with an apparent increase in the incidence of serious juvenile crime, have left the juvenile court with a clientele that is more violent, more disturbed, and less amenable to superficial therapeutic interventions than that envisioned by Jane Addams and her fellow child-savers. In the words of one juvenile court prosecutor:

[The juvenile court is] a very depressing, horrible place. It's completely not in the real world....How does this court think it's going to rehabilitate a fifteen-year-old who's lived in an entirely different society....The Family Court was made for the little newsboy who broke somebody's window with a stone. That's when the laws were made. Not for the fifteen-year-old who's into drugs and killing (8).

## PRACTICE IN THE JUVENILE COURT

Mental health professionals are called on by the juvenile court to perform evaluations, make treatment and program recommendations, and provide consultation and

expert testimony (9). It is important that the clinician who chooses to work in the juvenile court be familiar with its procedures and specialized terminology.

A juvenile delinquency case normally begins with an arrest, referred to as an “apprehension.” The juvenile is either released to the custody of his or her parent or held in a “detention center,” the equivalent of an adult jail for pretrial detainees. If the child is to be detained, he or she is provided a “detention hearing” before a judge. The standard for detention is customarily whether detention is necessary for the protection of the community or is otherwise in the child’s best interests. “Preventive detention,” the practice of incarcerating juveniles prior to trial in order to prevent them from committing further delinquent acts during the pretrial period, was upheld as constitutional by the United States Supreme Court in a 1984 decision, *Schall v. Martin*, on the ground that “juveniles, unlike adults, are always in some form of custody” (10).

Whether or not a juvenile has been taken into custody, the child and his or her parents will normally be summoned to an intake interview with a court probation officer. At that stage, a decision is made whether to proceed “nonjudicially” by diverting the child to a community resource for services or to advance the case to the next stage of the process. This normally requires that the youngster “admit” (confess) to the charges and agree to participate in the treatment or program recommended by the probation officer.

In some jurisdictions the child is brought before a judge, either before, after, or instead of the intake meeting with the probation officer, in order to “admit” (plead guilty) or “deny” (plead not guilty) the allegations contained in a “petition” (indictment or complaint) alleging that the child has committed specified acts that if committed by an adult would constitute a crime. The court official—probation officer or prosecutor (in some jurisdictions euphemistically referred to by some title such as “court advocate”)—who signs the petition, or the state, is called the “petitioner,” and the child the “respondent.” At that hearing, analogous to an adult arraignment, the judge informs the juvenile and his or her parents of their rights, including the right to counsel. If the family cannot afford to hire an attorney, the court appoints a public defender or private attorney paid by the court to represent the child.

If the juvenile is an older, repeat offender and is charged with a serious criminal offense, the probation officer or prosecutor may seek to have the case “waived” (transferred) to an adult criminal court on the ground that the respondent is “not amenable to treatment” as a juvenile. A hearing is then held by the court to determine whether the types of “dispositions” (sentences) available in the juvenile court are likely to be of benefit to the juvenile and sufficient for the protection of the community.

The juvenile delinquency proceeding itself is in two stages. First, there is an “adjudicatory hearing” (trial). If the child is found guilty, there is said to have been a “finding” or “adjudication” (conviction). Second, if there has been an adjudication, the case proceeds to the “dispositional” (sentencing) phase. Prior to disposition, a probation officer conducts a predisposition investigation and prepares a dispositional recommendation. At the disposition hearing the judge considers the recommendations of the probation officer, the child’s attorney, and any other relevant information and imposes a “disposition” (sentence), which may be supervised or unsupervised probation, including probationary conditions such as a curfew, school attendance, counseling, or psychotherapy; community service; or a residential program in a private residential school or treatment center or in a state “training school” (the equivalent of an adult correctional institution).

## THE MENTAL HEALTH PROFESSIONAL IN THE JUVENILE COURT

The rehabilitative ideal continues to inspire the rhetoric and guide the practice in the juvenile court. Individualized justice and nonpunitive, treatment-oriented dispositions intended to help juvenile offenders and, in so doing, to protect society are the articulated goals, if not the reality, of the juvenile court. Forensic child clinicians are regularly called on by the court to perform mental health evaluations and family assessments, recommend and refer juveniles to treatment, consult with court staff and the child’s advocate, and provide expert testimony.

Unlike the adult criminal justice system, where mental health professionals most frequently are called on to offer expert opinions on issues of competency, insanity, and diminished capacity, in the juvenile justice system the principal mental health issue is “amenability to treatment.” At preadjudicatory and postadjudicatory stages of a juvenile delinquency proceeding, the need for treatment, appropriate type of treatment, and likelihood of benefit from treatment are central concerns.

At intake the probation officer must decide whether to handle (“adjust”) the case “nonjudicially” by releasing the juvenile or diverting him or her to a community agency or file a petition and seek an adjudication of delinquency and court-imposed disposition. At this stage “disposition bargaining,” analogous to adult plea-bargaining, is common. The juvenile’s advocate or the juvenile may attempt to avoid the risk of incarceration or other restrictive disposition by admitting to some or all of the facts in exchange for some less intrusive form of community-based treatment.

If the probation officer is persuaded that the juvenile is amenable to treatment by a community agency, and the juvenile admits some or all of the facts alleged as the basis for the arrest and agrees to accept the referral to the community agency and cooperate in the proposed treatment, the juvenile will not be required to stand trial before a juvenile court judge and face the possibility of an adjudication and court-imposed disposition.

At the intake stage both the probation officer and the juvenile’s advocate may seek an expert clinical opinion regarding the respondent’s amenability to treatment as an alternative to incarceration or other court-imposed disposition.

The next point in the process at which amenability to treatment may be evaluated is the so-called “waiver hearing” at which a judge decides whether to transfer a juvenile to the adult court for criminal prosecution. Often, a juvenile considered for transfer will be an older, repeat offender, charged with a serious crime. The mental health professional asked to evaluate such a youngster’s amenability to treatment will need to consider the youth’s prior experience with juvenile court dispositions, whether there are appropriate dispositional alternatives available to the juvenile court that have not been tried, and the relative superiority of dispositional alternatives available in the criminal justice system to address the juvenile’s problems.

If the juvenile is not diverted or transferred from the juvenile court and he or she is adjudicated a juvenile delinquent, the mental health professional may be called on at the dispositional stage to assess the youngster’s amenability to treatment by performing a mental health evaluation and recommending a particular type of therapeutic or rehabilitative disposition.

The most common disposition is probation, involving periodic meetings with a juvenile court probation officer, and special conditions of probation, which may include psychotherapy. If the juvenile is determined not to be amenable to treatment in the community, then he or she may be incarcerated in a training school or “placed” in a residential treatment program. The court is frequently guided by the recommendations of mental health professionals in deciding which of these dispositions to impose.

Assessment of the need for and amenability to treatment and recommendation of appropriate dispositions is the principal function of the mental health professional in the juvenile court. This function can be carried out in different roles. The forensic child clinician may serve as an adjunct to the court, providing consultation (or testimony) for the judge, probation staff, or prosecutor. The clinician also may assist the defense. In either role, the clinician may be able to provide expert assistance in diagnosing mental health problems and their relationship to the particular behavior at issue, in supporting or rebutting claims that individual juveniles are or are not amenable to treatment, recommending treatment or other rehabilitative programs, and evaluating previous efforts to treat or rehabilitate an individual juvenile.

## EVALUATION AND TREATMENT IN THE JUVENILE JUSTICE SYSTEM

The term *treatment* is used broadly in the juvenile justice system. A child or adolescent may be considered to be in need of or amenable to treatment not only because of a psychiatric condition but also, and more often, because of behavioral problems that may not be treatable by psychotherapy alone, or at all. Consequently:

a thorough assessment of a juvenile’s amenability to treatment should usually include an evaluation not only of personality functioning, but also of cognitive, educational, vocational, and social needs in the context of the various systems (e.g., family, school, neighborhood) of which he or she is a part (11).

It is important for the clinician to appreciate that the juvenile court’s rhetorical commitment to individualized treatment and the rehabilitative ideal tends to mask a law enforcement function. As a practical matter, the juvenile court is concerned with community protection, notwithstanding its child-saving rhetoric. Judges, probation officers, and prosecutors want to know not only whether a child or adolescent suffers from a mental health problem for which he or she should receive treatment but also whether that problem is the cause of the misconduct and whether the proposed treatment is likely to prevent the juvenile from recidivating. Therefore, mental



health professionals must take care that they are not co-opted into a system that may misuse their expertise and induce them to exceed the bounds of their professional competence.

It is often not possible for the clinician to establish a clear connection between a juvenile's mental or emotional problems and the specific offense charged, or predict the efficacy of a treatment plan in preventing recidivism. Indeed, research on the effectiveness of "correctional treatment" shows that most interventions appear to have little positive effect on recidivism and many appear to exacerbate the problem ( 12). Nevertheless, the clinician should offer the court his or her expert opinion and clinical judgment about the child's treatment needs and leave it to the court to determine whether the recommendations, if followed, will strike the proper legal balance between promoting the best interests of the child and protecting the community from the child.

### Chapter References

1. J. Addams, et al., eds., *The Child, the Clinic and the Court* (New York: New Republic, Inc. 1925). Two excellent histories of the American juvenile court movement are E. Ryerson, *The Best Laid Plans: America's Juvenile Court Experiment* (New York: Hill & Wang, 1978); and A. Platt, *The Child Savers: The Invention of Delinquency* (Chicago: University of Chicago Press, 1969). In addition to its delinquency jurisdiction, the juvenile court also has jurisdiction over proceedings against parents alleged to have neglected or abused their children. Consideration of the court's neglect and abuse jurisdiction is beyond the scope of this chapter. See generally, *Institute of Judicial Administration and American Bar Association Juvenile Justice Standards Project: Standards Relating to Abuse and Neglect* (Cambridge, Massachusetts: Ballinger, 1981); Goldstein, Freud & Solnit, *Before the Best Interests of the Child* (New York: Free Press, 1979).
  2. Cook County [Illinois] Charity Service Report 253 (1920), cited in Twentieth Century Fund Task Force on Sentencing Policy Toward Young Offenders, *Confronting Youth Crime* 55 (1978).
  3. D. Hamparian, ed., *Youth in Adult Courts: Between Two Worlds* (Columbus, Ohio: Academy for Contemporary Problems, 1982), 97–100, 143.
  4. See, e.g., President's Commission on Law Enforcement and Administration of Justice, Task Force Report: *Juvenile Delinquency and Youth Crime* (1967); F. Allen, *The Borderland of Criminal Justice* (Chicago: University of Chicago Press, 1964); J. Polier, *A View from the Bench* (1964); P. Murphy, *Our Kindly Parent—The State: The Juvenile Justice System and How It Works* (New York: Viking Press, 1974); *Institute of Judicial Administration & American Bar Association, Juvenile Justice Standards Project: Standards Relating to Juvenile Delinquency and Sanctions, Dispositions, and Noncriminal Misbehavior* (Cambridge, Massachusetts: Ballinger, 1981). See also, Juvenile Justice and Delinquency Prevention Act of 1974, as amended, 42 US Code 5601 et seq., at 5601 (a)(2): "The Congress hereby finds that . . . understaffed, over-crowded juvenile courts, probation services, and correctional facilities and inadequately trained staff in such courts, services and facilities are not able to provide individualized justice or effective help."
- 4a. NY Family Court Act of 1962, L. 1962, CH. 686, Art. 2, Part 4.
5. 383 US 541, 554–555, 562 (1966).
  6. 387 US 1 (1967).
  7. 42 US Code 5633 (12) (1977).
  8. P. Prescott, *The Child Savers: Juvenile Justice Observed* (New York: Alfred A. Knopf, 1981), p. 168.
  9. For a detailed description of juvenile court practice, and of the mental health professional's role in the court, see G. Melton, et al., *Psychological Evaluations for the Courts: A Handbook for Mental Health Professionals and Lawyers* (New York: Guilford Press, 1987, chapter 11, "Juvenile Delinquency"). See also, E. Futterman, et al., "The Psychiatrist and the Juvenile Justice System, Report of the American Psychiatric Association Task Force on Juvenile Justice Issues, (revised version, 10/88).
  10. *Schall v. Martin*, 467 U.S. 253, 265 (1984).
  11. G. Melton, et al., op. cit. supra, at 300. See also Juvenile Justice and Delinquency Prevention Act of 1974, as amended, 42 US Code 5601 et seq., at 5603(15): The term "treatment" includes but is not limited to medical, educational, special education social, psychological and vocational services, corrective and preventive guidance and training, and other rehabilitative services designed to protect the public, including services designed to benefit addicts and other users by eliminating their dependence on alcohol or other addictive or nonaddictive drugs or by controlling their dependence and susceptibility to addiction or use.
  12. J.T. Whitehead & S.P. Lab, "A Meta-Analysis of Juvenile Correctional Treatment," *Journal of Research in Crime and Delinquency*, v. 26, n. 3, pp. 276–295 (1989).

# 112 MALPRACTICE

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## CURRENT SITUATION

All physicians feel the burden of malpractice insurance, and many will be sued at some time in their careers. Child psychiatrists are not sued as often as physicians who rely heavily on technology; however, their involvement in the hospitalization and medication of severely disturbed patients makes it likely that the profession will be a target for litigation.

Since 1980, the overall size of malpractice awards has risen by 12.7% per year, nearly double that of the yearly increase in the cost of living and more than double the average yearly rise in medical costs. This startling increase is the result of vigorous and ingenious legal advocacy ( [Rosenberg, 1988](#)). Although they are more difficult to win, medical malpractice claims produce higher awards than the usual civil liability suit. Currently, the national average, in the United States, for recovery in medical malpractice suits is 46%, compared with 57% in civil liability actions. The average monetary award in psychiatric malpractice suits is \$340,000, compared with about \$1,100,000 in all medical malpractice.

Traditionally, emotional injury was not compensable, largely because authorities feared opening the floodgate to dubious, unassessable suits. However, the courts have begun to regard emotional injury more favorably, particularly if the defendant's breach has been wanton, outrageous, or extreme. Moreover, the proposed limitation of monetary awards for "pain and suffering" has been struck down in several states as unconstitutional, and awards have been made in some jurisdictions for "hedonic damages" (i.e., for posttraumatic impairment in the capacity to enjoy life).

The potentially lucrative nature of malpractice litigation crowds the legal field and makes it intensely competitive. Able lawyers are current with the medical literature, brandish the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* in the courtroom, and grill clinicians on the meaning of such terms as "reliability" and "validity." The clinician would be wise to understand the basis of malpractice law and to take steps to limit liability. Conversely, despite the increasing regulation and legal scrutiny of medicine, it is imperative that the profession avoids defensive psychiatry with its interminable tests and therapeutic homeopathy: paradoxically, excessive caution could put the clinician at risk of an action for negligence ( [Simon, 1987](#)).

## ELEMENTS OF MALPRACTICE LAW

The term *malpractice* refers to an *act or omission* by a professional in the course of his or her *professional duty* that *causes or aggravates an injury* to a patient or client and is *the consequence of a failure to exercise a reasonable degree of prudence, diligence, knowledge, or skill*. In other words, to be held liable for malpractice, one must do something one should not or fail to do something one should, and the act or omission must represent a level of care that is demonstrably less prudent, less diligent, less informed, or less skillful than would be provided by the average practitioner of the same training, in the same circumstances. Furthermore, the substandard act or omission must cause the patient a demonstrable injury that is of a compensable nature.

To substantiate malpractice, the plaintiff must establish the following four points, by a preponderance of the evidence:

1. A relationship existed between the clinician and the patient such that the clinician owed a *duty of reasonable care* to the patient.
2. When judged by the standard of the average, prudent practitioner, the clinician *breached the duty of care*.
3. The patient sustained *compensable injury or harm*.
4. The said injury or harm was a *direct result* of the clinician's failure to exercise a reasonable standard of care; or, in other words, the clinician's dereliction was the *proximate cause* of the patient's injury.

### Duty of Care

#### CONTRACTUAL RELATIONSHIP

The clinician owes a duty of reasonable care toward a patient when a professional relationship exists between them. This relationship is formed when a clinician explicitly or implicitly agrees to provide care to a patient. The clinician thus enters into a contract that binds him or her to provide a reasonable level of care in return for a valuable consideration (the fee). Unless the clinician has unwisely promised a cure, he or she is not bound to provide more than a reasonable level of care.

The doctor-patient relationship cannot be imposed on a competent patient, nor can a doctor be forced to care for a patient. The most controversial situations arise when it is argued that a relationship has been implied by the physician's actions or words. For example, the discussion of a patient's condition by telephone before transfer to a different hospital has been held to imply a contractual relationship ( [O'Neill v. Montefiore Hospital, 1960](#)). Payment is not necessary; gratuitous services attract a duty. The clinician should be careful about giving casual advice at cocktail parties and the like, lest it be construed that a contractual relationship has been formed.

Conversely, a physician cannot be forced to treat patients who are unable to pay for services or to use a treatment that he or she is not competent to implement. The physician also has a legal (if not an ethical) right to refuse to give aid in an emergency. Good Samaritan laws have been enacted to protect from liability those physicians who do render emergency aid, unless they have been grossly negligent (e.g., abandoning a live patient who is still hemorrhaging).

After the termination of the contract, the physician owes no further obligation to the patient other than that of confidentiality. Physicians who terminate contracts unilaterally and without reasonable cause are at risk of actions for *abandonment*. The physician must give the patient due notice of termination and must ensure that necessary arrangements are made for alternative care. If the patient resists termination, failure to refer to another physician may be construed as negligence.

Clinicians may examine people on behalf of a third party, such as a government agency, to whom they owe a contractual duty. However, if the examination causes the examinee harm, for example, by failure to detect suicidality or the possibility of child abuse, the liability risk is ambiguous. Some courts have held that the clinician's duty is to the employer; others have held that, if the person being examined reasonably relied on the examination for diagnosis, a duty may be owed. The physician is



advised to inform the examinee that the purpose of the examination is not therapeutic.

#### VICARIOUS LIABILITY

In accordance with the doctrine of *respondeat superior*, a physician is legally responsible for the negligent actions of employees or supervisees. Thus, a psychiatrist may be held liable for the negligent or outrageous actions of interns, residents, or nurses in an inpatient service or office.

#### FIDUCIARY RELATIONSHIP

The clinician's obligations toward the patient go beyond the duty to provide reasonable care. The relationship between psychiatrist and patient is analogous to that between guardian and ward. The patient has a right to expect the physician to show good faith, that is, to act in the patient's best interest. This, the physician's *fiduciary duty* is especially onerous in psychiatry, because emotionally disturbed people share their most private experiences with their mental health clinicians and are thus very vulnerable. Improper sexual contact, invasion of privacy, breach of confidentiality, outrageous manipulation of the patient's emotions, and the exploitation of patients for financial gain are all examples of *double agency* and *breaches of fiduciary trust*. These *intentional torts* are discussed later in this chapter.

#### CONFIDENTIALITY AND PRIVILEGE

By ethical code and law, the physician is precluded from divulging private matters revealed by the patient in the context of the doctor–patient relationship. *Privilege* is the patient's right to bar the physician from disclosing confidential matters in a court of law. In most jurisdictions, testimonial privilege derives from statute. Physicians should consult their local statutes to understand the extent of this privilege and its exceptions. Confusion may occur when a clinician receives a *subpoena* to appear in court (with or without records). *A subpoena merely requires the clinician to appear in court. It does not compel him or her to testify about confidential matters unless the patient has specifically authorized the clinician to do so, or has waived privilege, or unless the clinician is ordered to do so by the judge.* Although the states vary somewhat in the exceptions allowed to the rule of privilege, the following are the most usual:

1. *Waiver of privilege.* The clinician should seek specific written authorization from the patient before disclosing confidential material in court or to other parties (e.g., other mental health agencies).
2. *The psychiatric evidence exclusion.* If a patient offers his or her mental health as evidence in litigation, he or she waives privilege concerning the specific issue in evidence. Similarly, if the patient pleads insanity as a criminal defense, the psychiatric examination conducted to evaluate that matter is not privileged (*Bremer v. State, 1973*).
3. *Evaluation for a reason other than psychiatric treatment.* For example, the psychiatric evaluation of a disputant in a child custody case is not privileged if the evaluation was conducted as part of the case. A similar exclusion applies to evaluations for the purpose of civil commitment.
4. *Duty to protect endangered third parties.* The *Tarasoff* exclusion applies when the community is endangered. It is discussed later in this chapter.

#### Standard of Care

In accordance with the contract inherent in the doctor–patient relationship, the physician is bound to provide a reasonable level of care. In other words, the physician contracts to provide reasonable, prudent, diligent, knowledgeable, and skillful medical care. Unless the clinician has unwisely promised a cure, the contract does not call for exceptional care, only a level of expertise equivalent to that exercised under similar circumstances by the average practitioner in the same field of medicine. *The clinician is not liable for an error of judgment unless the error represented a substandard level of care.*

The considerable variation among clinicians in methods of treatment has made standards difficult to establish, particularly in regard to psychotherapy. Conversely, the standard is tighter with regard to precautions against suicide or violence or the monitoring of medication.

#### Breach of the Duty of Care

Malpractice suits are founded in the legal theories of intentional and negligent torts. An *intentional tort* involves deliberate intent on the part of the wrongdoer or wrongful conduct that the wrongdoer ought to have known was unacceptable (in which case it is known as a *quasiintentional tort*). Examples of intentional torts are assault, battery, false imprisonment, fraudulent commitment, defamation, invasion of privacy, sexual exploitation, and the intentional infliction of emotional distress. Expert testimony is not required to substantiate an intentional tort, and malpractice insurance may not cover it. A *negligent tort* involves an unintentional error that reflects a failure by the clinician to exercise a reasonable standard of medical care. Expert testimony is required for proof of negligence.

The standard of care traditionally required that the clinician be judged by the professional standard in the locality. However, the emergence of national standards has caused the courts to move in this direction, with allowance for the paucity of resources in some areas. The standard of care is linked to the standard of professional practice at the time of the alleged breach of duty; clinicians are expected to stay abreast of developments. If a clinician practices medicine in a specialty area for which he or she is not trained, the clinician is likely to be held to the standard applying to that specialty.

#### Harm, Injury, or Damage

*Harm* may be physical or psychological. As previously described, the courts have been reluctant to award damages for psychological injury, unless the wrongdoer's actions have been wanton or outrageous. Physical harm or damage resulting from negligence can vary: for example, side effects of medication (e.g., tardive dyskinesia), physical injury incurred when a patient is improperly restrained, or homicide or suicide occurring when a patient is negligently released from a hospital. All these situations are discussed later in this chapter.

#### Proximate Cause

The plaintiff must substantiate that the defendant's wrongful act or omission directly caused or aggravated the patient's injury. In other words, it must be proven that, but for the wrongful conduct, the damage would not have occurred or would not have been aggravated or that a direct, uninterrupted link or foreseeable chain of events exists between the wrongful conduct and the injury or its aggravation. The legal concept of *cause* is analogous to the psychiatric concept of *precipitation* or *aggravation*.

If Dr. A misdiagnoses B's illness, B must prove that, if not misdiagnosed, he would have received treatment that would have alleviated his condition. If Dr. A is held negligent in prescribing treatment X rather than treatment Y, B must still prove that, more probably than not, treatment Y would have been successful.

#### Damages

If the defendant is found liable, the judge or jury may award damages. Damages are designed to be *compensatory*, that is, to recompense the victim for medical expenses, pain, suffering, loss of enjoyment of life, impairment of capacity, and future loss of earnings and to restore the plaintiff to his or her original position, so far as money is able to do so. If the defendant's behavior has been egregiously outrageous, malicious, or wanton, *punitive* damages may be imposed over and above compensatory damages. Psychiatric evidence may be required to inform the jury about the impact of an injury on the plaintiff's capacity to enjoy life. Injury to a parent or child may give rise to an action for loss of consortium, that is, loss of the care, comfort, and society of a spouse, parent, or child.

### CIRCUMSTANCES IN WHICH MALPRACTICE IS MOST LIKELY TO OCCUR

Child psychiatrists are most at risk of liability in particular circumstances. This section describes the most perilous situations and their attendant risks.

#### Hospitalization

Hospitalization and medication, which are often combined, present certain situations in which the clinician is at risk of liability.

## ADMISSION

### *Negligent Failure to Admit*

The physician may fail to admit a suicidal or violent patient who subsequently harms himself or herself or others. Such an error may be actionable if the physician had failed to evaluate the patient adequately, for example, by assessing mental status or by reviewing the patient's past history for dangerous behavior. The assessment of suicide or violence risk is discussed later in this chapter.

### Wrongful Commitment, False Imprisonment, and Fraud

A physician who causes a patient to be admitted to a psychiatric hospital without proper evaluation may be sued for *negligent diagnosis* or *false imprisonment*. A falsely imprisoned plaintiff may recover damages for *loss of dignity* and *emotional distress*. Ignorance of the Mental Health Act is no defense; failure to comply with statutory requirements could be actionable. For example, in [Johnson v. Greer \(1973\)](#), a patient recovered damages after being forcibly detained in hospital for several days beyond the 24 hours permitted on an emergency warrant. Malice, spite, ulterior motive, bad faith, or fraud during involuntary hospitalization could also result in damages for false imprisonment or *malicious prosecution*, as was the case in [Pendleton v. Burkhalter \(1968\)](#). Parents or guardians may admit unemancipated minors against their will, and health care providers should be able to rely on parental consent ( [R.J.D. v. Vaughn Clinic, 1990](#)). However, the psychiatric justification for admission should be carefully documented.

## NEGLIGENT DIAGNOSIS

The failure to distinguish organic disease from psychiatric disorder, to differentiate functional psychosis from other psychiatric disorder, or to discriminate among the major psychoses could be actionable if it resulted from inadequate or inept diagnostic investigation and if it caused harm to the patient.

## PATIENT MANAGEMENT

### *Failure to Protect or Control a Suicidal, Violent, or Sexually Aggressive Patient*

Hospitals assume a duty of care toward patients with a potential for suicide or violence. The psychiatrist must carefully assess the potential for danger and must ensure that the hospital staff takes adequate precautions to protect a suicidal or violent patient from doing harm to himself or herself or to others. Past medical records should be scrutinized concerning suicide potential, and referring agents and parents should be questioned. In accordance with the imminence of the risk, housing in a secure unit, confinement to a room, close observation, a search of clothing and personal effects, and removal of all dangerous objects (e.g., belts, mirrors), may be required. If the patient is medicated, staff members should check that medication is actually swallowed. It is essential that the degree and nature of risk be communicated to all staff who care for the patient. The importance of good medical record keeping cannot be overstressed. Timely, clear, legible, pertinent, thorough, dated, timed, and signed records are the key to communication and the best proof that the hospital and staff have exercised reasonable care. In [Abille v. United States \(1980\)](#), after a psychiatrist transferred a patient from a suicidal status to a less dangerous status, the patient committed suicide. The finding of negligence against the defendant hinged on the psychiatrist's failure to keep detailed records that explained his decision to transfer the patient, even though it was conceded that, under the circumstances, the decision may have been reasonable.

What if the patient refuses to cooperate with the admitting psychiatrist, who consequently does not elicit and diagnose an imminent suicide risk? In [Skar v. City of Lincoln, Nebraska \(1979\)](#), a recalcitrant patient injured himself in a suicide attempt. The court found for the defendant and held that the patient had a duty to cooperate with his physician as far as he was able. However, it is essential in such a case that the psychiatrist record the questions put to the patient and the patient's responses or failure to respond.

The prevalence of sexual abuse and its relationship to psychiatric disorder mean that many minors admitted to psychiatric hospitals are at risk of precocious sexual activity and unwanted pregnancy. Known perpetrators should be closely observed and housed in single rooms, if necessary. Actions may be brought against hospitals and physicians who fail to take reasonable precautions to protect other patients from sexual assault, sexual coercion, or illicit sexual activity. Similar strictures apply to a failure to take reasonable precautions to protect others from physically violent patients. However, the closeness of the observation possible decreases if the therapeutic environment is less restrictive, for example, in a residential treatment center.

### *Negligent Release or Discharge of a Suicidal or Violent Patient*

A patient may harm himself or herself or others while on pass in the grounds of the hospital, on leave with relatives or friends, after discharge, or after absconding from hospital. Was the tragedy foreseeable by a reasonably prudent psychiatrist? This is the question that the courts seek to answer. In doing so, they are aware that the safety of the public must be balanced against the need to rehabilitate patients, that reasonable, calculated risks must often be taken, and that *bona fide* errors of clinical judgment are unavoidable ( [Higgins v. State, 1965](#)).

Increasing pressure by managed-care organizations, Medicaid agencies, and insurers has raised the specter of premature discharge against medical advice forced by withdrawal of funding. The clinician should be aware that legal responsibility for any harm that consequently befalls the patient or community will be placed on his or her shoulders. Hospitals and private clinicians who collaborate with health maintenance organizations would be wise to draw the line. If premature discharge is forced against medical advice, the clinician should protest vigorously in the medical record and should appeal by letter before allowing it. The risk may be so great that the hospital should bear the cost of continued hospitalization.

### *Failure to Protect Endangered Third Parties*

If a patient absconds from the hospital and one has reason to suspect that a third person or persons are placed in jeopardy thereby, it is the psychiatrist's duty to take whatever steps are required to protect the community. This duty, which emanates from the *Tarasoff* decision, is discussed in more detail later.

### *Wrongful Injury, Assault, and Battery*

A patient injured by staff members who use excessive force to subdue him or her may have a claim against the hospital for battery or wrongful injury. Wrongful injury may also be claimed when one patient is harmed by another whom the staff could not control; however, the plaintiff would have to establish that the hospital was derelict in its duty to control the violent patient.

Seclusion and restraint present serious liability risks. They may be legitimate management techniques when the risk of harm is imminent and there are no alternatives; but they should not be used to compensate for understaffing. Physical control should be time limited, and the patient should be examined by a physician if the maximum permissible time (e.g., 1 hour) requires extension. Seclusion and restraint should never be ordered "as needed." Quality assurance tracking is required to ensure that the use of physical controls does not become excessive.

If the person causing the harm is a hospital employee, the hospital may be liable, particularly if it were known that the employee had a propensity for violence or sexual misbehavior ( [Samuels v. Southern Baptist Hospital, 1992](#)). The hospital may also be liable for the misbehavior of physicians, agency nurses, or others who work in hospital but who are not employed by it. State institutions may claim *sovereign immunity*, which precludes litigants from suing governmental institutions; however, most jurisdictions have greatly limited or abolished this doctrine.

## IMPROPER TREATMENT

Treatment may be administered against the patient's will in an emergency or if the patient has been adjudicated incompetent. In other circumstances, a mature minor and his or her legal guardian, or the legal guardian alone (in the case of immature minors), must give informed consent to treatment. This matter is discussed in more detail later in this chapter. Erroneous choice of treatment, the use of an unapproved treatment, and failure to monitor side effects are also discussed later in this



chapter. The hospital will be liable for failure to provide adequate staffing, equipment, and support to manage the problems associated with the procedures it offers.

## RECORD KEEPING

### *Improper Release of Information from Medical Records*

The comments made in this section are relevant to both hospital and outpatient practice. Traditionally, privilege has applied to all private communications between patient and doctor, including the medical record. However, in recent times, the confidentiality of the medical record has been “polluted” (i.e., invaded by a number of bodies). Third-party insurers, for example, have access to patients' records to ensure that medical costs are legitimate. Nevertheless, the unauthorized disclosure of confidential information from a patient's record could be actionable on the ground of breach of confidentiality. Medical records should be kept in a locked place in the ward or medical records department to bar access to unauthorized people. The patient or legal guardian must give written consent for the transfer of information to legitimate professionals or agencies (e.g., other involved clinicians, attorneys, hospitals, schools, social welfare departments, and insurance companies).

If a subpoena is served without the written consent of the patient or guardian, the clinician has three alternatives: to seek the patient's consent; to have a motion filed by an attorney to quash the subpoena; or to refuse to testify unless ordered by the judge to do so. If the clinician persists in refusing to testify despite the judge's instruction to do so, he or she may be held in contempt of court ([in re Lifschutz, 1970](#)).

### *Defamation*

*Defamation* involves communication by one party about a second party to a third party that damages the reputation of the second party. Defamation is most likely to occur in child psychiatry when carelessly written medical records are released to third parties. For example, the patient may have been described in the record as a “psychopath” or “malingerer,” labels that could be extremely damaging in the hands of employers or creditors. Clinicians who gossip about patients over coffee or in elevators put themselves at risk of liability on the grounds of defamation or breach of confidentiality.

Defenses against defamation include *substantial truth* and *conditional privilege*. For example, if Dr. A refers patient B to Dr. C, and Dr. C reports to Dr. A that B has antisocial personality disorder, Drs. A and C have a duty and interest to receive and report such matters. In other words, conditional privilege applies. No such privilege applies to those who have no such duty and interest. Testimony given in court or provided to the court in documents such as medical reports or medical records attract *absolute privilege* insofar as they refer to the broadly defined matter at issue. *Malice*, if proven, destroys conditional privilege. Malice is substantiated by the author's evident self-interest, excessive distribution of the defamatory material, reckless disregard of the truth, or the vituperative style of the documents involved in the case.

### *Liability for Failure to Communicate Records*

The courts have generally refused to find that patients have a property or constitutional right to their records. However, records may be made available on request for legitimate reasons such as litigation. Some states have enacted legislation to allow patients to have access to their mental health records. However, “personal notes” may be excised (e.g., confidential information, information potentially injurious to the recipient, and the therapist's speculations), thus limiting access to the official record only.

## Medication

The points in the medication of a patient at which negligence is most likely to occur are as follows:

1. The diagnosis of the psychiatric disorder
2. The adoption of an appropriate rationale for drug therapy
3. The choice of a particular drug to treat the patient's psychiatric disorder
4. Inquiry concerning a past history of excessive therapeutic response, severe side effects, or allergic reaction to the drug in question or to related drugs
5. The search for coexistent medical conditions that would contraindicate the medication in question or would indicate the need for caution in its use
6. Obtaining informed consent
7. The administration of an appropriate dose of the drug in question, by an approved route
8. The prescription of drugs in combination, or the addition of a drug or drugs to an existing medication regimen
9. The choice of a drug not approved by the U.S. Food and Drug Administration or the use of a drug in a way, or for a purpose, that deviates from that recommended in the package insert or *Physician's Desk Reference*
10. Monitoring the therapeutic effect and side effects of the medication
11. Ceasing the medication after a therapeutic effect has been achieved or maintaining long-term medication at the lowest effective dosage

## DIAGNOSIS

The physician must distinguish organic disease from functional disorders and, within the latter, must differentiate the major psychoses from each other and from other Axis I and II conditions. The physician who admits patients to a psychiatric hospital must be alert for signs that could indicate a toxic condition. In [Hirschberg v. State \(1997\)](#), for example, a patient who had taken an overdose of salicylates died after having been hospitalized without adequate physical examination, special investigations, or proper precautions. The hospital was found liable.

The physician who fails to diagnose the patient accurately (e.g., by missing psychosis when it is present or by confusing bipolar disorder with schizophrenia) may find treatment on a false premise and thus may be open to a liability suit. However, there have been few actions on these grounds. Lawyers have been reluctant to pursue such cases in view of the reputedly widely varying expert opinion concerning the most appropriate treatment for different psychiatric conditions. As child psychiatry becomes more empirically based, it is likely that such actions will increase in frequency.

## DECISION TO MEDICATE

### *Failure to Medicate When It Is Appropriate to Do So*

In [Osheroff v. Chestnut Lodge et al. \(1987\)](#), suit was brought against a private hospital for negligent failure to disclose all treatment alternatives. The plaintiff, who suffered from mixed affective and personality disorder, had been treated for several months with individual psychotherapy. After transfer to a different hospital, he responded to antidepressant medication within a few weeks. Several eminent psychiatrists testified for the plaintiff that antidepressant medication was the treatment of choice, and the plaintiff had had insufficient opportunity to consider it as a treatment alternative. The suit was settled out of court in favor of the plaintiff. It is likely that this case, which became an ideologic battlefield, foreshadows future similar actions.

### *Inappropriate Rationale for Treatment*

Psychotropic drugs are sometimes prescribed to control inmates in correctional or mental retardation institutions. These situations warrant close scrutiny; the physician may have been induced to medicate the patient by the urgings of harassed staff, rather than by the medical needs of the patient, a practice that has been specifically criticized in at least one class action suit ([Nelson v. Heyne, 1972/1974](#)) and one malpractice case ([Clites v. State of Iowa, 1982](#)).

### *Choice of an Inappropriate or Unapproved Drug*

This kind of error occurs when the physician orders a drug that is inadequate to treat the patient's disorder (e.g., a benzodiazepine for major depressive disorder) or when the physician prescribes a drug for which there are less risky alternatives (e.g., a neuroleptic for anxiety disorder). A different problem arises when the clinician prescribes a drug not approved by the Food and Drug Administration. Undoubtedly, the risk of malpractice is greater if approved guidelines have not been followed. The clinician should reserve unapproved drugs for cases in which conventional treatment has failed. In addition, he or she should document a risk-to-benefit analysis,

seek expert consultation, and obtain specific informed consent before prescribing the unapproved drug.

#### *Failure to Obtain a Medication History*

The patient's past medication history should be ascertained by interview, by review of medical records, and by telephone contact, if required, with other clinicians. The physician may be held liable for excessive side effects, allergic reactions, idiosyncratic responses, or drug interactions, if these had been foreseeable.

#### *Failure to Detect Contraindicative Conditions*

The physician may fail to check for conditions or disorders that would render the patient vulnerable to severe side effects. For example, preexisting subthyroidism may be overlooked when lithium is prescribed. Each class of psychotropic drug requires a standard workup involving medical history, physical examination, review of past medical records, and special investigations. If a contraindicative condition is uncovered but the medication is still considered potentially justified, a risk-to-benefit analysis should be documented, expert consultation should be obtained, and specific informed consent should be recorded. Hospitals should mandate, as policy, standard diagnostic workups for all psychotropic drugs.

#### *Failure to Obtain Informed Consent*

The doctrine of informed consent is founded on the constitutional right of the individual to control what is done to his or her own body ([Schloendorff v. Society of New York Hospital, 1914](#)). A patient cannot give informed consent unless he or she has (a) *sufficient information* on which to base a decision, (b) *mental competency* to make a rational decision, and (c) freedom to exercise *voluntary choice*.

The physician must therefore disclose sufficient information to enable the patient to weigh all material pros and cons. How much is that? The earlier "professional" standard ([Natanson v. Kline, 1960](#); [Aiken v. Clarey, 1965](#)) referred to *what a reasonable medical practitioner would disclose* under similar circumstances. Following two 1972 cases, [Canterbury v. Spence](#) and [Cobbs v. Grant](#), some jurisdictions may have adopted the "patient" standard, that is, *as much information as a reasonable patient would require* to make a rational decision under similar circumstances. [Canterbury](#) and other cases suggest that the physician should discuss the following matters with the patient:

1. The nature of the condition that requires treatment
2. The nature, purpose, and benefits of the proposed treatment, and the probability that it will succeed
3. The risks and consequences of the proposed treatment
4. Alternatives to the proposed treatment (including no treatment) and their attendant risks and consequences
5. The prognosis with and without the proposed treatment

The courts have held that *not all risks need be disclosed—only the material ones*. Unfortunately, there are no clear guidelines concerning what is "material," except to suggest that, even if the likelihood be slight, the more serious the risk, the greater the probability that it should be discussed. *A risk is material when a reasonable person would be likely to attach significance to the risk or cluster of risks*. Risk is linked not only to the procedure but also to the physician and his or her competence to perform the procedure (e.g., if the physician has alcoholism or suffers from human immunodeficiency virus infection). The plaintiff must prove that nondisclosure of material risk caused the injury of which he or she complains. Some courts require that the plaintiff prove that a reasonable person would have refused the procedure had the risks been disclosed.

*Therapeutic privilege* countervails the clinician's duty to disclose. The physician is obliged to protect a vulnerable patient from the emotional trauma that could be sustained if upsetting risks were prematurely revealed. However, therapeutic privilege should never be invoked merely because the clinician fears that, if apprised of the facts, the patient would reject a desirable treatment. If the clinician proposes to limit disclosure on the ground of therapeutic privilege, clear documentation and expert consultation are required concerning the patient's exceptional sensitivity.

Disclosure is not required for an emergency in which the harm from failure to treat is imminent and outweighs the potential danger of the treatment. However, if possible, a relative's consent should be obtained.

Patients beyond the age of majority are presumed competent to make decisions about their own treatment. Competence may also be extended to emancipated minors. When should an unemancipated minor be regarded as sufficiently mature to participate in health care decisions? A series of landmark decisions (e.g., [Planned Parenthood of Central Missouri v. Danforth, 1976](#); [Bellotti v. Baird, 1976](#)) extended to minors the right to make health decisions concerning abortion, and some states have enacted legislation with regard to mature minors and contraception, venereal disease, substance abuse, and emergency psychiatric care. The present situation is very confused ([Wadlington, 1983](#)), and the practitioner should ascertain the law on the matter in his or her own state. However, although it may not be legally required, the desirability of promoting a treatment alliance suggests that it is judicious to promote the understanding and cooperation of all minors mature enough to comprehend. For patients more than 13 years of age, it would be prudent to obtain formal assent. Other than in emergencies, when minors are treated, parental consent is also required. However, according to [Annas \(1975\)](#), no court in recent years has found a physician liable for treating a consenting minor who is more than 15 years of age in the absence of parental consent, provided the treatment was given in good faith.

The third element of informed consent, *voluntariness*, is easily compromised. True voluntary consent requires that the patient be free of coercion. However, even a mature minor can be susceptible to threat, cajolment, bribery, or false inducement by parents, physicians, or hospital staff. The inmates of correctional institutions and psychiatric hospitals are particularly vulnerable to therapeutic coercion.

## IMPLEMENTATION OF TREATMENT

### *Forcible Administration*

The involuntary commitment of a patient to a psychiatric hospital does not permit involuntary medication, except in narrowly defined circumstances. Forcible medication can place the practitioner at risk of an action for the intentional tort of battery. The legal doctrine most pertinent to this issue, *the right to refuse treatment*, has been most clearly articulated in two convoluted cases, [Rennie v. Kleir \(1978/1979/1981/1982\)](#) and [Rogers v. Okin \(1979\)](#). In [Rennie](#), a New Jersey case, it was determined that the mentally ill had a sufficient liberty interest to require due process before treatment was forcibly administered, but due process was satisfied by an in-hospital review. In considering this case, the United States Supreme Court deferred to the professionalization standard articulated in [Youngberg v. Romeo \(1982\)](#), in effect declining to uphold a constitutional right to refuse treatment.

[Rogers v. Okin \(1979\)](#), a Massachusetts case, evolved into [Rogers v. Commissioner of Mental Health \(1983\)](#) and was reviewed by the United States Supreme Court as [Mills v. Rogers \(1982\)](#). The Massachusetts Supreme Court held that involuntarily committed patients should be presumed competent to refuse treatment except in emergencies. An "emergency" was defined as a situation fraught with the need to prevent violence or associated with the likelihood that the patient's health would significantly deteriorate without treatment. In nonemergency situations, forcible treatment may be administered only after a judicial hearing in which the court approves a "substituted judgment" treatment plan. "Substituted judgment" requires the court to determine what the patient would have decided if he or she were competent.

The failure of the Supreme Court to clarify this matter means that each state must make its own determination of the boundaries of the right to refuse treatment. The legal situation with regard to children is unclear. The right to refuse treatment apparently extends to the legal guardians of minors and to emancipated minors. What of the mature minor? Arguably, except in emergencies, the clinician should seek the consent of both mature minor and parent before starting treatment. If the mature minor refuses treatment, and the treatment is regarded vital, a judicial determination of competence should be requested.

### *Inappropriate Dosage or Route of Administration*

The *Physician's Desk Reference* includes the information found in all official drug package inserts, together with manufacturers' information concerning drug products that have no insert. In [Mulder v. Parke Davis & Co. \(1970\)](#), the court held that a departure from *Physician's Desk Reference* guidelines represented a *prima facie* case of negligence unless the clinician could provide adequate reason for having done so. To protect themselves from liability claims, drug manufacturers publish information concerning all reported adverse reactions, however rare, and are conservative in the dosages they recommend. In some circumstances, it may be good clinical practice to regard the risk of particular adverse reactions as insignificant or to exceed the recommended dosage. However, before doing so, it would be



advisable for the clinician to document a risk-to-benefit analysis, seek a consultation, and locate references in the literature that would support the proposed medication regimen.

An intravenous or intramuscular route of administration carries an increased risk of excessive or adverse response. This is particularly likely if the patient is predisposed to side effects as a result of hepatic, renal, cardiac, or brain dysfunction. The clinician should be cautious if such conditions are detected. Parenteral administration should generally be reserved for emergencies or long-term depot treatment.

### *Polypharmacy*

Psychotropic cocktails increase the risk of adverse reactions. They are most likely to be prescribed when psychotic patients are slow to respond or in general hospitals and residential centers that have inadequate structure or psychiatric nursing care. Conversely, as [Simon \(1987\)](#) points out, drug combinations (e.g., of an antidepressant with a neuroleptic) are sometimes more efficacious than one drug alone. Nevertheless, the clinician should record a risk-to-benefit analysis before prescribing multiple psychotropic agents.

### *Failure to Monitor Treatment*

The case of [Clites v. State of Iowa \(1982\)](#) illustrates negligent failure to monitor psychotropic medication, in addition to other errors. The plaintiff, a mentally retarded patient, was admitted to a state residential facility at the age of 11 years. Between the ages of 17 and 22 years, he was treated with various psychotropic drug combinations, until tardive dyskinesia was noticed. The appellate court held that the hospital had failed to document behavior sufficiently aggressive or self-abusive to warrant neuroleptic treatment, that the medication appeared to be designed primarily for the staff's convenience, that the practice of prescribing polypharmacy was substandard, that the treatment had not been monitored adequately by a physician, that "drug holidays" should have been employed, that the staff had been too slow in recognizing the patient's dyskinesia, and that informed consent had not been sought from the patient's parents. Damages of more than \$750,000 were affirmed.

Adequate monitoring of drug effects requires baseline and regular mental status examinations, physical examinations, vital signs, and laboratory testing, in accordance with the pharmacology of the drug and its potential side effects. "Drug holidays" should be considered if long-term medication is required. Physicians run a serious risk if they write "as-needed" orders for potent drugs, if they telephone prescriptions into a pharmacy without examining their patients, or if they provide multiple repeat refills.

### *Suicide*

An action for wrongful death may be brought against the physician who prescribed a drug that was subsequently used to procure a suicide. The precautions that could prevent such a tragedy are discussed later in this chapter. When the risk of suicide is significant, less lethal drugs should be prescribed, in small amounts. Blood levels should be regularly determined, to ensure that the patient is not hoarding the medication.

### **Psychotherapy**

Psychiatrists are most at risk of liability when they terminate the treatment of patients without adequate safeguards, when they are unavailable and either no covering physician is provided or the covering physician has not been given sufficient information, or when adequate steps are not taken to protect endangered third parties. A therapist runs the risk of being held to have committed an intentional tort when he or she recklessly inflicts emotional distress on, has improper sexual contact with, breaches the confidentiality of, or defames a patient.

Unless the physician has behaved outrageously or recklessly, or unless the patient has committed suicide, it is difficult to prove harm as a result of negligent psychotherapy, because mental injury is hard to substantiate when the plaintiff was already emotionally disturbed before the alleged negligence. It can also be awkward to establish what is a reasonable standard of care, because there are so many schools of psychotherapy. Thus, most successful actions have alleged either negligence leading to suicide or outrageous conduct.

### **ABANDONMENT**

The physician has the duty to provide continued care, as long as needed, until the doctor-patient relationship is terminated. This relationship may be severed by mutual consent or by the patient. However, unilateral termination of care by the clinician, if abrupt or premature, may put him or her at risk of breach of contract should harm (e.g., suicide) befall the patient. Failure to provide a telephone number for a disturbed patient to call, failure to provide adequate substitute care during absences, or failure to convey adequate precautionary information to substitute physicians may all be construed as abandonment if the patient is found to have suffered harm as a consequence. The absolute duty to provide continued care does not apply to a consultant who has been asked by a primary physician to render an opinion, that is, unless it has been agreed that the consultant will treat the patient.

When is the therapist warranted in unilaterally terminating the relationship? Lack of cooperation or threatening behavior may justify termination; failure to pay the bill does not ([Smith, 1986](#)). If the clinician does decide to terminate the relationship, the following safeguards are required ([Simon, 1987](#)):

1. The reason for the termination should be discussed with the patient. The clinician should give at least 1 month's notice of termination, to allow the patient to locate another therapist.
2. The patient should be provided with the names and telephone numbers of alternative therapists or agencies.
3. The clinician should mail to the patient a certified letter (return receipt requested) reflecting this discussion and containing the reasons for termination. The letter should also convey the names and telephone numbers of alternative clinicians or agencies.

These problems are most likely to occur in highly ambivalent adolescent patients with personality disorder whose suicide risk is aggravated by loss, separation, or rejection.

### **FAILURE TO PROTECT ENDANGERED THIRD PARTIES**

This matter is discussed at this point, rather than under hospitalization, because it is with outpatients that the most problematic situations arise. The facts of the original case, [Tarasoff v. Regents of the University of California \(1974, 1976\)](#), illustrate the problems of deciding what to do when, during outpatient treatment, a patient threatens to harm a third person.

In the course of psychotherapy with a psychologist at a university health center, Prosenjit Poddar mentioned that he had fantasies of killing Tatiana Tarasoff, a young woman with whom he was infatuated. The therapist also learned from Poddar's roommate that Poddar had obtained a gun. After the patient unilaterally terminated treatment, the psychologist consulted a psychiatrist at the center who had previously evaluated Poddar. The clinicians decided to contact the campus police and informed them that Poddar met criteria for involuntary hospitalization on the ground of dangerousness. The campus police detained and questioned Poddar, concluded that he was not dangerous, and released him when he promised to stay away from Tatiana. Two months later, Poddar stabbed Tatiana to death. Tatiana's parents brought suit against the University of California; they alleged that the university was negligent in not hospitalizing Poddar and in not safeguarding the public by warning the Tarasoff family.

The trial court dismissed the suit on the ground that there was no duty to warn. The case was appealed on two occasions, known as [Tarasoff I \(1974\)](#) and [Tarasoff II \(1976\)](#). In [Tarasoff I](#), the California Supreme Court found that the defendants were not liable for failing to confine Poddar, but a treating clinician bears a *duty to warn threatened persons of foreseeable danger arising from a patient's condition*. Damages were awarded to the Tarasoffs.

Concerned by the serious implications for confidentiality of this judgment, the American Psychiatric Association pressed the appellate court to reopen the case. In [Tarasoff II](#), the issue of failure to warn was debated. The court decided that there was a *duty to protect* rather than a duty to warn: *if there is a serious danger of violence, the clinician must take reasonable care to protect the foreseeable victim*. The court failed to define what constituted "reasonable care" and how dangerous the patient must be before precautions should be taken.

*Tarasoff* spawned other cases. In [McIntosh v. Milano \(1979\)](#), a New Jersey case, the *Tarasoff* standards were applied even though the patient had never actually been heard to utter threats against his victim. The psychiatrist was described as having a broadly based obligation to protect the welfare of the community. In [Lipari v. Sears, Roebuck & Co. \(1980\)](#), the Nebraska court held that the duty to protect applies even when the potential victim is unknown or unidentifiable. It was not made clear how the duty to protect unknown third persons should be discharged, other than by detaining the patient or informing the police. In [Peck v. The Counseling Service of Addison County, Inc. \(1985\)](#), the Vermont court held that a community mental health clinic was liable for the destruction of a barn by a fire-setting patient, thereby extending the *Tarasoff* doctrine to nonmedical therapists and to property damage as well as personal harm.

Conversely, in [Thompson v. County of Alameda \(1980\)](#) and [Brady v. Hopper \(1983\)](#), the California and Colorado courts reaffirmed the *Tarasoff* decision by holding that the duty to protect was precipitated only by a *specific threat to an identifiable third person*, a decision upheld subsequently in other states.

In [Bellah v. Greenson \(1978\)](#) and [Cole v. Taylor \(1981\)](#) the California and Iowa courts, respectively, held that (a) the *Tarasoff* duty to breach confidentiality and to warn others did not apply when the patient threatened suicide, and (b) a psychiatrist was not liable to pay damages to a patient who killed her husband when the psychiatrist had allegedly failed to control the patient or to warn the victim. However, in a Pennsylvania case, [Hopewell v. Adebimpe \(1981\)](#), a psychiatrist who informed a third party considered to be endangered was found liable for breach of confidentiality. In those states that have a statute concerning privilege in the doctor-patient relationship, the requirement to maintain confidentiality may take precedence over the *Tarasoff* doctrine. The clinician should ascertain the situation in his or her own state.

In [Jablonski v. United States \(1983\)](#) and [Davis v. Lhim \(1983\)](#), the findings of negligence were based on such dubious evidence that it has been suggested that a doctrine of *strict liability* was applied ([Smith, 1986](#); [Simon, 1987](#)). In other words, if a patient causes harm, the clinician must be culpable. Such broad interpretations of the *Tarasoff* doctrine appear to be founded on the myth that mental health clinicians are reliable predictors of violence, that they have the means of preventing violence in patients not under their direct control, and that "warnings" or other unspecified precautions (short of hospitalization) would protect the community. In fact, in [Lundgren v. Fultz \(1984\)](#), the Minnesota court regarded warnings as problematic because they add to the stigma of mental illness while doing little to protect the community.

What, then, should a clinician do if a patient threatens violence? The following steps are recommended. First, undertake and document a violence risk-resource analysis. If the risk is serious, take precautions to protect endangered parties. Second, if the patient is already hospitalized voluntarily, consider whether civil commitment is required. Take precautions against elopement, consider limiting visitation and leave, and do not discharge the patient unless you are convinced that the risk of violence has diminished. Ask for a consultation if you are in doubt. Third, if a potentially violent hospitalized patient elopes or fails to return from leave, seek consultation from a colleague and from the hospital's attorney. If the risk of harm is significant, inform the local police and the police department of the area where the patient lives, by telephone and certified letter. If certain people (e.g., family, friends, or acquaintances) could be in danger, warn them by telephone, through the police, and by certified letter. Fourth, if you warn a third party by telephone or letter, take care to divulge only as much as necessary to let them know they are at risk. For example:

In the course of my professional duties, it has come to my attention that John Smith has feelings of anger toward you and that he has made threats to harm you. On (date), John Smith eloped from this hospital and is at large in the community. I have informed the police at \_\_\_\_\_.

Fifth, if the patient is an outpatient, attempt to deal with the problem in therapy. Seek consultation from a colleague, and consult your attorney. If you regard the risk of violence as serious, consider hospitalizing the patient on a voluntary basis or after an emergency commitment. Consider whether close observation at home is preferable to hospitalization. Document the rationale for your final decision. Finally, if the risk is serious but hospitalization is not feasible, inform the police and the endangered third party by telephone and certified letter. Let the patient know that it is your professional and legal duty to do so. Try to enlist the patient's cooperation by allying yourself with his or her ego controls. For example, if possible, let the patient be present when you telephone the police and the third party and allow him or her to read the contents of the letters sent to the endangered third party and the police.

In summary, the *Tarasoff* decision has created many problems for clinicians and the courts. The doctrine assumes that clinicians are reliable predictors of violence and that the threshold of risk that should precipitate reasonable precautions is clear, and that reasonable precautions are available and effective. The courts in different states have varied from requiring that a specific threat have been made against a foreseeable victim, to applying the doctrine when no specific threat has been made and the victim was unknown to the patient, to including property damage as well as personal injury, to holding that the doctrine of confidentiality takes precedence and eliminates *Tarasoff* obligations. Thus, contrary trends are operating in different jurisdictions, extending, limiting, or preempting the *Tarasoff* duty. [Simon's \(1987\)](#) recommendations can be summarized as follows:

1. In outpatient cases, the courts have generally held that, unless there is a foreseeable victim, the clinician's control over the patient is insufficient to create a duty to protect. The danger of harm must be substantial (i.e., involve bodily harm or death), and the evidence must be specific (i.e., involve threats or acts of violence towards a third person) for the *Tarasoff* duty to apply.
2. In inpatient cases, if harm results after release or elopement, liability may be created by the duty to control, regardless of whether or not a victim was foreseeable. Continued dangerousness necessitates continued close observation.

If the situation is unclear in the home state, the clinician would be well advised to act as though the broadest interpretation of the doctrine applies. However, as [Simon \(1987\)](#) points out, one should not allow concern about *Tarasoff* liability to interfere with sound clinical practice. Instead, the clinician should make reasonable efforts to control potentially violent patients before resorting to warnings; and if warnings are required, they should be incorporated into treatment, whenever possible.

#### IMPLANTATION OF ILLUSORY MEMORIES OF ABUSE

In some cases, a third party, usually the parent of a patient, sues a psychotherapist for implanting in the patient illusory memories of sexual abuse. For example, in [Ramon v. Isabella](#), the father of a patient who had remembered sexual abuse during therapy brought suit against a psychotherapist on the grounds of slander, negligent and intentional infliction of emotional distress, and economic loss.

The question of the extent to which a therapist owes a duty of care to someone other than the patient continues to exercise jurists. These cases raise legal and scientific questions that are the subject of much debate. Is it possible for a person to remember childhood abuse after a gap during which he or she had no memory of it? Should the statute of limitations be tolled in such cases? What is the scientific validity of the concepts of repression and dissociation? Do they satisfy the criteria for scientific evidence cited in [Daubert v. Merrill Dow Pharmaceuticals, Inc?](#) Would the imposition of a duty of care to third parties prevent patients from obtaining the treatment they need? The most egregious cases involve therapists who persuade their allegedly abused patients to seek retribution and who subsequently commit the cardinal error of appearing as expert witnesses on behalf of their patients.

In a variant of this genre, the patient sues the therapist for failing to recognize that dissociative identity disorder is confabulatory or for accepting uncritically the patient's recovered memories of abuse. For an extended discussion of these issues, see elsewhere ([Partlett and Nurcombe, 1998](#); [Simon and Gutheil, 1997](#).)

#### SEXUAL EXPLOITATION AND THE TORT OF OUTRAGE

Expert testimony is not required to substantiate the impropriety of sexually molesting a patient. As an intentional tort, *res ipsa loquitur* ("the thing speaks for itself"), and the burden of proof shifts to the defendant. In [Roy v. Hartogs \(1976\)](#), the court held that public policy protected patients from such malicious abuses of authority, power, and fiduciary trust. The perpetrator faces criminal sanctions for rape or aggravated assault, as well as civil actions for malpractice or battery (unconsented touching). He or she may be insured against the costs of such an action. Liability for improper sexual relations may extend beyond the patient to others involved, such as the spouses of patients so harmed. Residual transference has persuaded courts that cessation of treatment does not open the door to a sexual relationship. Indeed, *negligent management of the transference* is often adopted as the theory behind the action for damages.

The tort of *outrage* refers to the intentional infliction of severe emotional distress on a patient, as a result of reckless and intolerable behavior. For example, in [Abraham v. Zaslav \(1970\)](#), damages were awarded to a woman who had sustained bruising and renal failure after exposure to "rage-reduction therapy," an intrusive intervention involving several hours of physical restraint, poking, tickling, and verbal insult. Patients have a right to expect that clinicians will adhere to established methods of treatment. If exceptional methods are proposed, full informed consent is required, supportive consultation from a colleague would be advisable, and



adequate reference to the scientific literature should be made.

### Teaching, Research, and Publication

The torts of *invasion of privacy*, *breach of confidentiality*, and *defamation* are the liability risks most commonly incurred by teachers, researchers, and writers. The doctrine of informed consent is discussed earlier in this chapter.

These torts stem from the ethical, contractual, and (in some jurisdictions) statutory obligation of the clinician not to disclose private information that should be kept within the doctor–patient relationship. In invasion of privacy, the plaintiff alleges that details from his or her private life have been publicized. This may occur, for example, if a clinician publishes an article in which the identity of the subject is insufficiently disguised. It has been held that a patient undergoing psychotherapy cannot give proper informed consent to the publication of a book concerning the treatment ( [Jane Doe v. Joan Doe and Peter Poe, 1977](#) ).

A patient has the right to bar nonessential onlookers when he or she is being examined or treated or when his or her case is discussed. The teacher should seek informed consent before exposing patients to case conferences or discussing cases for teaching purposes. The use of videotapes for teaching necessitates written informed consent, the continuation of which should be requested on a yearly basis.

### LIABILITY UNDER NEW COST-CONTAINMENT SYSTEMS

The current national preoccupation with cost containment by means of managed care and capitation puts the clinician at risk, particularly in the care of suicidal or dangerous patients. In [Wickliffe v. State of California \(1986\)](#), a Medi-Cal reviewer allowed only 4 extra days of inpatient treatment after an arterial graft, rather than the 8 days requested by a vascular surgeon. The patient was duly discharged after 4 days, and complications cost her the leg. The patient sued the physician, the hospital, and the state of California. The court determined that the state, through Medi-Cal, owed the patient a duty of care in that it was bound by statute to evaluate the medical necessity of length of stay and level of care. However, in this case, no breach of duty was found. The surgeon was found liable for having acceded without appeal to the Medi-Cal determination. Conversely, in [Wilson v. Blue Cross of California \(1990\)](#), a suicidal patient was prematurely discharged from hospital, over his psychiatrist's protest. After the patient's death, an appeal court found the insurer liable in that the managed-care firm contracted by the insurer to conduct a utilization review had made a "medically insensitive" decision. However, in this case, the patient's insurance contract had contained no provision for managed care, and vexatious questions remain unanswered. Managed-care reviewers often preface their denials of further hospitalization by saying that the physician or hospital is at liberty to continue to treat the patient gratuitously; and, in many cases, the patient should be kept safe pending appeal. Physicians who receive financial incentives for diverting patients from specialized care should reveal this to their patients. Any economic constraints that could modify a physician's medical judgment should be disclosed.

The failure to regulate managed-care regulators is certain to generate future liability actions that will help to clarify some of the questions. However, managed care and capitation will continue. Physicians and hospitals should define their standards and should not be pressed to sink below them.

### Assessment of Violence and Suicide Risk

It is generally accepted that clinicians have little expertise in the long-term prediction of violence or suicide ( [Monahan, 1981](#) ). However, most of the research in this area comes from prison populations or from the long-term follow-up of involuntary psychiatric patients. There has been no research concerning the prediction of imminent risk, and such research will never be done. Faced with an uncontrollable, potentially self-destructive or assaultive patient in an emergency room, no clinician would be willing to assign the subject randomly to a no-treatment control group. Thus, although the accuracy of clinical predictions of imminent dangerousness is unknown, such predictions must and will be made. The following risk–resource analyses present a method of taking into account the factors that increase or mitigate risk.

#### VIOLENCE

A risk–resource analysis should be conducted whenever a patient presents a risk of violence. Such an assessment is most likely to be required in the following circumstances: when the risk of violence is raised during outpatient evaluation or treatment; when a potentially violent patient presents for admission to hospital; and when the leave or discharge from hospital of a potentially violent patient is being considered, or when such a patient elopes.

The factors to be considered can be grouped into the following areas: (a) demographic factors, (b) the violent threat, (c) past history, (d) psychological factors, (e) the social environment, and (f) the therapeutic alliance. Do not merely sum the positive and negative items; their weighting and combination differ in different cases.

#### Demographic Factors

*Late adolescent, male patients from disadvantaged ethnic groups with a cultural tradition of masculine defensiveness are at greater risk of violence.*

#### Violent Threat

Has the patient *directly expressed a violent threat* or threats toward another, by word or message? Does the potential victim have a *provocative relationship* with the patient that could provoke hostility? Does the patient have a *plan* for harming the victim, and does he or she have *access to lethal weapons*?

#### Past History

Is there a *past history of violent threats or actions*? Does the patient have a *history of being victimized* in early life by child abuse? Has the patient a *history of serious head injury, epilepsy, or neurologic impairment*? Does the patient have a *history of alcohol or drug abuse*?

#### Psychological Factors

Is the patient prone to cope with anxiety or hostility by *externalizing* or *projecting* it in the form of *impulsive, explosive actions, suspicious vigilance, or frank persecutory delusions*? Is there evidence of *command hallucinations* that instruct the patient to take violent action or that *threaten violence* to the patient or his or her family? Are the patient's *inner controls* against violent actions subjectively or objectively reduced? Is there a *strong inner urge to be violent*? Is the patient's *intelligence below average*? Is there evidence of *impairment of the sensorium*? Is the patient subject to *alcohol or substance abuse*?

#### Social Environment

Are the patient's *family or caretakers able and willing to control* the patient? Is there evidence of family psychopathology in the form of *rejection, neglect, physical or sexual abuse, or family violence*? Is *parental mental or physical health* impaired?

#### Therapeutic Alliance

Has the patient *lost or terminated a therapeutic relationship*? Is he or she *competent and motivated* to enter into one?

#### Resources

Factors that may protect against violence or enhance self-control are as follows: *younger, female, white, religious, middle-class* patients are at less risk. If there has been a threat of violence, a *threat without plan or identified victim*, in a patient *without access to lethal means*, is of relatively lower concern, particularly if the patient has *obsessional traits, low subjective urge, and above average intelligence*. A *secure family* without major psychopathology or mental or physical ill health is protective, as is a *positive relationship with a therapist*; or the *motivation and competence* to enter into one.

### Hypothetical High-Risk Case

The patient is an intoxicated 17-year-old, male, Hispanic youth from an impoverished family living in an urban ghetto. He was brought to the emergency room by police because he was acting belligerently in a local bar. During the interview, he threatens to kill his allegedly unfaithful fiancée and her lover by stabbing them to death. He has a history of several episodes of juvenile violence and is known to be a user of alcohol and street drugs. He has access to knives and other weapons. At the interview, he is highly emotionally aroused, threatening, hostile, and suspicious. He says that he is directed by an inner voice to take vengeance for the insult to his honor. His family is chaotic and afraid of him. He has no therapist and, at the interview, no desire to seek help.

### SUICIDE

A suicide risk–resource analysis is required when the clinician must decide whether the risk of suicide is *imminent* and *serious*. This decision is most often required in the following situations: when the possibility of suicide is raised in the course of ambulatory or inpatient treatment, when a new patient presents for voluntary or involuntary hospitalization, or when leave or the discharge of a potentially suicidal patient is being considered. In these circumstances, it is important for the clinician to record the pros and cons on which the clinical decision is based.

The factors to be considered incorporate 25 items in six sections. These items cannot be equally weighted because their relative valence and combination differ according to the particular circumstances and characteristics of each case. As with a violence risk–resource analysis, one should not merely sum the risks and resources, but rather consider their weight and combination in each case. The factors to be considered can be classified under six headings: (a) demographic factors, (b) the suicide attempt, (c) physical factors, (d) psychological factors, (e) the social environment, and (f) the therapeutic alliance.

### DEMOGRAPHIC FACTORS

*Older white men* are at greater risk.

### Suicide Attempt

Pay close attention to the suicide attempt, if one has occurred. *Lethal means* involve hanging, gassing, jumping from a height, drowning, knife or gunshot wound, using a motor vehicle for suicidal purposes, self-poisoning, major overdose, or other concerted attempts at self-destruction. Remember that, to a child who is ignorant of pharmacology, 10 aspirins may represent a lethal dose.

Greater risk attaches to suicide attempts that were *planned* and *concealed*, although *impulsiveness* (e.g., drinking and driving wildly after an upset) can also be very dangerous. The intent of the patient should be explored from suicide notes, prior conversations with family and friends, and the patient's recollection of the attempt. Did the patient truly *wish to die, sacrifice himself or herself, expunge shame, exit permanently from an intolerable existence, find peace, or be reunited with a lost loved one*? Was the suicide attempt a response to *command hallucinations*? Conversely, did the suicide attempt represent a communication of the *need for help* or a *desire to punish someone*? Is the patient *able to grasp the finality of death*? Does he or she fantasize that *resurrection* is possible?

### Past History

A history of a *previous attempt or attempts* adds to the risk of future suicide. The *means and intent of previous attempts* may also predict the seriousness of future attempts. Some patients have given up on the future as a result of *chronic illness or handicap*. *Alcohol and drug abuse* increase the risk potential of other factors because the intoxicated adolescent may take desperate risks or express suicidal impulses that would otherwise be controlled. Is the patient *intoxicated* at the time of the examination? Is he or she *likely to resort to drugs or alcohol* if you do not hospitalize him or her?

### Psychological Factors

Does the mental status examination reveal or confirm the presence of *depressive affect, despair, hopelessness*, or the *vegetative signs* associated with *melancholia*? Is there evidence of *psychosis* with mental disorganization, abnormal perception, or delusional thinking? Can you detect evidence of *command hallucinations*, which instruct the patient to kill or injure himself or herself or to take serious risks (e.g., to fly from the roof of a building)? Is there evidence of *mental confusion, obtundation, or delirium* suggestive of an organic confusion or acute psychotic condition? Does the patient's history indicate *poor internal controls* with impulsiveness or explosive behavior when under stress? Is the patient preoccupied with *suicidal ideation* or with the *desirability of death*?

### Social Environment

The resources and deficiencies of the family or caretakers are very relevant to the determination of suicide risk. Are the family members able to provide a *safe environment*? Are the parents able to cooperate with each other? Are they willing or competent to remove, or secure, all potentially lethal agents of suicide (e.g., prescription drugs, poisons, weapons)? Are they prepared or able to watch the patient closely and to be emotionally available to him or her in times of stress? Conversely, is there evidence of *parental rejection, neglect, physical or sexual abuse, mental illness, severe personality disorder, substance abuse, or physical illness or handicap* of a severity that would render the caretakers unable to provide adequate security and emotional sustenance to an acutely disturbed child?

Has there been a recent *loss or the severance of an important relationship*, such as the death of a close relative or beloved family pet, loss of contact with a noncustodial parent, or the departure of a close older sibling or special friend? Such an event may represent the "last straw" to an isolated, troubled child. One should pay special attention to *peer relations*. Particularly vulnerable is the isolated, alienated, persecuted, or rejected youngster or the patient who has withdrawn from contact with peers. *Has a love relationship been threatened or severed*? Does the patient feel intolerably *guilty* or *ashamed* before his or her family or peers, as a result of some action or dereliction? *Shame toward the family* is a particularly powerful emotion in Asian patients.

Is there a *family history of suicide*? A positive family history may reflect a genetically transmitted psychiatric disorder that increases suicide risk or a family tradition of committing or attempting suicide in stressful circumstances. Suicidal parents may unwittingly act as *suicide models*.

### Therapeutic Alliance

It is crucial to determine whether the patient has, or has lost, an adequate *therapeutic relationship*. Is the patient capable of forming an alliance? Do you believe the patient if he or she promises to cooperate? Has he or she been reliable in the past? Is he or she prepared to sign a "suicide contract"? Although such contracts have no legal status, they are a useful clinical index of the patient's cooperativeness.

### Resources

To some extent, the reverse of the risk factors can operate as mitigating factors. For example, a suicide attempt associated with the *desire to communicate a need for help* indicates that the patient is still potentially available to other people. A *competent family*, free of serious mental, physical, or personality disorder, is the most powerful protective factor. A *network of caring friends* who can be mobilized is also protective. The *capacity for internal control*, accentuated for example in obsessional patients, may impede direct suicidal action and may be activated if the patient has, or wants, a therapeutic relationship. Finally, an *existing adequate therapeutic alliance* and the *competence or motivation to seek one* are mitigating factors.

### Circumstances of Extreme Risk

The following stereotype describes an extreme case. The patient is a 17-year-old white male adolescent who was discovered making his second suicide attempt by hanging, while intoxicated, in a remote part of the woods near his home. His family is chaotic. The father has psychosis and alcoholism, and his mother is chronically physically ill. One sibling has died by suicide and one by accidental gunshot wound. The patient's suicide was planned, and he intended to do away with himself. He has no friends, is persecuted at school, and was recently greatly upset by the death of a beloved grandmother. Mental status examination reveals severe depression with agitation, despair, and vegetative signs. He is preoccupied with the idea of dying and joining his grandmother. At times, he hears the voice of his grandmother



begging him to join her. He has had no recent mental health contact, and rejects the offer of therapy.

### *Circumstances of Less Risk*

The patient is a 14-year-old girl who swallowed 10 aspirin tablets in the bathroom at home after an argument with her mother about permission to go out with a boyfriend on a week night. She told her mother about the overdose an hour after it occurred, and the mother brought her to the emergency room at once. The parents have been separated for 3 years, but both of them remain close to the patient, and neither suffers from serious mental, personality, or substance use disorder. The parents and child are in good physical health. The patient reveals no psychotic or melancholic symptoms. She says that she did not really want to die, but she was angry with her mother for not listening to her and for restricting her too much. She accepts the need for therapy, promises not to attempt suicide without calling you, and agrees to return for an outpatient appointment on the following day.

## CONCLUSIONS

Clinical practice is fraught with error, and error can have serious consequences. Confronted by the catalogue of litigation in this chapter, even the insouciant may quail. However, defensive psychiatry is no answer, because overly timid treatment can also put the clinician at risk of negligent malpractice. What, then, is the best way to avoid litigation?

The best precaution is to practice careful medicine while fully apprising the patient and family of the diagnosis, the treatment plan, and the progress of therapy. Many malpractice suits arise from a neglect of this simple principle. Hospitals provide many opportunities for failed communication, particularly when the attending physician delegates to other members of the team the responsibility for keeping parents informed of their children's progress. A good therapeutic alliance is the key to good medicine and the avoidance of lawsuits. Used for this purpose, informed consent can be transformed from an empty legalism into the foundation of a true collaboration.

*If it wasn't written down, it didn't happen.* The clinician should keep a good record of the rationale for treatment. Progress notes should be timely, regular, dated, and signed. Whenever a course of action is problematic, a risk-to-benefit analysis should be undertaken and documented, and a consultation obtained. If you disagree with the diagnosis, investigations, or treatment plan, in the record, note that you disagree and insert your amended diagnosis and plan. If you merely sign off on a trainee's notes, you could be concurring with erroneous observations, diagnosis, or treatment. One of the most common errors is to raise a diagnostic question but not follow through by investigating it or, having ordered investigations, to lose sight of the results.

Note any discrepancies between the nurses', the therapist's, and the physician's progress notes. If there is such a discrepancy, try to account for it. Do not allow a nursing progress note concerning suicide risk to go unremarked.

If you detect an error in your notes, do not erase it. Draw a line through the error, write "error," and date and sign your correction in the margin. Do not criticize or argue with other professionals or agencies in your notes, and avoid gratuitous or extravagant commentary (e.g., "This child has had an appalling home life").

Before a particular treatment is commenced, be careful to check that the patient harbors no conditions and is taking no drugs that would contraindicate it, and inquire about previous allergic or idiosyncratic reactions to drugs. Avoid polypharmacy. Monitor the progress of medication regularly. In patients taking long-term medication, "drug holidays" should be considered. Unless there are good reasons for departing from them, follow recommended guidelines for dosage and administration. Avoid prescribing medication "as needed" and automatic refills, and be careful of telephone requests for repeat prescriptions. If the suicide risk is serious, only small amounts of a potentially lethal drug should be prescribed. Hospitals should check that potentially suicidal patients are not cheating, concealing, and hoarding medication.

When a patient is admitted to the hospital, the degree of risk of suicide or violence should be assessed, and each degree of risk should be linked to a set of nursing precautions that are automatically activated when the clinician indicates the degree of risk. Every attempt should be made to obtain past records and to scan them for risk factors.

If you are unavailable in case of emergency during treatment, the name of a fully informed substitute physician should be provided to the patient. Do not terminate treatment unilaterally without preparing the patient, giving adequate notice, and providing him or her with the names of other clinicians or agencies who could help.

In outpatient practice, the duty to protect is precipitated when the patient makes a specific threat to harm a specified victim. When possible, incorporate the protection in therapy. Psychotherapy, hospitalization, and medication may be both more protective and more therapeutic than warning the foreseeable victim or alerting the police.

Do not record psychodynamic speculations in the clinical record; those unconscious incestuous strivings may come back to haunt you. You may consider keeping your psychotherapy *process notes* separate from the official record of therapy *progress notes*.

Be vigilant to avoid unauthorized disclosure of confidential information to external agencies, do not gossip about patients, and do not publish articles about patients without adequately disguising their identity. Obtain the parents' consent before releasing reports to external agencies. Blanket consent forms, obtained during the rush of admission, may not hold water, legally.

In conclusion, remember that the law has no wish to penalize physicians for honest errors. Its purpose is to protect patients from being harmed by reckless, careless, or incompetent clinical practice, and if they have been so harmed, to compensate them for injury. In most successful suits for negligent malpractice, the errors are glaring. Attention to the safeguards described in this chapter will protect clinicians from litigation while allowing them to practice nondefensive psychiatry.

## SELECTED READINGS

This chapter relies heavily on [Smith's 1986 \*Medical Malpractice: Psychiatric Care\*](#), which elaborates the legal principles of malpractice litigation and gives numerous case precedents, and on [Simon's 1987 \*Clinical Psychiatry and the Law\*](#), which contains a detailed examination of malpractice liability from the clinician's viewpoint. [Gutheil's 1980](#) article, "Paranoia and Progress Notes: A Guide to Forensically Informed Psychiatric Recordkeeping," is required reading for all psychiatric residents. Gutheil and Appelbaum's 2000 *Clinical Handbook of Psychiatry and the Law* contains an elegant analysis of the doctrine of informed consent within its chapter on malpractice.

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# 113 LEGAL ISSUES IN PROFESSIONAL LIABILITY

Angela R. Holder, LL.M.

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[Children and Adolescents and Consent to Treatment](#)  
[Consent in Emergencies](#)  
[Special Consent Statutes](#)  
[General Consent Statutes](#)  
[Emancipation](#)  
[The Mature Minor Rule](#)  
[Refusal of Treatment](#)  
[Freedom of Religion and Refusal of Treatment](#)  
[The Minor in the Mental Health System](#)  
[The Minor in the Outpatient Psychiatric System](#)  
[Commitment of the Minor Patient](#)  
[The Child Psychiatrist in Court](#)  
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Although child psychiatrists still are sued less frequently than physicians in most other specialties, in the past several years there have been an unusual number of very compelling suits against psychiatrists who were treating children.

The most frequent source of legal suits against psychiatrists (adult and child) remains drug reactions such as tardive dyskinesia ( 1). The issues in these cases tend to revolve around two questions: (a) Was the drug properly prescribed? In these cases, this usually means that the issue is whether the patient's illness was sufficiently severe to justify administering a drug with such serious side effects; and (b) Did the patient or the family understand the risks and consent to them ( 2)?

A second issue involving the prescription of medication arises in the context of a diagnosis of attention deficit/hyperactivity disorder (ADHD) and the use of methylphenidate (Ritalin) or other drugs to treat it. Although there have been reports in the press or a large number of preschoolers being treated for ADHD and professional differences of opinion about how the drugs should be used, somewhat surprisingly, there have been no malpractice cases arising from prescription of the drug. A very clear problem, however, could arise if the child psychiatrist (or other physician) assumed that the symptoms reported by parents or teachers were ADHD and did not investigate properly for other causes of the behavior, including brain tumors or other medical and nonmedical causes. If a medical condition is not diagnosed because a facile conclusion led to a finding of ADHD, liability for misdiagnosis would be hard to escape.

In this area of child psychiatry, the issue also arises of the child's or parent's right to refuse these drugs. In many instances, the child's diagnosis results from his or her disruptive behavior in the classroom and the psychiatrist sees the child as a referral from the school, not because the parent thinks the child needs to be treated. Even if the parent accepts the diagnosis (many do not), there may be many reasons, some of them quite valid, for why these parents refuse to permit their child to take these medications. Some parents, such as Christian Scientists, may reject any medications; others may question the long-term effects of giving their child these particular medications. Although private schools have a great deal more control over the conditions of admission and retention of their students than do public schools, it is unlikely that a private school could require medication if the parent found any licensed physician who agreed that the medication was not in the child's best interests. It is absolutely certain that no public school could require it ( 3). On the other hand, if there is a school nurse or other system for giving students any medications during the school day, a school nurse or principal cannot refuse to give Ritalin or similar drugs because they disapprove ( 4).

Physicians of all specialties, among others, are mandated to report child abuse to state authorities. In most cases, even if the diagnosis is incorrect, as long as it was made in good faith, the physician is statutorily protected from suit by the child or the family. In the past few years, however, courts in several states have held that diagnoses of child abuse must be made with the skill, care, and knowledge that would be required in any other diagnosis. If a child is negligently diagnosed as a victim of abuse and the parents are prosecuted, the child psychiatrist may be held to be liable to the parents. Courts are increasingly willing to find that the therapist owes a duty of care to the parent (not just to the child patient) when an accusation or report is made and that the techniques used to make the diagnosis of abuse must be those generally accepted in the profession ( 5).

Another area in which numerous malpractice suits against psychiatrists have succeeded is liability claims based on the patient's violent behavior, directed toward himself or others. In adult psychiatry, there is a clear duty to warn a person whom the psychiatrist believes may be in danger, as well as a duty to take reasonable steps to prevent the violence by committing the patient to a hospital or taking other such action ( 6).

In dealing with children as patients, a psychiatrist has every right to conclude that the standard of disclosure to parents of a possible threat by the child is very low. Virtually any concern about suicidality or any danger to others (particularly to the parents themselves) does not have to be proven credible before the psychiatrist discloses it to the child's parent or custodian ( 7). In fact, failure to disclose to the parent the possibility that a minor might be suicidal is sufficient, after the young person commits suicide, to impose liability on the psychiatrist ( 8).

In one recent and horrifying case, a supervising psychoanalyst knew that a child psychiatry resident at least had serious thoughts of being a pedophile. Moreover, the analyst suspected that the resident had probably engaged in such acts at some time. He did not report this to anyone at the hospital where the resident was in training. As might have been foreseen, the resident sexually molested a young boy in his care. The patient sued not only the hospital for the resident's acts but sued the supervisor as well. The analyst defended himself on grounds of confidentiality, but the court held that he had had a duty to speak up to keep the resident away from children ( 9). Physician-patient (or psychiatrist-patient) privilege never applies to situations with a potential for child abuse or confession of recent abuse of a child ( 10).

In the absence of any threats to self or others, however, what an adolescent patient says in confidence to a mental health professional is usually entitled to the same degree of confidentiality that an adult patient would expect ( 11).

## CHILDREN AND ADOLESCENTS AND CONSENT TO TREATMENT

At common law, a minor child, which meant anyone younger than 21, not 18 years, was effectively a chattel of his or her father. A father had the right to sue a physician who treated a child without his permission, even if the treatment was appropriate and had a good result, because such intervention transgressed on the father's right to control the child.

This still is the rule in a nonacute situation involving a young child. If for example, a 3-year-old is visiting his grandmother and she takes him to a plastic surgeon for repair of a birthmark his parents had decided to leave alone, the plastic surgeon would be at substantial risk if he operated without consent of the parents ( 12).

On the other hand, since at least the early 1950s, adolescents in our society have become increasingly independent, and courts have recognized and accepted this independence. It has been at least 40 years since a parent successfully sued a physician in this country for treating a minor 15 years of age or older without parental consent ( 13). It should be remembered, however, that, except in emergency situations, a parent is not liable for the cost of medical care for a minor unless the parent consented to having that care provided ( 14).

### Consent in Emergencies

In an emergency, a child of any age may be treated without parental consent if the parents cannot be located immediately ( 15). For this purpose, an "emergency" is any condition requiring prompt treatment, and not only a condition that may lead to the child's death or disability if untreated. For example, a small child who has an

earache or a cut foot and is brought to an emergency room by a teenage baby sitter would constitute an “emergency” for this purpose.

### Special Consent Statutes

Because physicians were concerned about treating adolescents without parental consent, beginning in the early 1950s state legislatures began enacting statutes permitting teenagers to consent to their own treatment for venereal disease (VD). It was perfectly evident to legislators as well as to physicians that, if parental consent to treatment were required, teenagers simply would not go for treatment, and the epidemic of VD in this population would get worse. All states have enacted VD treatment statutes (16); most, in fact, forbid the physician to tell the parent that the young person has been treated, and some specify that the parents may not be billed for the care (on the theory that, if the parent receives a bill, he or she will find out the purpose of the visits) (17). The next public health concern involving adolescents was drug and alcohol abuse. Most, if not all, states now have specific statutes covering consent to treatment for these problems. These disease-specific statutes do not have an age limit—a 9-year-old with a drinking problem is given the same right to confidential treatment as a 17-year-old with the same difficulty.

### General Consent Statutes

Many states also enacted statutes that provide that a minor of a given age—usually 16 years, but some statutes provide for consent as early as 14 years—may consent to any and all medical treatment as if she or he were an adult. Some of these statutes specifically prohibit consent to abortion or organ donation without parental consent, but a physician in a state with such a statute may, in keeping with any limitations the statute imposes, treat a teenager without discussing the situation with parents (18).

### Emancipation

For at least 200 years, courts in the Anglo-American system have recognized the concept of an “emancipated minor.” This means one who is living on his or her own (in the earlier days of the law, it was on *his* own), is self-supporting, and is not subject to parental control. The old concepts of emancipation included minors in the military (of which there were numerous when the age of majority was 21 years) (19) or married minors. The concept has evolved nowadays to include college students younger than 18 years of age, even when parents are completely responsible for paying the bills, or unmarried minor mothers (20), and in some states a pregnant minor is considered completely emancipated. A runaway also is considered an emancipated minor, no matter how young he or she may be.

### The Mature Minor Rule

In addition to the exceptions to requirements of parental consent discussed previously, courts in this country have bowed to modern reality, and decisions all over the country apply what has come to be known as the *mature minor rule* (21). The legal principle now applied is that if a young person (of 14 or 15 years of age) understands the nature of a proposed treatment and its risks and can give the same degree of informed consent as an adult patient, and if the treatment does not involve very serious risks, the young person may give a valid consent. The age and maturity of the patient must be considered, but the nature of the illness and the risks of therapy also are relevant. A 14-year-old might be perfectly capable of consenting to treatment of a sprained ankle; no oncologist would consider treating the same patient for leukemia without his or her parents' knowledge.

In almost all cases, the issue of treatment without parental knowledge arises in the context of ambulatory care. Most hospitals refuse to admit minors for nonemergency treatment unless the parent, who almost always is the person with insurance (on which children are carried as dependents), signs financial responsibility and insurance forms.

Thus, as we can see, in terms of consent to general medical care, courts are granting much more autonomy to increasingly younger adolescents. Preadolescents, however, do not have a right to consent (22).

## REFUSAL OF TREATMENT

If a minor has the right to consent to treatment, it is likely that he or she has the right to refuse treatment. It is therefore always a good idea, if dealing with an adolescent of 14 years or older, to obtain the patient's consent, as well as that of his or her parents, to a procedure. In any purely elective situation (such as cosmetic surgery) where a parent wishes an adolescent to be treated but the patient refuses, a prudent physician would decline to treat the patient. In any situation where a minor can be treated at his or her own request and without knowledge of the parents, it is likely that the minor has the right to refuse, at least if the problem is not life threatening.

Because a younger child does not have the right to consent, he or she also does not have the right to refuse.

It is clear that a pregnant teenager has the right to refuse an abortion that her parents wish her to have (23). In situations such as requests for court orders for treatment of a minor over parental religious objection, judges usually consult adolescent patients about their views on the matter before treatment is ordered, particularly if the patient's lack of cooperation could affect the outcome of the intervention (24). These cases, however, usually do not involve life-threatening problems. Only one case can be located in which a court allowed an adolescent to refuse life-saving treatment (25). A 16-year-old girl, who was the daughter of Jehovah's Witnesses, refused on religious grounds to have blood transfusions for leukemia. Her physicians and the hospital in which she was a patient asked the court for guidance on the legality of a minor's refusal of life-saving treatment. The judge ruled, and his ruling was upheld on appeal, that once he had determined that her religious views were her own and that she was not being coerced by her parents, her knowledgeable refusal should be respected to the same extent that it would be if she were an adult. It is unlikely that any judge would give such credence to the views of a minor younger than 16 years of age; it also is not at all clear that courts in other states will follow the same reasoning and allow any minor to decide to die where successful treatment of the problem is available.

### Freedom of Religion and Refusal of Treatment

A parent's failure to provide “adequate” medical care for a child is a criminal offense under all states' child neglect and abuse laws (26). Court intervention is an option in circumstances in which physicians and hospital administrators wish to invoke it. Until recently, court orders to treat were virtually automatic, and in almost all cases, that is still the case. Where, however, parental objection to treatment is based on a risk assessment of complex therapy or newly developed surgical intervention, courts appear to be willing to consider parental reservations. For example, if a court order for chemotherapy is sought by physicians and the parents' reason for refusing the treatment is that their child is, by all accounts, terminally ill and they wish to let her “die in peace,” it is unlikely that a court would order administration of the drugs. When a treating physician asks for court-ordered treatment, orders still are virtually automatic, but increasingly, when requests are initiated by welfare agencies, police, or school personnel under circumstances in which the physician taking care of the child agrees with the parents' decision, parents do seem universally to prevail. For example, in the *Babies Doe* cases involving handicapped newborns, when the physicians agreed with the parents that surgery on the infants was undesirable and state or federal governmental entities tried to intervene to ask the courts to order treatment, in every instance the parents and treating physicians prevailed (27).

Although it now is clear that a competent adult has the right to refuse life-saving treatment for himself or herself for religious or other reasons, in all states parents whose refusal to allow necessary treatment of their child is based on religious conviction still are found to be “neglectful,” even though their right to religious freedom is protected by the First Amendment (28).

All courts have held that a Jehovah's Witness whose child's condition requires blood transfusions has no right to forbid them on the ground of religious conviction (29).

## THE MINOR IN THE MENTAL HEALTH SYSTEM

### The Minor in the Outpatient Psychiatric System

Because minor treatment statutes and the mature minor rule apply to all forms of care provided by physicians, one must assume, although there are no cases on the point, that the same legal rules apply to an adolescent who goes to see a psychiatrist as apply to a visit to a gynecologist. If the psychiatrist wishes to treat the teenager, it is difficult to see how a parent could object. Because private psychotherapy is, however, extremely expensive, the issue is most likely to be raised when



the teenager goes to a mental health center or community counseling service. Where the community facility is one in which a qualified psychiatrist is on staff and other mental health professionals, such as psychologists, nurse clinicians, or psychiatric social workers, provide therapy as a team effort with physicians, there is little question that the minor has the right to seek treatment. There are, however, many so-called mental health facilities where no physicians are involved or available. In particular, many drug treatment centers provide therapists who are drug addicts themselves, and no medical backup is available. This may be a viable method of providing drug treatment or other specialized therapies, but it is not at all clear that the legal right of a minor to seek medical care necessarily applies to such facilities.

The intent of legislatures and courts who broadened the adolescent's right to seek medical care was to get young people to physicians, not to well-meaning but nonmedical counselors whose training may be questionable.

The issue of refusal of outpatient mental health treatment that the parent wishes the adolescent to have but the adolescent does not want is a legally fascinating one, but for very practical reasons not a real issue. If a parent takes a young person to a psychiatrist and the patient does not want to cooperate, all he or she has to do is refuse to talk to the psychiatrist. It is physically possible to hold someone down and administer medical care if one must do so; it is not possible to engage in psychotherapy with someone who refuses to talk.

## COMMITMENT OF THE MINOR PATIENT

Adult mental patients have certain legal protections against unjustified or malicious commitments to mental hospitals that minors do not necessarily have. An adult may voluntarily go to a mental hospital and may also leave it if he or she wishes to, unless the hospital concludes that the patient is so ill that involuntary commitment is necessary. In that case, the patient has a right to a judicial hearing. Commitment as an involuntary patient, if one is an adult, may occur only after a judicial hearing at which one is, among other things, entitled to counsel. The standard of commitment of an adult is that he or she is dangerous to self or others or is gravely mentally disabled, meaning that the person is too mentally ill to provide himself or herself with the basic necessities of life ( 30).

Minors, however, fall into an entirely different category. Many states allow "voluntary" commitment of minors by their parents, which means that although the young person cannot leave the hospital, he or she is considered a "voluntary" patient and has no right to a hearing at which a judge can rule on the merits of the commitment. If all families were healthy and happy, that might be a reasonable solution to the problem, but in any number of instances, minors have been "dumped" into mental hospitals by their parent, when the parent is probably the one who needed treatment ( 31).

Nowadays it seems that frightened parents, probably in good faith in most instances, are having teenagers in record numbers admitted to for-profit drug and alcohol hospitals and treatment centers in instances in which there may not be very clear indications that inpatient treatment is required and, if recent media reports are correct, where the quality of care and the training of therapists is open to very serious question ( 32). At least two states' courts have held that, because these facilities are not considered "mental hospitals," a minor admitted by parents has none of the due process rights or access to the legal system that would accrue to him or her if admitted to a psychiatric hospital ( 33).

Beginning in the 1960s, after a fair number of young people literally were committed to mental hospitals by parents who were upset with antiwar protests, long hair, and general hippiness, most states did enact statutes providing for judicial intervention in the commitment process when minors were patients. In other states, courts ruled that these minor patients had the right to some constitutional protections. In North Carolina, for example, it was held in a 1975 case that a 15-year-old boy who had been committed by his mother had a right to a hearing within 72 hours of admission, although the young person still could be signed in by a parent in an emergency ( 34). This decision was fairly typical of those in most other states' courts during this period.

Many of the states' new statutes provided that a child younger than 13 or 14 years of age could be admitted by parents without judicial intervention. Most, however, specified that minors over that age might continue to be admitted by their parents, but once in the hospital, the patients had a right to prompt hearings with the assistance of counsel if they wished to object to their admissions. Many statutes also provided that teenagers could admit themselves to mental hospitals as voluntary patients. If they did so, however, they had the same right as adult patients to discharge themselves unless the hospitals were willing to ask courts for orders of involuntary commitment ( 35).

On June 20, 1979, however, the Supreme Court ruled ( 36) that if a state legislature did not choose to enact procedural rights for minor mental patients, minors' federal constitutional rights were not violated if their parents admitted them to a mental hospital over their objection.

Thus, the rights of children and adolescents committed to mental hospitals are, at this time, exclusively derived from state law, and no federal constitutional protections are given them.

## THE CHILD PSYCHIATRIST IN COURT

A physician may appear in court as a witness for one party or the other, as a treating physician, or as an expert witness. A treating physician may be subpoenaed to testify about facts within his or her knowledge relevant to the case; however, expert opinion testimony may never be compelled from an unwilling witness ( 37). The expert, who may never have seen the patient but who has reviewed the medical records, usually testifies about his or her *opinion* of the situation—views on child placement, whether medical care met the requisite standard or was negligent, or the like.

In an increasing number of cases involving children, judges feel the need of objective expert medical advice and may use their discretion to appoint a physician to be an independent witness. The purpose of such an appointment may be to examine a party to the action or the children in a family, or, in most cases, to serve as a true advisor to the judge, providing assistance based on information already in evidence in the court and giving the judge professional analyses of that evidence. In any case in which a physician is a court-appointed witness asked to examine a party, his or her testimony about that examination is not covered by physician-patient privilege because he or she is not a "treating physician" for purposes of privilege statutes. His or her role as a court-appointed expert is to investigate, as "an arm of the court," those matters on which professional expertise is requested, and not to represent the interests of any party ( 38). If one is the treating physician of either parent or child involved in a custody case, physician-patient privilege is considered much less important than are the best interests of the child, so the physician usually will be obliged to testify. However, disclosure of confidential psychiatric information without being ordered to do so by a court may subject the psychiatrist to a successful malpractice suit. No information should be disclosed, particularly to the adverse party or his or her attorney, without consent of the patient or the patient's parent who has asked the psychiatrist to treat the child. If testimony is needed, a judge can order it to be given, and until that time arrives, no information should be disclosed without the patient's consent ( 39).

Another sort of issue is presented in some cases involving subtle forms of child abuse. Children's protective agencies usually are not staffed by particularly well educated workers, and even if they are, in all jurisdictions they are overworked and understaffed. Abuse that is primarily emotional in nature or, although physical, does not leave clear marks, fractures, or other undeniable signs on the child may, when reported, not impress the caseworker enough to result in any action. One sort of situation in which most child protective services workers are uncomfortable is in a case of Munchausen's syndrome by proxy, in which a parent is deliberately making a child ill in some way. (Actually, this is the wrong name for this situation. Munchausen's syndrome refers to a factitious illness; these children may be made critically ill.) Typically, the child with mysterious symptoms is admitted to the hospital, and it eventually becomes obvious to the caregivers that the child becomes worse when one or both parents are present and recovers when the parent or parents go away. Close observation short of witnessing the act may convince everyone at the hospital that the parent is harming the child, but the child protective services worker says "prove it" and refuses to go forward with court proceedings in the absence of objective evidence. Obtaining such evidence may, in rare cases, demand a hidden video camera, taping the child's room and all activities therein.

Although such taping raises clear-cut moral dilemmas about invasion of privacy, it is, when used as a last resort, legal. First, no such spying operation on a parent should be carried out in a hospital unless the suspected activity is or may be causing serious harm to the child. (If the child has been admitted to the hospital in the first place, however, the child usually is sufficiently ill that there is no question about the gravity of the situation.) Second, all other means of obtaining evidence should have been exhausted, and, third, the child protective services authorities should have been told of the suspicion and refused to act without the evidence provided by a tape. If all those factors are present, the important factor to remember is that the privacy rights involved are those of the patient (the child), not the patient's parent. Whatever privacy rights a young child may or may not have, they obviously are subservient to his or her rights to be protected from life-threatening or disabling abuse. From the constitutional perspective, the provisions of the Bill of Rights (such as protections from searches and seizures) apply only to state agencies and would not, therefore, be relevant to evidence gathered by a private hospital. Even where a public hospital is involved, however, a warrant to videotape would be granted on a showing of probable cause to believe that abuse is occurring within the hospital.

In many of these cases, before the evidence is gathered or, if gathered, before it is turned over to child protective services authorities, the pediatrician may ask a child

psychiatrist to interview the parent. The issue then becomes whether the parent should be told the purpose of the interview. It would seem that in general the parent does have a right to know that the interview is not intended to be directly therapeutic for the child and therefore she or he should be told that the interview is to assist in establishing whether the parent is making the child ill.

Because state statutes on evidentiary matters and state constitutions' provisions on such issues as videotaping vary widely, under no circumstances should a physician or a hospital engage in this sort of evidence gathering without advice from the hospital's local attorney. If a child may die if unprotected, a judge will be available to help with these situations.

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## 114 TESTIFYING IN COURT: A TRIAL LAWYER'S PERSPECTIVE

David N. Rosen, LL.B.

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What does it mean for a child psychiatrist to be an “expert witness?” On one hand, the phrase conjures images of a scientific demonstration by a chemist or fingerprint analyst. This conception is unlikely to seem appropriate because psychiatry does not lend itself to the seemingly unassailable conclusions of “hard” science. Alternatively, an invitation to testify may feel like being propositioned to be a hired gun and to do “junk science,” appealing only to those who enjoy fighting or being on display. In fact, a child psychiatrist who testifies in court can be both effective and true to his or her understanding of the reality of the case. In order to do this, the prospective expert witness should think about the structure of a trial. Trials create a framework within which the expert’s story will be told and evaluated, which creates a specific set of opportunities and hazards for an expert witness. This chapter discusses that framework and proposes that a child psychiatrist can be an effective witness by approaching testifying as an application or extension of his or her professional skills and activities. It is useful to consider separately direct examination and cross-examination, because these are very different activities and require the use of very different metaphors to understand and master. For direct examination, the first topic discussed in this chapter, a child psychiatrist must think about how to construct the most compelling story; whereas for cross-examination, he or she must cope with and learn to use the aggression inherent in the confrontation with the cross-examiner.

### PART ONE: TELLING A STORY IN DIRECT EXAMINATION

A trial is not a direct reflection of reality. It is a reconstruction of reality. It might more accurately be called a *construction* of a reality of its own, because it has its own purpose and structure, created by the law. Even a straightforward-seeming criminal case does not address a question as simple as “Did A kill B?” The question is, rather, “Has the state proven beyond a reasonable doubt that A murdered B and, if so, was it murder in the first degree, or perhaps manslaughter?” Changing the question from “what happened?” to “what has been proven?” and introducing legal categories that may have little connection to how anyone perceived events as they were occurring inevitably makes a trial different from, and more complex than, simply a search for truth. Additional complexity is added as the questions the trial seeks to address become forward looking, as in a custody case, where the issue is, “where will it be in the child’s best interest to live in the future?” In such a case there can be no pretense that the trial is simply finding a preexisting reality; however, in all cases the trial is a competition between versions or stories of what happened or will happen or should happen, a competition that is familiar to psychiatrists. Psychotherapy prominently includes formulating reconstructions of reality that ring true or are useful to the patient. Inevitably, such formulations are based on incomplete, perhaps fragmentary, evidence, but if they hang together and capture something important in the information the patient has been conveying, they can be persuasive and compelling; therefore, it is in a trial, where the adversaries present competing stories, or versions, to the fact-finder, who must choose between them based on necessarily limited information.

As there may be formidable barriers to hearing a patient’s story clearly and coherently, much about the structure of a trial makes it difficult to present a coherent narrative. One of the most striking features of most trials to any observer, and perhaps the most salient for a child psychiatrist contemplating testifying as an expert witness, is their fragmented, disjointed quality. Although trials make great subjects for stories and are often gripping drama, they are constantly subject to a variety of centrifugal forces that make them prone at any point to become incoherent. It is useful to identify the reasons trials are so much in need of the coherent unity effective expert testimony can provide because the expert’s role in direct testimony is above all to counteract that incoherence and provide order and a narrative thread to the case he or she is called to support.

Why are trials always in danger of becoming incoherent? First, because the adversary system entails by definition the clash of views. There are, at least, two sides to every story, and presentation of both sides almost invariably makes the narrative harder to follow. Moreover, in the adversary system, at every point confusion is likely to be in someone’s interest. Ordinarily, when one side is presenting part of its case, it is in the other side’s interest to disrupt the coherence of the presentation. As a result, one side is often doing what it can to interrupt the flow of the other side’s presentation—and the rules of procedure offer many opportunities to do so, from objections to cross-examination to legal claims.

In addition to being under constant threat from the very nature of the adversary system, coherence of narrative is also jeopardized by features of the rules of procedure and evidence. For example, trials proceed by questions and answers, which tends to give testimony a staccato, as opposed to lyrical, quality. The lawyer and the witness are performing a duet, and if either gets out of step the narrative will quickly become choppy, entirely apart from interruptions by an adversary claiming that questions are improper or answers are nonresponsive or contain inadmissible material. Indeed, the basic rule that evidence must be presented through witnesses, each of whom is limited in the scope of his or her testimony, makes it harder to sustain a narrative thread. The story is told in pieces that come from different witnesses; and in real life the contributions of each witness cannot be shaped entirely by needs of dramatic effect. The areas about which any given witness may testify are not always adjacent to each other in the logical sequence in which one side’s narrative unfolds. For example, in a trial for sexual abuse of a child, a teacher may be able to identify some relevant physical evidence such as the child’s clothing, testify extensively about the child’s behavior and condition at different times, and also testify about some contacts with the suspected abuser. The teacher’s testimony about all these subjects will almost invariably be presented at once, rather than at different times according to the demands of dramatic narrative.

Of course it is also the case that cross-examination of each witness follows the direct testimony of that witness, rather than, for example, beginning only after all witnesses have completed their direct testimony. Each witness, therefore, presents testimony that zigzags according to the competing dictates of the adversaries. Additionally, the scheduling difficulties and delay for which the legal system is justly notorious result in trials that are frequently interrupted, sometimes for long stretches, and in which witnesses cannot always testify in the sequence that would make for the most coherent narrative. Doctors in particular are often called “out of turn” to accommodate their schedules but, as a result, find themselves talking about evidence the court should logically have heard already but in fact has not. Finally, and most fundamentally, the story of a trial is told through witnesses, each of whom contributes only a part of the story. A case is built by weaving together the threads of the testimony of different witnesses or, to use another common metaphor, as a mosaic in which each exhibit and witness’s testimony adds elements that together form a coherent pattern. In every case, the task is to show that the elements form a coherent whole, and the danger is that they will simply be a jumble.

The trial lawyer’s response to this problem is to develop and emphasize a theme for the case. It is a truism of trial advocacy that every successful case has a theme, a narrative structure on which the advocate tries to hang all the information the fact-finder receives. A theme provides clarity to a form of presentation that otherwise can easily dissolve into incoherence. A theme is the essence of one side’s case boiled down to a clear, simple, attractive statement. In a murder case the prosecution’s theme might be that the defendant was a violent, possessive husband who killed his wife in a jealous rage, whereas the defense’s theme might be that a biased and sloppy police force fixated on the defendant and failed to perform the thorough investigation necessary to find the real killer—even more concisely: jealous rage versus rush to judgment. A theme is no less important in a civil case, such as a child custody dispute. One parent’s theme might be, “The child belongs with me because I was the one who raised her,” whereas the other parent’s theme might be, “The child belongs with the parent who is more stable and supportive now and promises to remain so in the future.” Although some might find this simplification frustratingly incomplete or even simple-minded, trial lawyers consider it distillation rather than distortion. The right theme captures the essence of one side’s view of the truth, and it is often true that a case that cannot be summarized in a brief, clear, forceful way is not a very good case.

The child psychiatrist who is going to testify in court needs to understand how his or her testimony fits into the themes each side is presenting. If he or she is allied in some way with one side, as is common, he or she will want not only to understand but also help shape the theme of that side’s case. Like an actor in a play, a child psychiatrist witness must understand his or her role in the drama of the trial. The psychiatrist can help the lawyer and the patient/client by collaborating on identifying and formulating the essential theme of the entire presentation because, unlike an actor, the child psychiatrist’s lines are limited not only by what is plausible but also but by what is actually true. In a child custody case, for example, the psychiatrist ordinarily gives an opinion about the ultimate issue in the case—what custody arrangement is in the best interest of the child. Before testifying, the psychiatrist should try to formulate the basis for her opinion as concisely as possible, ordinarily in

a single, simple sentence. Two examples were given in the preceding.

Others might be, "Honoring the child's expressed desire to be with one parent is critical to giving the child a needed sense of autonomy" or, "The child's most critical need is not to be separated from his or her half-sibling" or, "That parent is dangerously unstable." These examples may or may not even count as good bases for a custody decision to some readers, but by virtue of being clear and simple themes, they will have force in a trial. Once having chosen a theme, a trial lawyer will look for opportunities to repeat it and point to evidence that supports it throughout the trial. In an important way, the trial will become about the theme, or competing themes, and the psychiatrist's role will be to testify about which theme is closer to the evidence and—what is more challenging—to critique one of the themes as shallow or irrelevant, at least to some extent.

A child psychiatrist may protest that boiling the results of many hours of observation and years of training and experience into a phrase or a sentence trivializes any contribution he or she might make by substituting a slogan for an opinion. A trial lawyer would reply that simplicity is necessary because of the diffused and fractured nature of trials and also because the audience is either a lay jury or a judge who, whatever his or her experience with similar cases, is not a child psychiatrist. It is common also for experts to make a virtue of the necessity of simplicity in the courtroom. Boiling down opinions to their simplest and most direct expression can be a useful analytical as well as rhetorical device, and an expert who can summarize his or her most important point in a brief, direct sentence may find his or her ideas sharpened and improved as a result.

Having said this much about the virtues of simplicity and focus is not to say that there is no place for complexity; On the contrary, the issue is not whether complexity and nuance have a place in a trial and the expert's testimony, but rather how to formulate and present complexity. Here the expert's model, like the trial lawyer's, should be the story. Fact-finders, like any audience, have tolerance, indeed an appetite, for complexity so long as it is presented within a narrative framework that holds it together.

The expert witness is privileged by the rules of evidence to tell a more complete and coherent story than other witnesses are allowed to tell. Experts can testify not only to their conclusions but also, reasonably enough, to the basis for those conclusions. When explaining the basis for their conclusions, therefore, experts can draw on and restate other evidence. Consequently, they have a unique opportunity to draw the evidence together into a unified narrative. Moreover, in testifying about the basis for their conclusions, expert witnesses are not even limited to information that is admissible evidence. The rule is that they may rely on any information, even hearsay or other inadmissible evidence, so long as the expert affirms that the information is the type of material ordinarily relied on by experts in the field. Thus, the expert may testify about information he was given by a child's teacher even though the teacher does not himself come to court to verify the information. (It is important surely to take any reasonable steps to verify such information, particularly because it is a likely area for cross-examination.) The expert has an opportunity, then, to synthesize a lot of information and communicate and discuss that information on the witness stand.

Robert Coles, a child psychiatrist, has written of "the call of stories." His view is that the clinician does well to eschew theoretical formulations of patients' psychodynamics, or at least keep them in their place, and focus primarily on the narratives, the stories, the patients will tell about themselves to those who want to listen. He argues, and demonstrates, that "a good deal of the time our patients' comments tell their own story, one that can be interpreted by us in ordinary language with no loss of psychological nuance and subtlety" (Coles, 1989, p. 27). This perspective is readily adaptable to trials, and expert testimony in particular. A child psychiatrist could present her conclusions by following a theoretical schema or reporting first test results, then the findings of a clinical examination, then conclusions, and so on. Ordinarily, however, a more compelling and persuasive method of presentation is to start wherever possible with a framework of the patient's story and to relate the details of findings and conclusions to the framework of a story that illustrates the expert's theme. Clinical observations and findings can be reported using this approach as illustrations of the themes of the story. Thus, the expert might begin testimony about her findings and conclusions as follows:

**Q:** Doctor, did you reach any conclusions concerning what custody arrangement would be in the best interests of the child?

**A:** Yes, I did.

**Q:** What conclusions did you reach?

**A:** My ultimate conclusion was that it would be in the best interest of the child for her mother to have custody of her.

**Q:** What is the basis for your conclusion?

**A:** [Introducing the theme] To put it as simply as possible, I found that this child's mother is the most important person in the world for her, and she needs to live with her mother to feel secure and protected.

**Q:** Can you explain what led you to this conclusion?

At this point the expert can try as best as he or she can to narrate the child's story and illustrate the theme in a nontechnical way. Rather than proceeding deductively by announcing the principle that the adult with whom the child has formed the closest bonds or who is appropriately designated the "psychological parent" should have custody, the story can properly revolve around the child's relationship with that adult. From the specifics of the child's life and relationship with that adult, the expert can then move convincingly to the observation that the child's experience of depending on and needing a sustained central relationship with the particular adult is just what the expert would expect. In fact, the expert can continue, everything we know about the way children develop is consistent with what this child's needs appeared to be from looking closely at her life story. This form of narration ordinarily will feel more "right" and persuasive to either a jury or a judge than one that begins with the generalizations of theory and then moves to apply theory to the particular case at hand.

Psychiatrists are well trained to be expert witnesses because they are students of people and their life stories. By focusing on the fundamental point they are going to make in their testimony and how that point relates to the theme of the trial, and then allowing themselves to tell the story, with all its individual richness, that supports that theme, they can make a presentation on direct examination that feels right and consistent with their professional role and is at the same time highly effective in the courtroom.

## **PART TWO: THE USE AND MANAGEMENT OF AGGRESSION IN CROSS-EXAMINATION**

What awaits a child psychiatrist who is to testify in court? The experience, particularly cross-examination, inevitably seems somewhat surreal: Anticipating a serious and consequential discussion about clinical issues, the psychiatrist instead may find himself or herself being hectorated by an inquisitor who evidently has no interest in either the truth or the best interests of the child whose welfare is ostensibly the purpose of the proceeding. Equally disconcerting, the cross-examiner's questions may proceed from the assumption that the psychiatrist is dishonest or biased, leaving the psychiatrist feeling like a murder suspect protesting her innocence to a cynical police detective.

An important part of the difficulty many psychiatrists experience testifying in court is that they use the wrong model to understand it. A psychiatrist may imagine that testifying in court is more or less like giving an invited lecture. Using this model (or laboring under this misapprehension), the psychiatrist will be likely to regard cross-examination as uninformed, distracting, and impertinent. Exchanges like the following will ensue:

**Q:** How much time did you spend reviewing the report of the family relations officer before you formed your opinion regarding custody?

**A:** I am afraid you didn't understand my earlier testimony. The report of the family relations officer has very little significance for me because I performed my own independent evaluation and spoke with all the people the family relations officer spoke to. In fact, I deliberately did not study that report because I did not want my own views to be biased by another professional's opinion.

**Q:** So the answer to my question is "none." Is that right?

**A:** You aren't taking into account the reason I didn't examine the report at that time.

**Q:** Is the answer to my question, "none?"

**A:** Yes.

**Q:** Thank you.

The witness would have done much better to respond as follows:

**Q:** How much time did you spend reviewing the report of the family relations officer before you formed your opinion regarding custody?

**A:** None; I read the report after I formed my opinion.

Or, simply,

**A:** None.

Somewhat similar results may occur when a psychiatrist-witness regards cross-examination as a debate or an argument. He or she will invite being viewed as biased, belligerent, or both and will have missed the opportunity to be an effective witness. For example:



**Q:**How much time did you spend reviewing the report of the family relations officer before you formed your opinion regarding custody?

**A:**None at all, which is probably fortunate, since when I ultimately did review it I found it very misleading.

**Q:**Was it misleading when it said the child has lived with her father for the past 6 months?

**A:**No, the part I was referring to is ...

**Q:**[Interrupting] Please answer my question, Doctor. The answer to my question is, "No?"

**A:**Yes, but ...

**Q:**Thank you. Now, was the report misleading when it said the child is doing well in school?

And so on. Again, the witness would have done better simply to answer the question and trust that subsequent redirect examination by the lawyer who originally had called her to testify would give her the opportunity to correct any misimpressions left by the cross-examination.

A more useful reference point is another form of dialog with its own peculiar set of rules: the psychoanalytic session. Thinking about the similarities and differences between cross-examination of a witness and a psychoanalytic session is a helpful way to understand and therefore cope with the demands cross-examination places on a child psychiatrist testifying in court.

The lawyer's job, like the analyst's, is to lead a subject to the most difficult and painful topics and peel away layers of rationalization or dissimulation to reveal a truth within. That is, not only are both psychoanalysis and cross-examination investigations, but also they both begin with the assumption that people's testimony should not be taken at face value, nor should their honesty be presumed. Thus, cross-examination has been described, in a phrase Freud might have used about psychoanalysis, as the greatest engine ever invented for the discovery of truth.

On the other hand, although psychoanalysis depends on the patient free associating—saying whatever comes into his or her head without censoring any thoughts—the witness is entirely constrained in cross-examination and must answer precisely the question put by the cross-examiner. To a psychiatrist witness, used to a more discursive approach to the truth, this constraint can make cross-examination feel like the course in oral expression Holden Caulfield took in *The Catcher in the Rye* (1964), where each boy had to make a spontaneous speech in class:

And if the boy digresses at all, you're supposed to yell 'Digression!' at him as fast as you can. It just about drove me crazy.... What I think is, you're supposed to leave somebody alone if he's at least being interesting and he's getting all excited about something.

That is not what cross-examiners think. What cross-examiners think is that their job is not so much to *discover* the truth as it is to *demonstrate* the truth, as they have been hired to see it, so that the judge or jury may discover it. Accordingly, the first rule of cross-examination is, never ask a question to which you do not know the answer. What this precept means in practice is that virtually all questions on cross-examination are closed-ended, and witnesses are very often merely foils whose role on cross-examination is simply to admit facts the examiner wants the judge or jury to hear in the sequence the examiner has selected. For example, consider the following sequence:

**Q:**Doctor, how much time did you spend observing the mother and child interacting together?

**A:**One hour.

**Q:**And you have based your recommendation regarding custody primarily on your opinion about the relationship between the child and mother, isn't that right?

**A:**Yes.

**Q:**And that one hour was in your office, is that right?

**A:**Yes.

**Q:**And of course how people behave in your office when you are watching them may differ from how they behave at home when they are not being observed, isn't that right?

**A:**Yes.

**Q:**For example, the mother certainly may have been trying to impress you during that single one-hour session that you observed her with the child, isn't that right?

**A:**Yes.

**Q:**And that might affect her behavior, isn't that right?

**A:**I suppose so.

**Q:**So your only direct observation of the mother and child relating to each other was for a single hour, outside the home, under circumstances where the mother might be acting differently from the way she would before she came to your office and after she left it, isn't that right?

**A:**Yes.

And so on. Anticipating all of this, the psychiatrist may wish to respond by taking control of the conversation, for example, as follows:

**Q:**How much time did you spend observing the child and her mother together?

**A:**I don't think that is what is important in making an evaluation concerning child custody in this case.

She is not allowed to do so, however. If she does, the lawyer is entitled to say, "Please answer my question, Doctor," or even, "Are you willing to answer my question, Doctor?"

The psychiatrist cannot expose himself or herself to this line of questioning without damaging his or her credibility. The psychiatrist is not entitled to ignore the constraining rules of cross-examination and will score no points by attempting to do so. If the psychiatrist persists in trying to step outside the rules of cross-examination, she will be perceived, accurately, as breaking them.

Both court testimony and a psychoanalytic session are dialogs of a sort, but both are asymmetrical in important respects, in which the witness is analogous to the patient and the lawyer to the psychoanalyst. For example, they are asymmetrical with respect to disclosure. The central function of the witness or patient is to disclose information, freely and fully. On the other hand, the examiner and analyst need disclose nothing. This asymmetry can be frustrating in both settings; unlike an ordinary conversation, openness and candor in these settings cannot be expected to be reciprocated. Therefore, the witness, like patients, is vulnerable to feelings of powerlessness and loss of control.

These feelings may be intensified by the rule (or meta-rule) illustrated by the cross-examination excerpt in the preceding, but applicable to both settings, that the witness or patient cannot make the examiner or analyst step outside of his or her role. Thus, a patient who demands of an orthodox psychoanalyst, "What do *you* think?" about this or that may get for a reply only, "What do you think I think about it?" or silence. Likewise, a witness who demands of a cross-examiner, for example, "Don't you really think the child should stay with the parent who has been most involved with her up until now?" cannot expect to disarm the lawyer and get a straight, honest answer. Instead, the lawyer may reply, "Are you suggesting by that question, Doctor, that in your opinion the *character* of the parent is irrelevant to the best interest of the child?" The lawyer is *supposed* to respond only in whatever way he or she thinks is best calculated to advance a client's claim in the litigation, and this imperative typically will lead the lawyer to view a witness's appeal to step outside the game of the adversary system as simply another move in the game, calling for a countermove on the examiner's part.

These constraints on the witness suggest another, more fundamental asymmetry in cross-examination, which also has its counterpart in the psychoanalytic relationship. This is asymmetry with regard to aggression. Cross-examination is a weapon in the lawyer's arsenal within the adversary system. The lawyer uses it either to attack the witness or, perhaps, to turn the witness into a weapon directed at the adversary. But the witness is not supposed to be an adversary; the expert witness especially is supposed to be a representative in court of the commitment to truth. Thus, the cross-examiner properly regards the conversation, that is, the examination, as a form of warfare, but the witness cannot take this stance. The witness is required to treat the examination as a search for truth.

In this respect, the witness is analogous to the psychiatrist, not the patient, in the psychoanalytic relationship. Although the lawyer is permitted to give more or less free rein to his or her aggression, the witness is required, no matter what accusations are leveled or what hostility expressed, not to retaliate in kind but to be the anchor of reason and respond to hostility and aggression with understanding and without permitting himself or herself to be provoked to anger.

To understand why it is so important for the witness to preserve this asymmetry—not to respond in kind to the lawyer's aggression—consider that the expert witness, like the patient, is more knowledgeable about the subject matter of her testimony than the lawyer cross-examining her or the judge or jury evaluating her testimony. As the analyst, who has never met the patient's family and friends, evaluates the patient's narrative in part through careful attention to the patient's affect and with the aid

of generous amounts of intuition rather than logical deduction or reference to externally acquired information, therefore the judge or jury evaluating the psychiatrist cannot be expected to learn as much as the psychiatrist knows about psychiatry. They do not perform a kind of peer review of the psychiatrist's testimony. Judges and particularly juries are lay people, not experts in any of the fields about which they may hear testimony. Indeed, people with special expertise in the area in which an expert will testify frequently are excluded as jurors, in order to prevent them from using their knowledge in a way that is not open to scrutiny and control by the court. Of course, one must ask on what basis, then, are judges and juries supposed to evaluate the conflicting testimony of experts?

The answer is that fact-finders in court, and particularly juries, evaluate an expert's conclusions based primarily on their assessment of how the expert has handled the tension between the aggression of the cross-examiner and the witness's obligation to speak for scientific truth. That is, although judges or juries may well follow the substance of an expert's testimony carefully and attempt to evaluate it as a peer might, that is not the main thing they do, indeed not the main thing they are supposed to do. What courts and juries are designed to do is to evaluate the credibility of witnesses. And what this boils down to above all is evaluation of the witness's commitment to the value of truth when confronted with the aggression of the adversary system.

### **Coping with Aggression in Cross-Examination**

The way this process works in practice is that, to be persuasive, witnesses must accept and use the asymmetry of cross-examination. Like a psychiatrist with a belligerent patient, the witness must be careful not to be provoked by the aggression of the cross-examiner (and in this way the psychiatrist witness is not switching roles but may call on familiar skills). Because the cross-examiner is aggressive, the witness succeeds by being gentle; the jury understands that, as aggression and an adversary stance are companions, so their opposites, gentleness and fidelity to truth, also go together. Thus, the cross-examiner marks it a success when he or she succeeds in getting under the skin of the witness, or making the witness *blow up*. The successful witness, on the other hand, is one who *deflates* the cross-examiner.

Some examples of common lines of cross-examination show features of the adversary system in action and illustrate how the witness's use and management of the aggression inherent in the adversary system is the key to being an effective expert witness. Let us begin by imagining a case in which the psychiatrist who has treated a child for emotional problems arising from a physical injury is called to testify in a case seeking damages for the injury. Cross-examination begins as follows:

**Q:**You have seen the plaintiff as a patient for almost a year. Is that right?

**A:**Yes.

**Q:**In that time have you sent her family bills?

**A:**Yes.

**Q:**Have they paid the bills?

**A:**No.

**Q:**How do you expect to get paid?

**A:**They have agreed to pay me as they are able to do so.

**Q:**In fact, you expect to get paid out of the proceeds of this lawsuit don't you?

**A:**Well, yes.

**Q:**So you have a stake in the outcome of this lawsuit don't you?

**A:**I am being honest in my testimony.

**Q:**Isn't that for the jury to decide, Doctor?

**A:**Yes.

**Q:**My question was not: Are you being honest? It was: Don't you have a stake in the outcome of this lawsuit?

**A:**Well, not really.

**Q:**Don't you stand to get paid nearly \$5,000 if the plaintiffs win this lawsuit and little or nothing if they lose?

**A:**Yes.

**Q:**But \$5,000 is not a stake in this lawsuit, is that your testimony?

What is a witness to do about this sort of questioning? Clearly, the answer is not to say, for example, "Well, \$5,000 is not very much to me," or, "I wouldn't change my testimony for a million dollars." The first is condescending, the second defensive. This is the sort of questioning against which the best inoculation is not the witness's protestations of innocence but rather her *demonstration* of lack of bias in her answers. The psychiatrist could answer as follows:

**Q:**How do you expect to get paid?

**A:**I suspect I will either get paid out of the proceeds of this lawsuit or not at all.

By anticipating the lawyer's point in her answer, the witness has taken much of the sting out of it. And when the lawyer asks the inevitable question, "So you have a stake in the outcome of this lawsuit, don't you?" the better answer is simply, "To that extent I do, yes." By refusing to be put on the defensive—that is, refusing to resist a point that the examiner is going to make anyhow—the witness defuses the examiner's aggression. The point is *not* the witness's expectations regarding payment but whether the witness appears to be more concerned with accuracy than with preventing the examiner from scoring debating points.

Now consider an attack purportedly on the witness's expert knowledge, which again when looked at in action actually depends for its effectiveness not on the substance of the witness's answers but on how adversarial the witness is:

**Q:**Doctor, you diagnosed the patient as having posttraumatic stress disorder. What are the diagnostic criteria for posttraumatic stress disorder?

**A:**In my report I mention four basic diagnostic criteria.

**Q:**Can you name the criteria without looking at your report?

**A:**Well, (a) there must be a stressor, (b) the patient must reexperience the trauma, (c) there is a withdrawal from social engagement, and I am afraid that's all I can recall.

**Q:**You are not really an expert in the area of posttraumatic stress disorder, are you, Doctor?

**A:**Well, just because I don't remember all the criteria, that doesn't mean I'm not an expert. I have written in the area of schizophrenia and I couldn't recite all the diagnostic criteria for schizophrenia.

**Q:**But you have not written in the area of posttraumatic stress disorder, have you?

**A:**(Doesn't matter.)

The point about this line of questioning is that knowledge of the four diagnostic criteria may be elementary information or it may be arcane, but the jury does not know which it is, and even if they did know, they probably would not care. Their concern is with how the witness handles the questions, not with how high he or she scores on a pop quiz. The witness can defuse this line of questioning by refusing to take it as a challenge and at the same time staying within her role by not appearing to claim to judge the materiality of the question. For example:

**Q:**Can you name the four basic criteria without looking at your notes?

**A:**I don't think so.

By using a matter-of-fact, noncombative tone the witness has communicated all he or she needs to: This information is not something he or she needs to have memorized in order to feel capable of reaching whatever conclusions have been reached. Moreover, the witness is not acting like an adversary; implicitly, therefore, he or she is interested in the truth, not simply in winning. (If the psychiatrist can in fact name the criteria, the best answer is "Yes, I can." This answer is better than actually listing the criteria because it is not showing off and accordingly does not imply any agenda other than the accurate transmission of information.)

Likewise, to the inevitable question, "You are not really an expert in the area of posttraumatic stress disorder, are you?" a better answer is, "Posttraumatic stress disorder is among the psychiatric disorders that I treat, but it is not an area of special expertise," or (if true), "No, I believe I am an expert in the area of posttraumatic stress disorder."

### **Special Problems of Child Psychiatrists**

However, all this is not to say that the testimony of a child psychiatrist must be or appear to be without feelings. On the contrary, to be persuasive a child psychiatrist



must, like any expert, communicate commitment to the *field* and its values. In addition, as a child psychiatrist, the witness, unlike most expert witnesses, may properly, indeed must, communicate a form of partiality to the *welfare of the child* who is the subject of the court proceeding. Nevertheless, this is quite a different matter, indeed the reverse, of being adversarial or combative on one's own behalf or in order to score points. Consider the following exchange, from a child custody case before a judge without a jury:

**Q:**Doctor, if the mother is permitted to move far away with the child, it will be harder for the child to see her father, won't it?  
**A:**Not if the father moves too.  
**Q:**But don't you think if the mother wants to move away from the father now, she would just try to move again if he followed her?  
**A:**Not necessarily.  
**Q:**But you certainly aren't claiming that won't happen. Are you?  
**A:**I don't think it's likely.  
**Q:**But you really don't know one way or the other. Do you?  
**A:**Of course not; no one can know for sure at this point.  
**Q:**But if the court orders the mother not to move, then we would know for sure that the father will be nearby. Wouldn't we?

Here the psychiatrist, probably giving in to her frustration with the father, his lawyer, the legal system, or some combination of them, was unable in her answer to the first question to resist the impulse to be clever. Giving in to that impulse invariably gives the cross-examiner the opportunity to score points. If the witness had held her tongue, she would have had an opportunity to make her point not by being clever but simply by manifesting her commitment to the child's well being:

**Q:**Doctor, if the mother is permitted to move far away with the child, it will be harder for the child to see her father, won't it?  
**A:**It certainly may.  
**Q:**There is no "may" about it, is there, Doctor?  
**A:**Oh, I think it is far from certain that the mother's moving will necessarily have that effect. If the mother is permitted to move, she may feel more secure and be more open to the father's visitation; or, the father may even decide that being close to this child is so important that he will consider moving also.

The witness has in effect earned the right to give this answer by not jumping into combat with the examiner earlier. As a result, the effect of her testimony is fundamentally different from the effect of the first sequence.

Psychiatrists, particularly child psychiatrists, face a variety of other special problems as witnesses. The first of these is the plethora of experts who may be involved in a case. A child custody case may have testimony from psychiatrists retained by each of the parents, a court-appointed psychiatrist and/or one retained by counsel for the child, a court-employed family relations officer, and welfare department employees. To all these voices, frequently discordant, add those of counsel for the parents and the children, and the result can be that the competing opinions tend to cancel each other out, leaving the judge free to act on his or her impulses.

Moreover, confusion of roles is common in child custody cases ([Goldstein et al., 1986](#)). The child psychiatrist ordinarily is asked to testify concerning what custody arrangement is in the best interests of the child, the very same issue the judge ultimately will decide. (By contrast, an expert's testimony in a criminal case may establish that the bullet that struck the victim came from the defendant's gun, but that testimony is only one link in a chain needed to establish the defendant's guilt of a given crime.) As a result, it can be hard to distinguish the judge's role from the psychiatrist's. The psychiatrist may feel, and be treated by the parties, as if he or she is adjudicating competing claims. The psychiatrist accordingly may be subjected to criticism for not according one of the parties due process before deciding against that party, for example, by meeting with one side more than the other. The judge may also treat the child psychiatrist as the adjudicator and simply rubber-stamp his or her recommendation, particularly a psychiatrist retained by the court itself or by counsel for the child. More troubling from the point of view of the psychiatrist witness, the judge, like the parties, may evaluate the psychiatrist's work and testimony like a sort of court of appeals, asking not what is in the best interests of the child but whether the psychiatrist has been fair to all the parties. The psychiatrist, of course, is not trying to be fair to all the parties: At most, he or she is trying to determine what outcome will be in the best interests of the child. However, because the psychiatrist is confused with a judge, he or she is liable to be criticized for not taking into account the interests of the disappointed parent.

Moreover, the judge, particularly in child custody cases, very frequently has had so much experience deciding such cases that he or she is convinced that the opinion of a child psychiatrist is superfluous. That is, the judge, not to mention the lawyers and other professionals, confuses his or her role with the psychiatrist's because, after all, in a way it is everyone's job to determine the best interests of the child. This confusion is exacerbated by the fact that many people, including many judges, do not believe child psychiatry has much special expertise to offer. Child psychiatrists thus encounter triple skepticism—as witnesses, expert witnesses, and specialists in a field that is viewed with suspicion (as, say, surgery is not).

To be an effective witness under these circumstances, the child psychiatrist, even more than other expert witnesses, must not be inflamed by the aggression of cross-examination. The best defense of her expertise a child psychiatrist can offer as a witness is a demonstration of his or her therapeutic approach in the process of testifying. That is, the psychiatrist should let the court see how he or she operates as a therapist, not by practicing psychiatry in the courtroom but by showing a caring, gentle thoughtfulness rather than combativeness.

It is just this tone that makes for an effective witness: Any witness is more persuasive if he or she appears to treat the trial as a truth-seeking activity rather than as an arena. However, for a psychiatrist a gentle, noncombative tone serves the additional function of illustrating the maturity and thoughtfulness that a court will recognize as characteristics of a good therapist. For example, consider the following exchange:

**Q:**Doctor, did you perform any formal psychological testing on your patient?  
**A:**Certainly not. Psychological testing would have been a waste of time in this case.  
**Q:**Isn't psychological testing recognized as an important diagnostic tool?  
**A:**But I wasn't doing the kind of diagnosis that calls for psychological testing. I was investigating the question of custody, not doing a full-scale work-up of a psychiatric patient.  
**Q:**Wasn't this case just as important as a full-scale work-up of a psychiatric patient?  
**A:**As I just explained, it's not a question of importance, it's a question of whether psychological testing is going to be of any significant value in this context, and in my opinion it clearly isn't. What I did do was to talk at length with all the parties involved and observe the child interacting with each of her parents, and I think that was much more valuable than psychological testing could possibly be in this context.

Among the problems with this approach to testifying on cross-examination is that the witness does not sound like someone who is a gifted listener. A judge already somewhat skeptical of psychiatrists is unlikely to trust this witness. A better approach to the same line of questioning would be:

**Q:**Doctor, did you perform any formal psychological testing on your patient?  
**A:**No, I didn't.  
**Q:**Isn't psychological testing recognized as an important diagnostic tool?  
**A:**Yes, it is.  
**Q:**But you did not use that tool here, did you?  
**A:**No, I didn't.

The witness is being used as a foil in this sequence, and instead of fighting that role, simply allows the lawyer to make points, patient and secure enough not to need to correct every misimpression right away. Such a witness will get an opportunity to say what he or she wants to say, either on direct examination or perhaps even, as in the following example, on cross-examination:

**Q:**So inasmuch as you did not do any psychological testing, you did not really do a thorough evaluation, did you?  
**A:**No, I think my evaluation was thorough.  
**Q:**But it would have been more thorough had you done psychological testing, wouldn't it?  
**A:**No, the important issue that emerged in my evaluation was Mary's need to stay with her mother and her fears of losing the security her mother represents. And psychological testing would not really have added to my understanding of that issue.  
**Q:**But it might have added to your understanding of other issues, isn't that right?  
**A:**Yes, that is certainly true.

When and if the psychiatrist does get an opportunity like this one to say to the cross-examiner what he or she thinks is important about the case, the judge will be ready to listen, much more readily than if the psychiatrist mistakenly believed that his or her job was to say what was necessary to say no matter what. (And if the psychiatrist does not get to say it on cross-examination, the other lawyers or even the judge will give him or her an opportunity when the cross-examination concludes.)

In short, the judge will be most likely to notice the psychiatrist's virtues as a therapist if some of them are shown as a witness. Ultimately, the psychiatrist's professional skills—an ability to listen attentively and thoughtfully, care about the well-being of patients, and resist the urge to respond to aggression with aggression—will enable him or her to be a persuasive witness in court.

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# 115 ROLE OF THE CHILD EXPERT IN COURT-REQUESTED EVALUATIONS

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In this chapter, we explore the role of the child expert in court-contested child custody and placement conflicts. A *child expert* is a person who, by training and experience, has acquired and uses that body of knowledge referred to as clinical child development. No one discipline or profession is the exclusive source of such knowledge and its applications; the professions of pediatrics, child psychiatry, child psychoanalysis, social work, and clinical child psychology all require a mastery of the theoretical and practice aspects of this body of knowledge as defined by state and national standards of academic and supervised training. We assume that the knowledge used by child experts in the clinical evaluation, expert opinion, and recommendations conveyed to the court about what will serve the child's best interests is based on (a) the child's need for appropriate physical nurture and safety and (b) the child's need for a continuity of affectionate care by a limited number of adults who want to take care of the child on a permanent basis.

The child expert may be called upon to perform a clinical evaluation as part of a legal proceeding by agreement of the adult parties, by order of the court, or at the behest of one of the disputing parties. The legal conflict may stem from a custody or visitation dispute between divorcing parents or as part of a placement dispute between the child's parents and the state stemming from issues of neglect, abuse, or abandonment.

Divorce cases (issues of custody and visitation) are usually heard in family court, whereas placement cases are most often heard in juvenile court; guardianship and occasionally custody issues are determined in probate court. The most frequent request for clinical evaluations involves a legal decision about the child's custody and may include termination of parental rights. Although the principles of child development knowledge being applied are the same in evaluations regarding custody assignments and in state placement evaluations, the issues are considered separately in this chapter.

## DIVORCE

After a rapid increase between 1960 and 1980, family disruption in our society is believed to have stabilized such that about half of couples married after 1970 will divorce at some point. Half of all children are likely to experience the divorce of their parents and go on to spend an average of 5 years in a single-parent household ([Glick and Lin, 1986](#)). In 1999, the proportion of children younger than 18 years of age who lived with two parents was 68%, a decline from 1980, when 77% lived with two parents, and from 1970, when 85% lived with two parents ([U.S. Bureau of the Census, 2000](#)). Approximately 72% of divorced women and 80% of divorced men will remarry. The divorce rate for second marriages is greater than in first marriages. One of every 10 children will experience two or more divorces of the custodial parent before he or she becomes 16 years old ([Furstenberg, 1988](#)). In 1994, and in each following year into the foreseeable future, more than 1 million American children are likely to be reared in families disrupted by separation and impending divorce ([Maccoby and Mnookin, 1994](#)).

Many societal factors have contributed to the increased acceptance of divorce in our society. No-fault divorce represents a judicial shift from the "tender years" doctrine, favoring mothers, to a "best interests of the child" standard. The best interests standard not only places the child's needs at the center of custody decision making, but also it satisfies the requirements of our society for freedom from gender bias with respect to custody decisions. In the present divorce climate, dispute resolution through cooperation and mediation is preferred to litigation ([Levy, 1998](#)). Programs to assist divorcing parents in dispute resolution typically offer parents information on the legal process for determining the following: custody, visitation, and child support; the effects of divorce and separation on adults; and the effects of divorce and separation on children and how parents can help children to cope with this difficult transition ([Pruett and Pruett, 1999](#); [Schepard, 1993](#)).

Present knowledge about the impact of divorce on children suggests that it creates an immediate or short-term stressful challenge that may adversely effect the long-term development of the involved children. [Wallerstein and Corbin \(1991\)](#) note that "children of divorce are greatly overrepresented in outpatient psychiatric, family agency, and private practice populations relative to their presence within the general population." Confirming this view, [Maccoby \(1992\)](#) comments on the Hetherington study and states, "A central finding is that the children in intact families were doing better at each assessment period than those living with divorced mothers who had either remained single or remarried. This finding is consistent with a considerable body of literature pointing to the disruptive effects of divorce (and/or the familial conditions that precede divorce) on children's functioning." There is no way to predict which children will be more vulnerable. Many clinicians believe that the single most corrosive element in the child's experience when parents divorce results from emotions of unabated and intractable conflict. However, these inferences must remain tentative because there is no way at present of knowing whether these findings are associated with the divorce or are the influences of a child-disturbing marriage.

Nevertheless, many children and their reconfigured families will be able, over time, to achieve a progressive development and to restore an adequate sense of continuity and security. The parents' ability to resolve visitation and custody issues cooperatively either independently or with assistance (by family relations services of the court, mediation efforts, or voluntary mental health consultation) can be responsive to the child's needs for a sound unique relationship with each parent. Such effective collaboration after the divorce provides the conditions that enable parents *de facto* or *de jure* to create voluntary joint custody. The parents' ability, willingness, and motivation to cooperate enable the child to feel wanted and to trust and count on the parents to continue to provide nurture and guidance.

## ROLE OF THE MENTAL HEALTH EXPERT

Most divorcing parents make their own custody arrangements. Statistical data on the percentage of divorces in which contested custody issues arise are estimated to be 5% to 8%. In their California study, [Maccoby and Mnookin \(1994\)](#) found that only 1.5% of families actually relied on the judge to decide the custody or visitation issue; another 2.2% went to trial but were able to settle during the trial. In the same study, 75% of families experienced negligible or mild conflict, whereas 25% experienced substantial or intense conflict.

When a mental health expert is consulted by divorcing parents on a voluntary basis, the usual rules of confidentiality apply. Some therapists make formal signed agreements with potentially litigious parents to emphasize the therapist's willingness to assist the parents to come to an agreement on a voluntary basis. Conversely, clinicians may not be willing to serve as experts in court if they already are committed to voluntary, confidential therapy. Usually, a verbal understanding is sufficient. No agreement can completely shield the process from the possibility of a later subpoena by one of the parents. Most often in voluntary situations, the child is not seen; rather, the emphasis is on assisting the parents to maintain their relationship to their child in an affectionate and appropriately authoritative manner as the true experts on what the child needs.

When divorcing parents cannot agree about the custody and visitation arrangements for their child, the state temporarily becomes the parent in its *parens patriae* function. Once the parents cede their decision-making prerogative to the state and the court assumes the decision-making role, the atmosphere is likely to be charged with anger and conflict. In such instances, the parents' disagreements are usually heightened, and there is a greater risk that psychological war will lead to emotional wounds, to feelings of betrayal and bitter rage that too often remain unhealed even after the judge has made a decision about custody or visitation. It is in connection with such cases that the child expert may be called on to render an evaluation and recommendation that can be used in the court proceeding.

## WHAT THE CLINICAL CHILD EXPERT SHOULD KEEP IN MIND ABOUT LEGAL CONSIDERATIONS

The child expert is frequently called on in conflicted cases by one or both attorneys representing the parents. When the child has separate counsel, the child's lawyer may initiate or be involved in the designation of the child's clinical evaluation. Regardless of who hires the expert witness, the contribution of the child expert is as an advocate for the child's needs, that is, for the "best interests of the child." The child expert translates this legal standard into clinical terms, meaning what is best or least harmful to the child, given her or his age, developmental level, special needs, and relationship with each parent. The child expert's recommendation to the court is based on a clinical evaluation of the child's development and attachments and on what can best facilitate the child's developmental progress.

From the legal side, the best interests standard is given lip service in most states, especially as the court's adversarial process forces into view the competing interests of the adults. In reality, concerns of fairness to adults and personal value biases of the individual judge or the society often prevail. In this sense, all involved will verbalize their concern for the child's best interests, but only a few will transcend their identification with the contending adults in a manner that makes the child's interests and needs paramount.

Lawyers and judges complain about the vagueness and indeterminacy of the best interests standard. Although some experts advocate more rules and less discretion in custody decisions as a deterrent to litigation, the law as practiced requires a highly individualized determination of what is in the best interests of, or least detrimental to, each particular child ([Mnookin, 1975](#)).

Although the mental health practitioner is likely to be looked to by the court to assist in providing insight into the individual child's needs and the competing caretakers' capacities to meet the child's needs, increasingly the claims of these experts are being subject to question. [Homer and Guyer \(1991\)](#) believe "...by whatever processes of reasoning they use, clinicians predictably fail the most fundamental requirement of basic scientific inquiry, which is to achieve convincing demonstrations of interjudge reliabilities of perception and inference when the latter are applied to the same body of facts." We assume that experts disagree because their knowledge is not and cannot be precise. We respect the diversity of lifestyles and believe the community is best protected, not by experts, but by those who administer the rule of law, as imperfect as it is, to guide us in ensuring parents and children of their constitutional rights.

## DEVELOPMENTAL PRINCIPLES

[Goldstein et al. \(1996\)](#) define the central principle in custody and placement decisions, "the least detrimental available alternative," as "...that child placement and procedure for child placement which maximizes, in accord with the child's sense of time, the child's opportunity for being wanted and for maintaining a continuous, unconditional and permanent basis...relationship with at least one adult who is or will become the child's psychological parent." The authors further explicate the child's sense of time as varying with the urgency of his or her instinctual and emotional needs; for example, separation from a central nurturing person is less tolerable to an infant or young child or a child with particular vulnerability or identified special need than to an older or sturdier child. The child needs to be wanted by at least one adult who offers safety, affection, and appropriate stimulation. The child's developmental needs are best met when there is opportunity to experience such an attachment to an adult in a continuing unconditional and permanent relationship. These principles offer the clinician a conceptual guide and frame of reference but are not meant to be formulaic.

The restoration of emotional security for those few children who are the subject of continued litigated custody disputes in postdivorce conflict often requires the difficult acknowledgment that, for the time being, the child's future is best safeguarded in the hands of one primary, custodial parent. The least detrimental alternative in extreme cases when parents are addicted to fighting and cannot tolerate the contact required by visitation is to support and protect the child's relationship with the custodial parent. Despite the flaws and limitations in the child's relationship with the custodial parent, even in extreme situations the child can do reasonably well if the relationship and identification with at least one parent are ensured and if the child's best interest is given preference over equity for the contending parents. Intervention by the court or other experts can risk undermining the custodial parent's security, sense of control, and effectiveness and can often result in less cooperation between the parents ([Nordbaus, 1991](#)).

Noncustodial parents and those who consult with them would be well advised to emphasize how the noncustodial parent can persuade and make worthwhile voluntary agreements regarding visitation and other arrangements that will promote improved relationships of both parents in the service of the child's progressive development. For example, support by the noncustodial parent, not limited to money but including availability for child care, for doing errands, and for helping the custodial parent with various demands he or she confronts in maintaining the child's home, can be very useful if presented as a voluntary, nonthreatening option.

## CLINICAL EVALUATION

When the evaluation is for court purposes, what is the overall task of the child expert, and how is the task accomplished? The clinical evaluation in custody or placement issues, although similar to the psychiatric evaluation, is different in its goals. The purpose of a child psychiatric evaluation is to arrive at a psychodynamic formulation of the child's presenting problems, a diagnosis, and a treatment plan recommendation. Ordinarily, it is conducted on a voluntary and confidential basis. In a custody evaluation, the question is posed by the court. Usually, a custody conflict requiring a court-ordered evaluation signifies that the parents are unable to cooperate voluntarily in planning for custody and visitation arrangements. The questions are as follows: With whom should the child live? Which parent should have legal decision-making authority regarding education, medical decisions, religion, financial support, vacations, and so forth? How can the opportunities for the child to maintain a useful and sound relationship with both parents be optimized?

Intractable postdivorce conflict usually has a noxious and long-term negative impact on children ([Emery, 1982](#)). It is crucial that throughout the evaluation process the child expert support whatever capacity the parents have or can recover to resolve their own disputes, to diminish conflict, and to place themselves in the child's shoes in guiding themselves to be the best parents they can be for their child. Even experienced experts may find themselves unwittingly drawn into the family dispute because all adults, no matter how well trained, drift toward an identification with one of the conflicting adults. At times, the evaluator may experience a resurgence of her or his own childhood family struggles, especially if divorce is a significant experience in his or her own past.

The first practical step is to clarify as much as possible the court's questions. In complex cases (particularly foster care placement cases), it is advisable to obtain a copy of the motion for evaluation. The evaluator can request that the court furnish the exact questions to be addressed in the evaluation. This phase of the process is significant, because it initiates the attempt at a dialogue between the professionals in the two intersecting fields. The legal side usually values winning, establishing fault, being explicit, and meting out "justice"; mental health and developmental experts traditionally value helping and promoting understanding while tolerating ambiguity and ambivalence. During this phase, it may be necessary for the clinician to explicate the nature and limits of his or her expertise to the court.

Some clinicians will agree to be retained by one side or the other in a case. There are several reasons to insist that the court order the evaluation. It enables the expert to state effectively that he or she serves as an expert on behalf of the child and strives to be neutral with respect to the adult because the child's interests are paramount. Court-ordered evaluations discourage multiple evaluations, which constitute an undesirable intrusion and burden for the child. When the evaluation is ordered by the court, the report of the evaluation is sent to the court. Each court has its own policy with respect to making the report available to the lawyers in the case. Lawyers sometimes request that the consultant furnish them with the report even when this would be improper. When one is in doubt about any aspect of the legal proprieties, the court clerk is usually the appropriate person to consult.

Financial arrangements need to be attended to carefully. Many clinicians require a retainer fee before embarking on the evaluation because they have learned that, in adversarial settings, the child expert's bill may be overlooked or may be deliberately left unpaid by disgruntled parents even when a court order obliges them to pay the costs. Whatever the fee arrangements, they should be clarified at the outset, including the charges for expenses entailed in reviewing case materials, preparing the report, and testifying in court.

Outside materials should not be used without signed permission of those involved or through approval by the court. In divorce cases, the child's pediatric and school records should be obtained, because they provide essential information including the child's educational family, developmental, and health history. It is optimal if teachers or day care providers can write a paragraph about the child's school experience to supplement checklist forms that are used by many experts. If the child is or has been in counseling or psychotherapy, a brief report with the informed consent of the parents should be requested to include the presenting complaint, diagnosis, and progress or termination summary. If such an intrusion into the confidentiality of an ongoing treatment would significantly disturb the treatment, the question should be raised whether the additional information is warranted, considering the burden it would place on the child's or adult's treatment. All such reports should be in the form of written documents that can potentially be reviewed by anyone in the court proceeding. Verbal reports, that is, hearsay, do not carry the same weight in court. Drug and alcohol treatment, psychiatric hospitalization, and criminal records concerning the adults are pertinent for review.

Clinicians should be familiar with the guidelines developed by their professional disciplines. These include the following: "Practice Parameters for Child Custody



Evaluation" (*Journal of American Academy Child Adolescent Psychiatry*, October, 1997); "Guidelines for Child Custody Evaluations in Divorce Proceedings" (*American Psychologist*, July, 1994) and "Guidelines for Psychological Evaluations in Child Protection Matters" (*American Psychologist*, August, 1999).

## CLINICAL INTERVIEW

In preparing for the report and court testimony, the child expert evaluates the child's developmental and emotional level of functioning and describes what the child's needs are and whether there are special needs to be taken into account. The central issue is the child's relationship with the competing caregivers. As Anna [Freud \(1971\)](#) points out in her 1968 comment on the *Painter v. Bannister* case:

Clinical and analytic experience teaches us that children can fill many different roles in the emotional lives of their parents or of other adults. They may be no more for them than a piece of property which is valued egoistically as an extension of the adults personality, when this happens they serve the adult's needs, while their own developmental needs remain unconsidered. In contrast to this, they may be loved unselfishly, as persons in their own right, with their own needs of paramount importance. Further, they may be no more than a pawn in a game, with no importance of their own, except that possession or dispossession of their persons signifies victory or defeat for the warring factions.

The task of staying focused on describing the child's needs in a custody case is difficult for all adults, including the clinician. One significant source of difficulty is the adult's effort to identify with the child's sense of helplessness in the midst of unrelenting adult conflict. Because of the demanding nature of these cases, many clinicians pace themselves cautiously as to the number of custody evaluations they undertake. Specializing in such difficult work increases the risk that one's approach will become mechanical and superficial. Retaining a generalist's view lowers that risk and emphasizes that each case will be evaluated as unique.

In the consulting room with the family, it is useful and appropriate to review the purpose of the evaluation, its lack of confidentiality, the legal motives for the evaluation, and the requirement of submitting a written report to the court. Similarly, it is important to clarify that the report and testimony, where indicated, is designed to assist the judge because the judge, not the evaluator, has the authority and responsibility to make decisions.

The evaluator begins by meeting with the child's parents, usually individually. A sufficiently detailed history is taken to provide a general sense of the parents' own background, personality, and level of functioning. If severe character pathology or a psychiatric illness that interferes with parenting is evident, a more systematic mental status examination and diagnosis may be desirable. This may indicate the need for further interviews and formal psychological testing. The parents are each asked to describe the child's developmental history, to explain how they get along with their child, and to discuss what the parent believes would be the best arrangement for the child. A discussion of the cause of the breakdown of the marriage and an idea of why the parents cannot cooperate with each other may provide further insight by which to gauge the intractability of the conflict and to clarify realistic options for the child.

## EVALUATING THE YOUNG CHILD

The child's age and developmental level influence the interviewing process. The appropriate method for assessment of children younger than 5 years old is through play and the administration of a developmental examination. A systematic developmental evaluation for younger children, such as the Yale Developmental Scales, provides an examination of five domains: gross motor, fine motor, speech and language, adaptive, and social functioning (see [Chapter 40](#)). Such an assessment affords a reliable method of determining the child's level of functioning as well as a way of screening for deviations and vulnerabilities. Constitutionally and environmentally generated difficulties can be identified in formulating a child's special needs.

From the history, the developmental examination, and the play situation with younger children, useful inferences can be made about the child's patterns of attachment, separation, mood regulation, and bodily functions. In this systematic way, the clinical child expert prepares an inventory that becomes the basis for making recommendations to the court as well as for acknowledging the limits of what can be determined in such consultations. It is both tactful and useful in maintaining the primary focus on what is the least detrimental alternative for the child, to avoid "scapegoating" the parent who the expert feels should not be the custodial parent.

The developmental and clinical (play) assessments are also used to explore how well each contending party meets the child's needs for feeling wanted, for providing continuity of affectionate care, for uplifting expectations, and for safety. Having at least one adult who meets these needs of the child is essential in supporting the child's capacities for self-regulation and in establishing development-promoting attachments. In turn, these capacities enhance the child's ability to be comforted and soothed, to engage in age-appropriate contacts with peers, and to feel comfortable in the care of substitute caregivers.

## EVALUATING THE SCHOOL-AGE CHILD

Both the developmental and play assessments of a younger child usually occur in the presence of a parent, and this provides the opportunity to observe and differentiate the child's experience with each parent. The school-age and preadolescent child should be interviewed individually as well as together with each parent. Some evaluators find it useful to structure the interview with the parents to include a "task" the child and parent are asked to work on together. Leaving the interview unstructured can provide an opportunity to observe the child and parent in spontaneous interaction such as at play together, in conversation, in silence, and in conflict. In these observations, the quality of the child's relationship to the parent can be noted: Is he or she engaged, distant, apprehensive, responsive, avoidant? The play interview is generally considered the most useful process in assessing a child of this age who has not yet developed the cognitive ability to rely on conceptual thinking and on language for full expression.

Discussion with the school-age child of the reasons for the evaluation, including the courses interest in the custody or visitation issue, can be informative and fruitful when comfortable and handled sensitively. The interview may include drawings, thematically developed play, and occasionally the opportunity to evoke tactfully, often indirectly (e.g., "let's pretend"), the child's opinions and desires with respect to the custody and visitation issues. Play and drawings are phenomena that may reflect a child's imagination, fantasies, conflicts, and life experiences, as well as developmental level and intelligence.

Direct questioning of the child about a preference in living arrangements is risky because it often increases the child's anxiety about loyalty conflicts. Asking the child to choose one parent over the other may increase the child's fear of losing the other parent, thus involving a painful confrontation with the child's strong wish to have the parents stay together, albeit more as mother and father than as husband and wife. When the child's preference can be determined, it is taken into account as an important but not decisive factor. The weight given to the school-age child's preference should be assessed individually in each case.

## EVALUATING THE ADOLESCENT

Adolescents need not be seen routinely in custody assessments, because they are considered able to play an active and often decisive role in making their own choices. Occasionally, in special circumstances in which issues of physical safety, delinquent or behavioral problems, or a parent's mental illness are involved, an assessment may be required. Before the age of formal operational thought, which occurs around 11 years, children have limited ability to project the idea of themselves in the future and to sense how the past will influence the future. Beginning in early adolescence, cognitive and psychological development, including the push toward increased independence, will enhance the adolescent's capacity to be more active in custody decisions. With support, the older child and adolescent can usually begin to assess his or her own needs and to form sound judgments based on actual life experiences about custody and visitation arrangements (Schowaher, 1979).

## COURT REPORT

Once the clinical evaluation is completed, the report is prepared for the court. It reiterates the court's questions, cites all reviewed reports and documents, identifies all persons interviewed and specifies the dates and time spent in all interviews and reports, summarizes historical and clinical interview material, and concludes with a summary and recommendation. Court testimony is discussed in [Chapter 114](#). In child custody and placement cases, it is particularly important for the expert to review his or her testimony with the child's attorney when possible, to remain focused on the child's point of view, and not to go beyond professional expertise and knowledge. The court's interpretation of a New Jersey statute, for example, requires the expert to testify to a prediction that a child's removal will cause irreparable harm. This poses a dilemma for the expert because no one can predict with accuracy the long-term future outcome of such a placement change or the development of future relationships (1). As child experts, it is essential and valid to use the capacity for short-term predictions to indicate what immediate psychological injuries can occur if the child's best interests are corroded by the passage of time. The child should not be exposed to the risk of avoidable injury to put fairness to the contending

adults ahead of what would be least harmful to the child.

The nature of the child's relationship with each of the contending adults may enable the expert to document that one parent is more of a primary psychological parent than the other, despite socioeconomic consideration, or that each of the contending adults, for different reasons, is a primary psychological parent. In the latter instance, if the parents cannot decide and prefer that a stranger, the judge, decide, the expert should indicate that each of the parents is a primary psychological parent, equally competent for different reasons. If the parents or contending adults cannot decide or agree, the court should use the principle of least detrimental alternative to meet the best interest standard of making a decision and should not allow the passage of time to be corrosive to the child while adults debate how to be fair to each other.

## PLACEMENT EVALUATIONS

Unlike the desirability of private dispute resolution involved in divorce cases, foster care cases involving placement disputes are more regularly subject to court process and decision making. The court regulates the functioning of the state child welfare bureaucracy by imposing time limitations on temporary custody arrangements. Through its terminations of parental rights process, the court legalizes permanent placements for children who cannot return to the care of their biological parents. The questions are as follows: What is the best permanent placement for the child? Can the child ever be returned to the biological parents?

The child expert evaluating cases of abuse, neglect, or abandonment follows the previously discussed placement principles. The need for permanency and security is essential in these cases. Whereas divorce conflict cases pose the difficulty of raw familial passions, child welfare cases present a different challenge. Often, the children to be evaluated have already suffered grave psychological damage as a result of chronic deprivation, multiple placements, and other conditions noxious to the developing child. It may be frustrating to the evaluator to recommend an outcome for the child that, although it may be the least detrimental, seems so far from ideal. An evaluator's inability to tolerate the available least detrimental alternative for the child can lead to harmful delays and postponements and recommendations for an unknown or unrealistic solution. Such delays and indecision can inflict further psychological damage on the child. The child's sense of time should be taken into account in not delaying, beyond the younger child's tolerance, the finding of at least one qualified adult who can provide the continuity of affectionate care as described earlier. It is important to observe the child's current realities and to make the best of what is available.

Evaluations involving allegations of sexual abuse, when there is a finding of physical damage, require the child to be ensured of protection from further abuse ( [Solnit et al., 1992](#)). This means exclusion of the abuser, if that person can be identified, or a documented change in the child's circumstances that addresses the source and cause of the problem, as much as it can be determined. When the pediatric examination has been unable to document physical injury, the evaluator is unlikely to be able to know with certainty whether abuse occurred. Therefore, the mental health expert should not accept a request to determine whether sexual abuse has occurred, but should suggest the request be refocused to a psychological assessment of the child's general development and relationship with the caretakers for the purpose of determining safety, placement, visitation, or custody issues.

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### CASE ILLUSTRATION: *Ireland v. Smith*

In this Michigan case, a 15-year-old unmarried adolescent girl gave birth to a daughter who was raised in the mother's home with the mother as the custodial parent, assisted in child care by the maternal grandmother and the maternal aunt, while she completed her high school education. When the child was 1 year of age, the father, who had acknowledged paternity at birth, began to visit.

When the child was 3 years, 2 months of age, the mother filed a suit seeking the father's child support in preparation for her anticipated move to pursue a college education and to live independently with her daughter. The father then petitioned for custody of the child.

The judge found that the mother had established a custodial environment for the child, that is, that the child looked to the mother for guidance, discipline, and the necessities of life. He found that the day-to-day care of the child was provided by the mother and that the child was acquainted with her father but in a way that was of necessity limited by the structure of the visiting relationship.

### Comment

In this case, the judge described himself as attempting to reach a decision that would represent "the child's best interest." He followed the state statute in examining the relative abilities of each parent to provide affection, guidance, and other material needs. He found the central issue to be maintaining continuity, but he construed continuity to mean of physical environment rather than of the child's primary tie to the mother. He wrote:

The mother's program would require that the child be in day care. The child was in a program at the University of Michigan which apparently was appropriate and resulted in the child having a meaningful experience. It would be the mother's intention to continue this on until such time as she either graduates, as previously stated, or her marital circumstances somehow change. For the purpose of deciding this case, the Court would have to assume that it is the mother's intention to maintain the child at the University of Michigan for some considerable number of years. This issue is pivotal.

The judge decided to intrude into and violate the integrity of this child's ongoing adequate family and concluded that the child's best interest was to be moved to the father because, during the day when the mother was in class, the paternal grandmother, a "blood relative," would be preferable to a day care program for the child. Thus, while espousing the central importance of the principle of continuity in the life of a young child who is developing well, he rendered a decision contrary to the principle. The judge did not make reference to any involvement by a child expert whose opinion could have assisted him to accomplish his stated goal [\(2\)](#).<sup>3r</sup> (

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### CASE ILLUSTRATION: *Prost v. Greene*

In the *Prost v. Greene* case the parents separated after an 8½-year marriage, at first under the same roof and then 9 months later in separate domiciles when the mother received temporary physical custody with liberal visitation for the father. In a lengthy trial, each parent sought divorce, support, property, and custody of their sons 7 and 4 years of age. Even though the children were doing well in a custody arrangement that had been in place for more than 1 year, the judge reversed the custody and awarded sole legal and physical custody to the father. In her opinion, the judge wrote "...it is plain to the court (and should be to any other objective observer) that plaintiff [the mother] is simply more devoted to and absorbed by her work and her career than anything else in her life, including her health, her children and her family."

### Comment

For the child expert, it is instructive to observe that the judge, finding the expert testimony of the child psychiatrist "unpersuasive," complained that the expert "seemed overly focused on the parents' emotional makeup and on the relationship/conflict between them, rather than on the best interest of the children as evidenced by their current psychological status and relationship to each parent." In the absence of convincing testimony explicating the developmental needs of each child and the nature of the children's relationship with each parent, the judge relied on her own intuition, value judgments, and personal reactions to the parents' personalities.

Apparently, the needs of Matthew, age 7, and Jeffrey, age 4, were not credibly articulated in the courtroom and therefore did not form the central consideration in the decision. Rather, the judge in this case wrote:

...Children learn from the intensive presence, participation and involvement of a parent in their lives, from the frequent and sincere demonstrations of a parent's love and concern for them, from a parent's efforts to help them solve their problems, and from their observation that a parent enjoys being with them and, indeed, has fun with them. On all these fronts, the evidence is clear, defendant has it way over plaintiff.

The judge's language demonstrated how, in the powerful current of divorce conflict, there is a tendency to be drawn into taking sides and to focus on the adults. The judge further overstepped the bounds of her role when she chided the parents, "The parties should be ashamed of themselves, but it does not appear that they are." The judge's reproach of the parents prevented her from conveying the "experience, wisdom, and compassion...to fashion an appropriate [3](#).nedy" (

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### CASE ILLUSTRATION: BABY RICHARD

In this case, Richard's parents began living together in the fall of 1989. Daniella, Richard's mother, became pregnant in June 1990, and Richard was born March 16, 1991. Otakar, Richard's father, lived with Daniella and provided for all of Daniella's expenses during her pregnancy until late January 1991, when he returned to his native Czechoslovakia to attend his gravely ill grandmother. Daniella was informed by a member of Otakar's family that he had reinvolved himself in a romantic relationship with a former girlfriend. Daniella felt so betrayed and distressed by this news that she fled the apartment she shared with Otakar, avoided contact with him, and then informed him that their baby had died. As a single mother, she executed a consent for adoption 4 days after Richard was born. Daniella refused to furnish Otakar's name to the adoptive parents or to their lawyer, although she did inform them that she knew his identity.

When Richard was 57 days of age, Otakar learned that Richard was alive and had been placed in adoption. Otakar married Daniella and immediately began legal efforts to recover custody of his son. For the next 4 years, legal proceedings postponed the decision for Richard. First, the trial court ruled that Otakar did not show sufficient interest in the child in his first 30 days of life and therefore was found to be an "unfit" parent. As an "unfit" parent, Otakar's consent for adoption was not necessary. The appellate court then confirmed the trial court, by citing a best interest standard that at age 2 years, 5 months, Richard's adoptive parents were the only parents he had known. The judge wrote of Richard, "He has not touched or seen Daniella since four days after his birth and he has never spoken a word to her. Nor has he ever touched, seen, or communicated with Otakar. In fact, he is totally unaware of the existence of Daniella and Otakar." He further noted, "Since Richard was a newborn, John and Jane Doe have done everything with Richard that is the essence of being parents, and Richard has done everything with them that is the essence of being a son."

The Supreme Court of Illinois heard the appeal and reversed the decision of the trial and appeal courts, resulting in Richard's placement at 4 years of age with his biological parents, who were total str<sup>4</sup>gers (

### Comment

The psychologically distinctive role of the father begins early on with his anticipation of the child's birth. Through holding, feeding, protecting, and playfully interacting with the baby, he becomes a responsive and competent caretaker, central to the child. The father's presence is positively associated with the child's developmental progress [Pruett, 1987](#)). Separation from such a parent to whom the child is emotionally tied can cause shattering loss, regression, and disruption to the child's sense of stability and security.



In the case of Richard, Otakar could not have been considered Richard's "father" in any but a biological sense. The legal system, by its delay of 4 years and by its awarding a child rather than other compensation to Otakar, failed to uphold its best interest standard. The court recognized that a grave injustice had been done to Otakar—first by Daniella, who concealed Richard's existence from Otakar, then by the state, which concealed Otakar's identity and whereabouts from the adoptive parents and their lawyer, and finally by the failure of the adoptive parents and the various legal representatives to make a decision based on the child's best interests. This case illustrates the way in which the child's best interest comes to be subordinate to the court's priority of fairness to an aggrieved adult ( 4). The best guide for the child expert is: Put yourself in the place of the child.

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# 116 EPIDEMIOLOGY AND PREVENTION

David R. Offord, M.D., and Kathryn J. Bennett, Ph.D.

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The epidemiology and prevention of child and adolescent psychiatric disorders are closely tied together. On the one hand, data from epidemiology support the need for effective prevention programs and provide essential data for the prevention enterprise in child psychiatry. On the other hand, the results of prevention initiatives can add to our understanding of aspects of epidemiology such as the identification of true causal risk factors.

*Epidemiology* is concerned with the study of child psychiatric disorders in *populations* of children and adolescents (Lilienfeld, 1976). It differs from clinical work, which deals with children (and their families), usually one at a time, who actively seek help because of troublesome symptoms or behaviors and associated impairments. Epidemiology is concerned not just with the prevalence and distribution of child psychiatric disorders that can be obtained from cross-sectional studies, but through prospective studies, descriptive epidemiology is also concerned with the incidence or new cases of disorder, the temporal sequence of disorders, the discovery of causal risk and protective factors, and the developmental pathways along which risk and protective factors operate over time to produce healthy and unhealthy outcomes. Experimental epidemiology (Robins, 1978) moves into the prevention area, with its concern of establishing whether or not a risk factor is causal for a disorder. It accomplishes this by determining whether an intervention program aimed at reducing the incidence of the risk factor results in a reduction in the incidence of the disorder of interest. Figure 116.1 draws attention to the two uses of epidemiologic data, scientific and administrative or related to service (Earls, 1989; Robins, 1978). On the administrative side, the figure illustrates the differences among primary, secondary, and tertiary prevention by pointing out where in the development of the disease process the different strategies apply. Primary prevention comes into play before the initiation of the pathologic processes in which successful interventions can prevent the onset of disease. Secondary prevention focuses on altering the expression of disease after its onset, whereas tertiary prevention is concerned about minimizing the severity of disease and promoting recovery and remission (Caplan, 1964; Cowen, 1983). The figure also illustrates that, on the scientific side, epidemiology is concerned about causal risk factors involved not only in the onset of disease or disorder, but also in its subsequent course in terms of persistence, remission, and relapse.

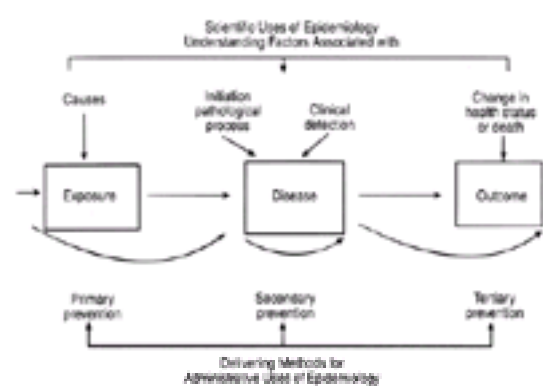


Figure 116.1. The uses of epidemiology.

The case for prevention of child psychiatric disorder rests on the limitations of clinical services to reduce the burden of suffering from these conditions. In the first instance, there is a mismatch between the number of children in the community with clinically important psychiatric disorders and the ability of clinical services to assess and treat them (Boyle and Offord, 1988; U.S. Congress, 1986). Findings from community surveys indicate that the prevalence of one or more psychiatric disorders varies between 17.6% and 22.2% (Costello, 1989; Costello et al., 1996). Further, in a more recent article of 52 separate studies with data on the prevalence of psychiatric disorder in children and adolescents in community populations, the mean prevalence was 15.8% (Roberts et al., 1998). The prevalence rate drops, of course, in one study to 11%, if the diagnostic criteria require significant functional impairment at home, at school, and with peers (Shaffer et al., 1996). It is estimated, however, that approximately 6 million to 9 million children in the United States have serious emotional disturbances (Friedman et al., 1996; Lavigne et al., 1996), and this accounts for 9% to 13% of all children (Friedman et al., 1996; Friedman et al., 1998). Given these prevalence estimates, it is very unlikely that a jurisdiction will be able to finance and staff the clinical services required to serve children with established psychiatric disorders adequately. To support this contention, many young people in the United States with a diagnosable mental disorder do not receive any mental health services at all (Burns et al., 1995; Leaf et al., 1996). A report in the mid-1980s (U.S. Office of Technology Assessment, 1986), indicated that approximately 70% of children and adolescents in need of treatment did not receive mental health services. A more recent report (Burns et al., 1995) indicated that only one in five children with a serious emotional disturbance used mental health specialty services, and most of these children failed to receive any services at all as reported by their families. A second limitation of the clinical enterprise centers on rates of compliance with assessment and treatment regimens. For example, many parents of children at high risk of emotional and behavioral problems do not enroll in parent training programs even when they are widely available (Cunningham et al., 1995; Cunningham et al., 2000; O'Donnell et al., 1995). Further, of families who begin treatment because of their child's emotional or behavioral problems, 40% to 60% terminate treatment prematurely (Kazdin, 1996). In addition, Armbruster and Fallon (1994) found that most children who enter outpatient treatment attend for only one or two sessions. Third, considerable time can elapse between when parents seek help for their child and when they actually receive assessment and treatment services. During this time interval, the child and his or her family can be expected to suffer, and perhaps the disorder and associated impairments will worsen and become more entrenched. Fourth and last, although considerable progress has been made in discovering promising treatments for child psychiatric disorders (Kazdin and Weisz, 1998), research is needed to demonstrate the effectiveness of treatments for children in actual practice settings as opposed to evidence of efficacy in controlled research settings (Weisz et al., 1995). In addition, there is no evidence that enriched systems of care for child mental health problems result in improved clinical or functional outcomes, (Bickman et al., 1997; Bickman et al., 1999). The considerable limitations of clinical work alone to reduce the suffering from emotional and behavioral problems in children has motivated not only researchers and policy makers but also clinicians to examine carefully the possibilities of developing and evaluating prevention efforts (Durlak and Wells, 1997; Institute of Medicine, 1994).

This chapter first covers selected aspects of epidemiology and then moves on to issues and findings in the prevention field. The epidemiology section begins with a



discussion of issues and then provides a summary of major findings.

## ISSUES IN EPIDEMIOLOGY

Using data on the epidemiology of conduct disorder, hyperactivity, anxiety disorder, and affective disorder, selected issues in child psychiatric epidemiology are now discussed.

### Measurement of Disorder

#### BOUNDARY BETWEEN NORMAL AND ABNORMAL

Two different approaches are used for identifying disorder in child psychiatric epidemiology ([Achenbach, 1997](#); [Jensen et al., 1999](#)). In the first, termed the *categorical approach*, the nosologic categories of psychopathology are chosen by a committee of experts who then decide what criteria are required to be fulfilled for an individual patient to qualify for a particular disorder. This strategy is viewed as a “top-down” approach, whereas the second strategy, the *empirically based approach*, works from the “ground up.” Statistical techniques are used to derive sets of items that occur concomitantly and thus can be viewed as syndromes. The validity or usefulness of the resulting diagnostic categories is judged by the extent to which the placement of a child in a diagnostic category provides some added information about the cause, course, prognosis, and response to treatment in the child with the disorder. These approaches mirror the debate about whether child psychopathology is better conceptualized as a categorical or a dimensional phenomenon. More recent work suggests that both approaches are useful ([Achenbach, 1997](#); [Jensen et al., 1999](#)).

Regardless of which of the two approaches is used, clinicians, administrators, and policy makers want to be able to know when a child qualifies as having a clinically important psychiatric disorder. This necessitates setting a threshold based on symptom frequency and severity that, when a child exceeds it, leads the clinician to state that the child is said to have a disorder; when the child is below the threshold, he or she is classified as not having a disorder. The setting and justification of a threshold identifying children with and without disorder are critical issues in child psychiatry. For example, changing a threshold for disorder even slightly can have noticeable effects not only on prevalence rates but on patterns of correlates and comorbidity ([Boyle et al., 1996a](#); [Robins, 1985](#)). The threshold problem extends beyond symptoms to other aspects of criteria for disorder such as impairment. Again, measures of impairment usually are on a continuum, and the issue of where the threshold should be set to identify clinically important impairment must be addressed. Unfortunately, there is a paucity of data justifying existing thresholds for disorder in child psychiatric epidemiology. This immediately raises the issue of the meaningfulness of cases identified in community surveys and, in turn, raises questions about the implications of the results of these surveys for estimating service needs.

#### COMORBIDITY

In cross-sectional epidemiologic studies of children in the general population, rates of concomitant occurrence of child disorders, far in excess of chance, have been reported ([Anderson et al., 1987a](#); [Bird et al., 1993](#); [Costello et al., 1988](#); [Costello et al., 1996](#); [Fergusson et al., 1993](#); [Offord et al., 1987a](#); [Velez et al., 1989](#)). In the Ontario Child Health Study, 67.8% of the children, 6 to 16 years old, with a diagnosis of at least one of the four psychiatric disorders had one or more additional diagnoses ([Offord et al., 1989](#)). More recently, in the Great Smoky Mountain Study, 100 of the 307 children with a diagnosis had more than one disorder, and of those with behavior disorder, comorbidity was 38% ([Costello et al., 1996](#)).

Certain factors can promote high rates of comorbidity ([Caron and Rutter, 1991](#)). They include the following: setting low thresholds so prevalence rates of individual disorders are high; detection artifacts; problems of classification, for example, using the same symptoms to define different categories of disorder; and when two disorders coexist in cross-sectional studies but one of them is an early manifestation of the other. The high rates of comorbidity have raised the issue of the extent to which individual disorders are separate entities, that is, with a distinctive origin, pattern of family history and associated features, prognosis, and response to treatment ([Robins and Guzé, 1970](#)). Although, as just noted, methodologic factors can contribute to high rates of comorbidity, current evidence strongly supports the idea that methodologic factors are not a major cause of comorbidity ([Angold et al., 1999](#)). Developmental psychopathologists use the concepts of homotypic and heterotypic continuity ([Angold et al., 1999](#)). This approach leads to two types of comorbidity, namely, one that examines comorbidity between disorders within a diagnostic grouping (homotypic; e.g., oppositional defiant disorder and conduct disorder), and another that deals with comorbidity between disorders from different diagnostic groupings (heterotypic; e.g., depression and conduct disorder). An elegant review ([Angold et al., 1999](#)) not only discusses the causes of homotypic and heterotypic comorbidity but summarizes the literature on comorbidity rates of different disorders. An important deficit in the comorbidity literature is the lack of knowledge about the sequence of psychiatric disorders in childhood, although evidence indicates that hyperactivity precedes conduct disorder ([Offord et al., 1992](#)).

#### DISAGREEMENTS AMONG INFORMANTS

There is a low agreement among informants about children's behaviors. This finding applies to both self-administered problem checklists ([Achenbach et al., 1987](#)) and lay-administered structured interviews ([Boyle et al., 1993a](#); [Edelbrock and Costello, 1988](#)), and it cannot be accounted for by poor reliability or lack of validity of assessment procedures. The correlations among informants are usually less than 0.30, and thus no one informant can substitute for all others ([Achenbach, 1997](#)).

A corollary of the lack of agreement among informants is that how many children and which children are identified as having a disorder depend on which informant has provided data for the assessment. For instance, in the Ontario Child Health Study, the prevalence of parent-identified conduct disorder for children 6 to 11 years old is 1.4%, whereas the prevalence of teacher-identified conduct disorder is 4.9%, and further, low family income has a strong independent relationship with teacher-identified conduct disorder, but not with parent-identified conduct disorder ([Offord et al., 1989](#)). Thus, both prevalence rates of disorder and their associated features will be affected by the informant who provides the data. Further, the predictive value of assessment data used to classify disorder has been reported to vary by informant ([Boyle et al., 1993b](#); [Loeber et al., 1991](#)).

These findings suggest that the identification of childhood disorder is influenced by both the contexts in which the assessments are carried out and the perception of informants. Unfortunately, the factors influencing informants' perceptions of children's behaviors are not well understood. For instance, whereas some studies have found that a mother's mental health (particularly her level of depression) is a strong predictor of mental health problems expressed by her children, the validity of the finding is in doubt because most of these studies have relied on maternal reports as indicators. However, a recent study ([Najman et al., 2000](#)) reports that anxious or depressed mothers of 14-year-old children tend to report more cases of child behavioral problems than not only their mentally healthy counterparts but also the children themselves. This finding raises questions about the validity of reports of child behavior by persons who themselves are experiencing emotional distress. It is not known which informants provide the best information for classifying children into groups that are valid and provide useful data on the usual criteria of etiology, prognosis, and response to treatment. Several strategies have been developed to deal with the dilemma that different informants identify different children as being disordered. They include the following: combining information, that is, giving value to any positive rating regardless of informant; giving priority to different informants about different kinds of behavior, for example, assigning priority to the child's report of internal mood and to the parent's report of behavior problems; assigning a confidence rating to each behavior proportionate to the amount of agreement among observers; and considering disorders informant specific and making no attempt to combine reports from different informants to provide an overall classification independent of informants ([Institute of Medicine, 1989](#); [Offord et al., 1989](#); [Offord et al., 1996](#)). Again, as with data from individual informants, it is not known which of these approaches of combining inputs from different informants results in the most useful and valid classification system.

#### INSTRUMENTATION

Three types of instruments are used to measure psychiatric disorder in community surveys: structured interviews (e.g., the Diagnostic Interview Schedule for Children) ([Shaffer et al., 1996](#)), semistructured interviews [Schedule for Affective Disorders and Schizophrenia for School-Age Children e.g., the (K-SADS)] ([Chambers et al., 1985](#)), and checklists (e.g., those used in the Ontario Child Health Study) ([Boyle et al., 1993a](#)). Checklists have a major advantage and disadvantage compared to interviews. Because they are self-administered, they are cheaper, but there is no opportunity to clarify wording or to probe responses, for example. Three studies compare the classification of psychiatric disorders by checklists versus structured interviews among children and youth in community populations, and in none of the studies was one strategy superior to the other, although they classified many different children as being disordered ([Boyle et al., 1996b](#); [Boyle et al., 1997](#); [Gould et al., 1993](#)). Further, the two kinds of interviews resulted in different prevalence rates of disorder ([Edelbrock and Costello, 1988](#)). Semistructured interviews focus on specificity, with higher thresholds and fewer children exceeding the cutoff for disorder, whereas structured interviews center on sensitivity, with lower diagnostic thresholds, resulting in more children reaching criteria.

Two major issues in this area are the difficulty in assessing psychopathology in young children and the unsureness of what to use as the “gold standard” in deciding

on the validity of cases identified in community surveys. Everyone agrees on the importance of obtaining the child's viewpoints of his or her emotions and behaviors, and there is an understanding that data from informants such as parents or teachers cannot substitute for information received directly from the child ( [Harter, 1982](#)). One problem is that it is difficult to obtain reliable interview data from children less than 11 years of age ( [Achenbach et al., 1987](#); [Edelbrock et al., 1985](#)) unless one uses a pictorial instrument such as the Dominic questionnaire ( [Valla et al., 1994](#); [Valla et al., 2000](#)). Concerning the second issue, establishing the standard for evaluating the validity of diagnostic categories identified by instruments in community surveys, usually the clinician workup is the standard that applies. Regardless of what data the clinician uses to arrive at a clinically important diagnosis, the procedure must have adequate reliability, both interrater and test-retest reliability.

### Measurement of Other Variables

All the issues that apply to the measurement of disorder pertain to the measurement of other variables. For example, as noted previously, the measurement of impairment is on a continuum, and what is needed, as with disorder, is a meaningful threshold that distinguishes children with and without clinically important impairments. Further, data on the presence, type, and severity of impairments are informant specific ( [Sanford et al., 1992](#)). There is a high degree of agreement between the presence of disorder and impairment within a particular informant and a low level of agreement between the two across informants. What is required are reliable and clinically important measures of impairment that are objective, that is, independent of data from parents, teachers, and children. One last point is that measurement error in correlates plays an equally important role to error in the measurement of disorder in distorting the strength and form of the relationship between a correlate and a disorder. Improving the reliability of measurement has been shown to result in larger associations between correlates and childhood psychiatric disorder, and it may increase the opportunity of revealing differential associations among variables ( [Boyle and Pickles, 1998](#)).

## FINDINGS IN EPIDEMIOLOGY

### Selected Correlates

#### INDIVIDUAL CHARACTERISTICS

##### *Age and Sex*

The prevalence of one or more psychiatric disorders in children and adolescents increases with age. In one review ( [Roberts et al., 1998](#)), the mean prevalence rates for preschool children, preadolescents, and adolescents were 10.2%, 13.2%, and 16.5%, respectively. Regarding sex differences, findings from community studies indicate that externalizing problems are more prevalent in boys, and internalizing problems are more prevalent in girls ( [Achenbach et al., 1991](#); [Anderson et al., 1987b](#); [Offord et al., 1987b](#); [Verhulst et al., 1985](#)). In the preadolescent age group, psychiatric disorder is more common in boys because of the high rate of externalizing problems in boys; in adolescence, girls have a higher rate of disturbance than boys, primarily because of the marked increase in internalizing problems among girls ( [Offord et al., 1987b](#); [Zahn-Waxler et al., 2000](#)). Studies have shown considerable stability of externalizing problems over several years ( [Cohen et al., 2001](#); [Feehan et al., 1993](#)), and recent evidence suggests that the stability of internalizing problems may be at least as high from childhood to young adulthood ( [Hofstra et al., 2000](#)). These findings support the contention that the early onset of problems is prognostic of chronic and serious problems in adolescence and beyond ( [Moffitt, 1993](#); [Rutter, 1997](#)). Because most of the studies of internalizing problems have focused on girls, and most studies of externalizing problems have focused on boys, little is known about gender differences on timing of onset of specific disorders and their subsequent mental health outcomes.

##### *Racial and Ethnic Minorities*

The prevalence of child psychiatric disorders has been reported to vary by race and ethnicity ( [Costello et al., 1997](#); [Roberts and Chen, 1995](#)). A survey of high school students in New Mexico found that depressive symptoms were more common in Mexican-Americans than Anglo students ( [Roberts and Chen, 1995](#)). A previous population survey of 10-year-old children in an inner London borough revealed that, based on teacher data, but not on parent information, there were elevated reports of behavioral problems, but not internalizing symptoms in West Indian children ( [Rutter et al., 1974](#)). However, not all groups of nonwhite children have increased rates of psychiatric disorders. For example, in a sample of 181 Chinese-American children, ages 5 to 17 years, attending a Chinese school in New York city, the rates of internalizing, externalizing, and total problem scores were significantly lower than Achenbach's American norms by age and sex ( [Chang et al., 1995](#)). Two major limitations of this work are the low response rate to the study (38%) and the inclusion of only one school. Prevalence rates of emotional and behavioral problems in Native North American children vary markedly among tribes and regions ( [Yates, 1987](#)). Finally, in a report from the public-use subsample of the National Longitudinal Study of Adolescent Health (Ad Health) ( [Bearman et al., 1997](#)), the authors compared the psychosocial adjustment of U.S. adolescents, grades 7 to 12, among three groups: single-race white (both parents white only), single-race minority (parents of same minority status), and multiracial (parents of differing racial or ethnic background) ( [Cooney and Radina, 2000](#)). Significant differences between the multiracial adolescents and the two groups of single-race adolescents were found on fewer than half of the school, behavioral, and psychological dimensions that were covered, and almost all the differences involving the multiracial adolescents were with the single-race white group. For example, for boys, multiracial adolescents were distinguished from their white peers by higher rates of counseling use, grade retention, suspension and expulsion from school, and depression scores. For girls, the same results applied, except for depression. In this study, the subsample of adolescents included all lived in households with both their biological parents, and thus the possible confounding effects of family structure on adolescents' adjustment were eliminated.

Two causal risk factors play a major role in placing children of certain racial and ethnic groups at increased risk of emotional and behavioral problems, namely, growing up in conditions of family adversity and difficulty in performing satisfactorily in school ( [Offord, 1990a](#)). Nevertheless, the causal risk factors for psychiatric disorder may vary for different groups. For instance, in the Great Smoky Mountain Study of Youth, family mental illness was associated with child psychiatric disorder for Native American and white children, but poverty and family deviance were linked with psychiatric disturbance only among the Native American children ( [Costello et al., 1997](#)).

##### *Chronic Health Problems*

Children with chronic medical illness, especially when the illness is associated with disability, have more than three times the rate of psychiatric disorders and social adjustment problems compared with their peers without chronic illness ( [Cadman et al., 1987](#)). Most children with chronic medical illness are free of psychiatric disorder ( [Cadman et al., 1987](#); [Perrin et al., 1987](#)), and no firm evidence indicates that a certain type of disease is associated with a particular pattern of emotional and behavioral problems. There is, however, broad agreement that diseases affecting cerebral functioning directly produce the highest prevalence rates of psychiatric disorder, and within that group, the highest susceptibility appears to be in cases of epilepsy ( [Hoare and Kerley, 1991](#)).

##### *Temperament*

Children from early on in their lives can differ from each other in behavioral patterns such as inhibition, biological irregularity, emotional intensity, and activity level, and there are stable differences between boys and girls ( [Martin et al., 1997](#)). These early differences in emotional reactivity and regulation can be risk factors for childhood internalizing or externalizing problems ( [Rothbart and Bates, 1998](#)). For example, series of studies have shown that early behavioral inhibition is a risk factor for later internalizing disorders ( [Biederman et al., 1993](#); [Rosenbaum et al., 1993](#)), and infants who are highly reactive to unfamiliar stimuli are more likely to be inhibited at age 4 years and more anxious by age 7 years ( [Kagan et al., 1999](#)). Further, it has been reported that "difficult" temperament in infancy and early childhood predicts externalizing and sometimes internalizing problems in preschool and middle childhood ( [Bates et al., 1991](#); [Prior et al., 1992](#); [Shaw and Winslow, 1997](#)).

The concept of *temperament* (the repertoire of traits with which each child is born) has supported the idea that the child is an active participant in creating his or her environment and is not simply a passive recipient ( [Plomin, 1986](#)). Temperaments can be modified during development, particularly by the interaction with the caretaker and the family. For example, a timid child can become more assertive with the help of parental encouragement ( [Kagan, 1984](#); [Kagan, 1989](#)), and the harmful effects of difficult temperament are increased significantly when children live in dysfunctional families ( [Maziade et al., 1985](#)). Many methodologic issues concerning temperament have to be sorted out ( [Prior et al., 1992](#)), including a major one of operationalizing the concept of temperament so it is separate from the measurement of psychopathology ( [Lengua et al., 1998](#)).

##### *Intelligence Quotient, Learning Disorders, and Educational Retardation*

Children who do poorly in school whether because of low intelligence quotient (IQ) or a specific learning disorder are at increased risk of a wide range of psychiatric disorders, including conduct disorder ( [Rutter et al., 1970](#)), hyperactivity ( [Hinshaw, 1992](#)), and overanxious disorder ( [Velez et al., 1989](#)), as well as delinquency ( [Lynam et al., 1993](#)). The mechanisms by which poor school performance leads to increased rates of psychiatric disorder have been investigated most fully for



externalizing disorders, especially conduct disorder ( [Hinshaw, 1992](#)). Four hypotheses have been identified: externalizing behavior leads to underachievement; the reverse is true; each leads to the other; and finally, underlying variables common to both (e.g., abnormal temperament, cognitive deficits) account for the relationship. Currently, none of the hypotheses can be eliminated, and additional research is needed to understand more thoroughly the causal process involved in producing the relationship between poor school performance and child psychiatric disorder.

## **BIOLOGICAL FACTORS**

Most child psychiatric disorders are thought to be caused by a combination of biological and psychosocial factors ( [Rutter et al., 1999](#)). There are two kinds of biological influences, namely, those that involve changes in brain structure and function and genetic factors. Investigators have recognized many examples of factors that lead to central nervous system dysfunction resulting in increased rates of mental disorders, including environmental exposure to lead ( [Needleman et al., 1990](#)), intrauterine exposure to alcohol ( [Nichols and Chen, 1981](#)), and prenatal trauma ( [Whitaker et al., 1997](#)). Children with brain disorder have markedly increased rates of psychiatric disorder ( [Rutter, 1977](#)), but the causal processes involved, including their relative strength and patterns of interaction, are not completely understood ( [Brown et al., 1981](#); [Wolf and Forsythe, 1978](#)). Genetic factors are covered in the section on parental psychopathology. Biological factors are thought to be especially important in several disorders including early-onset schizophrenia ( [McLellan and Werry, 2001](#)) and pervasive developmental disorder including autism ( [Piven and O'Leary, 1997](#)), and they probably also have an important etiologic role in Tourette's disorder ( [Leckman et al., 1997](#)), obsessive-compulsive disorder ( [Leonard et al., 1997](#)), and social phobia ( [Pine, 1997](#)).

## **PSYCHOSOCIAL FACTORS**

### *Early Rearing Environment*

Infants and preschool children who are not provided with adequate interaction and a stable emotional and cognitively stimulating environment are at risk of a wide array of poor outcomes in the cognitive, physical, and mental health domains ( [Committee on Integrating the Science of Early Childhood Development, 2000](#); [Janus and Offord, 2000](#); [McCain and Mustard, 1999](#); [Young, 1997](#)). Further, the security of attachment as an indicator of the quality of the relationship between the child and the caregiver is thought to be an important predictor of mental health outcomes in children ( [Rutter, 1995a](#); [van IJzendoorn et al., 1992](#)), such as depression, especially when the child is raised in an abusive environment ( [Sampson and Laub, 1993](#); [Toth, 1996](#)), and to later conduct disorder ( [Sampson and Laub, 1993](#)).

### *Parental Psychopathology*

Parents with psychiatric disorders have children with increased rates of psychopathology in general, and conversely, children with psychiatric disorders are more likely than their peers to have parents with psychiatric disorders ( [Rutter and Quinton, 1984](#)). An example of the former is that children of depressed parents are more than three times as likely as children of nondepressed parents to experience depressive disorder ( [Birmaher et al., 1996a](#), 1996b). An example of the latter is that between 20% and 50% of depressed children and adolescents have a family history of depression ( [Kovacs et al., 1997](#); [Puig-Antich et al., 1989](#); [Todd et al., 1993](#); [Williamson et al., 1995](#)). The mechanisms involved in the transmission of psychopathology from parents to children are thought to involve both genetic and environmental factors ( [Weissman et al., 1997](#)). On the environmental side, impaired parenting, marital conflict, and family dysfunction have been identified as variables mediating the relationship between parental depression and psychopathology in children ( [Cicchetti et al., 1998](#); [Cummings and Davies, 1994](#)).

There is mixed evidence about the specificity of the relationship between parental and child psychopathology. For example, parental depression increases not only the risk of depression in children but also the risk of anxiety disorder, conduct disorder, and alcohol dependence ( [Downey and Coyne, 1990](#); [Weissman et al., 1997](#); [Wickramaratne and Weissman, 1998](#)). However, another study gives some support for specificity. In a controlled family study, the children of parents with alcoholism were found to have elevated rates in particular of conduct disorder, attention deficit disorder, and oppositional disorder ( [Earls et al., 1988](#)). Finally, the childhood mental disorders most likely to have genetic components are autism, bipolar disorder, schizophrenia, and attention deficit hyperactivity disorder ( [National Institute of Mental Health, 1998](#)).

### *Family Factors*

Certain family factors have been associated with increased rates of psychiatric disorders in children. They include single-parent status, poor parenting, marital discord, family dysfunction, and large family size ( [Lipman et al., 1996](#); [Offord, 1990b](#); [Reynolds and Rob, 1988](#)). There are three important issues in this area. First, more information is needed to identify those correlates that are true causal risk factors. For example, marital discord and poor parenting are causal risk factors ( [Offord, 1989](#)). Second, a better understanding is needed about the mechanism or mediating variables by which the causal risk factors result in increased rates of child psychiatric disturbance. Third, and last, there has been little progress in demonstrating the specificity of causal risk factors, that is, that certain factors are associated with one disorder but not another ( [Bauermeister et al., 1994](#)). Most of the evidence indicates the existence of common causal risk factors for multiple outcomes. For example, parental psychopathology has been shown to be a causal risk factor for depression, conduct disorder, and anxiety disorders ( [Institute of Medicine, 1994](#)).

### *Socioeconomic Status*

The strength of the relationship between low socioeconomic status and child psychiatric disorder depends on certain factors, including the measure of socioeconomic status and the informant who provides the data on the presence of emotional and behavioral disorders, on whether the poverty is persistent, and on the age of the child. A strong relationship exists between low socioeconomic status and child psychiatric disorder when parental socioeconomic status is measured by the degree of economic disadvantage but not when it is determined by level of occupational prestige ( [Lahey et al., 1999](#); [Offord, 1990a](#); [Offord and Lipman, 1996](#)). The relationship between low socioeconomic status and externalizing disorder is stronger for teacher-identified than for parent-identified disorder ( [Offord et al., 1989](#)). Further, it has been reported that persistent poverty predicts internalizing symptoms, whereas only current poverty predicts externalizing symptoms ( [McLeod and Shanahan, 1993](#)). Finally, the relationship between economic disadvantage and child psychiatric disorder appears stronger in younger children than in adolescents ( [Lipman et al., 1994](#); [Rutter, 1981](#)). Two other points should be noted. First, the factors that place poor children at increased risk of psychiatric disorder have to do primarily with increased rates of parental and family characteristics associated with child psychiatric disorder rather than economic disadvantage itself. Second, the relationship between income level and psychiatric disorder is on a continuum or gradient with increased levels of income across the range associated with decreasing frequencies of psychiatric disorder ( [Offord and Lipman, 1996](#)).

### *Other Factors*

Space limitations preclude coverage of other correlates of child psychiatric disorders including stressful life events ( [Garber and Hilsman, 1992](#); [Garrison et al., 1997](#); [Jensen et al., 1991](#)), child maltreatment ( [Famularo et al., 1992](#); [MacMillan and Munn, 2001](#); [Main and Solomon, 1990](#)), and peer and sibling influences ( [Coie and Miller-Johnson, 2000](#); [Loeber and Farrington, 1998](#); [Patterson and Dishion, 1988](#)).

## **Protective Factors**

Protective factors operate to reduce the incidence or severity of children's emotional or behavioral problems among children at increased risk of developing these difficulties ( [Rutter, 1985](#)). They are products of both the attributes of the child and of the environment. They should not be seen primarily as existing in a cross-sectional snapshot, but should be understood as processes operating over the developmental years and perhaps having their effects in different ways at different points in development. These factors can exist in the child (e.g., above-average IQ, easy temperament), in the family (e.g., a high degree of cohesiveness within the family, a good relationship with one parent), and in the wider community (e.g., excellent schools and nonschool skill-development programs in the sports and arts) ( [Grizenko and Fisher, 1992](#); [Jones and Offord, 1989](#)). The mechanisms by which these protective factors have their effect are through the following main processes: reduction of risk impact, reduction of negative chain reactions, establishment and maintenance of self-esteem and self-efficacy, and opening up of opportunities ( [Luther and Zigler, 1991](#); [Rutter, 1987](#)).

## **Correlations and Interactions Among Risk and Protective Factors**

Risk and protective factors operate in different combinations over childhood and adolescence to produce different developmental trajectories ( [Hertzman, 1999](#)). Biological factors in the child can influence the child's environment (e.g., a child with a "difficult" temperament may promote irritability in the parents and reduce their parenting effectiveness), and aspects of the environment can become "biologically embedded" in the child (e.g., a violence-filled environment in early childhood can

lead to enduring changes in brain structure) (Hertzman, 1999). Similarly, there is evidence that social and environmental risk factors (e.g., early maternal rejection) may combine with physical risk factors (e.g., birth complications) to predispose children to violence (Raine et al., 1996; Raine et al., 1997; Raine et al., 1998). An important methodologic advance in studying the contributions of individual causal risk and protective factors to child psychiatric disorders is multilevel modeling in which the contribution of a variable in one domain (e.g., neighborhood residence) can be determined controlling for the effects of variables in other domains (e.g., child and family) (Leventhal and Brooks-Gunn, 2000). The challenge is to learn more about how risk and protective factors operate as mediating and moderating variables (Kraemer et al., 2001) to produce different developmental trajectories, in which, for example, some children's developmental trajectories begin with a biological risk factor and spiral downward toward morbidity, whereas in other children, who begin with the same biological disadvantage, the developmental trajectories lead to healthy outcomes.

## ISSUES IN PREVENTION

The issues that are covered include definitions, approaches, and selected contributions of developmental psychopathology to prevention.

### Definitions

A major problem with the widely used definitions of prevention initiatives (primary, secondary, tertiary) presented earlier in this chapter is that the distinction between primary and secondary prevention can be blurred. The reason is that determining the threshold indicating the presence or absence of disorder is difficult and tends to be arbitrary when the frequency and severity of symptoms are on a continuum (Offord, 1987).

An alternate classification system centers on who is offered the intervention (Institute of Medicine, 1994; Offord et al., 1998). The three types of programs using this strategy are universal, targeted, and clinical. A *universal* program is offered to all persons, for example, children (and their families) in a geographic area. In a targeted program, certain persons are singled out to be offered the intervention. Two types of targeted programs have been described, namely, *indicated* and *selective* (Institute of Medicine, 1994). In an *indicated* preventive initiative, the high-risk group is identified on the basis of biological or behavioral markers for increased risk of disorder, for example, children with mild antisocial symptoms who do not qualify for conduct disorder, but who are at increased risk of it (Lipman et al., 1998). In *selective preventive* programs, the target group is identified, not on the basis of biological markers or symptom patterns, but on the grounds that persons or a population subgroup are at increased risk of developing psychiatric disorder, for example, children and their families residing in a publicly supported housing complex (Offord et al., 1987a). In a *clinical* program, there is no active reaching out to children, but children (and their families) are referred for, or seek, services.

Each of these strategies has advantages and disadvantages, and they have all been described in detail (Offord et al., 1998). The advantages of a universal program include the following: no labeling or stigmatization; the involvement of the middle class, who will demand that the program be well run; and the "tilling of the soil" or sensitizing the setting for subsequent targeted interventions. The disadvantages of universal programs are several; for example, they will be seen as unnecessarily expensive and as having a small benefit to the individual, and they may have their greatest beneficial effect on those at lowest risk, thus producing the unexpected effect of increasing inequality between the high- and low-risk subgroups within the population.

The targeted approach has the advantages of addressing problems early and is potentially efficient if the targeting can be done accurately. The disadvantages are several. First is the possibility of labeling and stigmatization; second, there are difficulties with recurrent screening including the cost and maintaining a high degree of compliance, especially among those at highest risk; and third is the difficulty of targeting accurately. It is an arduous task to identify accurately in a community population those at increased risk of future disorder and thus the population that should be the focus of targeted interventions (Bennett, 2000; Bennett et al., 1998; Bennett et al., 1999). One strategy to increase the accuracy of the identification of high-risk groups of children is to have continuous risk assessment over time, rather than only one measure of risk status (Herlbrun, 1997). A fourth disadvantage of targeted interventions is that more cases of disorder will emanate from the large number of children at low risk rather than the relatively small number of children at high risk. An unwelcome implication of this finding is that one targeted program, even if effective, cannot be expected to have marked beneficial effects at the population level. The last of the three strategies, the clinical approach, has a major advantage in that the intervention is focused clearly on those children with disorder. The disadvantages to the clinical approaches are covered previously at the beginning of the chapter.

### Approaches

There are two somewhat different approaches to developing, carrying out, and evaluating prevention programs. The first, termed *preventive science*, is covered in the Institute of Medicine Report (Institute of Medicine, 1994). Five steps are outlined in carrying out a prevention initiative, beginning with identifying a problem or disorder, then specifying risk and protective factors, carrying out pilot efficacy studies, launching and evaluating large-scale effectiveness trials, and finally moving toward widespread dissemination of the program in a variety of settings. Participation by community members in planning and carrying out these studies is not emphasized, but academicians are viewed as experts and are usually in charge of the programs. The second model, termed the *collaborative community action research model*, is quite different. Community members are major participants throughout the whole prevention initiative (Kelly, 1988; Tolan et al., 1990). The resulting prevention initiative is community specific, especially in terms of the content and delivery of the program, and thus the program is not necessarily suitable for dissemination to other communities. Because of the limitations of the design, the strength of the scientific evidence supporting the effectiveness of these programs will not be strong (Canadian Task Force on the Periodic Health Examination, 1979). A review of the advantages and disadvantages of these approaches presents suggestions for melding the two of them (Weissberg and Greenberg, 1998).

## Contributions of Developmental Psychopathology

Developmental psychopathology integrates knowledge from several related disciplines including epidemiology, psychiatry, developmental psychology, and clinical psychology. It appeared in the 1980s, and it centers on factors that influence the onset and course of emotional and behavioral disorders in children and adolescents (Stroufe and Rutter, 1984). In this section, we summarize selected contributions of developmental psychopathology under four headings: risk and protective factors; multiplicity, specificity, and timing; theories of development; and life-course studies and causal models.

### RISK AND PROTECTIVE FACTORS

For a variable to be a risk factor for child psychiatric disorder, it must fulfill two criteria: be present before the onset of the disorder and increase the likelihood of future disorder in children exposed to the variable, compared with those who are not. The strongest evidence for determining whether a particular factor is a causal risk factor arises from experimental studies, but descriptive studies can also identify factors that are likely to be causal (Hill, 1965; Kraemer et al., 1997). It is necessary to distinguish causal risk factors from correlates, markers, and noncausal risk factors for disorder (Kraemer et al., 1997; Offord and Kraemer, 2000). Prevention interventions that focus not on causal risk factors, but on correlates, markers, or noncausal risk factors will likely have little effect on reducing the incidence of disorder. The same logic applies to protective factors in which the focus of prevention studies should be on causal protective factors.

Two important measures of the strength of the relationship between a risk or protective factor and a subsequent disorder are the odds ratio and the population attributable risk. The *odds ratio* provides information of the increased risk provided with a particular factor (Streiner and Norman, 1996), whereas the *population attributable risk* provides data on the percentage of all cases of disorder in a population that are attributable to a specific risk factor (Kelsey et al., 1996). Risk factors chosen for intervention in a prevention project should be causal, and, if possible, have both a strong relationship with a disorder (high odds ratio) and a high attributable risk. One last issue should be mentioned. A prevention model starts with a causal risk or protective factor, then a prevention intervention, and finally proximal and distal outcomes. One of the problems with prevention studies is that there is "slippage" at every point in the model that can only then weaken the effectiveness of the intervention (Baronowski et al., 1997). For example, if the causal risk factor selected to prevent conduct disorder is poor social skills, and the intervention program is social skills training, and the proximal and distal outcomes are mild antisocial behavior and conduct disorder, respectively, there will not be a one-to-one relationship at each point in the model. For example, not all conduct-disordered children will have deficient social skills, the social skills program will not be 100% effective, and many of the children with mild antisocial behavior will not go on to have full-blown conduct disorder. Each of these findings will serve to put limits on the effectiveness of a prevention program.

### MULTIPLICITY, SPECIFICITY, AND TIMING

The risk of psychiatric disorder in children depends on both the number of causal risk factors present at any one point in time and their accumulation over time (Hertzman, 1999; Yoshikawa, 1994). As noted previously, there has been little progress in demonstrating the specificity of causal risk factors for specific disorders. Finally, the importance of a particular causal risk or protective factor depends on the developmental stage of the individual. Poor parenting, for example, is thought to



have a stronger influence on producing psychiatric disorder in younger than in older children ( [Reid and Eddy, 1997](#)).

#### THEORIES OF DEVELOPMENT AND LIFE COURSE STUDIES

Longitudinal studies have made three important contributions to the prevention enterprise ( [Institute of Medicine, 1994](#); [Loeber and Farrington, 1997](#)). First, they revealed both continuities and discontinuities in the onset and course of psychiatric symptoms and disorder ( [Rutter, 1995b](#)). Second, longitudinal studies have forced investigators to change from reporting outcomes in terms of number of discrete events to describing developmental trajectories of outcomes over time ( [Kellam and VanHorn, 1997](#)). Third, and last, these studies have highlighted that the links between early risk factors and future psychiatric disorders may be indirect and may hinge on other mediating variables.

#### CAUSAL MODELS

The simple causal chains or pathways that have been used to explain the relationship between causal risk or protective factors and the onset of disorder are now viewed as oversimplified. Investigators currently are moving toward more complicated models that distinguish, for example, between risk factors that act as mediators and those that function as moderators ( [Baron and Kenny, 1986](#); [Baronowski et al., 1997](#); [Holmbeck, 1997](#); [Kraemer et al., 2001](#)).

### PREVENTION OF PSYCHIATRIC DISORDERS

In this section, a summary is presented of the current state of knowledge about the prevention of major disorders in child psychiatry, namely, depressive disorder, anxiety disorder, and conduct disorder. In addition, selected risk situations are covered, namely, child abuse, children of divorce, and environmental contaminants. Further, there are brief discussions of prevention in low-income countries and of the barriers to prevention. The chapter ends with some concluding remarks. For each prevention initiative reviewed, the following issues are discussed: the target population; characteristics of the intervention, including whether it was universal or targeted and whether the program was designed to prevent a specific psychiatric disorder or to promote competence in the cognitive and behavioral areas; the strength of the research design; and the findings to date. The design of almost all the studies included in the review is a randomized controlled trial. This design, when executed well, is the strongest in preventing biased estimates of program effectiveness ( [Jadad, 1998](#)).

#### Depressive Disorder

No universal programs have been shown to be effective in preventing the onset of depressive symptoms or depressive disorders ( [Harrington, 1997](#)). There are, however, targeted prevention programs, both of the selective and indicated subgroups. Beardslee and colleagues ( [Beardslee et al., 1993](#); [Beardslee et al., 1996](#); [Beardslee et al., 1997a](#); [Beardslee et al., 1997b](#)) have been involved for several years in the selective type of a targeted program in which the children, ages 8 to 15 years, are identified as being at increased risk of depressive disorder on the basis of being the child of a parent with a recent history of affective disorder. Children were randomly assigned to one of two psychoeducational interventions, clinician-facilitated or lecture-group discussion, both of which were planned to prevent childhood depression and other related problems through promoting resiliency and reducing the effects of risk factors. The lecture-group discussion consisted of two 1-hour lectures, whereas the mean number of clinician-facilitated intervention sessions was 7.7. The latter intervention had a strong cognitive approach, and a primary goal was to increase family members' understanding of the illness through individual sessions with the parents, family meetings, and an individual session with each child. In the most recent report ( [Beardslee et al., 1997b](#)) on the first 37 families, positive results, as would be expected, were more marked in the clinician-facilitated group. It will not be known for several years whether these interventions actually reduce the incidence of depressive disorder and other morbidities in these high-risk children.

A group of investigators carried out a series of indicated targeted intervention studies in which children at risk for depressive disorder were identified on the basis of self-reports of elevated levels of depressive symptoms ( [Clarke et al., 1995](#); [Gillham et al., 1995](#); [Jaycox et al., 1994](#)). In the most recent of these studies ( [Clarke et al., 1995](#)), 1,652 adolescents were screened in a two-stage procedure, and 150 were identified as experiencing depressive symptoms but did not meet the criteria for depressive disorder. The intervention consisted of a 15-session cognitive-behavioral intervention (three sessions per week for 5 weeks), and data were collected after treatment and 6 and 12 months later. The program emphasized training adolescents in cognitive-restructuring skills to increase their ability to reduce mild levels of negative cognitions before they reach depressive symptoms of clinical proportions. Overall, the incidence of either major depression or dysthymia at follow-up was significantly less in the treated group compared with the control group, 14.5% versus 25.7%, respectively.

#### Anxiety Disorder

The literature on the prevention of anxiety disorders is sparse ( [Spence, 1994](#)). Although there are no well-controlled experiments of universal interventions to prevent childhood fears, some programs are aimed at preventing fears in specific situations such as dental visits ( [Melamed et al., 1975b](#); [Melamed et al., 1975a](#); [Milgrom et al., 1992](#)), medical procedures ( [Jay et al., 1987](#)), test anxiety ( [Tryon, 1980](#); [Van der Ploeg-Stapert and Van der Ploeg, 1986](#)), and transition to a new school ( [Felner and Adan, 1988](#)).

The Queensland Early Intervention and Prevention of Anxiety Project ( [Dadds and Spence, 1997](#)) is a randomized controlled trial aimed at preventing the onset and development of anxiety problems in children ( [Dadds and Spence, 1997](#)). Of the total sample of 1,786 children in grades 3 to 7 (ages 7 to 14 years) in eight primary schools, 121 were selected for the trial on the basis of having anxiety symptoms (but not severe anxiety disorder) as a consequence of the results of a complicated and extensive four-stage screen that used data from teacher nominations and children's self-reports. Selected children were allocated to the intervention and control groups on the basis of school. The intervention was based on the *The Coping Koala: Prevention Manual* ( [Barrett et al., 1994](#)), and it consisted of 1- to 2-hour sessions with the children over 10 weeks (five to 12 children in a group) in which the children were taught strategies for coping with anxiety using a cognitive-behavioral approach. In addition, four parent sessions focused on teaching skills to manage their child's anxiety. The authors reported that the intervention was successful both in reducing rates of disorder in children with mild to moderate anxiety disorders and in preventing the onset of anxiety disorders in children with some anxiety symptoms. Furthermore, the differences between the experimental and control groups were present at the 24-month follow-up. The labor-intensive four-stage screen is an obstacle to wide adoption of this promising indicated targeted prevention program for childhood anxiety disorders.

#### Conduct Disorder

Most of the prevention programs for child psychiatric disorder focus on conduct disorder. Relevant programs are reviewed according to the age of the children involved: less than 5 years; 5 to 11 years; and 11 years and older.

#### PROGRAMS FOR CHILDREN YOUNGER THAN 5 YEARS OLD

Three major programs involve this age group. They have four characteristics in common: they begin early and are intensive; they focus primarily on markedly economically disadvantaged, commonly African-American preschoolers; the sample sizes are small; and the results are encouraging.

The Perry Pre-school project was designed in the 1960s for poor, black 3- and 4-year-old children to improve academic and social outcomes ( [Schweinhart and Weikert, 1988](#); [Schweinhart and Weikert, 1989](#)). The intervention lasted 2 years and consisted of a daily preschool program (90-minute classes 5 days per week) and a weekly home visit provided by trained teachers. The preschool element aimed to promote physical, academic, and social development, whereas the purpose of the home visits was to help parents reinforce the preschool curriculum. The program was evaluated in a randomized controlled trial (58 children in the experimental group and 65 in the control group) in which the children were followed-up into adulthood. Although the initial IQ gains in the experimental group disappeared, long-term follow-up at ages 19 and 27 years showed marked differences on measures of antisocial behavior including fewer lifetime arrests in the experimental group compared with the control group (31% versus 51%, respectively), and a reduced rate of being arrested five or more times (5% versus 35% in the experimental and control groups, respectively). In an economic analysis, the benefits of the program were found to exceed the costs ( [Barnett, 1998](#)).

The Carolina Abecedarian Project also focused on poor African-American children ( [Campbell and Ramey, 1994](#)). A very intensive intervention throughout the first 5 years of life consisted of a range of services offered through a day care center including an enriched day care curriculum, parent groups, home visits, and medical care and social services. Beginning in kindergarten and extending through grade 2, the parents received 15 home visits per year from a home-school resource teacher. The program was evaluated in a randomized controlled trial in which the outcomes of four groups of children were compared: group 1, birth to age 5 program plus kindergarten to grade 2 program (n = 25); group 2, birth to age 5 program only (n = 22); group 3, kindergarten to grade 3 program only (n = 21); and group 4, no program (n = 22). There were two major results of the study. First, the children who received the full program did better on measures of IQ and school achievement

than children in the other three groups. Second, the interventions that began in the first 5 years of life were more effective than the intervention that began in kindergarten, in terms of IQ and school achievement at ages 8 and 12 years. Although these investigations did not assess antisocial outcomes, the characteristics of the intervention and its positive effects on academic outcomes suggest strongly that it holds promise as an intervention strategy for antisocial behavior.

The third study provided nurse home visitation for poor unmarried pregnant women (Olds et al., 1998a). In a randomized controlled trial, children at age 15 years who received home visits by a nurse (during the mother's pregnancy and up to age 2 years) were compared with those who did not. Based on youth self-reports, the youths in the experimental compared with the control groups reported lower rates of arrests (56% reduction), convictions and violations of probation (81% reduction), running away (60% reduction), lifetime sexual partners (63% reduction), and alcohol consumption (56% reduction). Parents also reported lower rates of behavior problems associated with alcohol and other drugs (56% reduction). This same prevention program is reviewed later in this chapter, in the discussion of child abuse.

Two issues need to be addressed with these types of programs. First is a requirement to understand the mechanisms by which these programs exert their effects, and this necessitates the formulation of a more coherent and conceptual basis for these initiatives (Ramey and Ramey, 1998). Second is a need to test these approaches in other populations of children when the levels of economic disadvantage and associated risks are less extreme. Data on the costs and effects of interventions of this type on these latter populations are necessary to guide policy decisions about the extent to which widespread dissemination of these programs is desirable.

#### PROGRAMS FOR CHILDREN 5 TO 11 YEARS OLD

Most of the programs for children in this age group are offered in the school setting. Four different types of approaches are employed: single-factor, multiple-component, competence-enhancement, and school development.

Single-factor approaches focus on one risk or protective factor in one context, and when they are evaluated in randomized controlled trials, they have shown small to moderate effects on antisocial symptoms. Examples include social skills programs (Grossman et al., 1997), peer mediation (Cunningham et al., 1998), and a classroom-management strategy (Kellam et al., 1994).

Multiple-component strategies focus on multiple risk and protective factors, and they operate in more than one context. The Montreal Longitudinal-Experimental Study evaluated a targeted intervention for high-risk kindergarten boys (Tremblay et al., 1995). Two risk factors were addressed, one at the family level (parenting skills) and one at the child level (social skills training). Boys in 53 Montreal schools who showed increased rates of antisocial behavior on a teacher questionnaire and whose parents were French speaking and had less than 14 years of formal education were randomized to one of three treatment groups: a 2-year intervention program consisting of the social skills and parent training sessions, an attention-control group, and a control group. Initial results in elementary school showed that intervention-group boys did better on measures of school adjustment, but by age 15 years, 6 years after the conclusion of the program, intervention-group members were no different from those of the control group on teacher-rated disruptive behavior and frequency of involvement in the juvenile court.

The Tri-Ministry Helping Children Adjust Project was designed as a universal program to address three risk factors for emotional-behavioral maladjustment in childhood: poor reading skills, poor social skills, and poor parenting skills (Boyle et al., 1999). The initial elements of the intervention program were a reading partners program, a social skills program, and a parent management program. However, the uptake of the parent management program was so poor that it had to be dropped after the first year of evaluation. Thus, the programs compared in the randomized trial (in which the unit of intervention was the school) were reading partners alone, social skills training alone, and a combination of both reading partners and social skills training. Program duration was a minimum of two school terms, and the results after 1.5 to 3.5 years of follow-up revealed that the effects of the intervention were modest at best. Playground observations showed statistically significant increases in prosocial behavior and some reduction in externalizing symptoms (Hundert et al., 1999).

Fast Track is another multicomponent, multilevel approach (Bierman et al., 1999a; Bierman et al., 1999b). The intervention program focuses on multiple levels (child, family, school, peer group, community) and factors (academic skills, social skills, parenting skills, family support) and includes a universal and targeted components. The program begins in grade 1 and is planned to continue through middle school. Fast Track is currently being evaluated in a large, randomized controlled trial involving 54 schools in four separate geographic sites in the United States. Results based on outcomes at the end of grade 1 are encouraging, and it will be interesting to learn whether the Fast Track prevention initiative will result in reductions in conduct problems that are important in both clinical and policy terms, as the children move into adolescence and adulthood.

Competence-enhancement intervention strategies do not address emotional and behavioral problems specifically, but they promote social competence. An example of such a program is the Seattle Social Development Project, which consists of three components to increase social competence: teacher training, child social skills development, and parent training (Hawkins et al., 1999). The program is offered from grades 1 through 6. The results of a nonrandomized controlled trial showed that, at age 18 years, adolescents who had participated in the program self-reported fewer delinquent acts (19% reduction), less heavy drinking (40% reduction), and fewer sex partners (19% reduction) and pregnancies (35% reduction) compared with control students. Another promising competence-enhancing program is the Social Competence Program for Young Adolescents (Weissberg and Greenberg, 1998), which resulted in reductions in self-reported antisocial and delinquent behavior among middle-school students who received the 2-year program compared with those who did not.

The final strategy is school development, which has been applied in elementary schools and may result in reduced rates of antisocial behavior. The School Development Program, developed by Comer and colleagues (Comer, 1985), has three components: a school planning and management team, a mental health team, and a parent program. The focus here is on mechanisms or processes to support change, and the specific content of the program is not defined but is left up to the teams. The hope is that this strategy will improve school climate, thus promoting positive behaviors and minimizing negative health and social outcomes. The Comer approach, when evaluated in a nonrandomized controlled study, revealed superior school outcomes for children attending the program schools compared with those who did not (Comer, 1985).

In summary, it is clear that the school is a promising setting in which to launch prevention programs for conduct disorder. There are tradeoffs between single-component and multicomponent strategies: the former are simple and straightforward to implement but are unlikely to have a major impact on reducing the incidence of an etiologically complex condition such as conduct disorder, and the latter, although they address multiple risk and protective factors, are complex and expensive, and currently the magnitude of the benefit in relation to cost is uncertain (Hundert et al., 1999). Finally, the competence-enhancing and school development approaches could benefit from more rigorous evaluations.

#### PROGRAMS FOR CHILDREN MORE THAN 11 YEARS OLD

In older children, the emphasis on reducing the burden of suffering from conduct disorder is through treatment programs, not prevention initiatives. Two prevention programs are summarized here: one that shows promise and one that has been shown to be ineffective. The PATHE program (Positive Action Through Holistic Education) (Gottfredson, 1986) is a school development model focusing on this age group that has been shown to influence antisocial outcomes. The program consists of several components focusing on high-risk students: a school team composed of school and agency personnel and parents, development of school and classroom goals, schoolwide academic innovations, schoolwide climate innovations, and mental health and academic services. In an evaluation using a nonequivalent comparison group design, student reports suggested that the program results in fewer delinquent acts and lower rates of drug use and school suspensions. Further work is needed on this promising school reform strategy.

Finally, the Cambridge-Somerville Study is important because the results indicate that the intervention was probably harmful. In this program, which began in the 1930s, boys approximately 11 years old were randomized to be assigned or not to a social worker (McCord, 1992). The social worker's role was to build a close relationship with the boy and to provide counseling assistance to the boy and his family. Follow-up in adulthood, when the participants were 40 to 50 years of age, revealed that the treatment-group boys had worse outcomes in terms of death before age 35 years, conviction of a crime, and psychiatric disorder than the controls. The reasons that this program failed are not completely clear.

#### Risk Situations

There are markers or indicators of increased risk for child psychiatric disorders, and attempts have been made either to reduce the risk itself or to modify the effects of the risk. Some of the markers are intrinsic (within the child), such as low birth weight, developmental delay, brain damage, epilepsy, and chronic medical illness, and others are extrinsic (outside the child), such as parental psychiatric disorder, physical and sexual abuse, divorce, and residence in economically disadvantaged neighborhoods (Offord et al., 1998). Because of limited available space, only three situations are covered briefly, namely, child abuse, parental divorce, and



environmental contaminants.

### CHILD ABUSE

Prevention studies focus either on the prevention of physical abuse or neglect or on the prevention of sexual abuse and abduction ( [MacMillan et al., 1994b](#)). Universal programs to prevent physical abuse and neglect center on making available child development and parenting resource centers for children, from birth to 5 years of age, and their parents. The provision of these resources for selected groups of impoverished preschool children results in improved health and well-being ( [McCain and Mustard, 1999](#)), but the effects on reducing physical abuse specifically are not known ( [Leventhal, 1996](#)).

Targeted interventions have focused on home visiting, and evidence indicates that extended home visitation can prevent physical abuse and neglect among disadvantaged families ( [MacMillan, 2000](#); [MacMillan et al., 1994a](#); [MacMillan and Canadian Task Force on Preventive Health Care, 2000](#)). The most thoroughly evaluated home visitation program, the Prenatal and Early Childhood Nurse Home Visitation Program, has been evaluated in two randomized controlled trials in Elmira, New York and Memphis, Tennessee ( [Karoly et al., 1998](#); [Kitzman et al., 1997](#); [Olds et al., 1993](#); [Olds et al., 1997](#); [Olds et al., 1998a](#); [Olds et al., 1998b](#); [Olds et al., 1999](#)). Among the several positive results, rates of child maltreatment from birth through the child's 15th year were reduced by 79%. The essential ingredients of successful home visitation programs have been discussed ( [Leventhal, 1996](#)), as have been their limitations ( [Guterman, 1997](#); [Leventhal, 1996](#)).

The relatively small literature on the prevention of child sexual abuse reveals one central finding: educational programs, usually delivered in the school setting, can improve safety skills and knowledge, but no study has furnished results that educational programs reduce the occurrence of sexual abuse ( [MacMillan, 2000](#); [MacMillan et al., 1994a](#); [MacMillan and Canadian Task Force on Preventive Health Care, 2000](#)).

### CHILDREN OF DIVORCE

Children of divorce are increasing in numbers. In the United States, the proportion of children growing up in homes with two biological married parents fell from almost 90% in the 1960s to about 40% by the mid-1980s ( [Wadsworth, 1986](#)). In the United States in 1996, divorce and annulments involved more than 1 million children ( [National Center for Health Statistics, 1997](#)). The effects of divorce on the psychosocial adjustment of children have been reviewed ( [Hetherington and Stanley-Hagan, 1999](#)). A metaanalysis of 95 studies revealed that, on average, children of divorced parents are less well adjusted socially, emotionally, and academically than children of nondivorced parents ( [Amato and Keith, 1991](#)). However, not only do children's reactions to divorce vary widely, but also most children do not show severe or enduring behavior problems ( [Hetherington, 1993](#); [Hetherington et al., 1992](#)).

Reviews of interventions for divorced parents and their children show that adequate research in this area is scarce ( [Emery et al., 1999](#); [Grych and Fincham, 1992](#); [Hetherington and Stanley-Hagan, 1999](#)). Interventions for children usually occur in the school setting, involve groups of children of divorced parents, consist of both educational and therapeutic activities, and are of short duration. In the most thoroughly evaluated school-based program in which children in grades 4 to 6 from four suburban schools were randomly assigned either to an immediate 12-week intervention group or to a delayed intervention group, reports by teachers, group leaders, and the children themselves reported fewer anxiety symptoms and behavior problems in the experimental group compared with the control groups ( [Alpert-Gillis et al., 1989](#); [Pedro-Carroll and Alpert-Gillis, 1997](#)). Positive results were also obtained when the intervention was extended to urban children, with diverse ages, ethnicities, and socioeconomic backgrounds ( [Pedro-Carroll and Cowen, 1985](#); [Pedro-Carroll et al., 1986](#)). One child-focused program that has been widely disseminated showed few positive results in a single-group before-and-after design ( [Kalter et al., 1984](#); [Kalter et al., 1988](#)). Parent-focused group interventions have the goal of helping parents deal more effectively with the stresses of divorce, and only secondarily are they aimed at improving parenting and family relationships ( [Hetherington and Stanley-Hagan, 1999](#)). One of the few randomized controlled trials, in which the intervention was 14 parent-group sessions, indicated that mothers in the intervention group reported improvements in maternal discipline, and teachers reported improvements in children's behavior ( [Forgatch and DeGarmo, 1998](#)). A third type of intervention, focusing on legal issues, namely, mediation and coparenting, reveals no evidence that mediation compared with adversary settlement results in improved adjustment of children ( [Kelly, 1989](#)), and there are no consistent findings of an association between type of custody and children's adjustment ( [Grych and Fincham, 1992](#)).

The intervention literature on the children of divorced parents has major weaknesses. Well-done randomized controlled trials are rare, the outcome data are provided by informants who are not blind to the intervention status of the children, and the follow-up periods are short. In addition, most of the children of divorce do not exhibit adjustment problems, and thus for them the intervention is not needed, and indeed it may be harmful because of labeling and stigmatization.

### ENVIRONMENTAL CONTAMINANTS

Lessening or eliminating environmental contaminants is an important universal prevention initiative in the child psychiatric domain ( [Environment Leaders of the Eight, 1998](#)). Not only do children of poverty endure an inordinate impact from environmental health threats ( [Chaudry, 1998](#); [Mott, 1995](#)), but also children in general are especially vulnerable to the noxious effects of these environmental agents because of their early reliance on breast milk, which can become contaminated ( [Sonawane, 1995](#)), and because young children engage in crawling activities and spend most of their time indoors. There are several examples of the adverse effects of environmental toxins on children's development, including methylmercury ( [Rice, 1998](#)), phencyclidine hydrochlorides (PCPs) ( [Stern et al., 1994](#); [Ware et al., 1986](#)), air pollution ( [Brunekreef et al., 1997](#); [Raizenne et al., 1998](#); [Wang et al., 1997](#)), and, of course, lead. Lead poisoning not only produces deficits in IQ ( [Needleman, 1998](#); [Schwartz, 1994](#)), but also has been reported to result in attentional and language problems and perhaps social adjustment difficulties ( [Rice, 1998](#)). The removal of lead from gasoline in 1990 in the United States has been proclaimed as one of the public health triumphs of the 20th century ( [Needleman, 1998](#)).

### Low-Income Countries

The topic of the prevention of emotional and behavioral problems in children in developing countries is immense and important. It is included here not to provide, by any means, complete coverage of the subject, but rather to alert child psychiatrists and their mental health colleagues to the issue that prevention of child psychiatric disorders is a worldwide issue. Child mental health problems have probably worsened in low-income countries over the past several decades even though physical health and schooling have improved ( [Desjarlais et al., 1995](#)). It cannot be assumed that effective interventions in wealthy countries will necessarily be effective in poor ones ( [Dans et al., 1998](#); [Rahman et al., 2000](#)). Four universal interventions show promise in improving the quality of life of children in general and decreasing mental health problems in particular in these severely disadvantaged populations. First, there should be efforts to improve maternal education ( [Caldwell, 1986](#); [Hobcraft, 1993](#)). Possible mechanisms by which this improvement will lead to increased child well-being include later age of marriage and birth of first child, fewer children, and increased responsiveness to modern hygienic practices ( [Hobcraft, 1993](#)). Second, there should be better family planning. The greater the number and the more closely spaced the pregnancies, the greater is the risk of maternal and infant mortality and of poor developmental and mental health outcomes among the children ( [Desjarlais et al., 1995](#)). Third, the efforts to enrich the first 5 years of life of these children should be continued and expanded through the provision of early child development and parenting programs resulting in improvements in nutrition, cognitive stimulation, and parenting ( [McCain and Mustard, 1999](#); [Young, 1997](#)). Fourth and last, efforts should continue to increase the percentage of children enrolled in schools and to decrease school dropouts ( [Carnegie Commission, 1992](#)).

### Barriers

Two obstacles make it difficult for child psychiatrists to carry out prevention studies. The first is that, in most instances in North America, child psychiatrists are funded on a fee-for-service basis, and thus there is no remuneration for time spent in planning and carrying out prevention projects that do not involve direct clinical contact. A second barrier is that child psychiatrists are usually not trained to do prevention work ( [Group for the Advancement of Psychiatry, 1999](#)). However, one can make the case that child psychiatrists should know the prevention literature thoroughly so they can consult with groups planning and launching prevention projects ( [Group for the Advancement of Psychiatry, 1999](#); [Harrington, 1997](#); [Offord et al., 1999](#); [Rae-Grant, 1991](#)). A third barrier to prevention initiatives is the presence of an uncivic community ( [Offord et al., 1999](#)). When the setting in which the prevention program is to be located, whether it be a school, a geographic area, or an entire country is severely disorganized or dysfunctional, the chances of success are minimal. For example, initiating and maintaining a social skills program in a school that is disorganized and demoralized are not possible.

### CONCLUDING REMARKS

Although important progress has been made in the prevention of child psychiatric disorders, some remain. First, there has been little or no success in moving from successful small demonstration projects carried out under more or less ideal conditions (efficacious programs) to large-scale programs carried out in a variety of settings (effectiveness programs) ( [Tugwell et al., 1985](#)). Second, if efficacious programs are to be successfully disseminated, the fidelity of the intervention should be

monitored and maintained (Moncher and Prinz, 1991). Further, if the intervention is changed, for example, "watered down" to save costs, there is no assurance that it will be effective. Third are particular problems in demonstrating the effectiveness of universal programs, and they have been discussed in detail (Hundert et al., 1999). Fourth, effective programs tend not to be maintained, let alone disseminated (Offord, 1996), and, just as disturbing, prevention programs without proven effectiveness end up being widely disseminated (e.g., Kalter et al., 1984; Kalter et al., 1988). Fifth, and last, it is clear that meaningful reductions in the immense burden of suffering from emotional and behavioral problems will need the best cooperative efforts of researchers, clinicians, and teachers in both the treatment and prevention domains.

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## 117 MENTAL HEALTH DELIVERY SERVICES FOR CHILDREN AND ADOLESCENTS

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### [Chapter References](#) [Appendix/Questionnaire on Child Services](#)

It is appropriate at the beginning of the new millennium to take some stock of the state of mental health services for children and adolescents across a variety of national boundaries and within their different cultures. Expected would be variations reflective of ethnic, political, economic, religious, cultural, and social forces, and these findings also might represent something about the status and role of children and children's general health issues in a particular sociopolitical/cultural environment.

An editorial in the *New York Times* of May 20, 1971 states that the treatment of the mentally ill, the brain injured, and the mentally retarded “. . . is a test of how humane any society really is.” By this standard and by the limited data available, one could conclude that there are very few adequately humane societies if judged by the mental health, healthy development, and adequacy of clinical services for a large majority of the world's population of children and youth, which occupy a very low priority level, almost no matter what the type of government, culture, dominant religion, and socioeconomic circumstances.

Results are reported in this chapter from an informal survey of child psychiatrists from 10 countries around the globe conducted by the author, and from a more formal study of general mental health services and research in Arab countries, reported by [Okasha and Karam \(1998\)](#). For the informal survey, a questionnaire was completed by psychiatrists knowledgeable about the type, adequacy, and funding of services for children and adolescents in their respective countries. These countries include the United States, Canada, Sweden, Mexico, Argentina, Chile, Turkey, Israel, Egypt, India, China, and South Korea, not selected to represent any formally constructed sample, but rather countries around the globe representing both very different and overlapping populations, cultures, ethnicities, and forms of government. The questionnaire ([Appendix 117.1](#)) tapped information about each country's major population groups, type of government, sources of payment for child and adolescent health services, and whether patients had any choice in health care provider, and asked questions regarding availability and access to child and adolescent specialized psychiatric services.

Beginning with the United States as a model, the questionnaire reveals the following information. The population of 260 million is very heterogeneous in terms of ethnicity, religion, culture, and socioeconomic status. In order, white, Hispanic, African-American, and Asian ethnicities encompass a myriad of religious practices within Christian, Catholic, Jewish, and Muslim religious affiliations. All these ethnicities and religions are bound together in an elected democratic form of government. The most common and most important source of payment for health care for children and adolescents is insurance paid for by employers, but “managed” by contracted intermediaries; then, a combination of employer/employee contributions; followed by government-paid programs for the poor, such as Medicaid; and then paid for out-of-pocket (“self-pay”) but often reimbursed by individually purchased insurance. A significant portion, if not the majority, of the population has some degree of choice of health care provider, but often the choice is limited by contractual agreements or financial disincentives.

Child and adolescent psychiatry is an identifiable subspecialty of general psychiatry with accredited training programs readily available throughout the country, including Puerto Rico and Hawaii. These programs vary from highly competitive and generally sought after, to others with unfilled positions. A middle-class parent with concerns about a child's development/mental health/behavior would most likely initially consult a pediatrician or family practitioner, and in most urban areas also could seek help directly from a child psychiatrist or other mental health practitioner, often depending on type of insurance. Almost all indicated and recommended services would be available within a reasonable distance in most parts of the country, including inpatient, outpatient, residential, hospital consultation, school-based counseling, and special education programs.

Unfortunately, a wide divide has developed between availability and access; since the 1980s, access and benefits for all health care and especially for all mental health care have come under the increasing scrutiny and control of a so-called managed care intermediary or the limitations of a capitated carve-out. In both instances, the financial incentives reward delay, deferral, and denial of care, especially care provided by referral to a specialist (general psychiatrist) or subspecialist. It is paradoxical that the world's strongest economy, and by far the largest per capita child and adolescent psychiatry work force, has at the same time been unable to provide parity, has reduced access to services, and generated an uninsured population of approximately 45 million or 1 of every 6, largely among low-income families with a disproportionate share of children.

Moving to North America, Canada is the country closest to the United States in economy, population, and many aspects of culture. The population of Canada is 28 million, or approximately 1/10 of the United States; most are Anglo-Saxon, followed by French Canadian, Asian, and East Indian. The government is a constitutional monarchy led by a prime minister, with separation of church and state. Health care is dominantly provided by the government as single payor, with some coverage through employer/employee insurance or insurance paid by employers. The significant majority has a choice of physician/health care provider, but family physicians are the gatekeepers through whom referrals to specialists must be funneled. Child/adolescent psychiatry is a recognized practice entity with a number of special training programs, but only recently have “added qualifications in child psychiatry” been recognized by the Royal College of Physicians and Surgeons in Canada. If a family is concerned about a child's mental health or behavior, referral to a child psychiatrist depends on the gate-keeping function of the family physician or pediatrician. Such services then are paid for through the provincial governments.

Outpatient and hospital-based consultation services usually are available; all other services (inpatient, day-treatment, school-based) are either very scarce or unavailable. Child psychiatrists are very scarce outside the large cities, and even in these cities the waiting time in the queue is very long—months to years. Nonphysician mental health services are not included in the national system, so psychologists and social workers can be accessed without a wait if paid out-of-pocket.

Before moving southward to Central and South America, Sweden is included as a model of a democratically elected social welfare state. The population of 8.6 million is predominantly ethnic Swedes, with Lutheranism as the dominant but not very widely practiced religion. Because Sweden has a liberal immigration policy, there are numbers of families from the world's trouble spots.

Health care is provided by the government, everyone is covered, and doctors and hospitals are funded by public monies. There is a small out-of-pocket payment sector. The population does have reasonable freedom of choice of physician/health care provider within those programs funded by the government. Child and adolescent psychiatry is an established subspecialty with accredited training programs that tend to be highly sought after. A full range of services for child and adolescent disorders is funded by the government and is available and accessible, including inpatient, partial hospitalization, residential treatment, outpatient, and special education services. A special feature of the Swedish system is mental health services in the government-supported child day care programs, providing the opportunity for primary, secondary, and tertiary prevention. The success of Sweden's system reflects a relatively homogeneous and small population in a prosperous economy.

Three countries from Central and South American were surveyed—Mexico, Argentina, and Chile. Their commonalities include language, Catholicism as the dominant religion, and the makeup of their populations, including Native Indian origin, Spanish and other European immigrations, and mixtures (Mestizos). Since at least 1990, all three of these countries have had a popularly elected democratic form of government, although Argentina and Chile both have a prior long history of oppressive military dictatorships.

In Mexico, with a population of 90 million, the dominant form of payment for health services is employer/employee contributions to purchase insurance, followed by a public system in which physicians and hospitals are paid by government funds, and there are government programs for patients with acquired immunodeficiency syndrome, for the poor, and for children. But the significant majority of the population does not have a choice of physician or health care facility; this choice is available mostly to those with individual policies or who pay out-of-pocket.



Child and adolescent psychiatry is an identifiable subspecialty of psychiatry, and there is one special training program for subspecialization. However, services, practitioners, and training are concentrated in Mexico City, and training in child psychiatry is not very much sought after. A middle-class parent would most likely seek help from the family physician or pediatrician and much less, if not least likely, from a child and adolescent psychiatrist. Most child psychiatry services are paid for by government funds (i.e., are in the public sector). Outpatient evaluation and inpatient services usually are available for children, as are mental health services in general and in children's hospitals. All other services—especially for adolescents—are described as scarce to unavailable (e.g., partial hospitalization programs).

In Chile, the government is the dominant single payor for health care, followed by employer/employee combinations to purchase insurance from private companies. A significant majority of the population of 13.5 million has a choice of physician/health care provider in the government system; freedom of choice is more limited in the private insurance sector. Child and adolescent psychiatry is a recognized subspecialty; there is a program offering subspecialty training and this training is in general sought after. A middle-class parent would seek consultation preferably from a psychologist or a child and adolescent psychiatrist and less likely from a pediatrician or family physician.

In terms both of a severe shortage of trained child psychiatrists and a concentration of services in the capitol, Santiago, essentially all specialized psychiatric clinical services for children and adolescents range from unavailable to scarce. The only exception is the generally available special education services for the learning disabled and "emotionally disturbed."

Moving across the Pacific to the East, included are South Korea as a relatively small technologically advanced country, and the two most populous nations in the world, India, a parliamentary democracy, and China, a communist autocracy.

South Korea is a country of 45 million constituted almost entirely of one ethnic group and divided among Buddhist, Protestant, and Catholic religions. Its government is democratically elected and somewhat similar to the United States model. Essentially all health care is paid for and provided by the government, with some participation by large employers. The mechanism for this is the National Medical Insurance Corporation, which in essence provides every citizen with health/mental health coverage through employers, or by the Department of Social Welfare for the poor, disabled, and special populations. Most of the population does not have a choice of physician; this is determined by the government or employer. Child and adolescent psychiatry is a recognized subspecialty with special training programs available in the major University Medical Centers, and these programs are described as very sought after. Surprisingly, it is reported that a middle-class family with concerns about a child's development or behavior would be roughly equally as likely to consult with a child psychiatrist, psychologist, or pediatrician. As with general medical services, child and adolescent mental health services are funded by the government, with occasional out-of-pocket payment in a limited private sector. Outpatient and inpatient services are generally available. Partial hospitalization, school-based mental health services, and special education programs in schools are available but scarce. Residential treatment programs are unavailable.

China is of course the behemoth of 1.2 billion in population and the third largest after Russia and Canada in size (second largest, if Tibet is included). Over 90% of the population is ethnic Chinese (Han race), traditionally Buddhist but mostly nonpracticing, and with small percentages of Western religious denominations. The government is an autocracy with very tight central control by the Communist Party. Essentially all health care is paid for and provided by the government through government-run services. Even where there is a program of employer-sponsored insurance or health services, all significant production still is owned by the government. Choice of health care provider and location is limited within health care sectors. Child and adolescent psychiatry is recognized as a subsidiary practice entity in psychiatry; there are no formal residency training programs but a form of mentorship training, which takes place after medical school and job assignment. These mentorships in child psychiatry are considered not very desirable or sought after. Parents with concerns about their child might very well consult initially with the child's teacher, then a pediatrician or psychiatrist. Psychologists, social workers, and nurses are not a part of the provider chain of mental health services. Services are paid for through the hospital in the patient's health care sector. All psychiatric hospitals are government-owned public hospitals and are separated from general hospital services. Consultation is difficult to arrange in the general medical setting, and realistic concerns about confidentiality are a major issue. For all practical purposes of population and geographic size, all mental health specialty services for children and adolescents range from scarce to unavailable or nonexistent.

India dominates the Asian subcontinent with a population of 900 million, politically and religiously divided predominantly between Hindus and Sikhs, but also with significant Muslim and Christian populations. Since independence from Great Britain in 1947, the form of government has been a parliamentary democracy with two large dominant parties. Health care is provided in two ways: a private sector paid for largely out-of-pocket directly to the physician, and a system of government hospitals and clinics for those unable to afford private care funded by the government on a sliding scale fee basis. A more extensive employer/employee-based insurance system is in a relatively early stage of development and acceptance. Choice of physician, health care provider, and hospital are based on ability to pay. Although child and adolescent psychiatry is a recognized subspecialty, there are only a handful of special training programs (National Mental Health Institute in Bangalore, All India Institute of Medical Services/AIIMS in New Delhi, and the Post-Graduate Institute/PGI in Chandigarh), and these are not very sought after by medical graduates in terms of the linked considerations of status and stigma. Likewise, a concerned middle-class parent would most likely first consult a religious leader or respected family member and then a pediatrician or family physician, and very unlikely a psychiatrist or psychologist.

Bordering the Mediterranean, the three countries of Turkey, Israel, and Egypt, among others, are quite different in population, culture, government, and provision of health services.

Turkey is a large country of 60 million straddling the connection between Europe and the Middle Eastern countries, and has served as a "crossroads" for many centuries, encompassing many diverse ethnic and religious identities. Native Turks, both secular and Muslim, are the dominant population group, followed by Muslim Kurds, various Christian groups, and a historical Jewish population. The form of government is parliamentary democracy and, together with Israel, is unusual for its part of the world in having a freely elected parliament and Prime Minister. It also is a fiercely secular government and forbids any crossover between state and religion.

Health care for children and adolescents, as all other health care, is funded and provided by the government in combination with a "private" system of employer/employee-purchased health insurance. Employer-purchased insurance and direct self-pay are lesser parts of health care funding. Although most of the population has a choice of health care provider, the choice is limited for many, if not most, by geography (facilities, distribution, and quality are heavily weighted to the western half of the country) and paradoxically by the type of insurance determining which clinic or hospital can be accessed.

Child and adolescent psychiatry is an identified subspecialty with special training programs postresidency in general psychiatry. These programs are not highly competitive with other specialties for trainees. A middle-class family would most likely first consult a pediatrician or go directly to a child psychiatrist or psychologist. Outpatient specialty services, both evaluation and treatment, are generally available, as are hospital-based consultation programs. Inpatient and school-based mental health services are available but scarce to rare, likewise for day treatment and residential treatment programs.

By considerable contrast, Israel is a very small country of 4.8 million made up of 80% Jewish and 20% Muslim Arab populations. The government is a parliamentary democracy with extensive intermingling of state and church, the latter in the form of Orthodox Judaism. Health care is essentially funded by the government—both facilities and physicians—with some payment out-of-pocket directly to the physician. By contrast to almost all other government-funded programs, in Israel there is extensive freedom of choice of physician or other health care provider.

Child and adolescent psychiatry is an identifiable and vigorous subspecialty with special postresidency training programs. A middle-class family with concerns about a child's development or behavior has many options to choose from among a large and diverse number of health care providers, and is most likely initially to consult a pediatrician, then a psychologist, child psychiatrist, or general psychiatrist, in that order. Services are paid for by the national health insurance plan or out-of-pocket. All important services—outpatient, inpatient, day hospital, hospital consultation, and special education services—for children and adolescents are generally available, perhaps more so in Israel than in any other country surveyed except for urban centers in the United States.

Egypt, with a population of 55 million, is arguably the most substantial and influential country in the Arab world of nations. It is a predominantly ethnically homogeneous society where the main religion is Islam, with 6% to 7% practicing Coptic Christianity. The government is democratically elected but dominated by one party since the monarchy was overthrown in 1952. The major sources of payment for health care/mental health care for children and adolescents are predominantly out-of-pocket directly to the physician, followed by individually owned insurance and government funds, in that order. Most of the population has little if any choice in obtaining health care. Although child psychiatry is an identifiable practice entity in Egypt, there are no training programs in Egypt for this subspecialty. A concerned family would consult first with a pediatrician or family physician, very unlikely with a child psychiatrist. If used, such services would be paid for out-of-pocket. Through self-pay and government funding there are outpatient and inpatient services available, along with hospital-based consultation services. All other services, including

school-based and special education services, are essentially unavailable.

As the result of a survey of mental health services and research in the Arab world conducted by Okasha and Karam in 1998, more comparative information is available among countries in the Arab world than for any other large geographic area, such as Eastern Europe.

It is clear that among these countries, of which only Egypt and Lebanon have an elected government, there are very wide variations in per capita numbers of psychiatrists and other nonmedical mental health providers, ranging for psychiatrists from 1/45,000 population in Lebanon to 1/131,000 in Egypt and, at the lowest end, 1/500,000 in Yemen. This contrasts to approximately 1 psychiatrist per 5,000 population in the United States (and 1 child psychiatrist per 30,000 child and adolescent population). In the countries surveyed, several of their concerns and needs overlapped. A consensus agreed on the need for more extensive public education about mental illness, on increasing the number of psychiatrists, upgrading the training of all groups of mental health professionals, developing community mental health services, and the passage of a Mental Health Act. Child and adolescent psychiatry, geriatric, and especially substance abuse programs are in high demand.

The sample data make it abundantly clear that specialized services, programs, and facilities range from scarce to unavailable for the vast majority of the world's population of children and adolescents and their families. This is the case more or less independent of the form of government, culture, religion, economy, or source of payment. There are gradations, of course, from the relatively enriched circumstances in the United States and Israel to the abject absences or severe shortages characteristic of China and a number of Middle Eastern countries. This is so despite significant wealth in a number of the latter, so that the issue is not invariably the economy *per se*. What factors might be considered to explain the worldwide impoverishment of mental health services for children and adolescents (see [question 13](#) in the survey)?

An aphorism that comes to mind is "that nothing is too good for our children...except money." But, as hinted at earlier, money alone is not the entire story.

What, for example, might be the relationship between the form of government and the support for psychiatric and child psychiatric services? The more autocratic and dictatorial the regime, the more control there must be over what information can be available and limits on how much importance can be placed on the individual versus the state and on how much tolerance there can be for freedom of expression or of thought. Also, there must be limits to confidentiality and privacy, and placing trust in others is not a governmentally supported or safe activity, including even within the family. Under these circumstances, the government could support either incarceration of the mentally ill or the use of psychoactive medications, or the misuse of psychiatry to control thinking, but could hardly support the encouragement of self-exploration, increased autonomy, expanded freedom to feel and think, and independent judgmental capacity under circumstances of confidentiality and privacy. China would be an example, where psychiatry is almost exclusively biological and there is no entity or training program in child psychiatry.

Looked at another way, in those countries where the form of government reflects a society that most highly values the importance of individual rights and free expression of ideas over the importance of submerging individual rights for the best interests of the group (the government), there will be greater support for and access to psychiatric and mental health services for children and adolescents. That correlation is best exemplified by the United States, Canada, and Israel.

Another perspective is that afforded by the extent of overlap versus separation of church and state. The smaller the degree of separation, the more conservative, orthodox, and absolute the religious influence is likely to be, and the more, once again, there would be little value placed on or encouragement given to the development in the child of strivings for autonomy, independence, and challenge to or questioning of authority. Only one belief system can exist. Under such circumstances, such as in countries whose governments and cultures are dominated by a fundamentalist religious orthodoxy, either Eastern or Western, one finds state-sanctioned gender discrimination, severe punishments for deviation, strict control of communication, and little opportunity for child psychiatric services to be valued or supported.

The worldwide child and adolescent psychiatric community, led by the American Academy of Child and Adolescent Psychiatry and by the International Association of Child Psychiatry, has both an opportunity and a responsibility to support the development of mental health services for children, adolescents, and their families to the maximum extent possible and wherever possible. Many initiatives already have been undertaken, but perhaps not in as organized, systematic, and strategically planned a way as is needed and could be undertaken. Various possibilities could include offering consultation to the health planning authority in targeted countries, initially *pro bono* with monies raised independently to support such an initiative, and support to send leading child psychiatrists on lengthy visits or sabbaticals to undeveloped countries to offer educational programs and assistance in introducing the core aspects of child psychiatry and psychopathology into the health care system.

### Chapter References

Okasha A, Karam E: mental health services and research in the Arab world. *Acta Psychiatr Scand* 98:406-413, 1998.

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## Appendix/Questionnaire on Child Services

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Name: \_\_\_\_\_

1. Date: \_\_\_\_\_
2. Country of Report: \_\_\_\_\_
3. List four major ethnic and/or religious groups making up the population, in order of importance in the country:
  - a. b.c.d.4. Type of government: Please provide a brief description of the current form of government, and if different, the form of government during most of post-WWII history:
5. What are major sources of payment for health care for children/adolescents, (rate 1 to 6, with "1" being the *most* important, "6" being the *least* important):
  - a. \_\_\_\_\_ Doctors and hospitals paid for by government funds for everyone (e.g., single payor)
  - b. \_\_\_\_\_ Insurance paid for by employers
  - c. \_\_\_\_\_ Paid for by individually owned policies with payment directly from the purchaser
  - d. \_\_\_\_\_ Paid for by combination of employer/employee contributions to insurance
  - e. \_\_\_\_\_ Paid by patient out-of-pocket directly to physician
  - f. \_\_\_\_\_ Government programs for specific groups, for example: children, elderly, poor, disabled, and, if so, which groups
  - g. \_\_\_\_\_ If none of the above are adequate to describe the most important, please give a brief description

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_
6. Does a significant majority of the population have a choice of physician/health care provider?



Yes \_\_\_\_ No \_\_\_\_

7. Is choice limited in some way?

Yes \_\_\_\_ No \_\_\_\_

If "Yes," please comment:

8. Is child/adolescent psychiatry an identifiable specialty or practice entity in this country?

Yes \_\_\_\_ No \_\_\_\_

9. If "Yes," are there special training programs for child psychiatry in post-medical school residency training?

Yes \_\_\_\_ No \_\_\_\_

10. If "Yes" are these programs:

- a. Very sought after
- b. Generally sought after
- c. About average
- d. Not very much sought after

11. List, by order of likelihood, who would a middle-class parent consult regarding a concern about a child's mental health, development, behavior, etc., with "1" being the *most* commonly considered option:

- a. \_\_\_\_ Family doctor
- b. \_\_\_\_ Pediatrician
- c. \_\_\_\_ Psychiatrist
- d. \_\_\_\_ Child/Adolescent Psychiatrist
- e. \_\_\_\_ Psychologist
- f. \_\_\_\_ Social Worker
- g. \_\_\_\_ Nurse
- h. \_\_\_\_ Other

12. How are child psychiatry services typically paid for? \_\_\_\_\_

\_\_\_\_\_  
\_\_\_\_\_

13. Number which of the following services are:

- 1. Generally available
- 2. Available but scarce
- 3. Rare or unavailable

- a. \_\_\_\_ Outpatient evaluation
- b. \_\_\_\_ Outpatient treatment
- c. \_\_\_\_ Inpatient services
- d. \_\_\_\_ Partial Hospitalization (include day-treatment)
- e. \_\_\_\_ Residential Treatment Programs
- f. \_\_\_\_ Mental Health Services in general and/or children's hospitals
- g. \_\_\_\_ Mental Health Services in schools
- h. \_\_\_\_ Special Education Services in schools for learning disabled and/or "emotionally disturbed."
- i. \_\_\_\_ Other (Please Specify)

14. Comment. (Please feel free to do so. Please add any description of the system which you would consider important and want to include.)

\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

Thank you for your help.

# 118 NATIONAL POLICIES FOR CHILDREN, ADOLESCENTS, AND FAMILIES

*Edward F. Zigler, Matia Finn-Stevenson, and Emily M. Tanner, M.Sc.*

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## [Focus on Mental Health Disorders in Children](#)

### [Scope of the Problem](#)

### [Contributing Factors](#)

### [Preventing Mental Health Disorders: Policy Directions](#)

### [Contributions from the Research](#)

### [Other Examples of the Possibilities Inherent in the Integration of Research and Policy: Program Development and Evaluation](#)

### [Family Support Programs](#)

### [Children and Violence](#)

### [Problems in the Use of Research in Policy](#)

### [Strategies for Change](#)

### [Chapter References](#)

Since the 1970s, researchers and clinicians in psychiatry, developmental psychology, and other disciplines related to mental health have become increasingly involved in the shaping of policies and legislation designed to address the mental health problems of children and youth. Although the focus of legislative action on behalf of children is not new, the presence of researchers and clinicians in the debate adds a different dimension and offers new opportunities for interaction between research and policy. Mental health professionals are directing their work toward the understanding of how contemporary social problems contribute to mental dysfunction in children, and they are not only reporting their research but also underscoring the policy implications inherent in their findings and suggesting a course of action. By participating in the policy process and by conducting studies relevant to social issues, they are contributing to the accumulation of knowledge and are thus enhancing their understanding of development as well as improving the nation's capacity to address the needs of children.

In this chapter, we discuss some of the social changes we experience in our society, their impact on children and families, and policy responses to address these. We also discuss the role of mental health professionals in the policy arena. It will become apparent in the course of the chapter that there are benefits as well as problems inherent in the use of mental health research in policy settings. Numerous opportunities exist for mental health professionals to contribute to the development of policies for children and families. However, their effectiveness in this regard is dependent not only on their knowledge of scientific principles and findings from mental health research but also on their familiarity with the social policy process and their ability to work with policy makers.

## **FOCUS ON MENTAL HEALTH DISORDERS IN CHILDREN**

Interest in social policy among mental health professionals was precipitated by several developments. One of these was the implementation during the 1960s and 1970s of federally sponsored social programs such as Project Head Start ([Zigler and Muenchow, 1992](#); [Zigler and Styfco, 1993](#); [Zigler and Valentine, 1979](#)). The proliferation of such programs, and the funds made available for them, enabled researchers and clinicians to apply their knowledge and training to such areas as program development and evaluation, which had not previously received their attention ([Phillips, 1987](#); [Salkind, 1983](#); [Takanishi et al., 1983](#)). A related development, noted by [Zigler \(1998\)](#), is that to secure funding for research, it is often necessary to demonstrate the practical application of findings and their potential to address societal needs.

Another development that fueled the interest in social policy was the recognition that children develop within the social context; they are influenced by various aspects of their immediate environment as well as by the more remote social institutions such as the school, the workplace, government, and the mass media, areas over which children and parents have little, if any, control ([Bronfenbrenner, 1979](#)). This realization gave impetus to ecologic studies and the compilation of information on children's behavior, achievement, and physical and mental health ([Garbarino and Kostelny, 1995](#)). On the basis of data generated by these efforts, it has become apparent that ever-growing numbers of children and adolescents in the United States face serious problems that often result in mental dysfunction ([Miringoff, 1993](#)).

### **Scope of the Problem**

The magnitude of the problem has been noted for some time: a committee of the [Institute of Medicine \(1989\)](#), convened at the request of the National Institute of Mental Health, studied the mental health status of children and adolescents. It found that at least 12% of children less than 18 years of age (7.5 million children) have a diagnosable mental illness, and many other children exhibit broader indicators of dysfunction, including substance abuse, teen pregnancy, and school dropout, which the committee defined as consequences of or risk factors for developing mental disorders. These findings are echoed in more recent studies. One of these studies estimates that 20% of youths aged 9 to 17 years have a diagnosable emotional or behavioral disorder, and 9% to 13% of them suffer from serious emotional disturbances that interfere with their daily functioning ([Manderscheid and Sonnenschein, 1996](#)). Another study, by [Kazdin \(2000\)](#), focuses on even younger children, indicating that 10% of 3- to 17-year-old children and adolescents in the United States receive treatment for emotional and behavioral disorders.

That so many children are affected by mental disorders suggests that the problem is of national concern. The costs involved in treating mental health disorders are difficult to estimate, in part because of comorbidity with other problems such as substance abuse, a situation that makes it difficult to separate the costs of care associated with each disorder. Additionally, the information needed to calculate the personal, social, and other costs of childhood mental disorders has not been systematically collected, thus rendering any cost analyses conservative estimates at best. Nevertheless, the studies that are available suggest that the costs of childhood mental disorders are staggering. [Rice et al. \(1990\)](#) found that treatment services for mentally ill children aged 14 years and younger exceed \$1.5 billion a year. Other investigators suggest that the costs of mental illness in children are much higher because, besides treatment costs, there are indirect costs and costs for nonhealth services, which are borne by families, the schools, the juvenile justice system, and other social institutions ([Office of Technology Assessment, 1986](#)). As an indication of the costs involved, the estimated direct and indirect cost of mental illness for the total population of the United States was \$150 billion in 1996, the latest year for which data are available ([U.S. Department of Health and Human Services, 2000](#)). Clearly, more definitive analyses are needed to establish the actual costs of childhood mental disorders, and such information is important if we are to have a context within which to make decisions about the care of mentally ill children and the allocation of funds to address their needs.

This is a critical issue, given the widely held belief that many children do not have access to mental health services. Two major points are noted in this regard. First, only a small proportion of the overall health budget is directed toward children ([Deal et al., 1998](#)). Second, the health care reforms that have replaced fee-for-service care with managed care have had both positive and negative impacts on mental health care for children and adolescents. According to the National Health Care Reform Tracking Project, a 5-year study of the impact of managed behavioral health care, access to health services in general has increased for young people ([Stroul et al., 1998](#)). However, with the emphasis on brief, problem-oriented approaches, it has become more difficult for children with serious emotional disorders (the "high users") and the uninsured to obtain the care needed. Additionally, 90% of health care expenditure for children is consumed by the 15% of children who have chronic illness and disability, and this leaves little for mental health care. In budget allocation decisions that are made, for example, when Medicaid funds are decreased, mental health services are often eliminated.

### **Contributing Factors**

Perhaps even more significant than the findings on the prevalence and potential costs associated with childhood mental disorders are the findings on the factors that contribute to the development of such disorders. More research is needed to unravel the causes and determinants of childhood mental illness. However, much progress has been made since the 1980s, including multiple lines of evidence that suggest that various biological, psychological, social, and environmental factors are involved as causal agents, and, in some cases, an interaction among these factors exacerbates vulnerability to mental disorders. Of significance is that in increasing numbers of children, social and environmental risk factors are implicated in the onset of mental dysfunction ([Galston, 1993](#); [Tuma, 1989](#)). Included among these risk factors are prolonged separations between the parent and child ([Tennant, 1988](#)), physical or sexual abuse ([Rutter and Smith, 1995](#); [Widom, 1998](#)), poverty ([Brooks-Gunn and Duncan, 1997](#); [Garmezy, 1985](#); [Rutter, 1976](#)), marital discord ([Wallerstein, 1988](#); [Wallerstein and Corbin, 1996](#)), parental psychopathology ([Beardslee et al., 1998](#)), instability in the family environment ([Rutter, 1987](#)), and various other stressors related to family life ([American Psychiatric Association, 1987](#); [Institute of Medicine, 1989](#); [Tuma, 1989](#)). [Rutter \(1980\)](#) points out that children who experience one of these risk factors may not be any more likely to suffer serious



consequences than children with no risk factors. However, the more risks or stressors that are present in children's lives, the greater is the probability of damaging outcomes.

In addition, some risk factors compound other problems, such as low birth weight and central nervous system difficulties, which, when they occur in isolation, may have no negative effects. Infant central nervous system difficulties, for example, may be overcome if the child is reared in a stable and supportive environment, but they are exacerbated if the child is raised in an unstable, poorly educated, low-income, or otherwise stressful family environment ( [Hughes and Simpson, 1995](#); [Sameroff et al., 1987](#)). Likewise, premature babies of low birth weight, who are more vulnerable to environmental insufficiencies than are full-term babies, may experience developmental problems if they are reared by unresponsive adults, but these infants may suffer no negative consequences if they receive appropriate care ( [Hack et al., 1995](#)).

#### *CHANGES IN FAMILY LIFE*

These findings raise concerns because many children today experience potentially damaging experiences that stem from difficult conditions in family life. Since the early 1970s, the society of the United States has undergone vast economic and social changes, which have transformed the structure of the family and the roles and responsibilities of men and women. These changes have created stressful conditions for children and adults.

Consider, for example, the increased fragmentation and isolation of the family. Currently, one of every four children in the United States lives in a single-parent family, and among African-American children, the rate is one of every two children ( [Children's Defense Fund, 1998](#)). The growth in the number of single-parent families is particularly disturbing because it is often associated with multiple stressors for both parents and children. The presence of a female head of household, young children, poverty, and maternal depression are characteristic traits (although not ubiquitous ones) of single-parent families ( [Kagan and Fuller, 2000](#); [U.S. Bureau of the Census, 1988](#)). The rise in single-parent households is attributable largely to the increasing number of births to unmarried mothers ( [Blank, 1997](#); [Hernandez, 1998](#)). Although the birth rate for married women has declined since 1980, the birth rate for unmarried women increased 49% between 1980 and 1990 and remained constant through to 1995, a finding reflecting the growing preference for unmarried status ( [U.S. National Center for Health Statistics, 2000](#)). The percentage of nonmarital births to African-American women is particularly high, at 69.7% ( [Blank, 1997](#)).

Some single-parent families are the result of divorce. Although the rate of divorce has remained constant after a significant rise in the 1960s and 1970s, roughly 40% of children will nevertheless experience the divorce of their parents ( [Behrman and Quinn, 1994](#)). The rate of divorce is higher among African-American families ( [U.S. Bureau of the Census, 1992](#)). Although divorce is not necessarily a disaster for children, stresses arising from the disruption and subsequent remarriage of parents often lead to psychological difficulties among children ( [Emery, 1999](#); [Twaite et al., 1998](#)).

A related change in family life is the relative isolation and lack of social support that have occurred because of the increasing mobility of people in search of employment and other opportunities, as reported by McCullough, in testimony presented to the U.S. Congress ( [U.S. House Select Committee on Children, Youth and Families, 1987](#)). As a result, many families no longer live near or have access to the support and assistance of friends and relatives. Referred to by some as a decrease in "social capital" ( [Coleman, 1987](#)), the lack of social support is notable, because having access to a support system often mediates the negative consequences of stress ( [Brown and Harris, 1978](#); [Garnezy, 1985](#); [Gore, 1980](#)).

#### *IMMIGRATION AND ETHNIC DIVERSITY*

Another important societal change has been the growth in the proportion of immigrants and ethnic minorities in the United States. Currently, 20% of children less than 18 years of age who are living in the United States are immigrants, and this percentage is likely to increase ( [Blum and Berrey, 1999](#)). The profile of immigrants in the United States is changing; they are arriving from increasingly diverse countries of origin and are experiencing more limited economic opportunities upon arrival in the United States than was the case with previous generations of immigrants. Research indicates that, despite the great diversity between and within immigrant groups, many share particular needs arising from the experience of stress, depression, and isolation in the years after their arrival in the United States ( [Board on Children and Families, 1995](#)). Furthermore, because many immigrants lack full access to education, health, and social services, the healthy development of children is jeopardized. There has also been a significant rise in racial and ethnic diversity within the U.S. population, and this development also raises concerns regarding the availability, access to, and nature of social services. It is projected that by 2030, 50% of children living in the United States will be nonwhite ( [Hernandez, 1998](#)). It is thus increasingly important that services for children are tailored to the cultural diversity of the population. The increased diversity also poses a challenge for policy makers to design effective antipoverty strategies because the poverty rate is higher among some racial and ethnic groups than in the general population.

#### *POVERTY*

Poverty is a grave concern because increasing numbers of families with young children are experiencing serious economic problems. This is in part because of the growth in the number of single-parent households, which are the largest and fastest growing family type ( [Blank, 1997](#)). According to the Current Population Survey ( [U.S. Bureau of the Census, 1999](#)) in 1998, children younger than 6 years old who were living with a single mother were five times more likely to be poor than children living with both parents. Other contributing factors are cuts in public assistance and the decline in the real value of family income ( [Blank, 1997](#); [Children's Defense Fund, 1993](#)). The United States provides far less public income assistance to single-parent families than do many other industrialized nations ( [Blank, 1997](#)). Instead, welfare reform initiatives emphasize labor force participation as the route out of poverty. However, whereas in the past, economic prosperity and employment were effective means of reducing the poverty rate, they are no longer sufficient as an antipoverty strategy for single-parent families because of the decline in wage rates, particularly for less-skilled workers ( [Blank, 1997](#)). For example, in the 1980s, each 1% expansion in the aggregate economy correlated with a \$0.32 decline in weekly wages. Comparing two strong economic years, high school dropouts earned 22% less in 1993 than in 1979, whereas high school graduates earned 12% less. This compares to a rise in income of 10% for college-educated men and 22% for men with postcollege degrees ( [Blank, 1997](#)). For two-parent families, the wage decline has been partly offset by the entry of increasing numbers of women into the labor force, but for single-parent families, the escape from poverty is more difficult.

The decline in real income affects adults and children. However, for children the consequences are particularly serious because, as noted earlier, many families in poverty are those with young children. Indeed, even though the overall poverty rate has declined in recent years, poverty among children less than 18 years old remains high at 18.9%, which is 13.5 million children. Children less than 6 years old are particularly vulnerable, with a poverty rate of 20.6% ( [U.S. Bureau of the Census, 1999](#)).

The ramifications of living in poverty are numerous and include assaults on children's physical and mental health ( [Wolfe, 1995](#)). [Klerman \(1991\)](#) found that poor families have no access to health care, and other conditions associated with poverty, such as lack of money to spend on health-promoting activities, hunger, lack of transportation, and inadequate housing, further exacerbate the problem. As a result, poor children experience more health problems and have a higher mortality rate. Several other studies indicate a powerful, albeit indirect, link between poverty and mental health disorders, thus leading mental health professionals to the conclusion that poverty is one of the major risk factors in such disorders ( [Albee, 1986](#); [Rutter, 1976](#); [Webster-Stratton, 1998](#)). Although at one time mental dysfunction, low achievement, and other problems associated with poverty were discussed in terms of assumed negative traits of poor children, researchers now realize that the major sources of psychopathology associated with poverty stem from environmental stresses and feelings of powerlessness and frustration ( [Albee, 1986](#); [Albee and Gullotta, 1997](#)). Moreover, poor families have a high incidence of poor prenatal care, low birth weight, and malnutrition ( [Brown, 1998](#); [Chomitz et al., 1995](#)), which are known to contribute to children's vulnerabilities to environmental stress ( [Institute of Medicine, 1989](#)). Parental depression and substance misuse are also heightened among poor families, and these factors increase the risk of child neglect and abuse and contribute to mental health disorders ( [Johnson and Leff, 1999](#); [U.S. Department of Health and Human Services, 2000](#); [Widom, 1998](#)).

#### **PREVENTING MENTAL HEALTH DISORDERS: POLICY DIRECTIONS**

These stressful conditions are just a few examples of the changed circumstances under which many children live. Other potentially damaging conditions that result from marital discord, maternal employment, and the increasing reliance of families on child care are discussed later in the chapter. Although most families are affected by these changes in family life, social policies in the United States have not kept pace with societal changes. Our society is, as a result, in a state of disequilibrium wherein social policies are not in synchrony with the realities of family life.

#### **Contributions from the Research**

This disequilibrium is creating difficulties for families. Not all mental health disorders in children stem from such difficulties. However, the stressful conditions under which many children live place a burden on children's ability to cope with the demands of school, the family, and relationships with peers. The problems that emanate

from the changing conditions of family life touch on economic realities, traditions, and institutional structures, so solutions may be slow to evolve ( [Zeitlin, 1989](#)). Nevertheless, mental health professionals can have a positive effect on family life in several ways. For example, they can alert policy makers and the general public to how stress in families negatively affects the very core of society. They can also call for further studies of the conditions under which children live and children's responses to these realities. This focus on research is important. It can deepen our understanding of how children are affected by different conditions and of why some children are able to cope with difficulties in their lives whereas other children succumb ( [Garmezzy, 1985](#); [Gunnar, 1996](#)). The research can also enhance our understanding of the ways children cope with problems so we can devise useful strategies for intervention and prevention. To illustrate how scientific research can be a constructive force in the policy process, we discuss some of the studies related to major societal changes as well as associated policy developments that have had a profound impact on family life.

### WELFARE REFORM

The most dramatic piece of legislation for children and families in recent years is the Personal Responsibility and Work Opportunity Reconciliation Act of 1996. By replacing Aid to Families with Dependent Children with Temporary Assistance to Needy Families, the government effectively reduced public entitlement to cash benefits. States are now granted greater flexibility in the use of welfare funds, although they are obliged to impose work requirements and a 5-year lifetime limit on receipt of federal assistance.

What has been the impact on children and families? The plethora of research studies on the impact of welfare reform reveals success in achieving a 40% reduction in the numbers of dependent families, along with a substantial increase in labor force participation by mothers ( [Blum and Berrey, 1999](#)). However, these positive indicators may mask unmet needs. First, a primary implication of increased employment is the need for child care. Research indicates that many parents lack access to high-quality care for their children, either through poor provision or because it is too expensive. These findings were highlighted in the first wave of results from a study of the impact of welfare reform in California, Florida, and Connecticut ( [Kagan and Fuller, 2000](#)). The study noted that most children were in home-based care, and only 13% of this care was deemed to be of good or excellent quality. (The importance of high-quality preschool care for children's development is discussed later in the chapter.) A second implication of the welfare reform emphasis on employment is that some parents who have difficulty in maintaining employment may be rendered ineligible for financial assistance, thus increasing the poverty of their family. For example, the long-term unemployed are likely to require support to maintain their jobs; furthermore, some poor people, through depression, domestic violence, addictions, or mild mental retardation, may be unable to sustain employment. The figures indicating reduction in welfare dependency do not account for those who have been diverted from welfare assistance without entering employment ( [Blum and Berrey, 1999](#)). Third, welfare and immigration legislation has reduced access to noncash services such as Medicaid and Food Stamps, and it has affected 20% of the children living in the United States ( [Blum and Berrey, 1999](#)). [Aber et al. \(1995\)](#) further indicate that overall welfare reform does not appear to have reduced child poverty, despite the increased levels of parental employment. However, the researchers note that some specific groups, such as children of teen parents, may fare well as long as they are provided with sufficient support services to mediate any negative impact associated with welfare reform.

### MATERNAL EMPLOYMENT

Another complex societal change affecting all children, regardless of family income, has been the entry of large numbers of women into the labor force. This phenomenon is especially apparent among women who have children. For women with school-age children, full-time employment has been relatively common for about three decades; more than 70% of such mothers now work out of the home. Among women with infants and preschool children, more dramatic changes have taken place. In 1973, 30% of mothers with children younger than 6 years old and 50% of mothers with school-age children were working. By 1997, these figures had risen to 65% and 77%, respectively ( [Children's Defense Fund, 1998](#)). Among mothers with infants, the rise has been even more dramatic. In 1987, fewer than 30% of women with infants 1 year old and younger were working. By 1998, 55% of mothers returned to full-time work within a few weeks of the baby's birth ( [Children's Defense Fund, 1998](#); [U.S. Department of Labor, 1997](#)).

Research on the effects of maternal employment on children finds that, in and of itself, maternal employment is not necessarily associated with either negative or positive effects ( [Greenberger, 1989](#); [Hoffman, 1989](#); [Zaslow and Emig, 1997](#)). Rather, parental attitudes to the mother's employment are more significant in their effects on children than is employment itself ( [Hoffman, 1986](#); [Hoffman, 1989](#)). However, researchers point out that although maternal employment appears to be benign in its effects on children, in many dual-worker families, both the parents and the children experience an inordinate amount of stress. In general, women have assumed new roles in the workplace, but they have not abdicated their traditional responsibilities to family life and child rearing. This situation has resulted in role conflict and guilt ( [Hoffman, 1989](#); [Moen and Dempster, 1987](#)), as well as in changes in lifestyle and difficulties that permeate the whole family system. Studies have found that almost 40% of employed parents, both women and men, indicate that they experience severe conflict, guilt, and stress ( [Friedman, 1987](#)). For children, this state of affairs means that not only do they have less time with their parents, but also they are affected by the fact that their parents are under stress from trying to do too much.

Other institutions besides the family are affected by the increase in women's participation in the out-of-home labor force. Employers, concerned about worker productivity, are raising questions about women's juggling work and family responsibilities, and some employers are also beginning to realize that they may be losing valued female employees when child rearing conflicts with full-time work. There are also pressures on institutions such as the school, which have to implement changes to accommodate the needs of children not only during school hours but also before and after school ( [Finn-Stevenson and Zigler, 1999](#); [Zigler, 1989](#)). Additionally, changes have occurred in some professions that were previously associated with flexible work schedules that enabled mothers to work and at the same time rear their children. Teachers, for example, are finding that they have to extend their work day and thus disrupt their own family life because many of their students' parents are working and are unavailable for parent conferences and other school events during the day (Zeitlin, 1989).

### Increased Demand for Child Care

Although numerous societal changes are associated with women's participation in the labor force, none are as significant as the unprecedented demand for child care services. Today, child care is one of the most widely recognized social problems. Virtually everyone, from working parents to chief executives of major corporations, is discussing the lack of good-quality, affordable child care services. Child care is also the subject of debate at state and local level governments, where the need for child care services is noted. The attention to the child care issue is not surprising, given the increase in the number of infants and children who need child care. However, it belies the fact that it has been a major social problem for more than three decades. At the 1970 White House Conference on Children, the need for child care services was noted as the number-one priority for the nation to address. However, two obstacles—ideologic arguments against the use of child care and the lack of public awareness of the need for child care services—stood in the way of policy action on the issue ( [Nelson, 1982](#)). As a result, the problem worsened and reached crisis proportions before it finally attracted national recognition.

Notwithstanding the attention the issue now receives, child care continues to be regarded as an individual family problem to be addressed by parents. This is evident in that most businesses do not make provisions to ease the stresses associated with balancing work and family life, and despite the enactment of the Family Support Act and the Child Care and Development Block Grant, which provide needy families with financial assistance for child care, the nation is still far short of having a comprehensive solution to the problem ( [Finn-Stevenson and Zigler, 1999](#); [Zigler and Lang, 1991](#)).

The child care problem has numerous facets, one of these being the high cost of services. This is a major concern for parents, some of whom choose a child care facility solely on the basis of cost. This point is made by [Hofferth and Wissoker \(1992\)](#), who found that parents not only choose child care on the basis of cost, but also switch facilities if the price increases. Precise data on what families spend on child care are not available, but it is known that child care costs are anywhere between \$1,500 and \$10,000 a year, depending on the quality of care and the age of the children. It is estimated that full-time child care for preschoolers costs an average of \$3,000 a year, and for infants, the costs can exceed \$9,600 a year. With the cost of care being so prohibitive, it is not surprising that it is one of the major factors in choosing child care.

From a policy perspective, the high cost of care is significant for at least two other reasons. First, child care costs are a major expenditure for families, and the amount of money families spend on child care is directly related to their income ( [Future of Children, 1996](#)). Low-income families spend less on child care in absolute terms than do higher-income families, but the proportion of the family budget that is taken up by child care costs is greater among low-income families, who have to allocate as much as 27% of their earnings to child care ( [Kagan and Cohen, 1997](#)). Even for middle-income families, the burden of child care costs are great, because these families do not qualify for state subsidies and pay higher taxes on their earnings ( [Betson and Michael, 1997](#)). Second, there is a relationship between the cost and quality of care, with good-quality care costing substantially more than poor-quality, custodial care. This being the case, there are inequalities in the quality of child care children experience, depending on their family's income. The finding that good-quality care is a privilege that only some children are enjoying is of concern because the child care setting is an environment in which children spend a large portion of every day. As such, it has significant effects on children's development and well-being. For example, research highlights the link between educational child care programs and reduced rates of violence and crime ( [Newman et al., 2000](#)).



In an attempt to define good-quality care, mental health professionals make a distinction between developmentally appropriate care that is responsive to the needs of children and care that is merely custodial ( [Phillips, 1987](#)). The determinants of good-quality care have been found to be an age-appropriate ratio of staff to children and group size, as well as the presence of providers who have knowledge of and training in child development ( [Arnett, 1989](#); [Roupp et al., 1979](#)). Training in child development, rather than years of experience working with children, sets apart nurturing providers who respond to the varied and individual needs of young children from providers who are unable to provide children with appropriate experiences ( [Arnett, 1989](#)).

Researchers have used this information in important ways to address the needs of children and to promote national awareness of the lack of quality child care and its impact on children. For example, in the Cost, Quality, and Outcomes Study ( [Helburn, 1995](#)), researchers examined child care centers in four states and looked at the relationships among the cost of child care, the actual experiences of the children in the centers, and the effects of these experiences on children's development. The study noted several disturbing findings, the major one being that although child care centers vary widely, most centers are mediocre in quality and are "sufficiently poor to interfere with children's emotional and intellectual development" ( [Helburn, 1995](#)). Forty percent of the infants and toddlers in center-based care were found to be at risk as a result of poor health and safety standards. In the follow-up to this study, it was confirmed that children in poor-quality care do indeed experience developmental problems that persist when the children are of school age ( [Peisner-Feinberg et al., 1999](#)). Another study focused on the quality of care in family day care homes and in the homes of relatives, which are popular child care choices among parents, especially low-income parents and parents of infants and toddlers. The study found that only one in nine family day care homes offered good-quality care, and that the quality of care by relatives was even worse ( [Galinsky et al., 1994](#)).

Researchers used child care quality indicators in other ways as well, for example, in the development of training programs and accreditation for child care providers and child care facilities ( [Bredenkamp and Copple, 1997](#); [Ward, 1976](#)) and in the establishment of guidelines for the operation of child care programs ( [Provence, 1982](#)) and evaluative tools to assess the quality of child care facilities ( [Harms et al., 1998](#)). [Young et al. \(1997\)](#) used quality indicators to determine whether the standards governing states' child care licensing requirements are adequate. These investigators found that although state regulations represent a basic minimum in terms of ensuring the health and safety of children, the regulations in most states fall below even that minimum. In other words, a child care facility may be licensed by a state, but that does not mean that it provides good-quality care. The researchers not only analyzed states' regulatory standards, but also recommended changes states can make to upgrade the quality of child care.

These and other similar efforts underscore two important points about the link between research and policy: (a) research findings can be brought to bear on social problems and can serve as the impetus for appropriate action ( [Zigler and Finn-Stevenson, 1987](#)), and (b) a thorough understanding of the problem—in this case, the factors that influence quality care—is essential if mental health researchers are to be able to make recommendations for appropriate action.

The research on child care, discussed only briefly in this chapter, has also focused on the effects of child care on infants as well as other issues such as supply and demand and the availability of child care services. The research has been important and provides indications that although the supply of child care facilities for preschoolers has increased since 1977, there are still regions in the United States where demand exceeds supply ( [Kisker, 1991](#)). Studies also suggest that the demand for child care is going to increase, given the growing presence of women in the labor force as well as other demographic factors and employment trends ( [Child Care Action Campaign, 1988](#); [Hernandez, 1998](#)). Additionally, it has been found that in two segments of child care, namely, child care for infants and toddlers ( [National Center for Clinical Infant Programs, 1988](#)) and child care for school-age children, the demand far exceeds the supply ( [Seligson, 1989](#)). On this latter point, the research has led to policy action, with the federal government allocating substantial funds for the establishment of supervised programs for children during time when they are out of school. This is important because, for adolescents, the peak time for violence, crime, and sexual activity is after school hours, between 3 and 6 PM, when many are unsupervised by an adult ( [Besharov, 1999](#)).

#### *Changes in the Workplace to Support Family Life*

The high cost of child care and the lack of good-quality care is an issue not only because of the possible harm to children, but also because it is a source of stress for parents. This situation has prompted discussions of the need to make changes in the workplace to create conditions that are supportive of family life. Several suggestions have been made, including corporate support of child care services. Although a few companies have developed on-site child care centers for their employees, and others partially offset the cost of child care for their employees or provide other supportive services, such as information and referral, the need for child care is too large for corporations to address alone. Corporate involvement can thus be conceived, at best, as only one part of a comprehensive solution to the child care problem.

However, there are other ways for corporations to support family life. For example, some corporations may be able to implement flexible schedules to allow employees time for child-rearing responsibilities ( [Bureau of National Affairs, 1986](#)). Some corporations provide this option to their employees, but most do not ( [Friedman, 1987](#); [Kamerman, 1983](#)). In some businesses, flexible work schedules may prove to be counterproductive and therefore unworkable as a means to enhance family life. However, among some corporations, such changes can be effectively implemented if steps are taken to alert the corporate world to the need and importance of such changes in the workplace. It has been suggested that mental health researchers can help by engaging in relevant studies. Among the questions that need to be addressed, [Stipek and McCroskey \(1989\)](#) note that little research is currently available on the different work schedules that support employees in their role as parents, on the increased productivity, if any, associated with different work schedules, and other important questions, such as how do different work schedules for parents affect the frequency with which children are sent to school ill or are examined by a doctor? This latter research question is important in part because even when parents have good child care arrangements, they need to be at home with their children when the children are ill. Parents' inability to take time off from work, however, has resulted not in any workplace policy changes but rather in the development of child care facilities that specialize in the care of sick children. This is disturbing because children who are ill need to be with their parents. No studies are needed to establish this fact. In fact, the American Academy of Pediatrics indicates that studies substantiate that children's ability to overcome illness and to benefit from medical treatment is directly related to their being with their parents during the illness. However, because of lack of advocacy on the issue, society's response to the care of sick children has been child care for sick children rather than the institution of flexible work schedules for parents.

#### *Parental Leave*

Related to flexible work schedules is the need for parental leave policies that would enable parents to spend time with their infants during the first several months after birth. Any student in child psychiatry is well aware that the first few months of life represent a critical period for the development of attachment between parents and the infant, and within the context of a secure parent–infant relationship, the growing child thrives and is encouraged to become more autonomous ( [Ainsworth et al., 1978](#); [Cicchetti et al., 1990](#)). The first few months of life also represent a very stressful period of life that necessitates the adjustment of all family members to the newborn ( [Brazelton, 1985](#)).

A Blue Ribbon Committee on Infant Care Leave studied the issue and found that increasing numbers of women return to work very shortly after the birth of a baby. The committee recommended that, in the interests of infant mental health as well as parents' well-being, one of the parents should be given the option of a paid leave of absence from work for the first 6 months after the infant's birth ( [Zigler and Frank, 1988](#)). Although the importance of such leave is noted on the basis of medical and social science research ( [Hopper and Zigler, 1988](#); [Zigler and Frank, 1988](#)), adequate parental leave is not available to most parents in the United States.

This lack is despite the passage of the Family and Medical Leave Act in 1993, which granted 12 weeks of unpaid leave in any 12-month period to employees in companies of 50 or more workers. The impact of the federal legislation was limited for three major reasons. First, 34 states had already passed laws before the Family and Medical Leave Act ( [Finn-Stevenson and Trzcinski, 1991](#); [Trzcinski and Finn-Stevenson, 1991](#)). Second, as many as 40% of workers were ineligible for leave because of the size of their companies, because most workers are in small businesses ( [U.S. Department of Labor, 1996](#); [Wisensale, 2000](#)). The business community is adamantly against any interference with their policies and has thus lobbied to exclude small companies from eligibility. Third, the leave granted was unpaid, and research shows that many workers have been discouraged from taking leave for financial reasons ( [Waldfogel, 1999](#)). However, the Family and Medical Leave Act is significant as the first national move toward parental leave, and it has prompted discussions about paid leave at the federal level and in some states, such as Connecticut. Again, the issue of parental leave is instructive, because it reveals how findings from research can influence policy.

#### *DIVORCE*

Another reality for many families is divorce. The rate of divorce, particularly among families with children, rose dramatically between 1965 and 1979. Since 1979, the rate of divorce has declined and seems to have reached a plateau ( [Hernandez, 1988](#)). Nevertheless, it is estimated that half of all first marriages in the United States will end in divorce ( [U.S. Bureau of the Census, 1992](#)). The presence of children is not a deterrent: roughly 60% of all current divorces involve children ( [U.S. National Center for Health Statistics, 1995](#)). Most children will experience the remarriage of one or both of their parents and will live in reconstituted families ( [Emery, 1999](#)).

This is illustrated by the following data. Although 71.8% of U.S. children live with a mother and father figure, the percentage of children living with their biological parents is much lower: 64.9% of white children, 23.6% of African-American children, and 53.4% of Hispanic children ( [U.S. Bureau of the Census, 1992](#) ).

Controversy surrounds the impact of divorce and remarriage on children. [Emery \(1999\)](#) draws four salient points in his review of the research. First, divorce is a source of great stress for children, whether through loss of contact with a parent or because of economic hardship. Second, divorce makes psychological problems up to twice as likely for children: even though children may appear to adapt well initially, there may be delayed negative effects ( [Wallerstein, 1988](#) ; [Wallerstein, 1991](#) ; [Wallerstein and Corbin, 1996](#) ). Third, despite the risks, most children from divorced families function as well as those from married families, a finding demonstrating resilience ( [Twaite et al., 1998](#) ). Fourth, resilience does not preclude vulnerability: children experience difficult feelings despite coping well. The outcomes of divorce for children depend on many factors including age, gender, ethnicity, and socioeconomic status. [Tschara et al. \(1990\)](#), for example, find that children have difficulty in adjusting to the divorce if they are older, have had prior psychological problems, and have parents with more marital conflict. [Hetherington et al. \(1989\)](#) find that the long-term effects of divorce and remarriage appear to be related to numerous factors, including the following: the child's developmental status, gender, and temperament; the quality of the home environment; and the availability of support systems, both to the parents and the child.

The number of stressors the child experiences is also a factor because, as noted earlier, a single stress typically does not carry with it appreciable psychiatric risk, but multiple stressors increase the risk of mental dysfunction ( [Rutter, 1980](#) ). Particular concern is noted for children whose custodial parent experiences extreme economic difficulties for an extended period or whose noncustodial parent fails to pay for child support ( [Emery, 1999](#) ; [Haskins et al., 1985](#) ). In 1991, only 54% of single parents with children had awards for child support, and only half of these awards were paid in full ( [Emery, 1999](#) ; [U.S. Bureau of the Census, 1995](#) ). Even when awards were paid, the average amount was small, at \$3,011. Part of the problem is that the noncustodial parent also may have a low income, but often, noncustodial parents simply refuse to acknowledge financial responsibility for their children.

Children whose parents suffer emotional and psychological difficulties as a result of the divorce are also likely to experience multiple stressors. Researchers have found that parents whose distress is acute fail to attend to the needs of their children, do not recognize the children's painful experience with the divorce, or burden the children with their own adjustment difficulties ( [Kurdek and Blisk, 1983](#) ).

Also at substantial risk are children who are involved in prolonged custody fights. These are the most vulnerable of children of divorce because custody battles can continue indefinitely. Judges attempt to make custody decisions on the basis of the best interests of the child. However, neither judges nor lawyers are prepared for the arduous task of determining the best interests of the child, nor are they trained to interview the child, consider his or her needs and concerns, or weigh the urgency of the child's condition and circumstances ( [Wallerstein, 1986](#) ). Recognizing the child as the hidden client in divorce proceedings, Goldstein et al. attempt to provide guidance to lawyers and judges by incorporating legal considerations within the framework of principles drawn from developmental psychology and psychiatry ( [Goldstein et al., 1973](#) ; [Goldstein et al., 1979](#) ). They recommend that decisions regarding child custody be made quickly, that an effort be made to avoid prolonged proceedings, and that whatever decision is made have a final effect that is not reversible. They further recommend that judges award full custody of the child to one "psychological parent." There is controversy surrounding this last recommendation. Some psychiatrists emphasize the psychological value for some children of maintaining a close relationship with both parents, even those involved in a bitter dispute over custody issues ( [Guidibaldi et al., 1983](#) ). Although some of their recommendations are controversial, Goldstein et al. pave the way for other psychiatrists to think about the use of knowledge and theoretical principles in establishing criteria for practical decisions that involve children.

#### *Mediating the Effects of Divorce*

The need for mental health professionals to consider the policy implications of their work is underscored by Wallerstein ( [1986, 1988](#) ). Wallerstein notes that although increasing numbers of policy makers and legal professionals are seeking guidance from the mental health professions, the accumulation of psychological knowledge has not kept up with rapid changes that have occurred in family law. [Hetherington and Camara \(1984\)](#) make a similar point, by indicating that the knowledge about the effects of divorce on children and on postdivorce parent-child relationships is still fragmentary, with several important questions remaining to be addressed.

Although more research is needed, sufficient information currently exists for us to appreciate the widespread implications of the research and the opportunities that exist to mediate the consequences of divorce. In this regard, the knowledge of how divorce affects children and parents should be disseminated not only among mental health professionals but also among other professionals who work with children. Teachers, for example, need to be alerted to these findings so they can be sensitive to any changes in children's behavior and can offer them and their parents support and counsel about ways they can cope with the changes in their lives ( [Kurdek, 1981](#) ; [Miller et al., 1999](#) ). Additionally, as noted earlier, the legal profession needs to be made aware of the research and its implications. Ideally, psychological support for parents and children should be made available immediately when the divorce proceedings begin. One important policy development has been the use of mediation services in divorce cases. These services are staffed by mental health professionals who have access to legal advice. Although initially begun as a way to curtail the high costs of divorce, families who have used mediation services note that one of the major benefits of the services is the availability of psychological support ( [Bahr, 1981](#) ).

The importance of psychological support for children of divorce should be made known to policy makers, who can make it a national priority to ensure that these children have access to support services. Several successful support programs for children of divorce have been developed in schools across the United States ( [Weiss, 1989](#) ). However, they are few, and they meet the needs of only a small percentage of the children who stand to benefit from such programs. Given the number of children who need such support, these programs should be made available in all schools, or at least in some schools in every community. The federal government can take a leadership role by making available funds that would finance the development of such programs.

#### *PARENTAL PSYCHOPATHOLOGY*

Divorce is only one circumstance under which parents may be unable to fulfill their parenting responsibilities adequately. A substantial body of literature supports the observation that children of psychiatrically impaired or substance abusing parents have an increased risk of developing mental health problems compared with children of normally functioning parents ( [Beardslee et al., 1998](#) ). It has long been recognized that maternal depression is widespread, particularly among families in receipt of welfare ( [Brown and Harris, 1978](#) ). [Kagan and Fuller \(2000\)](#) observed in their sample of 948 single mothers with young children that the incidence of depression was three times higher than the national average, and maternal depression resulted in disengaged parenting practices likely to result in poor development.

Another area of increasing concern is parental substance misuse. An estimated 11 million children less than 18 years old have alcohol-dependent parents, and the number of children whose parents abuse drugs is unknown ( [Johnson and Leff, 1999](#) ). Negative influences are transmitted to children through a variety of mechanisms. One leading area of research addresses the consequences of prenatal exposure to cocaine and identifies disruptions to development such as impaired arousal regulation, which lowers the threshold for coping with stressful conditions ( [Mayes et al., 1998](#) ). Other scholars document the relationship between parental substance abuse and subsequent psychopathology in children and emphasize the role of mediating factors such as family conflict, lack of family rituals, poor home management, ineffective parenting strategies, physical violence, abuse, isolation, stress, and frequent family moves ( [Johnson and Leff, 1999](#) ; [Widom, 1998](#) ). With the rates of drug misuse increasing, it is important to provide medical and social support to families to ensure healthy child development.

#### **OTHER EXAMPLES OF THE POSSIBILITIES INHERENT IN THE INTEGRATION OF RESEARCH AND POLICY: PROGRAM DEVELOPMENT AND EVALUATION**

The importance of support services is noted for many families who encounter different types of stressful life events. Many families, for example, those who have premature or handicapped babies ( [Field et al., 1980](#) ; [Goldberg and DiVitto, 1983](#) ; [Shiono and Behrman, 1995](#) ) or who experience the illness or death of family members, have difficulty coping and are in need of some kind of support. Likewise, many people need assistance with child rearing: many parents need help in gaining the ability to nurture and discipline children simultaneously, or they need assistance coping with transitional problems encountered during different stages of their children's development ( [Albee and Gullotta, 1997](#) ).

Support services are also needed to prevent chronic juvenile delinquency. [Yoshikawa \(1994\)](#) notes an increase in juvenile crime in the United States. There was a 60.1% increase over a 10-year period in arrests of youths less than 18 years old for murder and manslaughter ( [Federal Bureau of Investigation, 1991](#) ). Juvenile crime is linked to the violence that has become part of the daily lives of families, especially those who are poor; it is experienced by increasingly younger children ( [Ososky and Fenichel, 1994](#) ). [Yoshikawa \(1994\)](#) documents several factors that predict chronic delinquency: low socioeconomic status, criminally convicted parents, low intelligence, poor parental child rearing, troublesomeness, and conduct disorders. His review of the research further suggests that programs that combine early family support and education and include, among several aspects, a parent-focused informational and emotional support component, represent a promising direction for the



primary prevention of early-onset, chronic delinquency. Other studies ( [Berrueta–Clemant et al., 1984](#); [Zigler et al., 1992](#)) suggest that early childhood intervention and support programs are linked to the prevention of juvenile delinquency.

## Family Support Programs

In response to the widespread need for such programs, many family support services have been developed and implemented. The programs range from informal, grass-roots, self-help services such as Parents Anonymous and Parents without Partners ( [Whittaker and Garbarino, 1983](#) ), to more formal types of services that include professional assistance ( [Gullotta, 1997](#) ). These programs have been referred to as a “new breed” of programs in that they are rooted in the premise that the most effective way to create and sustain benefits for children is to improve their families and communities. However, this premise is hardly new and can be traced to Project Head Start ( [Zigler and Freedman, 1987b](#) ). Project Head Start, along with other social programs, was initiated in the 1960s in an effort to enhance the lives of young children. It was, and continues to be, an innovative program that includes a cycle of experimentation and revision that helps to ascertain which types of services are best suited for and have the most impact on children. As a result of this cycle of experimentation and revision, and because of research interest in the ecologic study of children, the conventional wisdom about how to address the needs of children has shifted from child-centered programs to programs that focus not only on the child but also on the family as a whole ( [Bronfenbrenner and Weiss, 1983](#); [Zigler and Berman, 1983](#) ).

Although the development of family support programs is conceptually traced to previously developed social programs, these newer programs differ in numerous ways. Most important, many family support programs are nongovernmental initiatives. Rather, they began as grass-roots efforts, initiated and sustained by individual persons in response to stressful situations in their lives, and in the absence of any other form of social support. Although several states have initiated family support programs ( [Powell, 1991](#); [Weiss, 1989](#) ), such programs are still characterized by the lack of government support. Another characteristic of family support programs is that although the programs are varied in the type of services rendered and in the population served, they share a commitment to provide emotional, informational, and instrumental assistance to family members and thus enable persons to cope with whatever problems they may have ( [Weiss, 1995](#); [Zigler and Weiss, 1985](#) ).

### Primary Prevention

In helping in these ways, family support programs exemplify a primary prevention strategy that focuses on the prevention of mental health disorders ( [Caplan, 1974](#); [Albee and Gullotta, 1997](#) ). As noted earlier in this chapter, there is a clear and consistent relationship in research findings between adaptive difficulties and heightened levels of stress ( [Bloom, 1979](#); [Cowen, 1980](#); [Hamburg, 1982](#) ). Other studies have established that both personal and situational variables may mediate this reaction and may enable persons who are vulnerable to become better able to cope. One such variable is social support. Social support has been found to improve a person's ability to withstand stress ( [Albee, 1986](#); [Cassell, 1976](#) ), to mediate the consequences of life crises ( [Gore, 1980](#) ), and to enhance general adjustment and well-being. Social support systems, according to [Caplan \(1974\)](#), should not be conceived of as the propping up of someone who is in danger of falling. Rather, they refer to efforts to augment someone's strengths to facilitate mastery of the environment. [Caplan \(1974\)](#) further points out that social support as a means of primary prevention in mental health should not be perceived as a one-time intervention but rather as an enduring pattern of continuous or intermittent ties that help to maintain the psychological and physical integrity of the individual person.

Families, like individual persons, have a certain life course in which, at particular points, stresses and crises are a natural state of affairs. At those times, support programs can be invaluable in helping family members to use their strengths and to rally to cope with the problem, thus warding off severe family dysfunction and mental health disorders ( [Riessman, 1986](#) ). Although family support programs have this primary prevention potential, the degree to which they are effective in preventing mental disorders is not yet known, because the growth and proliferation of family support programs have not been matched by evaluations of their efficacy ( [Elias, 1997](#) ). The reason for this may be that the programs are grass-roots efforts that were not known to or recognized by many researchers in the field of mental health until recently ( [Weiss, 1984](#); [Whittaker and Garbarino, 1983](#) ). An additional reason is that many such programs are difficult to evaluate. For example, it is often difficult, if not impossible, to select individual persons for participation in a program at random—a basic requirement needed to meet scientific standards.

However, the lack of evaluation data is a characteristic not only of family support programs but also of other types of primary prevention programs. [Cowen \(1986\)](#) notes that evaluation studies are needed to separate some of the good and effective prevention programs now being tried from others that are simply “maintained by inertia or falsely placed conviction.” Addressing the problem caused by the lack of evaluation data, [Zigler and Freedman \(1987a\)](#) observe that although family support programs have proliferated, they have done so without any clear indication concerning the direction their course of growth should take. Moreover, those evaluation studies that do exist are varied in quality of design, appropriateness of data collection measures, and validity of conclusions ( [Roth et al., 1999](#) ). On a more practical level, [Cowen \(1986\)](#) notes that the future of prevention programs in general and family support programs in particular depends on evaluations of their effectiveness, because the funding and therefore the continued existence of many programs often depend on the answer to a single question: Are these programs beneficial and cost effective?

The evaluation of family support programs, although important, is not a simple task. In many cases, the programs have no explicitly stated goals, and this renders an evaluation difficult ( [Weiss and Jacobs, 1988](#) ). Additionally, many of these programs are in the formative stage, too early to evaluate results accurately ( [Rossi, 1998](#) ); these and other problems associated with the evaluation of programs are not insurmountable. Indeed, they are being addressed by many researchers in mental health who are finding that by participating in program evaluation, not only do they contribute to the development of more valid evaluation methods ( [Green and MacAllister, 1998](#) ), but also they contribute to a theoretical understanding of children's development and thereby open up new vistas for research and practice. For example, on the basis of a review of existing evaluations of family support programs, researchers have found that those that are successful in addressing the multiple needs of families have four common features: they are comprehensive; they are flexible; they are located in the community; and they are results oriented, with their ultimate goal to strengthen families ( [Carnegie Corporation of New York, 1994](#) ). These findings about family support services are useful because they provide directions for implementation and the replication of the services on a wider scale ( [Elias, 1997](#); [Schorr, 1997](#) ).

## Children and Violence

Another area of particular concern for researchers and policy makers alike is the “epidemic of youth violence” ( [Marans and Schaefer, 1998](#) ). Although exact numbers are difficult to quantify, there is little doubt that large and growing numbers of children are exposed to violence in their homes, schools, and communities as victims, observers, or perpetrators. For example, a study in New Haven, Connecticut, revealed that 41% of sixth-, eighth-, and 10th-grade students in public schools reported having witnessed at least one violent crime in the past year, and almost all the eighth graders knew someone who had been killed through violence ( [Schwab–Stone et al., 1995](#) ). The findings of such studies are troubling not only because of the threat to children's safety, but also because of the short- and long-term impact on their healthy development. It is not unusual for children exposed to violence to experience disruptions in sleeping, eating, and toileting and to display generalized fear and flashbacks. Repeated exposure increases the likelihood of depression, anxiety, posttraumatic stress disorder, low school attainment, and high alcohol use ( [Marans and Schaefer, 1998](#) ).

### Domestic Violence

Many children also experience violence in their home. Although the issue of domestic violence has been on the policy and research agenda for several decades, the impact on children has received less attention. It is estimated that between 3.3 million and 10 million children in the United States are exposed to domestic violence each year ( [Future of Children, 1999](#) ). Although such violence cuts across the social strata, it is more prevalent among families living in poverty and is associated with multiple stressors including substance abuse and other forms of violence. According to research, in 30% to 60% of families experiencing domestic violence, child maltreatment is also present ( [Edelson, 1999](#) ). Possible consequences for these children include behavioral problems and depression, and in adulthood they may develop low self-esteem and may resort to violence and criminal behavior ( [Future of Children, 1999](#) ). Programs do exist in health care, child welfare, mental health, and law enforcement agencies, but rigorous evaluations have not yet measured their effectiveness.

### VIOLENCE IN SCHOOLS

Media attention has focused on violence in schools ( [Aber et al., 1999](#); [Brener et al., 1999](#) ). Although the high-profile incidents of schools shootings are relatively rare, many young people are exposed to violence of varying levels ( [Schwab–Stone et al., 1999](#) ).

Statistics from the National Crime Victimization Survey and the Youth Risk Behavior Survey indicate a decline in rates of violence that may be partly the result of the proliferation of prevention programs, but rates of violent crime among youth remain high ( [Brener et al., 1999](#); [Federal Interagency Forum on Child and Family Statistics, 1999](#) ). The results from one study indicate no decrease in feeling too unsafe to go to school, being threatened or injured with a weapon on school property, or having property stolen or deliberately damaged at school ( [Brener et al., 1999](#) ). In addition to the immediate consequences of exposure to violence, the longer-term

mental health of young people may be affected. In a survey of 1,100 youth in an urban school, there was a strong correlation between exposure to violence and the development of internalizing and externalizing disorders ( [Schwab–Stone et al., 1999](#)). Children in middle school were found to be particularly vulnerable.

However, although the picture looks bleak, more recent violence prevention strategies have yielded promising results. One of the largest and longest-running school-based violence prevention programs in the United States—the Resolving Conflict Creatively Program—is currently implemented in more than 60 New York City schools and in 12 other school systems across the country ( [Aber et al., 1999](#)). The skills taught in the classroom include communicating clearly and listening carefully, expressing feelings and dealing with anger, resolving conflicts, fostering cooperation, appreciating diversity, and countering bias. A 3-year evaluation of the program indicates positive outcomes when 25 lessons, on average, are taught in a year, according to child and teacher reports and objective measures.

Important policy implications arise from research into the prevalence and effects of exposure to violence among children and youth. Studies of resilience indicate that a key protective factor is a relationship with a caring, responsible adult, usually a parent ( [Gunnar, 1996](#)). However, in some instances, parents may be emotionally or practically unavailable to their children, either because they themselves are the victims or perpetrators of violence or because they are numbed by the exposure to violence in their communities. Therefore, the resources of other adults in the community need to be tapped. Research reveals great potential for school-based programs to lead the way in reducing violence among young people and to promote resilience. Other agencies are also well placed to intervene. One example is the Child Development and Community Policing program (CD-CP) in New Haven, Connecticut, which fosters collaboration among schools, mental health services, and the police department and includes training professionals to work within a developmental perspective ( [Marans and Schaefer, 1998](#)). An evaluation of the Child Development and Community Policing program is currently under way, but anecdotal evidence suggests that the program has reduced fear of crime, has improved relationships between the police and community, has increased referrals of children to mental health agencies, has reduced rates of violence, and has improved adjustment among children ( [Marans and Schaefer, 1998](#)).

## PROBLEMS IN THE USE OF RESEARCH IN POLICY

Mental health professionals have made numerous contributions. For example, school-based programs have been developed by psychiatrists such as James Comer in an effort to prevent affective disorders in children and to ensure responsivity to children's mental health needs. We have also developed a school-based early care and family support program that has been implemented in more than 700 schools across the country ( [Finn–Stevenson and Zigler, 1999](#)). Indeed, as a result of their involvement in program development and evaluation, mental health researchers have accumulated a vast amount of knowledge that “totally transforms the nation's capacity to improve outcomes for vulnerable children” ( [Schorr, 1997](#); [Schorr and Schorr, 1988](#)). This knowledge, derived from more than 3 decades of program development and evaluation, includes evidence of the effectiveness of numerous programs that reduce the burdens of risk factors in childhood and thereby reduce the probability of later damage ( [Price et al., 1988](#); [Schorr, 1997](#)).

Mental health professionals further note evidence indicating that it is not necessary to change everything—the structure of opportunity, the neighborhood environment, and other aspects of the child's life—to make a crucial difference for children at risk ( [Schorr, 1997](#)). However, this knowledge is not being used to alter the life path of many of the children who are growing up under stressful conditions, as noted by Hamburg, in testimony before the U.S. Congress ( [Schorr, 1997](#); [U.S. House Select Committee on Children, Youth and Families, 1987](#)).

The failure to use the knowledge from the research stems from several problems. One problem is that the information on effective programs is generally not shared with the public or with policy makers ( [White, 1988](#); [Zervigon–Hakes, 1998](#)). Thus, these programs, many of them at the demonstration stage, fail to be replicated on a larger scale. Even when programs' potential benefits are known, there is skepticism that such programs, once they are replicated, will continue to be effective. Although this is a valid concern, [Schorr \(1997\)](#) notes that successful programs can be built on if we can attract and train enough skilled and motivated persons, if we devise various replication strategies, and if we resist the lure of replication through dilution. This last point is noted, given that in efforts to serve as many children as possible or because of lack of sufficient funds, programs are diluted, thus diminishing their quality and potential benefits. [Zigler and Berman \(1983\)](#) note that the inclusion of more children at the expense of program quality has occurred even in such well-known programs as Project Head Start. They suggest continued monitoring of programs as a means of ensuring their effectiveness.

This point needs to be conveyed to policy makers and others who are in charge of the allocation of funds for program development and replication. However, although increasing numbers of mental health researchers are working in the policy arena, there is still a rather uneasy relationship between them and policy makers ( [Zigler, 1998](#)). [Maccoby et al. \(1983\)](#) note that policy makers often regard researchers as impractical. From their perspective, they may be skeptical of policy recommendations coming from researchers who do not seem to understand the complexities of achieving a consensus among rival constituencies. Researchers, conversely, seem to regard policy makers as disingenuous and too willing to compromise even when research evidence does not justify such action. [Meltsner \(1986\)](#) also observes that part of the tension and mistrust between policy makers and mental health researchers emanates from the assumption that knowledge from research is value free, whereas policies are made in a value-laden context. However, this characterization of research and policy is misleading. Often, scientific research takes on the values of the investigators, as is evident in the questions asked, the methods employed, and the interpretation and presentation of the findings.

Problems such as these impede the use of research in policy settings. The problems are further exacerbated by the situation that researchers are often perceived as unable to provide clear answers to policy questions, or, looked at from another perspective, that policy makers are unable to ask questions in a way that would lead to valid and reliable research ( [Maccoby et al., 1983](#)). In part, this problem stems from the unrealistic expectations among policy makers and the inability of many of them to appreciate that single studies cannot, in and of themselves, provide definitive answers to questions.

However, researchers also contribute to the problem. [Sheldon White \(1988\)](#) notes that often researchers are unfamiliar with the policy process or are unable to “read” political issues. They hold to long, slow standards of proof and refutation that are, in the policy arena, “obstructive and nihilistic.” [Thompson \(1993\)](#) makes a similar point and notes that, often, policy issues do not lend themselves easily to research, and research findings are often limited in their applicability to policy because of sampling and measurement issues. Although it is imperative that researchers uphold their professional standards and credibility as scientists ( [Zigler and Finn–Stevenson, 1987](#)), there are times when findings from the research, even if they are not entirely conclusive, can nonetheless provide a direction for policy. For example, the early research on the effects of child care on children's development was controversial, yielding conflicting findings that served to confuse the public and policy makers ( [Clarke–Stewart, 1989](#)). Although research on the topic continues, researchers were able to convene and come to a consensus that indicated that as long as young children are in a good-quality child care settings, they will not be adversely affected by their experiences in child care. This led to a policy recommendation for efforts to monitor the quality of care children receive and to ensure that all children receive care that is conducive to optimal development ( [National Center for Clinical Infant Programs, 1988](#)). It is apparent that in certain circumstances, such as the increasing number of children in child care, awaiting definitive conclusions from the research is counterproductive, especially when action can be taken at the same time that research on a particular issue is continuing.

A different problem occurs when research findings are misused by academics or the media to support a vested policy interest. An example is the controversial issue of the importance of the early years in child development. The research on brain development is a case in point. Magnetic resonance imaging and other techniques that were once confined to medical diagnoses have been used to examine the development of the brain. The findings on brain research have been illuminating, although in many cases preliminary, and they have shown the developmental importance of the first 3 years of life. Such studies have captured media and policy attention. Some researchers, excited by the window of opportunity for action on behalf of children, made exaggerated and distorted claims not substantiated by the research. These claims were refuted by [Bruer \(1999\)](#), who noted that some researchers, in emphasizing the potential for brain development in the years between birth and 3 years, ignored other critical periods through to adulthood. However, Bruer himself went to the other extreme, by denouncing the importance of the first 3 years of life and ignoring findings from social science that have shown that prevention and intervention in the early years are critical. The debate has been taken up by the national media, with viewpoints expressed through sound bites, undermining the complex scientific findings of decades of research. In the process, there is danger that the polarization of the issue could result in a policy retreat from children's services and continued misunderstanding of the research on brain development ( [Zigler et al., in press](#)).

## Strategies for Change

Understanding what impedes the use of research in policy is important if mental health researchers are to have an impact in the policy arena. [Lindblom \(1986\)](#) identified four general guidelines for researchers to follow to encourage the use of research in influencing policy directions: (a) that researchers be concerned in a nonpartisan way with the values and interests of society in general and children in particular, (b) that they take a practical approach and suggest policies that are feasible and have a chance of attracting widespread political and public support, (c) that they respond to the needs of policy makers and provide them with recommendations for action on the basis of research findings, and (d) that they become cognizant of and responsive to the policy process.

It is also suggested that researchers make serious attempts to disseminate the findings from the research, not only to policy makers but also to the general public. No



society acts until it has a sense of the immediacy of the problem (Zigler and Finn, 1981). The Great Society programs of the 1960s illustrate this point. During that time, social issues were covered in major newspapers and were in the forefront of national attention. There were daily stories on welfare mothers, reports on poverty, and expositions on hunger in the United States. Hence there was sympathy for the poor and support for the War on Poverty (Zigler and Valentine, 1979).

Although for a time thereafter interest in issues pertaining to children and families was appreciably less, there are indications that this is changing. First, developmental psychologists, psychiatrists, and other mental health professionals are becoming aware of the need for public education on the needs of children (McCall et al., 1984). In a departure from their past practices, many mental health professionals are no longer satisfied with simply sharing information with one another. Rather, they disseminate their knowledge not only by presenting their findings directly to policy makers but also by taking steps to ensure that the information is covered in the popular media. Indeed, the dissemination of research in the context of the popular media has come to be accepted as an important aspect of the training received by some professionals in the field of mental health (Zigler, 1998).

Many mental health professionals are also receiving training in the integration of child development research and social policy, to learn not only about the policy process but also about some of the ways to merge their knowledge with that of policy makers in the formation of programs and policies for children. For example, with support from the Bush Foundation in Minnesota, several training centers were established, one of which is the Bush Center in Child Development and Social Policy at Yale University in New Haven, Connecticut. The Bush Center has prepared doctoral students and postdoctoral fellows in a variety of disciplines related to mental health to apply their knowledge in the policy arena. Many graduates of the Bush Center have gone on to work in the policy arena or in other universities, where they have established courses and programs on the integration of child development research and social policy. The success of the training centers is evident not only in the increased number of researchers who apply their work to the policy arena, but also in the numerous issues, such as child care, parental leave, and the need for family support services, that only a few years ago were not discussed but that now command national attention. The success of these efforts is further evident in that increasing numbers of policy makers are now acknowledging the importance of knowledge from the research in the formulation of policies and are actively seeking the collaboration of professionals in the field of mental health. If mental health professionals and policy makers continue to work together in this spirit of collaboration, we will be able to bring about much needed changes that will assist family life.

## Chapter References

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# 119 INTERNATIONAL CHILD AND ADOLESCENT MENTAL HEALTH REVIEW

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## GOAL

This chapter delineates the areas of concern in international child and adolescent mental health and focuses on areas of particular import to child and adolescent psychiatrists and other child mental health clinicians. The goal is to provide a perspective on child and adolescent mental health and to identify areas of particular interest as a reference for further study.

## OVERVIEW

International child and adolescent mental health embraces the diversity of the world view on the place of children in society, the appreciation of diverse behavioral styles and the identification of psychopathology, and the priority setting for the use of scarce resources. Child and adolescent mental health is influenced by the economics of countries and societies within countries, by the internal and external displacement of children and adolescents through war and catastrophe, by the role of the child in the family, and by the place of women in society. New knowledge and greater recognition of the impact on children of exposure to trauma, sexual and physical abuse, inhumane living and working situations, inadequate health care, and drug abuse have heightened interest in approaches to ameliorating the impact on child and adolescent health and mental health of these potentially pathogenic influences. It is a challenge to child and adolescent psychiatrists and allied professionals to be active participants in understanding the nature of the problems faced and in being a part of the solution ( [Sugar et al., 1992](#)).

The overall health and well-being of children are international concerns. More than 179 countries have ratified the 1989 United Nations Convention on the Rights of the Child ([United Nations, 1989](#)). It commits countries to "ensure that all children have the right to develop physically and mentally to their full potential, to express their opinions freely, and to be protected against all forms of abuse and exploitation." It is striking that some democratic societies have chosen to not ratify the treaty out of concern with intrusion on their sovereign rights and others because of traditional views of the child in a dependent position in society. Likewise, in some countries that are party to the treaty, the affirmation of the rights of children has not resulted in benign policies toward the protection of children from harm or the fostering of positive development.

No longer can those interested in international child mental health focus solely on psychopathology as viewed from a Western perspective. Given the diversity of cultural representation in most Western countries, the same caution may apply as a universal maxim. It is simplistic to state, but meaningful to understand; that what may appear pathologic in one country or society may be deemed normative or adaptive in another. This does not imply that it may not be helpful to have a consensus about a frame of reference regarding psychopathologic conditions, but the interested party must keep an open mind in attributing cause to behaviors, interpreting responses to events, or judging parental or familial interactions with children. The complexity of understanding children and adolescents embraces anthropologic, social, psychological, political, and rights dimensions.

In many developing countries, educational institutions represent the most coherent system embracing children and adolescents and provide the primary venue for health-related interventions. As never before, the value placed on education in societies is being emphasized as agrarian pursuits have become commercialized or made nonviable. In developing countries, the impact of technology is differentially affecting parts of society. On the one hand, technologic advance offers an unprecedented opportunity to the educated, but on the other, it accelerates inequality with the less educated. Urbanization combines with the technology revolution further to challenge accustomed ways that may stress individuals and families ( [Rahim and Cederblad, 1984](#)). Children and adolescents, as students or as part of a family, experience new stresses that convey either advantage or disadvantage, depending on access, intelligence, and resources. In response to these changes in society, resilience-building programs in schools, along with primary care health programs in communities, form what is recognized as international child and adolescent mental health programming.

The role and responsibilities of child and adolescent psychiatrists and other child mental health professionals vary in developing countries. The competencies of the child and adolescent psychiatrist must fit the needs of the society in which they exist. For example, epilepsy and mental retardation clearly fall within the expected clinical competencies of child and adolescent psychiatrists in developing countries, but they are not expected competencies of child and adolescent psychiatrists in developed countries. When child psychiatry is a very scarce resource, there may be the opportunity only for a consultative role, limited diagnostic capability, and an inability to be part of or stimulate discussion of national policy. At the same time, child and adolescent psychiatrists brought to developing countries may play a vital role in educating other professionals in medicine, psychology, education, social work, nursing, and the volunteer community.

In understanding the impact of child and adolescent psychiatric disorders, it is not sufficient to understand diagnosis alone. Significant gains have been made in raising the consciousness about the mental health of children and adolescents, as well as adults, by bringing attention to the "burden" of mental illness ( [Desjarlais et al., 1995](#)), when the global burden of disease is measured in disability-adjusted life-years. This approach makes possible a more standardized assessment of the burden of disease as measured by lost opportunity, diminished function, and the cost of treatment and rehabilitation, and it has gained a supportive response from policy makers. From the child mental health perspective, disability-adjusted life-years have limitations in that they do not quantify negative or positive effects of behaviors but only address outcomes. As a result, the importance of behaviors that start during childhood and adolescence but result in disease and death only later in life may be underestimated by this approach.

The importance now attributed to mental disorders and mental health is reflected in the focus of the World Health Organization (WHO) on mental health in its 2001 World Health Report and in a mental health focus for World Health Day in 2001. In addition, the World Bank now considers mental health among its priority areas of concern.

An understanding the culture of the individual is important. For example, [Murthy \(2000\)](#) reports that studies have found that suicide rates among immigrants are more closely aligned to the rates in the country of origin than to the rates in the country of adoption. Generally, suicide rates of immigrant populations are higher than in the country of origin. The methods of suicide are those used traditionally in the culture of origin. [Murthy \(2000\)](#) found that suicide by burning was nearly 10 times more



common in girls and women from the Indian subcontinent than in the overall female population in England and Wales ( [Raleigh and Balarajan, 1992](#)). [Canino et al. \(1991\)](#) also documented the persistence of the importance of culture-bound syndromes.

## EPIDEMIOLOGY

Determining the epidemiology of childhood mental disorders in Western society is a challenge. On the international scene, the ability to determine the precise magnitude of mental disorders is even more complex. Reporting systems are inadequate, the definition or recognition of disorders varies or has variable interpretations, and the cultural component of what constitutes a disorder is only now being more fully appreciated by epidemiologists and researchers. Of significance in developing countries is that any measure of mental disorder takes place against a background of child and adolescent mortality and morbidity that makes the epidemiology of psychiatric disorder not only inaccurate, but often of a lower priority. Thus, in studying the epidemiology of psychiatric disorder in children and adolescents in developing countries, it is important to define not only the prevalence and incidence of the disorders, but also the associated burden of disease as measured in cost to society of lost productivity, cost of care, and lost human potential. No single study or consistent set of independent studies on the epidemiology of child and adolescent disorders since 1980 can be identified as definitive or relevant across societies. Those studies carried out in the 1980s reflect the deficiencies noted earlier and certainly do not reflect the current realities of the countries from which the data were reported ( [Hackett and Hackett, 1999](#); [Odejide et al., 1989](#)).

When one is faced with the realities of developing countries, as noted, there is the danger of becoming a diagnostic nihilist in attempting to understand mental disorders in youth. Responsible investigators have clearly identified disordered mental functioning that meets a set of defined criteria ( [Tadesse et al., 1999a](#)). There is clear evidence that depression, psychosis, and mania can be defined and treated. The problem arises when one considers the context for the presentation of child and adolescent mental disorders. Is a hallucination during a ritual a disturbance in need of treatment? If the hallucination persists, should it be treated? What diagnostic label is appropriate? It is important to bear in mind the study of [Giel and Van Lujik \(1969\)](#), who found, counter to prevailing belief, that mental disorders were diagnosed more frequently than infectious diseases in the health centers in Africa that they studied. Until reporting is adequate and accurate, it cannot be assumed that the current state of mental health in the developing world actually supports the too prevalent minimalist and optimistic view. This sense is supported by the finding from WHO studies of primary care clinicians that showed that many patients seeking care had mental disorders, and their communities were aware of the problem ([Harding, 1980](#)).

### Prevalence

Although it is interesting to consider reports of more esoteric disorders, these are a distraction from the significant burden of disease that needs to be addressed in the mainstream of care. In most studies, the methodologic inadequacies and other constraints do not permit these studies to be of use for program planning or needs assessment. Most countries today have access to appropriate epidemiologic study guidelines, and it is a matter of setting a national priority and allocating resources to ascertain the data. For example, a study in India by [Malhotra \(1995\)](#) used a sophisticated three-stage assessment of the epidemiology of disorders in school children aged 4 to 12 years. In this study, assessments by teachers, parents, and clinicians were compared. The teacher assessment on the Rutter B scale, a generally accepted measurement instrument, had a low concordance rate with the clinical assessment. When children tested positive on both the teacher and parent assessment, there was a diagnostic rate of 92.3% on the clinical assessment. The evidence pointed to a prevalence rate of psychiatric disorder ranging between 7% and 20%. The diagnostic possibilities included enuresis, mental retardation, and epilepsy, among others. Overall, the most conservative estimate of severe psychiatric disorder in India is 10% of the population younger than age 14 years, representing 35 million children ( [Malhotra, 1998](#)).

[Giel et al. \(1981\)](#) demonstrated in four countries (Sudan, Philippines, Colombia, and India) that between 12% and 29% of children aged 5 to 15 years showed mental health problems. The types of disorder identified in these developing countries were reported as being no different from those encountered in industrialized countries. However, recognizable diagnoses were not given in the article. Mothers readily reported the symptoms that made possible the diagnosis of disorders. [Thabet and Vostanis \(1998\)](#) report a pattern of anxiety symptoms and disorders among children living in the Gaza Strip comparable to previous epidemiologic research in Western societies. There are high rates of anxiety disorders and school-related mental health problems. Thabet and Vostanis find the same prevalence rate (21%) of anxiety-related disorders as do [Kashani and Orvaschel \(1990\)](#). [Thabet and Vostanis \(1998\)](#) state that their findings do not support the commonly held belief that, in non-Western societies, anxiety and other mental health symptoms are predominantly expressed through somatizing symptoms. Citing [Nikapota \(1991\)](#), they state that child mental health symptoms do not differ significantly across cultures, and culture-specific mental health disorders are rare. Most recently, [Tadesse et al. \(1999a\)](#) report a prevalence of childhood behavioral disorder of 17.7%. The behavioral disorders are more frequent in boys than in girls. These latter data were gathered with a version of the Reporting Questionnaire for Children developed by WHO ( [Tadesse et al., 1999b](#); [World Health Organization, 1977](#) ). Studies of [Hackett et al. \(1999\)](#) and [Bird et al. \(1989\)](#) find an excess of male patients with externalizing disorders. The findings reflect the Western view of a male predisposition to externalizing disorders. These views of comparability with Western epidemiologic data are at odds with older studies and may reflect new social and economic realities.

What of the disorders that now occupy considerable attention in developed countries such as attention deficit hyperactivity disorder, autism, and anorexia nervosa? There is little doubt that these disorders are seen, but what resources need to be invested in the treatment of these disorders in countries that have little access to the medications or programs that may be indicated? The diagnosis and treatment of these disorders highlight both a weakness and a strength of having an international perspective. The recognition and labeling of disorders come as a result of improved international communication. However, the process of assessment is most complex, taking into account cultural concepts of what is normal or abnormal and how parents perceive the presence or absence of a diagnosable disorder ( [Hackett and Hackett, 1999](#)). In the case of eating disorders, there is clear evidence that the incidence may be affected by Western influences ( [Becker, 1995](#)). Increasingly, the pharmaceutical industry provides local education of providers in countries throughout the world and thus may establish a market for the medication to treat a disorder. This may provide an indirect incentive for the overdiagnosis of disorders such as attention deficit hyperactivity disorder or anxiety. The scrutiny of these practices is quite variable throughout the world.

## SPECIFIC PROBLEMS WITH MENTAL HEALTH IMPLICATIONS

The priority concern of international child and adolescent mental health is often the acute and continuing tragedies that involve youth in armed conflict or the aftermath of conflict. Eighty percent of the victims of war are reported to be children and women ( [Lee, 1991](#)). The result of armed conflict is often displacement. The problems of displacement from homes, families, communities, and countries affect children in a host of ways. [Zivic \(1993\)](#), in a study of Croatian children during war, found significantly higher depressive and phobic symptoms in displaced refugee children than in local children in stable social conditions. [Laor et al. \(1996\)](#), in a developmental study of Israeli children exposed to Scud missile attacks, found higher externalizing and stress symptoms in displaced children as opposed to those able to maintain family and community connections. Children in these circumstances may find themselves without the protection and support of parents at critical junctures in their lives. Children are forced to act in more mature ways far earlier than normal development would dictate or allow. Displaced children are faced with exposure to war and violence that may have seen family members murdered. Less often, but even more horrific, some children have been forced into being the murderers of their family or conscripted to serve as child soldiers. Others find themselves either displaced to other countries or internally displaced and left to fend for themselves. Street children engage in survival tactics that include criminal activity and prostitution. In an effort to find a context for survival, the formation of youth gangs is increasingly evident, especially in societies where there is a lapse in government organization and control. More often than not, the children are the victims rather than the perpetrators.

### Posttraumatic Stress Disorder

It is a challenge that as mental health professionals we know so little about the consequences of exposure to these assaults on healthy development. The literature for adults is consistent in demonstrating persistent negative functional consequences from exposure to trauma. The child and adolescent literature reflects conflicting views on the impact of the traumas noted earlier ( [Mollica et al., 1997](#); [Pynoos et al., 1987](#); [Sack et al., 1999](#); [Weine et al., 1998](#)). The resiliency of children over the long term seems to be a consistent dominant finding, but individual investigators identify specific consequences, with depression, externalizing behaviors, and posttraumatic stress disorder (PTSD) as evident consequences ( [Laor et al., 1996](#)). Diagnostic status does not relate to functional status according to [Sack et al. \(1995\)](#). [Sack et al. \(1999\)](#) not only show persistence of PTSD but also demonstrate a sometimes delayed onset of symptoms. Even in those youth demonstrating delayed PTSD, symptoms of depression diminished over time. So whereas the study by Sack and colleagues notes a persistence of the PTSD diagnosis in youth, over time these children appeared able to function well. [Terr \(1983\)](#) demonstrates persistent effects on children from traumas with lasting functional deficits. Studies related to children from Kuwait ( [Abdul-Khalek, 1997](#)) and Iran ( [Almquist and Brandell-Forsberg, 1997](#)) show the persistence of PTSD. [Nader et al. \(1993\)](#) report moderate to severe PTSD in 70% of Kuwaiti children after the Gulf War. [Thabet and Vostanis \(1998\)](#) find, in the Gaza Strip, that the prevalence among children ages 6 to 11 years of at least mild PTSD is 73%, and 39% present with moderate to severe PTSD symptoms. [Ahmad \(1992\)](#) reports that 25% of displaced Kurdish children evidence PTSD, and [Weine et al. \(1998\)](#) find similar rates in Bosnian adolescents who moved to the United States during the war. [Hussain et al. \(1998\)](#), reporting on the impact of the siege of Sarajevo, note that it is not the exposure to sniper fire, but rather the loss of a family member and deprivation of food, water, and shelter that

have a severe adverse impact on the children. The clinical manifestations of the trauma are avoidance and reexperiencing symptoms.

## Malnutrition

Too many societies are now unable or are only marginally able to feed their populations. Malnutrition is pervasive among preschool children in countries such as Bangladesh, Botswana, Nepal, Zambia, and parts of the Caribbean region ([Galler et al., 1983](#); [Jazairy et al., 1992](#)). The World Bank estimates that the total direct and indirect damage from malnutrition is a least 20% to 25% of the disease burden in children ([World Bank, 1993](#)). The consequences of malnutrition on brain development with delayed cognitive development are known, and more subtle behavioral manifestations of malnutrition are manifest with attentional problems and learning disabilities ([Agarwal et al., 1989](#); [Chavez and Martinez, 1982](#); [Galler et al., 1983](#)). Some studies suggest that the behavior disorders associated with malnutrition are secondary to impaired maternal capacities and not to the malnutrition itself because evidence indicates that malnutrition does not contribute to behavioral disturbance later in life ([Galler and Ramsey, 1989](#); [Miranda et al., 1996](#)). Studies are needed to determine more precisely the consequences for the mental health of children who survive these hardships. Is lowered cognitive functioning the dominant finding? What of survival with other impairments that limit the potential for achievement and create a society unable to be mobilized under improving circumstances?

## Suicide

Suicide in youth is a pervasive world mental health problem. In Western cultures, suicide is overwhelmingly associated with defined mental illness. Elsewhere in the world, it is very difficult to identify the mental illness associated with the suicidal act. Studies of suicide in the West have focused on risk factors associated with cognitive distortions, substance use, and familial factors ([Shaffer, 1988](#)). In trying to assess the high rates of suicide in some developing countries, it appears that the balance in determining suicidal risk may rest with environmental stressors and the perception of "no way out." Expectations may often be more of determinant of suicidal angst than reality (Bertolote J: May 11, 2000, personal communication). The traditional protective effect of religion in certain cultures seems not to operate among the younger generation ([Murthy, 2000](#)), and as noted elsewhere, suicide may be condoned in certain cultures.

It seems more apparent that suicide is viewed by those without demonstrable mental illness as a solution to social and personal dilemmas that bring with them thwarted expectations for a happy or successful life. For example, from this perspective, in India and in other developing countries, the focus of the suicidal individual is not on achieving some exalted goal, but rather on being able to have enough of a dowry to be married, to not be isolated because of rape, or to be successful in passing a school advancement examination. This relative alteration in emphasis is important in the consideration of intervention strategies and in the training of workers to perform triage and to treat suicidal children and adolescents.

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### CASE ILLUSTRATION

In India, four sisters aged 16 to 24 years committed suicide by hanging after an evening during which they bought sweet cakes and samosas and played word games. The context was that they were part of a once prosperous family in which the father died of tuberculosis for lack of medical treatment. Now they periodically were without sufficient food, but with a mother too proud to ask for help. This family was socially isolated because of parental marriage across religious lines, and they had suffered an unexpected financial downturn as a result of a road-widening project that took their once fertile land. Suicide rates in India, although less than the international average of 16 per 100,000, have been steadily rising. Psychiatrists believe this is in part a result of the accelerating pace of social and economic change. Whereas the biggest risk factor in the West is mental illness, studies in India have consistently found that the dominant risk factor is a combination of social and economic issues. Farmers in debt take pesticide, and ostracized women immolate themselves ([Dugge, 2000](#)).

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### FAMILY SUICIDE

In Hong Kong, amid the impressive high-rise buildings and fancy stores, reside families barely able to subsist. In this context of economic hardship, the result in part of massive economic adjustment in the Far East, the phenomenon of family suicide exists. Chan reports that families come together and, in a well-planned manner, seal themselves their small apartments and light a charcoal heater (Chan, May 11, 2000, personal communication). In a relatively brief time, the members of the family are asphyxiated. This has become an acceptable form of suicide in that the bodies remain intact and have an attractive appearance because of the monoxide poisoning. To the extent that it has been possible to determine the psychological state of the family before the suicides, major psychiatric disturbance has not been reported.

## Displacement

The global problem of displacement from family, home, community, and country are of enormous importance to the mental health of populations. Displacement by war resulted in approximately 21.5 million refugees in 1999. An additional 30 million, 80% of whom are children and women, were displaced internally. [Fullilove \(1996\)](#) emphasizes the importance of "place" in the healthy development of individuals. [Sampson et al. \(1997\)](#) specifically address the importance of the community as a mediator and contributor to the impact of violence on children and adolescents. The delineation of the importance of collective efficacy in communities is an important concept when one considers the impact of imposed poverty, housing disruption, and displacement in ethnic conflicts affecting previously closely aligned groups. In developing countries, the notion of "place" and community are of equal or greater importance. The disruption of traditional communities by war, famine, and natural disaster leave children and adolescents in vulnerable situations that affect mental health. Internal displacement by war and famine leads to the breakup of families, months and years of uncertainty, disruption of education, and physical illness. Forced emigration and the loss of parents and relatives in war often mean abandonment or orphaning of children and adolescents. Although these stressors may serve to demonstrate the enormous resiliency of youth, they often lead to depression, suicide, and a range of stress responses.

### "Child Soldiers" and Sexual Slaves

In the turmoil of some developing countries, children are now being forced to become "child soldiers," and other others are drawn into the conflict as sexual slaves. These horrific experiences place an as yet undefined burden on the psychological development of the victim. Understanding these experiences may shed additional light on the extremes to which resiliency may allow future healthy development, but perhaps more likely it will demonstrate the more permanent scarring evidenced in disturbed interpersonal relationships, distorted defenses, heightened aggression, reduced empathy, and self-destructive behavior. The data are not yet available to ascertain whether these young people evidence PTSD in the classic sense or whether, because of the early age of induction into the culture of war, they develop in a different way as a survival response.

## Human Immunodeficiency Virus and Acquired Immunodeficiency Syndrome

In sub-Saharan Africa, Russia and parts of Asia, acquired immunodeficiency syndrome (AIDS) is now a pandemic. Special attention needs to be given to the consequences of AIDS on children and youth. The direct impact on children and adolescents is evident in India, other parts of Asia, and Africa, where sexual exploitation has led to a high incidence of youth infection with the inevitable outcome of death as a result of lack of available treatment. An estimated 1.5 million children less than 15 years old are living with human immunodeficiency virus (HIV) infection or AIDS ([UNICEF, 2000](#)). More than one-fourth of the young population in sub-Saharan Africa is infected. Among the 10 most affected countries, all in sub-Saharan Africa, approximately 6,000,000 children younger than the age of 15 years lost their mother or both parents to AIDS. Those infected but struggling with the illness face the prospect of having to adjust to declining physical and mental functioning and often living isolated lives. Thus, the mental health consequences of AIDS as a chronic and pervasive illness must be considered. There is the obvious concern with the direct effect of AIDS on the youth with manifestations of neuropsychological dysfunction including dementia, depression, and other disorders, which go largely untreated. These children and adolescents living as orphans or in stigmatized environments are vulnerable because of the loss of parent figures, malnutrition, and disenfranchisement from societies that have a stigmatized view of AIDS-affected and HIV-infected persons.

The mental health consequences are similar in the international arena and are well documented in U.S. studies ([Belfer and Munir, 1997](#)). The caution in developing countries is that recognition of the neuropsychological consequences will be overshadowed by the totality of the devastation. This lack of recognition of depression, dementing illness, and other consequences of HIV infection may contribute to the continuing spread of the epidemic. As documented by [Carlson and Earls \(1997\)](#), whether through social policy as evidenced in the Leagane children of Romania or as the consequence of the pandemic of AIDS, the rearing of children in orphanages or in other situations that deprive children of appropriate stimulation and nurturance has potentially long-lasting consequences for societies.

## Substance Abuse



Substance abuse in children and adolescents is a worldwide problem ( [Belfer and Heggenhougen, 1995](#)). In developing countries, the problem is of no less importance than in Western countries and exacts a tremendous toll in terms of morbidity and mortality. Illicit drugs, psychoactive substances not defined as drugs of abuse such as khat, inhalants, and alcohol are used by youth regardless of economic circumstance or religious prohibition (1995). Remarkably, in some Muslim countries, alcohol use and abuse are significant contributors to psychological morbidity. Khat is used extensively in East Africa and the Middle East. In Somalia, Ethiopia, and Kenya, khat is used at all levels of society from about the age of 10 years ( [Alem et al., 1999](#)). Khat may induce a mild euphoria and excitement that can progress to hypomania. In youth, khat use, especially if it is combined with the use of other psychoactive substances, may lead to psychosis.

Solvent and inhalant use is associated with poor economies. In South America, inhalant use is a dominant factor in the presentation of youth affected by psychoactive substances. In Sao Paulo, Brazil, up to 25% of children age 9 to 18 years abuse solvents ( [Carlini-Cottrim and Carlini, 1988](#)). In the Sudan, gasoline is the inhalant of choice, whereas in Mexico, Brazil, and elsewhere in Latin America, paint thinner, plastic cement, shoe dye, and industrial glue are often used. Solvent use is also found among the aboriginal group in Australia and on Native Canadian reservations ( [Cameron and Debelle, 1984](#)). In Mexico, three of every 1,000 people between the ages of 14 and 24 years use inhalants on a regular basis ( [Belasso, 1978](#)). These figures do not include two high-risk groups, the homeless population and those less than 14 years old, whose rates of inhalant abuse are much greater. Several community studies carried out in different parts of Mexico show that starting ages are as young as 5 or 6 years ( [Belasso, 1978](#)). Data suggest that the percentage of young people using inhalants decreases as age increases, and other substances such as alcohol and marijuana are substituted. Inhalant use decreases as educational level increases ( [Cravioto et al., 1992](#)).

### **Mental Retardation and Epilepsy**

Mental retardation and epilepsy are major disorders that often dominate the services of child mental health and pediatric professionals in developing countries. In the 1980s, prevalence rates in developing countries were estimated to be in the range of 8 to 12 per 1,000 for children aged 3 to 10 years ( [Belmont, 1984](#); [Narayanan, 1981](#); [Tao, 1988](#)). Mental retardation and epilepsy are the most common mental disorders in India ( [Lal and Sethi, 1977](#); [Malhotra and Chaturvedi, 1984](#)). The rate of serious mental retardation in some developing countries ranges from 5 to 16.2 per 1,000 population ( [Stein et al., 1986](#)), significantly higher than the rate in the West. Cerebral palsy and postnatal causes of mental retardation are much more common in transitional societies than in developed countries. Untreated epilepsy limits a person's potential to participate in society. Unfortunately, although the cost of medication is relatively low, access to care is often limited. The care of the mentally retarded varies widely in developing countries. In some countries, special effort is made to provide for productive lives with meaningful vocational education, especially in agrarian economies. All too often, the moderately and severely retarded are housed in minimal care institutions where premature death and illness are common.

### **Culture and Assessment**

The concern with accounting for the cultural contribution to the understanding of psychopathology is very great in the West ( [Canino et al., 1991](#)). However, the picture is more complicated in international mental health. There is the danger that attribution to culture of symptoms representing treatable mental illness can deflect energy from the development of effective treatment and prevention efforts. This view has to be balanced with the understanding of less severe psychopathology, in which the observation of [Neki \(1976\)](#) holds true. [Neki \(1976\)](#) states that ethnodynamics determine the psychodynamics. In India, where the cultural ideal of an independent adult is not an autonomous adult, dependency is inculcated from childhood through a prolonged dependency relationship between mother and child. Dependency has a negative, pejorative connotation in Western thought, which is not so in the Indian context. The fostering of dependency is coupled with a high degree of control, low autonomy, and strict discipline, enforced within the broad framework of the family system. When this is identified by clinicians as representing a degree of pathology, decreased emphasis on the expression of thoughts and emotions in children could explain the greater preponderance of neurotic, psychosomatic, and somatization disorders ( [Malhotra et al., 1992](#)). Thus, culture can influence the definition of normalcy or disorder. It proscribes the values and ideals for the behavior of individuals, it determines the threshold of acceptance of pathology, and it provides guidelines for the handling of pathology and its correction ( [Malhotra, 1998](#)).

### **Cultural Differences and Research**

The foregoing illustrates the dilemma of attempting to understand patterns in non-Western, developing countries of child rearing or assessment of pathology through a Western lens. [Nikapota \(1993\)](#) underscores the importance of determining "culture-appropriate" criteria to permit consistency in diagnosis. In doing research, it is important to consider the characteristics of the interviewer as well as the informant. [Munir and Earls \(1992\)](#) articulate a set of ethical parameters for research that must be considered in doing research among children and adolescents of developing countries. To apply a different standard justified by the difficulty of implementing protocols would violate the very support of a rights framework so essential for progress to be made on behalf of children in developing countries.

An area of great interest is the development of assessment tools that incorporate the diversity of cultural parameters. Increasing numbers of instruments have been translated and back translated for use in cross-cultural studies. Instruments exist for the assessment of depression, anxiety, PTSD, quality of life, and other conditions. It remains for there to be a sufficient body of cross-cultural research with modern standards for the conduct of the research that yields information on the reliability and validity of the instruments in their revised versions. Few instruments meet an agreed standard for use across all cultures in their current form.

### **Service Gaps**

Developing countries lack the child mental health personnel to mount large-scale programs of treatment with fully trained staff members. The prospect of training large numbers of child mental health workers remains a continuing goal, whereas the training of child and adolescent psychiatrists to meet the potential need is beyond the realm of possibility. In the interim, what can be done to provide a way to intervene for the promotion of child mental health? Obviously, one focus is on developing prevention programs in general health and education systems. Second, training primary care practitioners from numerous disciplines is needed to provide basic child mental health services. Basic assessment and treatment are possible, with triage of the most severely disturbed.

In some countries, the lack of child mental health personnel has stimulated some remarkable efforts to train persons from diverse backgrounds to be effective in identifying and intervening to ameliorate child mental health problems. In Alexandria, Egypt, child counselors have been trained to develop sophisticated interventions in schools ( [El-Din et al., 1993](#)).

## **PROGRAM ILLUSTRATION**

In Alexandria, the Department of Community Medicine has supported the development of a cadre of school counselors. These counselors come from the ranks of volunteers, social workers, and psychologists. Without prior child mental health training, the workers are provided with course work on common child mental health problems and then are supervised in field placements. The counselors work with parents around children identified by both the school and parents as having some type of behavioral problem. They also serve as contact points for the school, parents, or pediatricians to bring children with more severe behavioral disturbance to the attention of the few fully trained mental health professionals.

Leaders in child mental health programming in developing countries are emphatic when faced with the reality of program implementation that Western models of care by specialists are neither feasible nor desirable. Indigenous methods and models of care need to be developed that are not dependent on specialists. Conversely, the development of these models that use parents, teachers, pediatricians, and others can be informed by the best thinking of child psychiatrists and other specialists. This has led to an emphasis on the training of primary care practitioners. Furthermore, short-term, focused training in specific areas related to diagnosis or intervention can be provided by specialists or through specialized child psychiatric centers that have a broad regional or national area of responsibility.

The development of child mental health training for primary care practitioners is well established. [Murthy \(1998\)](#) describes the use of primary health care providers in developing countries. In India, with 1.5 billion people, there are only 5,000 mental health providers of whom only a fraction are psychiatrists, 35,000 psychiatric beds, and a dearth of emergency services. The primary care provider when adequately trained is a valuable and essential point of contact and treatment for the mentally ill. In India, it has been demonstrated that primary care providers can provide a level of professional care that reduces morbidity and mortality. However, without appropriate training, primary practitioners have been shown to have a poor record of recognizing mental disorders. [Giel et al. \(1981\)](#) report in their study in developing countries that primary care practitioners identified only 10% to 20% of the disorders that the researchers were able to diagnose. WHO has devoted considerable effort to the development and distribution of training manuals to aid primary care practitioners in the recognition of mental disorders ( [Graham and Orley, 1998](#); [Nikapota, 1993](#)).

Moreover, India has developed the *anganwadi* system to provide basic nutrition and educational support in villages. This is both an appropriate preventive

intervention and a way to assess youngsters presenting with disorders (Mathur et al., 1995). The *anganwadi* system focuses on providing essential services to very young children. Like Head Start, the program provides nutrition, basic education, socialization, and a venue for more specialized intervention for children perceived to be at risk or in need of additional services (Jazairy et al., 1992). There must be a concern in the development of these indigenous systems of care that not too much dependence is placed on family structure and support at a time when urbanization and industrialization are eroding the traditional family structure. With the absence of security, and often living at a subsistence level, the new nuclear family faces previously unknown challenges and may be particularly vulnerable over this and the next generation.

Some programs are international in scope. For instance, WHO, as part of its Programme on Mental Health, has fostered the use of life skills education ( [World Health Organization, 1997a](#)). The goal of the Life Skills Program is to foster psychosocial competence. For children and adolescents, the Life Skills Program is taught in schools. The program itself as promulgated by WHO is based on the social learning theory of [Bandura \(1977\)](#). Many similar models are operative throughout the world. A training the trainer component affects the overall resource of a community to provide for the mental health needs of some children and adolescents who are at risk. Among the obvious limitations in the developing world are the absence of universal education and the capacities of the teachers to go beyond essential educational tasks. [Kapur and Cariapa \(1980\)](#) demonstrate that the training of teachers as counselors, in India, was effective.

The prospect for the future of child mental health practice in developing countries is tied to economic growth, health literacy, and reduction of stigma. The creative efforts to develop programs to reach children and adolescents in developing countries need to be supported. Child and adolescent psychiatry will remain a scarce resource to be used in ways that will have a duplicative impact. This means that the training of volunteers, the training of peers, the support of family intervention programming, and the use of community-based early intervention need to be the focus of attention.

## PREVENTION

It appears that prevention of mental disorders is the way to approach the problem of reducing the toll of mental illness in developing countries. However, mental health initiatives in developing countries often depend on outside sponsorship. Such sponsorship, whether in the form of grants from donor countries or foundations, most often requires evidence-based intervention with a definable outcome. The field of prevention in both developed and developing countries offers little support for evidence-based programming for the prevention of mental disorders. Compounding this problem is the investment needed to adapt promising prevention programs for implementation in developing countries. The increased emphasis on mental health and mental disorders in World Bank and WHO planning and program support is some cause for optimism that additional financial and programmatic resources may facilitate the implementation of prevention programming.

Life skills education promoted by the WHO is the backbone of prevention programming in many developing countries ( [World Health Organization, 1997a](#)). Life skills training provides in the context of the school curriculum a program to enhance psychosocial competencies. The training focuses on basic, generic skills such as decision making and problem solving, creative and critical thinking, communication and other interpersonal skills, self-awareness and empathic skills, and coping with stress and coping with emotions. The aims are to promote mental well-being and to enable children to take more responsibility for their lives and feel more effective ([Graham and Orley, 1999](#)). In a collateral program, WHO's Mental Health Program has supported the development and dissemination of a checklist to promote "child-friendly schools" ([World Health Organization, 1997b](#)).

## RESEARCH

[Earls and Eisenberg \(1991\)](#) highlight three areas for enhanced research activity in relation to child and adolescent mental health. First is research to understand how the changes in contemporary society are reflected in the prevalence and incidence of mental disorders. Second is research to enhance the understanding of how different child-rearing methods affect normal and deviant behavioral and emotional development. Third involves research on the design and delivery of mental health services.

As described by [Earls \(1987\)](#), compulsory schooling leads to the need to understand the impact of learning disorders better. The appropriate diagnosis and remediation of these disorders are first-order priorities in countries where technologic advance places a premium on knowledge acquisition and use. As yet, research in this area has not been implemented in developing countries, but the pressure to implement such studies is mounting.

Research into the understanding of the differential impact of child rearing methods should be an area of collaborative inquiry between those interested in mental health and those concerned with the role of women and the family in evolving societies. It is not evident that any one method of child rearing is superior to another. Perhaps developing and developed societies can learn from one another about the optimal methods for child rearing in the presence of the evolution of individual societies. The experience with the effects of urbanization, industrialization, changing roles of women, and increased survivorship of children in developed countries may form the basis for translation into the programs for developing countries. Conversely, the healthy development of youngsters growing up in adversity in developing countries may provide information on how to enhance the understanding and interventions to develop new interventions for children at risk in developed countries.

The issues related to the development of mental health services are addressed elsewhere in this chapter. The diversity of cultures, the acceptance of change, and the continuing availability of resources for program implementation are issues that need to be confronted in international health services research. The development of pilot programs that are not sustainable does not promote infrastructure development, but it does promote a cynicism that erodes the potential for the success of future efforts.

## SYSTEMIC ISSUES

Throughout the world, it is rare to see child mental health incorporated in national health policy. In many countries, developed or developing, no coherent health policy exists that would provide a framework for program development. In countries with a health policy, child mental health rarely rises to a prominent position. Until child mental health becomes integrated into health policy, stable budgetary support for child and adolescent mental health programs will not be realized.

Advocacy for child and adolescent mental health is evident throughout the world, but competition with other interests often forces the issue off the policy agenda. When crises involve children, such as the child soldiers in the Sudan, female genital mutilation, or the case of Elian Gonzalez in the United States, the issue of child mental health for a time gains the spotlight. Unfortunately, the advocacy and concern diminish with time and rarely find a sustaining constituency.

Nongovernmental organizations play an important role in promoting child mental health, in disseminating information, in providing a forum for professional exchange, and in advocating for specific causes. The constituency base of these organizations differ, but they generally have broad representation and provide an opportunity for interested persons to learn more about specific topics or develop ideas in a context of knowledgeable individuals. Many of the nongovernmental organizations have regional affiliated regional organizations that permit ongoing local involvement. The following are some of the more established international nongovernmental organizations: the International Association for Child and Adolescent Psychiatry and Allied Professions, the World Association for Infant Mental Health, and the International Association for Adolescent Psychiatry.

As noted previously, there are many innovative models of integrating mental health in school settings. Whether it is through life skills-related curriculum development or consultative services, programs in the developing world have at hand effective models. The limitation for program implementation is the availability of trained persons for leadership and "training of the trainer." Further, there is the need for these programs to be able to access tertiary diagnostic and treatment services for those with manifest psychiatric disorder.

## LESSONS TO BE LEARNED

It would be very wrong to focus only on the areas where it appears that more could be done to enhance child and adolescent mental health services. Western mental health professionals and program developers can learn from the programmatic necessities and innate capacities of individuals and families in developing countries.

Family participation in the care of the mentally ill or retarded child in developing countries is impressive by any standard. The acceptance by communities of the special needs of affected families is often dramatic. Likewise, the willingness and ability of families to care for children, including the appropriate use of medication, for children with epilepsy and other disorders challenge Western concepts of continuity of care and the role of providers.



Finally, the West has flirted with the enhanced use of primary care providers in the delivery of mental health services, but in developing countries, necessity has led to impressive models for the training of primary care practitioners, as noted earlier. This is true in many countries, notably India and Thailand. Primary care training for specific mental health interventions is also part of a WHO strategy.

Conversely, there is a global trend toward the imposition of managed care on mental health services. This is occurring in countries, such as China and Eastern Europe, that have hardly met their child and adolescent mental health needs. The need is felt to control the cost of mental health services. Perhaps uncritically, economies throughout the world are adopting managed mental health care. From a Western perspective, it is obvious that although all mental health services suffer in a managed-care environment, child and adolescent mental health services are often most vulnerable to reductions and the use of the lowest-common-denominator service. Research into managed care and health services has not had the beneficial impact of stimulating the development of some innovative systems of care, but unfortunately the investment needed to foster these systems of care and the social network needed to provide "wrap-around" services do not exist in developing countries. It remains to be seen whether traditional systems of care can be integrated into a meaningful continuum.

## CONCLUSION

International child and adolescent mental health is no longer an exotic topic for theoretical discussion. With our global village, knowledge of child and adolescent mental health problems throughout the world is an important part of the education of all child and adolescent psychiatrists and allied professionals. The perspective gained from appreciating the stressors of children and adolescents in parts of the world embroiled in conflict and the nature of the responses offers the opportunity to learn more about the resilience of children and adolescents and about what we must do to develop more effective intervention programs.

The dearth of trained child and adolescent psychiatrists and allied professionals in developing countries challenges us to find the most effective means for inculcating knowledge and providing meaningful services. It is unrealistic to assume that any effort will meet the needs of child and adolescent psychiatry as determined by conventional planning assumptions. As services evolve in developed countries, there is probably much that can be learned from the way in which less developed countries have found the means to support families and individual persons to be relatively self-sufficient even when they are affected by mental disorders.

Given the enormity of the challenge to extend child mental health in a meaningful manner globally, the establishment of regional centers of excellence should be considered. These centers would incorporate resource libraries, have access to consultants, support training, and in some instances provide clinical diagnostic functions. Ultimately, the goal is to establish a sufficient cadre of child mental health professionals trained to an acceptable standard with the capacity to relate in a culturally appropriate manner to the mental disorder of children and adolescents and to be able to support the healthy development of children and adolescents.

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# 120 SCHOOL CONSULTATION

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For many compelling reasons, child psychiatrists should engage actively in school consultation and in related school-based activities that support the mental health of children. The most pressing is that mental health services delivered in traditional clinic, hospital, and private office settings do not reach most young people who suffer from psychiatric disturbance. School consultation provides a unique avenue for helping many children who would not otherwise come to psychiatric attention. It also extends knowledge to educators and other school-based professionals to help them work more effectively with psychiatrically disturbed and psychosocially stressed children. In addition, school consultation can serve as an entry point for the child psychiatrist who is curious about the functioning of schools, the social institution that, second only to the family, bears responsibility for promoting socialization, as well as for other key aspects of development. Finally, through such engagement with schools, the child psychiatrist may have the opportunity to foster ways of thinking and teaching that will, in turn, promote the physical and mental health of young people.

Despite the compelling argument of need and the potential for mutually interesting interchange, the relationship between educators and mental health professionals was described, for many years, in terms such as tenuous ([Mattison, 1993b](#)) or ambivalent and uncertain ([Comer, 1992](#)). The paucity of collaborative research between educators and child psychiatrists and the lack of cross-fertilization by way of publication in each others' journals attested to a relationship that was more distant than collaborative ([Mattison, 1993b](#)). This may have been the result of differences in theoretical orientation, because educators have generally been trained to use behavioral paradigms and psychiatrists, psychodynamic ones ([Mattison, 1993b](#)). Moreover, residual ill or ambivalent feelings may result from the period in the 1960s and 1970s when community mental health centers were mandated to provide consultation to public agencies, an arrangement that led to the sense of psychiatric consultants as uninvited "strangers bearing gifts" to other community agencies ([Borus, 1984](#)). More recently, however, newer models of mental health involvement with schools have emerged ([Rappaport, 2001](#)), and public attention has focused on difficult school problems involving emotional and behavioral disturbance, such as violence in schools ([Mattison, 2000](#)). Whatever the reason or combination thereof, child psychiatrists are being called on increasingly to advise on classroom management, appropriate educational programming for psychiatrically disturbed children, and prevention and early intervention efforts in schools.

In what appears to be a rapidly improving climate for working relations between educators and mental health consultants, it is timely for this chapter to (a) review models that apply to school consultation, (b) consider some unique features of schools that should aid child psychiatrists in consultation work in schools, and (c) discuss the tasks of the child psychiatric consultant in some detail with reference to a frequently requested type of consultation or evaluation for special education services.

## HISTORICAL PERSPECTIVE

In the two decades after World War II, awareness of the expanding population base and the numbers of untreated mentally ill stimulated a sense of urgency to provide community-based psychiatric services. In Caplan's words: "An outstanding challenge which today faces mental health specialists is connected with our realization that the number of actual or potential mentally disordered persons in the population exceeds our current capacity for remedial action" ([Caplan, 1970](#)). With President Kennedy's message to the U.S. Congress on mental health and mental retardation and the subsequent passage of Public Law 88-164, the Community Mental Health Centers Act of 1963, community mental health consultation became, for a time, an obligatory basic service ([Caplan, 1970](#)). Changes in the organization of the mental health care delivery system further stimulated the opening up of mental hospitals and the expansion of psychiatrists into a variety of community settings. The Community Mental Health Centers Act spawned the rapid growth of community mental health consultation, as well as ideas about a range of consultation models.

The discovery of the prevalence of mental illness in the United States did not initially advance the practice of school consultation because the few mental health workers who interacted with schools were pressured to work in places of greater need, such as Veterans Administration hospitals ([Alpert, 1976](#)). Over time, it became clear that, to address the problem of mental illness in the community, intervention in schools would be essential. Through the leadership of Caplan, Sarason, Berlin, and others, a literature on school consultation emerged, documenting the theoretical basis for and common clinical issues encountered in the growing field of school consultation.

## MODELS OF CONSULTATION

After conducting a multidisciplinary literature search, [West and Idol \(1987\)](#) reported finding 10 different models of consultation. In most of these, consultation was conceptualized as an indirect method of problem solving that provided remedial services for current problems and increased the consultee's ability to handle such problems in the future ([Gutkin and Curtis, 1982](#); [West and Idol, 1987](#)). Traditionally, the indirect nature of consultation was considered to be critical to success. However, more recent paradigms have proposed forms of more direct involvement ([Rappaport, 2001](#)): helping school systems to plan on-site interventions (e.g., mental health services in schools, as described later), system and organizational level interventions, and involvement in prevention and early intervention programs.

In both traditional and emerging models, the mental health consultant is often able to reach many more children than would be possible through direct service. Estimates of need for mental health services have ranged in the order of 8 million children and youth in schools, of whom less than one-fourth receive appropriate services ([Kestenbaum, 2000](#); [Mattison, 1999](#); [Rappaport, 2001](#)). Hence, the statement of urgency and level of need articulated by Caplan, even in 1970, are as relevant today as then.

Models of consultation include the classic ones—the mental health model, the behavioral model, and the organizational model—as well as a set of emerging, school-specific models. Although the mental health model in various forms has been most commonly applied, it is also clear that the emerging, broader role engages the child mental health practitioner as collaborator, expert consultant, and partner in prevention, early intervention, and service delivery efforts.

### Mental Health Consultation

Caplan defines *consultation* as "the process of interaction between two professional persons—the consultant, who is a specialist, and the consultee, who invokes his

help in regard to a current work problem with which the latter is having some difficulty . . . . The problem involves the management or treatment of one or more clients of the consultee, or the planning or implementation of a program . . . (for) such clients” ( [Caplan, 1963](#)). An essential feature in this conceptualization of consultation is that the consultee (in this case, the teacher) retains professional responsibility for the client and, in this capacity, is free to follow—or not—the recommendations of the consultant. Inherent in this is the nonhierarchical nature of the relationship between the consultee and the consultant. In the usual arrangement, this is facilitated by the consultant’s being of a different profession from the consultee and by the consultant’s “visiting” rather than membership status in the consultee’s institution ( [Caplan, 1970](#)). Caplan also stresses that the consultant’s goals are both to be helpful with the problem that led to the consultation request and also to impart new knowledge and skills so the consultee will become more capable of handling such problems in the future.

Caplan delineates four basic forms of consultation ( [Caplan, 1963](#); [Caplan, 1970](#)): client-centered case consultation, program-centered administrative consultation, consultee-centered case consultation, and consultee-centered administrative consultation. In client-centered consultation, the consultant brings expertise to bear on problems the consultee is having in working with a client (or group of clients); in school consultation, this is usually a student. Although the consultant may deal primarily with the consultee (school staff), the focus nevertheless is on the client (student). In the program-centered administrative form of consultation, the consultant typically meets with a group of consultees (again school staff) to help reform troubled administrative programs or to advise in the development of a new program or policy. The last two forms of mental health consultation, consultee-centered case and consultee-centered administrative, are parallel in content (case versus program) to the first two forms. They differ in that the consultant focuses on the problems that the consultee (school staff) has with either the client (student) or program rather than on the client or program *per se*. In consultation work in schools, an example of the consultee-centered case approach would be the consultation to a teacher about how he or she may be contributing to problems in working with a student ( [Alpert, 1976](#)). Central to this approach is the idea of theme interference ( [Caplan and Caplan, 1993](#); [Caplan et al., 1995](#)). *Theme interference* refers to problems the consultee is struggling with that interfere with the ability to work constructively with a student client. The consultant focuses on reducing theme interference by addressing the problems of the school staff rather than of the student.

[Caplan \(1963\)](#) proposes four types of problems that can be addressed in consultee-centered consultation: (a) lack of understanding, (b) lack of skill, (c) lack of objectivity, and (d) lack of confidence. As [Caplan \(1970\)](#) and [Alpert \(1976\)](#) point out, in real life the clean distinctions of the four-part typology often break down, with shifts in focus occurring in the course of the consultation process. [Fritz \(1993\)](#) further notes that, in the current climate for consultation work, consultee-centered consultation is difficult to fund, rarely requested as such, and problematic for the maintenance of the egalitarian relationship between consultant and school staff.

Applying principles of dynamic psychiatry to a broad range of consultation situations, Caplan’s writings are filled with rich descriptions of case material and of mechanisms (e.g., simple identification, transference, characterologic distortion) by which the teacher’s work with a student becomes problematic or stymied ( [Caplan, 1963, 1970](#)). Consultation skill lies in the accurate recognition of these processes and in the ability to work constructively with school teachers and administrators toward the goal of increased effectiveness, that is, more effective work on the part of the teacher in the service of the child or adolescent student. Caplan is careful to delineate the major distinctions between consultation work and psychotherapy, which are quite fundamental and involve, for example, the nature of the contract and relationship between the consultee and the consultant, as well as the very different goals of these two activities (improving the teacher’s work with his or her students versus personal cure).

Berlin, another pioneer in school mental health consultation, notes the potentially unique opportunity that the mental health consultant has “to communicate and sometimes to demonstrate mental health principles to teachers,” that is, to teach the teachers ( [Berlin, 1962](#)). Applying principles of ego psychology to the consultation process, Berlin focuses on teachers, with the goal of reducing anxieties and tensions that hinder effective classroom work with disturbed students. He notes that, in sensitive areas, it may be particularly useful to find strategies for externalizing the situation, such as through the use of examples from other contexts. Joint exploration of difficulties in work with a particular student helps the teacher to identify critical choice points and to consider new, more adaptive solutions.

[Sarason et al. \(1966\)](#), recognizing the potential of schools for preventive mental health work, applies a social ecologic approach to mental health consultation. Extrapersonal features of the institution, along with intrapsychic phenomena, are taken into account in examining problems between consultees and clients (students) ( [Alpert and Meyers, 1983](#); [Tindal et al., 1990](#)). Like the approaches of Caplan and Berlin, this approach commonly focuses on changing the teacher’s perception of a problem with a student. Particular emphasis, however, is placed on the role of teachers in schools and on difficulties in relationships within the school as an institution ( [Alpert, 1976](#)).

The school mental health consultation will thus vary somewhat depending on the consultant’s theoretical orientation, but in today’s world it generally focuses on the problems that a teacher is having with a student, with a teacher’s concern about the psychological well-being of a student, or with issues of educational programming for a given student’s educational progress, such as the need for special education. Although consultee-centered consultation is rarely requested using the traditional psychodynamic, ego psychological, or social ecologic models, questions about institutional or program difficulties are generally addressed using models for organizational consultation (see later).

## **Behavioral Consultation**

Bergan broadly defines *behavioral consultation* as “the application of behavioral theory and research in consultation services” ( [Bergan and Kratochwill, 1990](#)). Like behavioral therapists, behavioral consultants focus on changing the behavior of teachers through a problem-solving process designed to attain behaviorally defined goals ( [Bergan and Kratochwill, 1990](#)). Different behavioral consultants subscribe to slightly different problem-solving processes, but most processes are fundamentally similar to that described by Bergan, which involves the following four stages: (a) problem identification, (b) problem analysis, (c) plan implementation, and (d) problem evaluation ( [Bergan and Kratochwill, 1990](#); [Kratochwill et al., 1995](#); [Osterweil, 1987](#); [West and Idol, 1987](#)).

During the problem identification stage, the consultant and teacher determine whether there is a problem and, if there is, how to define it. A problem occurs when the student’s observed behavior is not what is desired and expected by the teacher or other school personnel ( [Bergan and Kratochwill, 1990](#)). When the consultant and the consultee have defined a problem by establishing that a discrepancy exists between current and desired behavior, they establish goals for the resolution of the problem ( [Bergan and Kratochwill, 1990](#)). The consultant is first mainly responsible for defining the problem and for formulating it in behavioral terms.

During the stage of problem analysis, the consultant and the school staff generate hypotheses about factors that influence the behavior and design a plan to solve the problem ( [Bergan and Kratochwill, 1990](#)). Again, school personnel play an integral role in problem analysis and plan development. Next, the plan is put into action. During the plan implementation stage, the consultant and school staff collect data to assist in the final stage, outcome evaluation. During outcome evaluation, the consultant and teacher (or school staff, more generally) determine whether the goals have been attained, whether new problems have arisen, and whether plan implementation should continue, be changed, or be terminated ( [Bergan and Kratochwill, 1990](#)).

Behavioral consultation is popular, especially among educators, and it is the model of choice for many school psychologists. There are several distinctive features of the behavioral model: the consultee (teacher) is viewed as a problem solver who contributes to all stages of the problem-solving process; the client (student) is encouraged to develop problem-solving skills; the goals of consultation involve the teacher’s observation and measurement of incidents of behavior; and the model defines the problems of individuals as related to the environment ( [Bergan and Kratochwill, 1990](#)). There is also a greater focus on evaluation in the behavioral model of consultation than in the mental health model; moreover, evaluation of outcome may be easier because the consultation process involves the specification of certain student behaviors that the consultant and teacher endeavor to change. Behavioral consultants have worked to develop standardized measures to evaluate consultation outcomes ( [Bergan, 1995](#)).

This model, emphasizing observed and measurable behaviors, fits well with educational practices and has been applied in consultation to many special education classroom settings. The behavior-modification plans that are often the product of this type of consultation can be applied to a wide range of student problems, from the more prevalent disruptive behavior disorders to some profound disturbances. For example, a nonverbal 7-year old girl, who was unable to separate from nearly constant physical contact with her mother, was assessed by a behavioral specialist who devised a behavioral program for the child–mother unit. The initial goals involved proceeding through a set of steps toward separation. The specialist’s analysis and explanation of reinforcers and related mechanisms were integral to recruiting school understanding and support for the program, which was administered with slow but clear progress by a special education teacher.

## **Organizational Consultation**

*Organizational consultation* focuses on schools as systems and seeks to facilitate improvement in their functioning through the application of behavioral science concepts and the involvement of system members (e.g., administrators and teachers) in the process of organizational change. According to this model, mental health and learning difficulties are in part symptomatic of a dysfunctional educational system ( [Christ, 1991](#)). [Schmuck \(1995\)](#) notes that systemic problems such as communication breakdowns and ambiguity about responsibilities are common in many schools. These problems can cause a great deal of anxiety and frustration



among school staff.

Achieving optimal organizational functioning by introducing systemwide change is seen as essential to improving the quality of life and facilitating the productivity of individual members. Overall, the goal of organizational development is to build "self-renewing schools, schools that are able to adapt to current changes within the student body, community, and world while continuing to maintain an effective educational program" (Schmuck, 1976). Self-renewing schools are viewed as adaptive and changing and thus are not bound to particular structures and procedures.

An important feature of the organizational consultation process is the involvement of system members (e.g., teachers, administrators) in the assessment, diagnosis, and transformation of the organization (Schmuck, 1976). Consultants work with and advise teachers and administrators to help them change their interpersonal and group methods of interaction. Thus, this model of consultation focuses on the organizational context, including the mode of communication and the quality of relationships within the school. School "culture" is viewed as a powerful influence that must be understood and worked with to ensure the success of educational innovation and reform efforts (Schmuck, 1976). For example, in the late 1960s the consultation and collaboration process described in Chapter 106, known as the Comer School Development Program, examined governance and management in two inner-city, low-achieving schools. Application of ecologic and child development perspectives led to school restructuring and change in the school culture and climate, with positive results in the areas of student self-esteem, motivation, and achievement.

### Emerging Models and the Pressing Need for Service Delivery

Increasingly, there has been a move toward the provision of mental health services in schools. Concurrently, emerging models of school consultation extend beyond the traditional limits of the consultant's role and seek instead a broader involvement, including team membership, as a focus of consultation, or perhaps more correctly, of collaboration (Rappaport, 2001). Thus, it is important for the consultant to be knowledgeable about methods of psychiatric intervention that can be put into place in schools, as well as of prevention and early intervention programs that have been developed for school settings.

School-based mental health services may be offered in school-based clinics that offer a spectrum of health care services to students. It is also possible to provide mental health services in schools following a model involving collaboration between a treatment center and a school.

An example of this type of program is one created as a direct outgrowth of work carried out by a university-affiliated children's outpatient psychiatric clinic in an urban community (Armbruster and Lichtman, 1999; Armbruster et al., 1997; Armbruster et al., 1999). Involvement with the community and with youth-related programs had impressed members of the outpatient clinic with the number of unserved children. A study of attrition in the clinic had found that the inner-city family is at risk for dropping out during the first few weeks of contact with the clinic (Armbruster and Fallon, 1994; Armbruster and Schwab-Stone, 1994). In response to these observations and with the support of the public school system, the clinic developed a school-based mental health program with the goals of providing effective access and outreach to underserved, disadvantaged children and of collaborating more closely with the schools.

Initially, mental health services were established in four inner-city schools, increasing to 37 or almost 80% of the schools over the next 5 years. In addition to responding to clinical need, this project was built on the legacy of school collaboration instituted by a school reform program, the School Development Program, developed by James Comer (1985, 1988) (Chapter 106) and implemented for many years in the local public schools. The Comer project addressed issues of school climate; the clinic provided direct services. The World Health Organization, Division of Mental Health (1994) noted that effective school intervention can implement an environment-centered model (e.g., the Comer project), a child-centered model (e.g., the clinic's service), or, optimally, both. In both cases, the programs worked in concert to provide supplementary services to the school.

Children and families seen in the schools are considered clinic cases. They are ensured the same evaluation and treatment they would receive if they entered the central child guidance clinic. All children seen in the schools are assigned to the central child guidance clinic's weekly interdisciplinary clinical treatment team and hence are subject to the identical review and quality assurance program as cases seen in the central child guidance clinic. In addition to the central clinic's weekly interdisciplinary treatment team, a school-clinic team meets bimonthly. This school-clinic team promotes clinic integration of school-based services, both within and outside the central clinic, because it comprises school-based clinicians in the schools, consultants from the university's clinical departments, and representatives from schools and community agencies. A child psychiatrist provides medication consultation at the school sites and participates in the central interdisciplinary school-clinic team.

Another function of the school-based clinicians is to refer children for medical care, specialized or acute care, and also for recreational activities and tutoring. Hence, the mental health providers may become the informal gatekeepers and conduits to a range of services (Dowden et al., 1997). Furthermore, with the availability of mental health services based in schools, referrals can be made directly and expeditiously after a traditional psychiatric consultation has been conducted. The central clinic also has an immediate access service for visits at the clinic within 24 hours, if the family chooses not to receive services in school during a crisis. A visit to the immediate-access clinic often serves in place of the less efficient and much less appropriate hospital emergency room visit.

Certain elements of this and other innovative models for school-based mental health service delivery have been identified (Rappaport, 2001). These include the following: (a) increased access to services for disadvantaged and underserved youth; (b) systemwide collaboration; (c) the potential for effectively triaging acute psychiatric incidents, thus preventing the need for acute psychiatric intervention; (d) service as gatekeeper for, and conduit to, more acute or specialized care, when needed; (e) systematic program evaluation; (f) training in working with a range of systems; (g) increase in providers' cultural competence and understanding of diversity; and (h) outreach and community-based care. Thus, school-based mental health service is a highly promising strategy for treatment delivery, but research data for therapeutic and cost effectiveness are not yet available (Armbruster and Lichtman, 1999). Nevertheless, evaluation efforts to date have shown that, in this example, school-based services are reaching the targeted disadvantaged population (Armbruster and Lichtman, 1999).

## SCHOOL CONSULTANT AS ADVISOR ON SCHOOL POLICY AND INTERVENTION PROGRAMMING

In an article reviewing research on interface issues between child psychiatry and schools, Mattison (2000) notes that, increasingly, mental health professionals are asked to offer their opinions and advice on a wide array of new programs and policies. In an attempt to aid such consultations, (Mattison, 2000) highlights the psychiatric and consultation implications of four such school-based problems: absenteeism, disciplinary issues, retention, and school dropout.

### Absenteeism

Epidemiologic findings on *absenteeism* point to mental health precursors. It has been shown that nearly half of chronic absentees have a psychiatric disorder, with most expressing either disruptive behavior disorders (greater truancy) or high levels of anxiety (school phobia) (Berg et al., 1993). Despite these substantial associations between absenteeism and mental disorder, very little research has focused on prevention and treatment. From the research available, findings have shown that cognitive-behavioral treatment and educational support therapy can lessen anxiety-based school refusal (King et al., 1998; Last et al., 1998). Psychopharmacology research has failed to draw conclusive findings, but it has been suggested that imipramine may lessen depressive symptoms and may improve school attendance (Bernstein et al., 2000). Although the prevalence of conduct disorder is higher in chronic absentees than is anxiety, research has yet to identify an effective treatment for delinquent truancy.

With respect to excessive absenteeism, Mattison (2000) suggests that the most effective advice to schools is early detection. Determining which students show high levels of absenteeism and then proactively addressing the needs of these children should have the greatest impact. Unfortunately, proven programs to combat truancy are unavailable and often require the coordinated efforts of the broader community.

### Disciplinary Issues

Although *problems with authority* may be a developmentally appropriate behavior that discipline can address, studies have shown that most adolescents who are suspended from school have academic or social deficits (Morgan-D'Atrio et al., 1996) that are unlikely to be improved by suspension. Proactive identification of students at risk (using indicators such as consistently poor grades, high rates of absenteeism, and ongoing disciplinary problems) may help schools to prevent suspensions and expulsions. Through adequate academic and psychiatric evaluation, schools should be able to determine which students should be referred for disciplinary action and which will more likely benefit from therapeutic intervention or extra academic support (Mattison, 2000).

## Retention (Nonpromotion)

Although methodologic issues (e.g., lack of appropriate controls, *post hoc* analysis, and lack of baseline information) make it difficult to assess the impact of *retention*, most researchers have concluded that there is no long-term academic benefit when retained students are compared with students of similar dysfunction who have been promoted (Mattison, 2000). Despite these contrary-to-fact findings, most teachers regard retention positively, and, indeed, national epidemiologic studies show that cumulative totals for retention range between 14% and 19% (Byrd et al., 1997; Meisels and Liaw, 1993), with much higher rates in many urban centers (Alexander et al., 1994; Silver et al., 2001). Very few studies have assessed intelligence quotient, mental retardation, and learning or language disorders among retained students, but it has been shown that students with a newly diagnosed learning disorder frequently have been held back in school (McLeskey and Grizzle, 1992). Thus, without adequate programs and interventions, holding a student back may only delay his or her difficulties in school. Mattison (2000) notes that low intelligence quotient or mild retardation, learning disorders, psychiatric disorders, family dysfunction or stress, and developmental variations are among the factors that characterize the diverse group of retained students. Hence, it is not surprising that the practice of simply “recycling” the child through the same academic curriculum for a second time does not appear to help many children (Mattison, 2000).

## Dropping Out

Although many of the precursors to *dropping out* have been established (low socioeconomic status, minority status, living with a single parent, behavioral problems, pregnancy, chronic absenteeism, weak academic achievement), specific psychological precursors have been assessed only rarely. Relatively little is known about whether the early presence of a learning disorder, psychiatric diagnosis, or concomitant family stressor will germinate into a greater likelihood of dropping out of school. One study of graduating eighth graders who eventually dropped out of high school showed that having a preexisting psychiatric disorder significantly increased the odds of dropping out (Kessler et al., 1995). Barrington and Hendricks (1989) show that poor academic achievement as early as third grade can predict eventual school dropout. Clearly, early identification of students at risk can play a part in lowering dropout rates. Mattison (2000) suggests that such interventions not only should begin early, but also should continue throughout schooling. These efforts should attempt to institutionalize success by creating “curricula relevant to the varying, unique needs of such students.” Although many of the risk factors for dropping out cannot be addressed in the school setting (low socioeconomic status, weak family involvement), academic and psychological risk factors should be identified early and addressed proactively.

## EFFECTIVENESS OF SCHOOL CONSULTATION

Assessing the effectiveness of school consultation is difficult because the practice of consultation tends to be naturalistic, it has varying goals, and consultations are rarely performed in a standardized manner (Brent and Howell, 1983). As a result, consultants often assess effectiveness anecdotally, and empirical attempts frequently suffer methodologic problems (Erchul and Martens, 1997). Nevertheless, some research reviews and results of two metaanalyses have shown school consultation to be an effective means of intervention (Medway, 1979; Sibley, 1986; see also Medway and Updyke, 1985, a metaanalysis of psychological outcome studies that used 54 controlled studies predominantly, but not entirely, limited to school consultation.) One comprehensive review of 46 school outcome studies of all types of consultation models concluded that 67% had reported positive results, in 28% results were neutral, and in 5% the outcomes of all types of school consultation were negative (Sheridan et al., 1996). The two metaanalytic studies compared effectiveness across *different models* of consultation and concurred in the finding that there was *no* evidence for the superiority of any particular model (Medway and Updyke, 1985; Sibley, 1986; West and Idol, 1987).

Studies investigating relative preference for different models of school consultation have found that teachers prefer behavioral consultation to mental health consultation (Jason and Ferone, 1978; Medway and Forman, 1980), although school psychologists view mental health consultation as more effective (Medway and Forman, 1980). Teachers may prefer behavioral consultation because it provides them with a specific and directed plan of action that seems more readily applicable to the classroom setting. West and Idol note that preference for a particular model may depend on the *stage* of the consultation process, a finding lending support “to the concept of consultation as a complex, multi-dimensional process” (West and Idol, 1987), which warrants continued and more sophisticated efforts at evaluation.

## COLLABORATION IN SCHOOL CONSULTATION

A central guiding principal in all models of school consultation is the importance of a collaborative relationship between the consultant and the teacher (Truesdell and Lopez, 1995). However, the meaning of collaboration has never been well operationalized in the literature (Erchul, 1999). After a series of studies in which observational analyses of consultant–teacher dyads led to a questioning of the efficacy of collaboration in school consultation (Erchul, 1987; Erchul and Chewing, 1990; Witt et al., 1991), researchers were forced to reevaluate their long-held assumptions about the collaborative nature of school consultation. Subsequent research has indicated that collaboration in school consultation is a multidimensional construct consisting of at least two continua: one ranging from “coercive” to “collaborative,” the other ranging from “nondirective” to “directive” (Gutkin, 1999). Collaborative–directive consultation is characterized by interactions in which the consultant uses his or her psychiatric expertise to take the lead in prescribing a course of action to the teacher, while at the same time being receptive to the teacher’s input. Such a relationship promotes teamwork between consultant and consultee by highlighting the contribution of each other’s expertise in different fields. Gutkin (1999) argues that the empirical research on school consultation underscores the strength of such a directive–collaborative approach.

Caplan et al. (1995) state that the mental health consultation relationship should be coordinate and nonhierarchical, such that the consultee is free to accept, or not, the recommendations of the consultant. Within this model, however, these researchers maintain that the consultant’s full and expert understanding of the presenting situation should be used and applied. For example, a teacher may displace some of his or her problems onto a particular situation or may harbor unconscious feelings about a student. In such a situation, the consultant may use the student’s problems to address the teacher’s issues in an indirect and nonconfrontational manner (Caplan et al., 1995).

In sum, the relationship so important in school consultation may vary in terms of its directiveness and degree of responsibility and authority. Caplan et al. contrast the characteristics of the traditional mental health consultation model and those of the mental health collaboration model (Caplan et al., 1995). They note that the collaborative model is the most appropriate mode of working together when the mental health specialist is “in-house,” that is, a staff member who as a treating expert also bears responsibility for the client, whereas the mental health consultation model has distinct advantages when the specialist is truly an outside consultant. Regardless, it is imperative that there be understanding and respect between consultant and consultee for each other’s expertise and that the assumptions of the model be clear, so both parties can work together as an effective team.

## PRACTICAL ASPECTS OF SCHOOL CONSULTATION

From the foregoing review, it is apparent that the novice consultant, preparing to embark on school consultation work, has at hand a diverse set of theoretical approaches on which to draw and relatively little evidence from the research literature to constrain his or her work. In many ways, it is up to the consultant to fashion a strategy for this undertaking and to contribute to the evolving process of the consultation relationship.

### Schools as Social Institutions

In engaging with a school or school system to provide consultative services, the consultant is entering a *social system* that has well-specified and unique functions. These functions have developed historically to address society’s needs for socializing and educating its young. Comer says: “The school, like the family, is charged with preparing the young to become successful adult workers, members of families, and citizens” (Comer, 1992). This is a large mission, and accordingly children spend a considerable amount of time in schools each year through the span of the childhood and adolescent years.

Not surprisingly, to accommodate the goals that have been set for them, schools have developed into complex social institutions. They bring together people of different disciplines, levels of preparation, age, and experience (Comer, 1992) to function together to address the developmental needs of children. Viewed this way, an important aspect of the consultant’s preparation is to become knowledgeable about and comfortable moving in this system. For example, schools have traditionally been hierarchical and authoritarian in structure. Although innovative models of school reform are challenging the necessity and efficacy of that type of structure (Comer, 1993), it is nevertheless not uncommon for the consultant to encounter a fixed structure with explicit lines of authority and relatively low tolerance for transgression. It may be useful for the consultant to consider why schools may have developed in this manner, that is, to institute externally a degree of structure that may be necessary to learn at a time when the child’s internal capacities for self-structure and regulation are still developing. At a practical level, this makes it easier to understand what may superficially appear to be unnecessary constraints on times available for meetings with staff or students or the necessity of communicating a seemingly straightforward request through a specific chain of authority. In addition, an understanding of the structure and rules of school life can facilitate the



consultant's ability to work effectively without unproductive conflict and to appreciate the culture of school life as perceived through the eyes of the child.

### School Climate

Although it is useful for the consultant to become familiar with the structural and organizational aspects of school life, it is also important to evaluate the process aspects of its functioning. *School process* refers to the nature and style of relationships and flow of information within the school ( [Purkey and Smith, 1983](#)). The resulting *climate* of the school involves numerous components that have been variously conceptualized and include, for example, dimensions such as orientation toward learning, level of conflict, promotion of independence, and student involvement in school decision making. ( [Kasen et al., 1990](#)). Research suggests that school climate influences both the academic achievement of students ( [Purkey and Smith, 1983](#); [Rutter et al., 1979](#)) and their emotional development ( [Kasen et al., 1990](#)). However, although there is general agreement that the climate of a school has an important impact on its students, it is less clear which specific factors are most influential. In a review of the literature, [Purkey and Smith \(1983\)](#) argue that many of the studies of effective schools focus on very simple factors that have little effect on the development of students (e.g., class size). Important factors deeply rooted in the school's "culture" are slow to change even when sweeping changes in policy are made. Such a process is well-described in [Chapter 106](#) by Comer et al., who detail the mechanisms through which staff and parents engage with the ultimate goal of changing the culture and climate of the school to promote development, teaching, and learning.

From the perspective of the school consultant, evaluating school climate provides essential information that will enhance the consultant's understanding of the experiences of both students and teachers (consultees) who study and work in the school. Over time and with the development of trust, the consultant may have the opportunity to influence decisions and attitudes that will sustain a positive and supportive context for student learning and development.

### School Staff

The *school system staff* that the school consultant typically encounters includes teachers, guidance counselors, school psychologists, school social workers, and administrators (principals and assistant principals). Teachers spend the most time with and know the students best and as a result are an extremely valuable resource for the consultant ( [Mattison, 1993b](#)). Teachers' roles differ depending on the age of the student. In elementary school, students spend most of their day with one teacher, whereas in middle and high school, students usually spend an hour or less with each of their teachers per day.

Guidance counselors, usually former teachers who have degrees in guidance counseling, are often the first school staff members contacted by either student or teacher when a student has a problem ( [Mattison, 1993b](#)). Guidance counselors frequently have access to mental health resources, but their roles and training vary. Although they are familiar with psychological principles, the impact of psychosocial stress, and fairly normative adjustment issues, they may not actually have much experience with psychiatric diagnostic thinking and nomenclature.

School psychologists have degrees in psychology and have probably been trained in the diagnosis and treatment of behavioral problems ( [Mattison, 1993b](#)). School psychologists often perform psychoeducational testing and may be involved in behavioral management or counseling. In working with children with learning disabilities and complex profiles of cognitive deficiencies and strengths, the consultant will often need to rely on the school psychologist's expertise in describing the impact of such conditions on the child's school functioning.

School social workers work with the families of students but may also share with the school psychologist the function of providing psychological interventions, usually in the form of group or individual treatments. The school social worker may be the key link to the family. The likelihood of achieving success with a classroom intervention may be considerably heightened by engaging the social worker to gain the parents' understanding and support for it. School social workers vary widely in their training and experience with psychiatric diagnostic and treatment principles.

Finally, administrators are often alerted to problems with a student, and they may serve as a liaison between the school and the family. A principal's opinion concerning a student or his/her capacity for involvement in the consultation process can determine the course of action taken by a school ( [Mattison, 1993b](#)).

### Students

In beginning consultation work at a school, it is useful for the consultant to spend some time observing the *students* in the school. This can be done by walking the halls, observing classes across the school's grade range, watching children play on the school grounds during recess, visiting the cafeteria, and attending art, music, or other enrichment activities. In particular, the consultant should get to know the special education classes, both the teachers and the students. This background work is essential for developing a sense of the normative experience of growing up and going to school for the population from which consultation referrals will be generated. It provides an opportunity to begin assessing the school climate, as well as educating the consultant about the range of intervention options that exist within that school setting.

The consultant brings to the consultation process his or her expertise in the diagnostic assessment of psychopathology in children and adolescents and experience in identifying and working with dynamic issues. Although the range and specific types of problems that lead to consultation requests are not reviewed here, it is useful to have a framework for thinking about clinical presentations in the school context.

Briefly, a framework that has been found useful focuses on the developmental tasks of childhood, how schools support a normative progression along developmental lines, and why students who cannot meet the challenges posed at certain stages come to attention for special psychiatric help or educational services. Although a full discussion of this model is beyond the scope of the current chapter, a few examples illustrate the general approach.

In the United States, most children are in school by at least age 6 years. Although today kindergarten is a fairly universal experience, and increasingly now children are attending day care and preschool, entry into first grade has generally been considered the beginning of formal schooling, with a more academic curriculum and, in many places, the start of full-day programming. The child entering school faces a fairly extensive set of demands on his or her capacities across developmental lines. These include, for example, the requirement to separate from primary caregivers and to maintain a sense of sustenance and comfort that is sufficient to get through the day relatively emotionally intact. The child must tolerate and comply with what is probably a greater level of structure than has been previously imposed on his or her day. Activities requiring sustained attention are required. Whereas the average 6-year-old is ready cognitively, emotionally, and socially for these challenges, it is also apparent that some children are not so neatly on schedule. Some may be compromised in specific areas, for example, by difficulties with attention or activity regulation and impulsivity. The challenge of separation may overwhelm others with anxiety, or the stress of coping with peer interactions may send the socially immature child into behavioral outbursts. Some, albeit fewer, children, such as those with pervasive developmental disorder, will be unable to function across all or nearly all domains. It is common then for the consultant to become involved with children characterized by a lack of developmental readiness for the challenges at hand. For some children, this is constitutionally based; for others, it may be rooted in stress from home and family issues. Although the pervasively affected child often requires a special classroom and supportive services, those for whom the "fit" problem is more limited may improve with therapy, medication, or merely clarification and reassurance for the teacher and some adjustment of classroom practices. Often, sharing a developmental formulation with the teacher or other school staff is immensely useful and leads to constructive and creative ideas about the classroom management of the student's problem.

Generally, school transitions are points of particular vulnerability because the new setting usually poses a new set of developmental challenges. Again, these challenges are ones that the child who is developing in a roughly normal manner will be able to negotiate with reasonable success. Those who are not ready may come to the consultant's attention over the course of the year after the transition. This can be seen in the transition to middle school, which occurs for most children at age 10 or 11 years. The transition to middle school brings the expectation that the student will function with more autonomy. Children change teachers and classrooms, more homework is required, and expectations are heightened for the student to take a serious and independent interest in learning and schoolwork. Not surprisingly, at this stage, the consultant hears of children who are suffering the cumulative effects of learning failure because they have been referred too late or because successful interventions were not found in their elementary school years. For other children, the family and home have not provided the degree of structure and support needed to develop the internal motivation to apply themselves in the more challenging and less personal middle school setting. For such youth, the lure of street culture may become irresistible for its excitement and promise of easy access to material rewards. The sooner such transition casualties are caught, the more likely they can be helped back onto the track. In addition to the obvious clinical role that the consultant can play in helping school staff work with and refer such youth, the consultant can, over time, educate consultees to adopt a developmental mode for conceptualizing student problems and for initiating timely consultation requests and referrals for treatment.

## SPECIAL EDUCATION CONSULTATION

An important and common type of school consultation for the child psychiatrist is the consultation to determine eligibility for *special education*. The Education for All Handicapped Children Act, Public Law 94-142, was enacted in 1975 and established the right of children with any number of handicaps to an appropriate education and to related services needed for the child to benefit from that education. ( [Chapter 121](#) has discussion of legal background and issues relating to special education.) When a student's emotional or behavioral difficulties appear to be seriously interfering with his or her educational progress, a referral is made for an evaluation for special education. The labels applied in these situations vary; [Mattison \(1993a\)](#) uses perhaps the most common terminology, seriously emotionally disturbed.

On referral for special education, the student enters the special education administrative structure, which prescribes a set of procedures for evaluating eligibility and for determining the services to be provided to the student. In brief, a multidisciplinary team is convened to conduct the appropriate educational, psychological, medical, social work, and other evaluations. When the evaluations are complete, the team meets together with the parent or parents to review the results. The team determines whether the child meets the criteria for handicap on the basis of serious emotional disturbance (and also for other categories of handicap if relevant), and, if so, an appropriate individual educational plan is developed. This plan includes goals to be met, the time expected to meet them, the educational setting that is appropriate, the amount of time to be spent in a separate classroom and in regular classrooms, the related services that are recommended, and other considerations that the team may wish to note formally. In accordance with Public Law 94-142, students who are seriously emotionally disturbed must be placed in the "least restrictive," most mainstreamed setting possible.

[Mattison \(1993a\)](#) notes the need for greater standardization in school consultation and, in particular, cites the need for a formalized approach to the consultation for determining special education eligibility. He emphasizes that these evaluations usually involve children with complicated, serious symptoms who also often have families suffering from major concurrent stressors. The following is an outline of a consultation procedure used for these evaluations. It is very similar to the excellent model described by [Mattison \(1993a\)](#). The reader is referred to this reference for a detailed description of the model and for very useful examples of forms, interview outlines, and a sample final consultation report.

## **Special Education Case Evaluation**

### *Review of Referral Packet*

In the usual case, the consultation is initiated with a referral summary from the school, a copy of the parent's signed consent for psychiatric evaluation (obtained by the school), and results of previous evaluations. Of particular use are the recent and past reports from the school psychological evaluation. The consultant should develop some ability to understand test scores, particularly for basic tests of intelligence and achievement. Of particular importance is the presence of a learning disability, which should be described in the psychologist's report. A social work assessment may be present and may in some instances include an extensive developmental history. Various teacher reports and documentation of special education programming may also be provided. What is missing at times, however, despite the presence of a wealth of information, is documentation of the chronology of events leading to the current clinical state and referral for consultation.

### *Meeting with the Multidisciplinary Evaluation Team*

This meeting allows team members to review their knowledge of the case with the consultant before his or her interviews with the student and parents. For the consultant, it provides an opportunity to piece together the history if, as noted earlier, the chronology was not apparent from the materials in the referral packet. In this meeting, the consultant can also learn about the concerns or worries that are too "soft" or unsubstantiated for staff to write about in their formal evaluation reports. The meeting also provides an important opportunity for the consultant to gauge staff feeling about the student and to assess whether certain programming outcomes are desired by various members of the team.

### *Interview with the Parent or Parents*

The goals of this interview generally resemble those of parent interviews in other types of evaluation. The consultant seeks to gather history about the difficulties that have led to the consultation and to obtain history about other emotional and behavioral problems and treatments (past and present). Also to be covered are a review of psychiatric symptoms, medical conditions, developmental history, family history (including psychiatric conditions and learning problems of extended family members), assessment of the student's current social adjustment, and parental assessment of the student's strengths and positive aspects of coping. Additionally, the consultant should evaluate the parent's understanding of the special education evaluation process and hopes or expectations about the outcome. The level of trust that the parent feels toward school staff may be critical to the ultimate success of an intervention or referral and should be assessed at this time.

### *Classroom Observation*

When one is evaluating an elementary school-age child, it is often useful to schedule a classroom observation, and it is usually best to do this before the individual interview with the child. Such an observation can yield considerable information about the student's capacity to function in the current classroom setting, about his or her peer relationships, and about the teacher's current strategies for dealing with the student's difficulties. It is usually not helpful to observe an older student in his or her classroom, and it can be unduly embarrassing for the student to be observed.

### *Interview with the Student*

This student interview is similar to the general psychiatric interview in covering an explanation of the nature of the interview and the "kind of doctor" the consultant is, a discussion of current and past difficulties, a review of relevant areas of symptoms, inquiry about physical health and health concerns, and a discussion of home, friends, favorite activities, and feelings about school. A mental status examination should be conducted, and [Mattison \(1993a\)](#) notes the value of a systematic minineuroeducational examination covering coordination and basic educational skills (e.g., reading, mathematics, spelling). It is also useful to assess the student's hopes and expectations about the outcome of the current evaluation. An important feature of this type of consultation is that, beyond the conclusion of the evaluation, there is no expectation of a continued relationship with the consultant. As in any psychiatric evaluation, it is important to ask about anything that could shed light on the nature of the student's problems or be of potential use in disposition planning; however, uncovering techniques and extensive probing in affectively sensitive areas should be curtailed if their yield is not of direct relevance to the purpose of the consultation. There will not be an ongoing therapeutic relationship with the student, and he or she must return to school and cope with multiple academic and social demands for the rest of the day.

### *Team Meeting for Presentation of Consultant's Report and Collaborative Planning*

After completing data gathering through the stages outlined earlier, the multidisciplinary team reconvenes to hear the results of the consultant's evaluation. At this meeting, the consultant should present an overview of the history (with the level of detail determined by the group's general knowledge of this). It is extremely useful for the consultant to synthesize and summarize information contained in the initial packet along with the perspectives of the various team members; however, this should be done in a succinct manner that generates a sense of accord about the definition of the problem. Then, results of the parent interview, student interview, and classroom observation are presented, followed by the consultant's formulation. If the team agrees on the formulation, the consultant's program recommendations can often serve as a springboard for the discussion of an educational plan for the student. This phase should be viewed as a collaborative process among professionals with different areas of expertise. The consultant's unique contribution will be in conceptualizing and communicating about potential strategies for dealing with emotional and behavioral issues in the school setting. Together the team will agree on a specific plan and on how it will be implemented.

### *Feedback to the Parent or Parents and Student*

In some places, the parent is present at this team meeting. Agreeing with [Mattison \(1993a\)](#) that it is far preferable for feedback to be given by the consultant accompanied by the educator who will have continued involvement with the student and family rather than in a larger group context, it is nevertheless the case that in some systems there is a strong precedent for having parents at the team planning meeting. When the parent is present, the consultant must present findings in a style that is comprehensible, devoid of jargon, and recognizable to the parents as the story they have told the consultant. When the parent is not present, a separate feedback session should be scheduled relatively promptly, that is, before the team members begin talking about and implementing aspects of the plan. In some instances, depending on the age of the student and the nature of the problem and plans, it will be important to include the student in the feedback session.

### *Consultation Report*



Often schools prefer to see a draft copy of the report at the time of the team meeting but allow some further work afterward, so it can reflect planning decisions that were made collaboratively at the meeting. An excellent model for such a report is provided by [Mattison \(1993a\)](#).

### Referral

In the traditional school consultation process, appropriate clinical services can be recommended to school personnel, who, in turn, can refer the student and family for extended evaluation, possibly leading to treatment. Sometimes the consultant is the best person to make the referral, but it is important to work out the protocol for this with school personnel. In some instances, school staff members fear that if the referral is made in the context of a formal special education proceeding (see later), the school will be held financially responsible for the treatment, a burden that would unfairly and quickly drain special education resources. Indeed, the child is often referred for emotional, behavioral, or developmental difficulties that are not uniquely school matters and that should be handled financially in the same manner as pediatric care. Frequently, it is quite possible to discuss the referral as a noneducational recommendation and to meet after the formal educational proceeding has concluded to discuss privately with the parents (and youth, depending on age and clinical appropriateness) the reasons for referral and suggestions for appropriate clinical settings. What is important is that the clinician show sensitivity to this issue and work out an agreed on procedure with school personnel.

## SCHOOL CONSULTATION: EVOLVING ISSUES

[Jellinek \(1990\)](#) identifies four evolving trends affecting schools in the United States that, in various ways, concern child psychiatrists practicing school consultation. The first is the increasing demand for special services in the face of limited resources to pay for expensive, albeit often needed, services. The second issue is that of rising parental expectations for academic achievement and the expanding role of the school as an institution bearing increased responsibility for nurturing children and for preventing harmful behavior (e.g., substance use, suicide, high-risk sexual behavior). Given this situation, consultants may find it useful to promote avenues for outreach to families, both directly through the consultation process and indirectly through work with consultees (teachers and other school staff). The third issue involves the aging of the faculty of school teachers whose personal pressures and needs are new to school administrations accustomed to a younger faculty and higher rate of turnover. In fact, in the next decade, an estimated 2.2 million new teachers will be hired, according to the [U.S. Department of Education \(1999\)](#). The effective psychiatric consultant will recognize such situations and will engage, educate, and stimulate teachers. Although many aspects of this situation will need to be addressed systemically by schools themselves, the consultant can make a contribution on an individual-by-individual basis through awareness and sensitivity to these social trends and their ramifications. Such efforts may lessen the disaffection of older teachers as well as provide the consultant with the added insight of experience.

The fourth issue is the burden created by the interaction of the foregoing, as well as other complex stresses of modern life. Behavioral issues such as dropout and absenteeism are increasingly being seen as problems for which the mental health consultant may have useful input, thus opening new inroads as consultants are called on to advise on practices and policy, as well as on the needs of individual students.

The increasing recognition of need for mental health expertise in schools, combined with concern about cost effectiveness, is leading to an expansion of the traditional consultation model to a more collaborative model in which mental health experts work from within the bureaucratic structure of schools, not as outsiders ([Caplan et al., 1995](#)). This move toward collaboration involves consultants more deeply in the school system by (a) assigning them official roles in the bureaucratic hierarchy and (b) holding them more accountable for the outcomes of consultation ([Caplan, et al., 1995](#)). When this occurs, the consultant may gain a better grasp of the pressures on the local administration, as well as of the optimal avenues for positive growth and change. Conversely, the mental health professional's role with regard to school personnel differs structurally from that of the traditional consultant, and the basic assumptions of the model for working together differ as well, factors the mental health expert must incorporate in his or her manner of practice.

[Jellinek \(1990\)](#) notes that it is critical for the child psychiatrist to understand the evolving economics, values, and pressures on local school systems, because these affect the lives of the children and families with whom he or she works. Further, these trends reflect the enormous pressures on an essential social institution, but they may also signal a unique opportunity for the school consultant to contribute in new and creative ways to the mental health of many children and to the viability of many schools in our communities.

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# 121 SCHOOL CONSULTATION: THE INDIVIDUALS WITH DISABILITIES ACT

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The Individuals with Disabilities Education Act (IDEA), originally enacted as the Education for All Handicapped Children Act (EAHCA) in 1975 and popularly known as Public Law 94-142, embodies the federal statutory requirements that govern the provision of educational services to children qualifying for special education. The Act provides all children with disabilities the right to a free public education, including special education and related services designed to their needs. The underlying premise of the Act is that all children with disabilities can learn if their education is designed to meet their unique needs ( [1](#) ). Promising individuals with disabilities an opportunity for full participation in the life of their communities, and the right to be as independent as possible and meaningful access to economic self-sufficiency became a national policy ( [2](#) ).

## HISTORY

The Education for All Handicapped Children Act [EAHCA] (popularly known as Public Law 94-142) was enacted by Congress in 1975 in response to the recognition that, of eight million disabled children nationwide, nearly half were not receiving the appropriate educational services to establish equality of opportunity, and one million were excluded from the public school system altogether ( [3](#) ). Children with emotional disabilities were among the most poorly served of disabled students; studies revealed that the educational needs of 82% of children with emotional disabilities were not being met ( [3](#) ). Further impetus for EAHCA were federal lawsuits that challenged the refusal of appropriate education to children with disabilities on the grounds that refusal violated the precedent set by the Brown vs. Board of Education decision ( [4](#) ). The EAHCA, originally applying only to eligible children ages 5 to 21 years, was amended in 1986 to include children 3 to 5 years old.

EAHCA became IDEA in 1991; it was revised mainly in response to a congressional finding that children with serious emotional disturbance (SED) remained severely underserved. Fewer than half the nation's children with SED were being identified and provided with appropriate services ( [5](#) ). Among other changes, IDEA ensured a smooth transition for children moving from early intervention programs (covered in another statute for at-risk infants and toddlers, ages zero to 3 years) to preschool special education programs ( [6](#) ). IDEA stressed the importance of family support and involvement in early intervention and preschool programs. Research indicates that this is a critical provision of the bill, because one of the most consistent factors in successful preschool programs is family support and involvement ( [6](#) ).

Congress added significant amendments to the IDEA in 1997, citing a growing disparity in educational opportunity between white and minority children. In its preface to the substantive changes, Congress cited the increasing need for special education of minority students as the minority population grows relative to the white population. It further noted difficulties in accessing educational opportunity faced by minority children who lack role models in the schools and who are challenged by social disadvantage and poor language skills ( [2](#) ).

## THE INDIVIDUALS WITH DISABILITIES EDUCATION ACT

The IDEA as amended in 1997 defines "children with disabilities" to mean children with mental retardation, hearing impairments, speech or language impairments, visual impairments, orthopedic impairments, autism, traumatic brain injury, other health impairments, specific learning impairments, or emotional disturbance and who, by reason thereof, need special education, but not related services ( [2](#) ). The 1997 amendments granted states the option of extending special education and related services to students aged 3 to 9 years, if it is determined that they are experiencing developmental delay but do not fall explicitly within the definition of "children with disabilities." The areas of delay include physical development, cognitive development, communication development, social or emotional development, or adaptive development ( [2](#) ).

IDEA defines "emotionally disturbed," previously called "serious emotional disturbance," as a condition having one or more of the following over a long period of time, to a marked degree, and which adversely affects educational performance: an inability to learn that cannot be explained by intellectual, sensory, or health factors; an inability to build or maintain satisfactory interpersonal relationships with peers and teachers; inappropriate types of mood or behavior under normal circumstances; a general mood of unhappiness or depression; a tendency to develop physical symptoms or fears associated with personal or school problems ( [2](#) ).

"Related services" under the Act includes transportation, developmental, corrective, and other supportive services as may be required to assist a child with a disability to benefit from special education, including the early identification and assessment of disabling conditions in children ( [2](#) ). Included among the specified services are speech-language, audiology, psychological services, physical and occupational therapy, recreation, social work services, counseling services, and orientation and mobility services ( [2](#) ). Medical services are covered for diagnostic and evaluative purposes only ( [2E](#) ).

"Supplementary aids and services" refers to aids, supports, and services provided in the regular education environment that enable children with disabilities to be educated with nondisabled children to the greatest extent possible (i.e., least restrictive environment) ( [2](#) ).

The definition of "free appropriate public education" (FAPE) contained in the 1997 amendments tracks the language of the EAHCA of 1975. It provides that a free appropriate public education must conform to state educational agency standards and include students from preschool to secondary education ( [2](#) ). The United States Supreme Court has interpreted FAPE to mean such special programming as is "sufficient to confer some educational benefit on the handicapped child" ( [7](#) ).

## THE INDIVIDUALIZED EDUCATION PROGRAM

In order to arrange appropriate education for eligible children, IDEA requires the state to find and evaluate all children with disabilities residing within its borders. The state must see that an individualized education program (IEP) is devised for each eligible child by a multidisciplinary team involving the child's parents, teachers, a local educational representative, and people who have special knowledge about the child.

The IEP must describe the child's current educational performance, and the support he or she is receiving. Annual and short-term goals must be made to address the child's educational needs resulting from the disability ( [2](#) ). The IEP must also stipulate the special instruction and related services that will enable the child to meet those goals ( [2E](#) ), how his or her disability influences involvement and progress in the general education program, and to what extent the child will not be educated in the regular education program ( [2](#) ). Evaluations must be conducted in the language used by the child and may not be racially or culturally discriminatory. The IEP team is required to reevaluate the IEP once every 3 years, or at the parents' or teachers' request, or if conditions warrant, to determine how the child is progressing in the program and whether modifications are needed ( [2](#) ).

When appropriate, children with disabilities are expected to participate in state or district-wide assessments to the best of their abilities. The IEP must posit assessment administration modifications that permit the child's participation; if the child will not participate or the assessment is inappropriate, the IEP must state why the assessment is inappropriate and how the child will be assessed.

The IEP must see that each disabled child's FAPE occurs within the least restrictive context possible ( [2E](#) ), meaning that a child with disabilities is to be educated with

nondisabled peers unless this cannot be achieved, even with supplementary aids and services. A child with disabilities is to be educated in the most residentially proximate public school that he or she would attend if not disabled; however, if the child's particular needs prevent this, another placement may be selected ( [1](#) ). This provision is particularly relevant to children with emotional disturbance who have been removed from their educational settings owing to behavior problems that may or may not be related to their disabilities.

## DISCIPLINARY AMENDMENTS IN 1997

Under the provisions of the pre-1997 IDEA, a great deal of confusion developed with regard to the discipline of children who qualified for special education, especially for those who were identified as having a "seriously emotional disturbance." Courts throughout the country ruled inconsistently on such issues as whether students could be unilaterally removed from school if their behavior is considered dangerous by school authorities, whether students could be removed for conduct that is considered to be dangerous but "related to their disability," and whether schools are responsible to continue educational programming for students who are removed from school for behavior unrelated to the disability.

### Dangerous Behavior

One of the major procedural protections contained in the pre-1997 IDEA was a provision that required educators to conduct an IEP team meeting before initiating a change in the placement of a child. Under prior law, if a disagreement over the proposed change of placement developed between the parents and school officials, the child's last placement remained the placement until a final resolution is reached after a due process hearing and any ensuing court actions. Unless the school authorities obtained a court order enjoining the continued attendance at school by a child whose behavior was considered dangerous by school officials, the child could be suspended for a cumulative total of 10 days, but thereafter the maintenance of placement provision applied and the child had to be returned to his or her last placement ( [8](#) ). That provision of the law was modified by the 1997 amendments.

The authority of school officials was significantly increased under the new amendments. Administrators may immediately remove and place in an alternative setting a child who brings a weapon to school or to a school function, or who sells, uses, or attempts to buy drugs at school or at a school function. The alternative placement is decided by an emergency IEP team meeting ( [2A](#) ). The amendments also include a provision for dealing with other conduct considered to be dangerous. School officials can suspend a child for 10 days and in the interim ask for an expedited administrative hearing at which an order changing placement can be obtained on a showing by "substantial evidence that maintaining the current placement of the child is substantially likely to result in injury to the child or to others" ( [2B](#) ). This process can be repeated during a formal dispute by parents over the change of placement or of the appropriateness of the alternative placement.

The school must conduct a "manifestation determination review" on determining that a change of placement is necessary in response to the behavior of a child. This consists of an IEP team meeting where "all relevant information," including information supplied by the student or the parents, is considered. The team must determine whether the student understood that his or her behavior was inappropriate, whether the student could control the behavior, and whether the IEP and placement had been appropriate ( [2](#) ). If it is determined that the misconduct was not a manifestation of the child's disability, the school may apply the same disciplinary sanctions to that child as apply to children without disabilities ( [2C](#) ). If the conduct is found to be a manifestation of the disability, the school is obligated to continue programming, embodied in an IEP, in the alternative setting ( [2C](#) ). Children subject to discipline, but who have not been evaluated for and/or identified as eligible for special education, but who are suspected of having qualifying disabilities are entitled to the same protections as children already found eligible.

## THE ROLE OF THE CHILD MENTAL HEALTH CLINICIAN

There are many potential roles in the special education process for child mental health consultants ( [Chapter 120](#) ). Child psychiatrists and behavioral pediatricians may be asked to submit diagnostic assessments and medically based recommendations about a child's educational needs. Mental health consultants also may serve an important role in pulling together and interpreting the assessments and recommendations of other medical or psychiatric evaluations. The consultant may suggest specific strategies for the classroom to address the IEP objectives and help parents understand the nature of their child's disturbance, including its implications for functioning at school. School psychologists may be integral in developing creative educational techniques and in conducting functional behavioral analyses, in addition to taking part in manifestation determination procedures ( [10](#) ).

## PARENTS AND CHILDREN'S RIGHTS AND RESPONSIBILITIES

Beginning at age 14 years, the IEP must describe each child's transition service needs, with a focus on the course of study and plan for successful integration into mainstream society. Competent children must be aware of the transfer of rights on age of majority under state law. At age 16 years, the IEP must modify and reiterate necessary transition services and any appropriate interagency responsibilities ( [2](#) ).

Although the IDEA promises all children with disabilities a FAPE that meets their unique needs, this promise is sometimes unfulfilled because of ambiguities in the law. A substantive standard prescribing the level of education that each child should receive is noticeably absent from the law. The law only requires a "free appropriate public education" to meet the unique needs of a child with a disability so that the child may *benefit* from the instruction, but not necessarily reach his or her fullest potential ( [2](#) ). The lack of any clear guidelines regarding what constitutes educational "benefit" has resulted in much school-parent conflict and generated a great deal of litigation. What is clear is that unless the state law provides otherwise, a school does not have the obligation to provide services that will maximize the potential of the child with a disability. The actual benefit level that must be achieved by a child in order to constitute a FAPE is determined on a case-by-case basis and generally requires a meaningful gain and not merely trivial progress (see e.g., Board of Education of East Windsor Regional School v. Diamond) ( [10](#) ).

The IDEA provides extensive procedural protections to parents of children with disabilities, including "the right to participate in the development of the IEP, the right to independent evaluations, the right to inspect educational records" and "the opportunity to present complaints with respect to any matter relating to the identification, evaluation, or educational placement of the child, or the provision of a free appropriate public education to such child" ( [2D](#) ). Such a proceeding, referred to as a "due process" hearing, requires a neutral adjudicator, a right to counsel at the parent's expense, the right to present evidence and cross-examine witnesses. If dissatisfied, either party has a right to judicial review in the appropriate state or federal court. Relief necessary to assure that the child receives a free appropriate public education may take many forms, including reimbursement to parents who unilaterally place their child in a private placement and compensatory education in the form of educational services beyond the age of 21.

## RELATED LEGISLATION

In addition to IDEA, there are other legislative requirements enacted to protect the rights of disabled individuals. The Americans with Disabilities Act of 1990 (ADA) and Section 504 of the Rehabilitation Act of 1973 are civil rights laws that prohibit discrimination on the basis of disabilities. Although the ADA does not focus specifically on FAPE, it requires that accommodations be made to allow students with disabilities to perform essential functions of a job and thus relates to job training/placement and community activities that are components of a student's special education plan ( [11](#) ). Section 504 of the Rehabilitation Act of 1973 protects persons with physical or mental impairment that substantially limits one or more life activities. It requires a free, appropriate public education, which may be either regular or special education programming. Thus, related services may be provided for children with impairments who do not meet eligibility requirements for special education under IDEA ( [11](#) ). Section 504 requires information to be obtained from various sources, decision-making by a knowledgeable group, development of a plan, periodic reevaluations, parental notification, and the opportunity for parents to participate in impartial hearings when they disagree with the process or planning decisions ( [11](#) ).

## RESOURCES

Familiarity with basic aspects of the laws pertaining to special education and their application strongly enhances the child mental health clinician's effectiveness in the consultation process. A comprehensive and thoughtful discussion of these issues can be found in the chapter entitled "The Legal Rights of Children with Disabilities to Education and Developmental Services" by Miriam Berkman ( [12](#) ). Another good source of information is the United States Department of Education at <http://www.gov.ed>. This Website also provides easy access to information about the Americans with Disabilities Act of 1990 (ADA) and Section 504 of the Rehabilitation Act of 1973. Other resources include the National Information Center for Children and Youth with Disabilities (P.O. Box 1492, Washington, DC 20013-1492, 1-800-695-0285) and in most states, the Department (Board) of Education. Susan Etscheidt and Larry Bartlett provide a guide to determining what aids and services to include in a child's IEP in "The IDEA Amendments: A Four-Step Approach for Determining Supplementary Aids and Services" ( [13](#) ).



## FEDERAL RESTRICTIONS AND STATE PREROGATIVES

The Individuals with Disabilities Education Act (IDEA) is a federal statutory scheme that provides federal money to assist states and local agencies in educating children with disabilities. To receive a federal grant, a state must demonstrate to the Secretary of Education that the state eligibility requirements listed in IDEA have been fulfilled and that they have in place "child find" mechanisms for children with disabilities who reside in the state. Infant and toddlers and youth ages 3 to 21 years are included in the 1997 Act in Part C for children's services and in Part B for infants and toddler services.

The federal requirements under both of these sections are flexible in order to address the needs of individual states. Specific programs and policies necessary to attain the goal of IDEA are ordered, but often the methods for carrying out these programs and policies are left open to the states. IDEA provides a list of the lawful uses for grant funds awarded to a qualifying state, but the state may distribute the funds among these uses according to need. The law also allows for states to use IDEA funds to execute school-improvement plans for helping troubled children who do not necessarily qualify as disabled but who are having difficulties in school.

States applying for a federal grant under the IDEA must also guarantee certain procedural safeguards to children with disabilities and their parents ( 2). For example, due process must be guaranteed in the event that a dispute arises, evaluation and placement procedures must be administered in the child's native language, evaluations must use a variety of assessment tools and strategies, personnel doing the evaluations must be trained in those evaluation procedures, and evaluations must be conducted in complete confidentiality ( 2).

States may apply for a grant under the IDEA by submitting a state Improvement Plan, maximally integrated with the Elementary and Secondary Education Act of 1965 and the Rehabilitation Act of 1973, and based on state and local needs for meeting the goals of the state-wide system. Federal law requires that the improvement plan include: analyses of data indicating the academic performance of children with disabilities and employment rates compared to nondisabled children; the state's personnel development needs; analyses of findings relating to improving results for children with disabilities; and analyses of available information about the efficacy of the state's early intervention, special education, and general education services in accommodating the needs of children with disabilities. States must specify paths to achieving federally set goals and plans for achieving general collaboration with institutions and agencies. However, within the federal conditions for a statewide system, states may write their own definitions of developmental delay to be used in appropriate programs, develop personnel according to state certification or professional requirements, and make a number of specific decisions pertaining to the administration of special education programming.

## IDEA AMENDMENTS OF 1997: CURRENT OPINION AND RESEARCH

The practices of the special education reform movement have been characterized as follows: focusing on the child and family's strengths, rather than their deficits; promoting achievement through high expectations and proven instructional techniques and evaluation methods; encouraging collaboration across service-providing agencies in order to reduce bureaucracy; and granting access to these services without labels ( 9).

The finding that prompted IDEA 1991 persists today ( 9), in that emotionally disturbed children and adolescents are still underserved in special education programs. In IDEA 1991, a "child with disabilities" could be one who was "seriously emotionally disturbed"; however, IDEA 1997 dropped the adjective "seriously" ( 2). In one study, adjectives such as "serious" or "severe" were the single negative predictor of the number of children receiving special education service for emotional disturbance ( 14); other studies have argued that definition does not significantly influence identification patterns ( 9). Although results are inconclusive, without the adjective "serious," there might be a substantial increase in the number of students classified as emotionally disturbed ( 9).

States have been allowed to extend the nonspecific category of "developmental delay" to children aged 9 and below since IDEA 1997 was passed. States are free to define the category and specify the criteria that will be used. Some authorities claim that the extension aids young children's access to specialized interventions and strengthens communication between schools and families ( 9). Others believe that this change encourages the local educational authority (LEA) to label students as disabled and serve them as such when their needs might well be met in the general education setting ( 9).

The requirement in IDEA 1997 that children with disabilities participate in state- and districtwide assessments, although promising to advocates of special education accountability, is complex ( 9). Students who are unable to participate fully in the general assessments may take an alternate assessment designed by the LEA. Alternate assessments create the possibility of a "second class" of testing to go along with a lower standard of teaching, the existence of which would be incompatible with the goal of improving educational results for all students ( 15). States must analyze the students with disabilities' results separately from the regular students, which assures support for equality and academic improvement because the results will not be misread in a sample or lost ( 9).

A controversial aspect of the 1997 amendments to IDEA is the inclusion of parents on the evaluation team. One study indicated that as parents of children with disabilities were provided with more information about their children's schools, they developed more negative opinions of those schools ( 16). However, in the same study, frequent and involved parent-educator interactions were correlated with more positive views about school. Some note that the hierarchical system and social organization of schools constitute barriers to successful family-school collaboration ( 12), and it has been suggested that schools adopt a structure where parents, educators, and administrators are equal in decision making ( 17).

IDEA 1997 permits the IEP team to conclude that existing data are adequate for deciding whether a child with disability will continue to be eligible for services. Some argue that this provision helps school districts by eliminating pile-up of documents and advances a reform agenda aimed towards intervention-relevant assessments ( 18). Others reason that relaxing the reevaluation procedure could decrease the surety of placement decisions ( 19). Studies analyzing the effect of reevaluation on categorization and programming have brought forth inconsistent results ( 9).

The broadened IEP requirements of IDEA 1997, including members of the IEP team and IEP document inclusions, are promising to advocates of special education reform, especially regarding experimentally validated instructional approaches and student achievement. On the other hand, some general education teachers may consider the terms overly demanding and refuse to cooperate ( 15). Recent studies indicate that general educators are becoming more actively involved in the IEP process than they were in the past ( 20). The multitude of factors influencing learning that must be considered by the IEP team, although appropriate for special educators, may not be relevant to a general educator's knowledge and constitute a distraction from teaching ( 9,21). Several studies have concluded that the contents of the IEP document and the activities of the school day are not consistent, thereby rendering the IEP impractical ( 22). It has been found that teachers in general education settings are less likely to deliver IEP's agreeing with special education policy than are teachers in other, more exclusive settings ( 23). Some question whether the IEP requirements are sufficient to bring about the goals of IDEA, in terms of special education delivery and student success ( 9).

The discipline amendments were added to IDEA to address misconduct in students with disabilities. Behavioral analysts have known the benefits of functional behavioral approaches to discipline for over 35 years, without widespread acceptance in schools ( 9). It also has been suggested that perhaps the only way to persuade schools to accept proven behavioral techniques is to place experts in applied behavior analysis in schools in order to influence teachers, parents, and government workers ( 24). Because of the emphasis on education rather than punishment in the discipline amendments, schools also may be encouraged to explore more creative and collaborative approaches; for example, alternative schools, to address the needs of students who have behavioral problems ( 9). Although in certain respects, current special education practices and the 1997 amendments to IDEA are a long way apart, it is hoped that increased attention to special education policies and practice will stimulate a new generation of special education practices and research ( 9).

In conclusion, it is important to remember that the overriding goal of the IDEA initiative is provision of free and appropriate education for all children. To best achieve this, each student needs to be in the least restrictive environment that is compatible with learning and educational progress. Understanding the least restrictive environment entails understanding the arguments, benefits, and trials behind both inclusive and special education settings. Supporters of inclusion contend that a separate education denies children with disabilities the opportunities afforded to regular students. The same inclusion advocates can provide data contending that students with disabilities who are educated in general education settings achieve higher social and academic standards than equivalent students in noninclusive settings ( 25). On the other end of the spectrum, opponents to inclusion warn that simple physical presence in a regular classroom fails to lead to full participation in a classroom's intellectual and social life ( 26). Opponents contend that at times, special education programs are superior to general education programs for certain individuals. ( 27). The unique and individual nature of development must be appreciated to insure that special education programming is responsive to the needs of each eligible child in assessing the relative advantages of the various settings and modalities of special education.

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# 122 HIGH-RISK CHILDREN, ADOLESCENTS, AND FAMILIES: ORGANIZING PRINCIPLES FOR MENTAL HEALTH PREVENTION AND INTERVENTION

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[High-Risk Children, Adolescents, and Families](#)  
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The complex, continuous, and reciprocal interactions between environmental, biological, intrafamilial, and individual factors common to multiproblem families may place children at high risk for poor developmental, psychological, and educational outcomes, including disruption of primary attachments, placement out of the home, and entry into the juvenile justice system. Prevention and intervention efforts that focus on the developmental needs of children in the context of addressing the needs of other family members are consistent with the goals of Public Law 105-89, the Adoption and Safe Families Act of 1997 ( [McCarthy et al., 1999](#)). These goals, which focus on the promotion of safety, permanency, and well-being for children and families, inform multiple contemporary federal and state initiatives. This chapter describes organizing, developmentally based principles for working with families in which children are at high risk, and provides a case example derived from an intervention designed to address the previously stated goals and outcomes.

## HIGH-RISK CHILDREN, ADOLESCENTS, AND FAMILIES

Although there does not appear to be a common description of families in which children and adolescents are at high risk, these families often are described as *at-risk*, *multiproblem*, *troubled*, *low-functioning*, or *families in crisis*. Most authors acknowledge that multiple factors contribute to the inability of these families to function adequately ( [Bowlby, 1953](#)). Factors frequently cited in association with breakdown of the family include parental substance abuse, mental illness, chronic physical illness, domestic violence, cognitive deficiencies, constitutional inadequacies, and social isolation significant for lack of familial and community support ( [Deutsch, 1983](#); [Rutter and Quinton, 1984](#)). When these factors are joined with environmental stressors such as poverty, educational failure, joblessness, homelessness, and racial or ethnic discrimination, the risk of family disintegration increases. Families who share these combined characteristics commonly are identified as multiproblem families ( [Kaplan, 1986](#); [Spencer, 1963](#)). [Kilpatrick \(1999\)](#) argues that these families lack the leadership and control within the family necessary for meeting basic nurturing needs and providing adequate protection for family members.

In recent decades, the ability of families to provide adequately for the needs of their children has been compromised by the convergence of adverse environmental, psychosocial, psychological, physical, educational, and economic factors ( [Ford Foundation Executive Panel, 1989](#)). The prevalence of these stressors on families appears to be increasing in the United States. Between 1979 and 1994, the number of children younger than 6 years of age living in poverty grew from 3.5 million to 6.1 million. During this same period, the percentage of children living in families with incomes below the poverty line rose 39% overall. Between 1979 and 1998, the percentage of U.S. children younger than 18 years of age living in poverty increased by over 3 million, representing an increase in the national child poverty rate from 16.7% in 1979 to 18.7% in 1998 ( [Bennett and Lu, 2000](#)). A report of the National Center for Children in Poverty (1996), based on data from each of the 50 states, found that young children living in poverty are more likely to experience high levels of interpersonal conflict in their homes, be exposed to violence in their neighborhoods, and experience delays in their physical, cognitive, linguistic, and emotional development, which in turn affect readiness for school and ability to learn. Minority young children were found to be five times more likely to be poor than white children; the rate of poverty among young Hispanic children was increasing more rapidly than among other racial and ethnic groups ( [National Center for Children in Poverty, 1996](#)).

The effects on children of growing up in these families are profound ( [Taylor and Newberger, 1979](#)). They are more likely to present with symptoms of neglect and nonorganic failure to thrive. Their health care is more likely to be episodic and illness oriented. Developmental delays, language difficulties, behavioral problems, and school failures are common. For some children, attendance at school may be disrupted frequently owing to family moves or repeated incidents of homelessness. [Klerman \(1991\)](#) reported that poor children experience more of many types of health problems than children from families with more adequate incomes. Rates of infant mortality, sudden infant death syndrome, unintentional injuries, child abuse, and infectious diseases, including acquired immunodeficiency syndrome (AIDS) and human immunodeficiency virus (HIV) infection, are higher among poor children. Poor and near-poor children also suffer disproportionately high rates of problems related to nutrition, asthma, otitis media, and other infectious diseases, as well as increased incidence of dental decay ( [Lewit and Kerrebrock, 1998](#)) and lead poisoning. Economic adversity also is associated with an increased incidence of prenatal risks, prematurity, and accident. The existence of multiple stressors often increases exponentially the impact of any particular stressor and heightens the eventual likelihood of central nervous system dysfunction ( [Kavale and Forness, 1985](#)). Poor children on inadequate diets are likely to experience more frequent infectious disease than the nonpoor, to be sicker longer and be affected more severely by their illnesses ( [National Center for Children in Poverty, 1996](#)). Inadequate nutrition also may affect cognitive development and social behavior; undernourished children may be more apathetic and less responsive in social situations ( [Adnopoz, 1994b](#)). [Kilpatrick and Holland \(1999\)](#) suggests that the best, generally descriptive term of these families is *impoverishea*, where poverty is more complex than a lack of financial resources. These families typically live in a world that is lacking in many resources that others take for granted, such as stability in housing, preventative health care, and, in more recent times, information technology.

Many families, who require additional support or specialized services, are thought to demonstrate similar characteristics ( [Geismar and Lasorte, 1964](#); [Kaplan, 1986](#); [McCroskey and Meezan, 1998](#)). [Kaplan \(1986\)](#) found that these families have “a number of problems that cut across many dimensions of family life.” These families have been found to evidence poor coping skills in intrapersonal, interpersonal, and environmental areas and have demonstrated difficulty in negotiating the interrelationships among these three spheres of functioning. [Belsky \(1980\)](#) proposes a developmental–ecologic model for understanding high-risk families. In this model, risks are present at four levels: individual, family, environmental, and cultural. The individual level includes issues of parental self-perception such as self-esteem, impulse control, and behavioral and health history; individual factors also include the child's personality traits, the fit between the parent and the child, and other special characteristics. The family factors may include interpersonal conflicts or violence, the capacity of the family to provide support, safety, and protection for its members, the nature of the extended family relationships, and the dynamics of family functioning. Environmental influences extend to the family's interactions with external institutions and systems. The cultural context relates to the family's belief system, its values, and its attitudes. The constant interaction of these factors affects the family's ability to parent safely and appropriately and determines the relative risk to the children in the family's care. In this view, in the social ecology of the family's environment, problems transcend a single level and often appear as a “web” or cluster of issues that may eventually threaten to overwhelm the family.

Some believe that these families contribute to the burgeoning costs of social services through their inappropriate use of the programs and systems that have been developed to provide care for them. Some politicians have redefined poverty as a problem that results from the immoral and irresponsible behaviors of individuals rather than an unfortunate economic condition resulting from external social and economic factors, with an emphasis on the need for individuals to change their behaviors ( [Kammerman, 1996](#)). Similarly, [Spencer \(1963\)](#) places the responsibility on the family for its frequent reliance on crisis care, remedial services, and public support. He concludes that chronic dependency on public programs is not only costly but represents an inefficient use of available resources. [Selig \(1976\)](#) suggests that effective intervention can be designed for these families. He suggests that the term *multiproblem family* is a misnomer that places blame undeservedly. He believes that the problem lies within a “multi-problem delivery system” that offers fragmented social services. Selig identifies issues of accessibility, ownership, caregiver attitudes toward inadequately functioning families, lack of coordination between providers, gaps in the service continuum, insensitivity to social and cultural distinctions, and lack of continuity of caregiving as central contributors to a troubled service system. [Overdorff \(2000\)](#) is explicit in describing adversarial relationships that may develop among agencies serving this population. Ineffective serial interventions in the form of uncoordinated, isolated events may result in community “impotence” in changing dangerous situations for children.

## MULTIPROBLEM FAMILIES: PROBLEMS IN INTERVENTION

The association of such major societal problems as poverty, racial and ethnic discrimination, inadequate and substandard housing, and underclass status with families requiring public assistance and support discourages caregivers from working intensively with them. The behaviors of service providers suggest a lack of confidence in both the family's ability to institute change and in the service system's capacity to intervene positively. The providers' cynical attitudes toward these families are unwittingly supported by the families themselves, who because of past unsuccessful, and possibly demeaning experiences with service providers may be unable or unwilling to give expression to their need for help. These behaviors may cause families to appear disinterested in the interventions offered to them, lead them to be labeled as noncompliant, and result in their punitive treatment. Negative caregiver attitudes may be perpetuated by the frequency with which issues of distrust, noncompliance with treatment regimens, and failure to keep appointments arise in working with these families. Families are blamed by providers for behaviors and attitudes that are in actuality rudimentary attempts by families to defend against the attitudes and behaviors of providers who are expected to serve them, rather than symptoms of the family's inability to accept responsibility and respond appropriately to the needs of its children. For example, a mother who fails to visit regularly her hospitalized child may be made to feel like a negligent, inadequate parent by the floor staff when she does come on the ward. Her failure to visit regularly may represent an attempt to protect her own fragile sense of competency and self-esteem in the face of what she perceives as the uncompromising and judgmental demeanor of the staff, rather than an uncaring, neglectful attitude toward her child. The frustration of providers, who recognize that the child in their care has a deep wish to be cared for by his or her parents, may interfere with their knowledge that parents also have a need to be supported by others and made to feel competent in the parental role.

There are additional barriers to successful intervention; some families may be resistant to interventions provided by "outsiders" who come from cultures, races, or socioeconomic groups from whom they feel alienated. Services may be inaccessible, especially for parents who have no alternative source of child care and must travel unassisted, with several small children. Language barriers may make communication between families and caregivers difficult. Caregivers may have little if any knowledge of the cultural differences, customs, and strongly held beliefs of the families they treat. Significantly, the service system may focus on the family's failure, without assessing the family's strengths and attempting to build on them. Although families do share responsibility for their situations, blaming the family for being in trouble is not useful.

Traditional social service delivery systems have proven inadequate to meet the complex needs of inadequately functioning families. Unidimensional interventions designed to address specific problems in single spheres of functioning have not provided an effective means of confronting the multidimensional problems that categorize these families. Recent legislative, child welfare, and mental health initiatives have challenged service providers to find innovative ways to address the multidimensional needs of troubled, difficult-to-serve families. The Adoption Assistance and Child Welfare Act of 1980 (Public Law 96-272) required states to make "reasonable efforts" to keep children from out-of-home-placement; the Education for All Handicapped Children Act of 1975 (Public Law 94-142), rewritten in 1990 as the Individuals with Disabilities Education Act, requires that placement decisions be made on the basis of the least restrictive setting; the Child and Adolescent Service System Program (CASSP) of the National Institute of Mental Health, created in 1983; and the Mental Health Services Program for Youth (MHSPY), initiated in 1989 by the Robert Wood Johnson Foundation in 1989, all support the development of integrated, comprehensive service systems ( [Solnit et al., 1997](#)). The failures of the foster care system, combined with a rise in the number of children referred for placement ( [Knitzer and Cole, 1989](#)), placed increased pressures on policy makers and professionals to find new models of service capable of responding to the needs of children and families.

In August 1993, the United States Congress enacted Subpart 2 of Title IV-B of the Social Security Act, entitled Family Preservation and Support Services. This legislation strengthened the Adoption Assistance and Child Welfare Act of 1980, and authorized funding to serve as an added stimulus for states to engage in broad-gauged consensus building, collaboration, and systems reform by providing family support and family preservation services ( [Adnopolz, 1994a](#)).

New programs emerged that were designed to reduce the barriers to effective use and change the systems by which services were to be delivered. These programs embody principles first articulated in the CASSP and are expected to be family oriented, child centered, strength based, developed in partnership with the family, delivered in the child's home and community, and provided through collaborative service networks. Crisis intervention, case management, skill building, respite, mentoring, and concrete services are standard core treatment elements. Services are informed by the presumption that permanence, safety, and a consistent relationship with a caregiving adult are essential to the healthy growth and development of all children. As a result, the most appropriate placement for a child is thought to be an adequately functioning family, preferably the child's biological family, to ensure continuity of caregiving and maintain the child's ties to his family's culture, history, and belief system. When this is not possible, adequate care of the child relies on the ongoing support of the appropriate service systems to ensure a committed, permanent alternative placement. Assisting isolated, disaffected, alienated families to function well enough to care adequately for their children or to support the child's permanent placement elsewhere has been found to require a flexible, enlightened, and integrated service system whose purpose is clear and unambivalent. [Weber \(1998\)](#) argues that this integrated system should be community based, relying on "a network of services and supports offered by partnerships involving multiple sectors of society" (p. 129).

Other factors in the environment have had a negative impact on attempts to meet the needs of children who are at risk but who can, with adequate family supports and intervention, remain with their families. The unfortunate, well publicized deaths of children who had been allowed to remain in their homes by protective service workers attempting to support the integrity of the family has engendered an adverse response to family preservation ( [Gelles, 1996](#); [Murphy, 1993](#)). However, thoughtful, clinically informed risk taking has been successful in preventing placement of some children, whereas in other cases, clinical judgment has led appropriately to removal and placement. The significant cuts in federal spending for low-income children and their families and the devolution of primary responsibility for low-income children and families from the federal government to the states ( [Kammerman, 1996](#)), coupled with the serious constraints of managed care, particularly in relation to mental health services, place children and families at elevated risk of poor developmental outcomes.

## TWO ORGANIZING PRINCIPLES FOR WORKING WITH MULTIPROBLEM FAMILIES

The doctrine of the best interests of the child constitutes the first of two organizing principles that have guided a home-based, family support and preservation intervention that was developed by the authors for families in which a child is at risk of placement. The second principle recognizes the strengths of the ego support lent to the parent by the therapeutic team, whose role is to engage with the family and build a relationship through which the path to behavior change can be negotiated ( [Lewis and Stark, 1966](#); [Soule et al., 1993](#)).

### The First Principle

The family unit is the primary means through which the needs of the child can be addressed. [Anna Freud \(1965\)](#) has stated that the best interests of the child are satisfied when the child's needs for affection, stimulation, and object constancy are met. The family unit is the institution society considers best suited to respond to these needs and to guide the child from dependence to independence ( [Caplan, 1978](#); [Provence, 1979](#)). Some families are unable to assume these responsibilities and to provide an environment supportive of healthy child development. Children need adults (usually parents) to attend to their physical needs, provide nurturance, protection, and guidance, and demonstrate that they are prized and respected.

Parents also have needs. [Solnit \(1968\)](#) has described these as needs for evidence of their competence as parents and for effective, esteem-enhancing assistance from their community in times of crisis. In the best of cases, children's needs are met in relatively strong, relatively self-sufficient families by parents or other adults who are capable of nurturing and committing themselves to them. In the best of cases, parents' needs are met in turn by familial and social networks and by other support systems. When parents are able to meet children's needs, children are more likely to feel secure and valued. They are more likely to be equipped for the challenges of school and better able to make successful adaptations throughout life. Importantly, they are more likely to grow into adulthood with the ability to nurture others.

However, children and families who experience a confluence of parental inadequacies and environmental stressors are at greater risk of being distanced from the mainstream of familial and societal supports and the positive reinforcement that such support elicits. The resultant social isolation may lead to pervasive feelings of futility, impotence, and desperation. [Erikson \(1964\)](#) has noted that

Defenseless as babies are, they have mothers at their command, families to protect the mothers, societies to support the structure of families and traditions to give a cultural continuity to systems of child care and training. All of this is necessary for the human infant to evolve humanly, for his environment must provide that outer wholeness and continuity which, like a second womb, permits the child to develop his capacities in distinct steps, and to unify them.



If a child's environment does not provide that "outer continuity," the child's chances of growing and developing in a healthy way are diminished. When families lack the resources and capacities to satisfy the needs of individual family members, they cannot perform their crucial function as socializing agents for children. If they cannot perform that function, the individual family and society as a whole suffer important losses ( [Schorr and Schorr, 1988](#)). The situation is particularly egregious for the children of poor families ( [Kaplan, 1986](#)).

The argument for interventions that support the structure of the family is made even more compelling by the human and economic costs involved. The inability of families to meet the basic needs of their children may lead appropriately to foster care placement and a consequent break in the child's relationship with the primary object. For some children, out-of-home placement may be clinically indicated. For most, however, the first separation can be expected to provide an easy pathway to additional placements and the loss of any possibility of maintaining stable, affective ties to their caregivers. The current shortage of foster homes and the inability of public child protection programs to ensure continuity of placement once a child is removed make any removal of a child a significant risk, to be balanced carefully against maintenance of the child within the family. In addition, inadequately functioning families use public services such as crisis health care, police, special education, and mental health programs more frequently than other families. Therefore, efforts to stabilize these families and to improve their capacity to manage have important social and economic implications as well. [Tracy and Whittaker \(1987\)](#) and Tracy (1990, 1991) have demonstrated that identifying families appropriate for intervention and developing social support resources with those families has helped to stabilize and improve their capacity to manage with potential social and economic benefits.

### The Second Principle

The centrality of relationships in human functioning constitutes the second organizing principle for effective intervention. The relationships between parents and children, between the family and the community, and between the family and the service systems are influential and diagnostic. The achievement of behavioral change in the family unit may be accomplished best through the development of a working alliance with a clinician able to engage with the parent. However, families may be reluctant to accept assistance of any kind initially and introduce barriers intended to frustrate attempts at intervention. The process of engagement frequently begins with the clinician's demonstration of willingness to accept the family as they are and to withstand the family's repeated tests of personal commitment. The empathy, support, acceptance, and understanding extended to the parent by the clinician, perhaps for the first time in her history, enables the parent to enter into a trusting relationship, even if tentatively, and make the initial move toward a higher and more appropriate level of functioning ( [Soule et al., 1993](#)).

## INTERVENTION MODELS FOR WORKING WITH MULTIPROBLEM FAMILIES

As previously noted, pressures to reduce out-of-home placements and other expensive services have increased as the numbers of children requiring protection and support continues to rise. The striking increase in children affected by parental substance abuse and HIV/AIDS has overtaxed the foster care system and created a placement crisis ( [Nelson, 1996](#)). The number of family support and family preservation programs designed to help inadequately functioning families parent more effectively has increased dramatically. The CASSP principles of integration, collaboration, and coordination have been used to bring families and providers together to develop community-based programs and strategies to help families cope more successfully in stressful environments. Because a negative synergism often exists in the complex, dynamic interrelationships among the family, its members, and the environment, interventions that are focused only on one problem area may be futile. Practice has come to reflect the clinical knowledge that effective program strategies address the interrelationship of problems in intrapersonal, interpersonal, and environmental domains. For example, programs designed to address the needs of substance-abusing women and help families to provide safety, stability, and permanence for their newborn children may provide parent education, health and family planning, infant day care, developmental assessment and evaluation, transportation, job training, individual treatment, and legal services in addition to access to formal drug treatment ( [National Abandoned Infants Assistance Resource Center, 1999](#)). Attempts to engage parents in meeting the needs of their children may be unsuccessful if long-standing parental issues such as marital or partner conflict, past child abuse or neglect, incest, alienation, or absence of supportive personal relationships are not addressed. In addition, a history replete with persistent failures of social service interventions, limited access to and availability of services, and institutional racism and sexism may present barriers to engagement and family involvement.

Several of the programs designed to serve inadequately functioning families make use of a generic model of family-centered, home-based services that seek to accomplish the goal of preserving the family while addressing its interlocking problems ( [Edna McConnell Clark Foundation, 1985](#)). [Kaplan \(1986\)](#) offers descriptions of several exemplary home-based, family-centered programs that have proven to be successful with multiproblem families. These programs have several common elements:

1. The family system is seen as the primary unit of treatment ( [Kaplan, 1986](#)).
2. Services are provided to the family in their own home.
3. It is assumed, according to a growth model, that change in the family is possible; family strengths, not weaknesses, are pursued ( [Kaplan, 1986](#)).
4. Services are provided on a goal-oriented, time-limited basis.
5. Services focus on keeping families together and preventing unnecessary foster placement of children ( [Edna McConnell Clark Foundation, 1985](#)).
6. Services are intensive; caseloads are low.
7. Concrete services and child and family advocacy are included in the range of offered services.

Although clinicians may draw on a number of theories to guide the intervention with families, Grigsby ( [1993, 1994, 1995](#)) argues that social attachment theory ( [Bowlby, 1969, 1973, 1980, 1988](#)) is most useful as a guide for working with families of this type.

A number of these principles were incorporated into one of the earliest home-based family preservation programs, The Homebuilder Program, first implemented in 1972 in Tacoma, Washington. The program was designed to avert out-of-home child placement of children, most of whom were in acrimonious disputes with their parents, by offering intensive, short-term, home-based therapy and casework to families experiencing a crisis of placement, using a single, master's-level therapist as the in-home worker. The program model spread across the country rapidly and has been implemented on both coasts, although its efficacy only recently has been tested experimentally. Other models of intervention have been developed as well, with similar strategies but with somewhat different programmatic elements. The duration of the intervention may vary from 4 weeks to 6 months; services may be provided either by individual clinicians, therapeutic teams, or paraprofessionals, and time in the home may vary from 5 to 18 hours weekly. Nelson (1990a, 1990b) and [Nelson et al. \(1990\)](#) offer descriptions of various service models and discuss their effectiveness.

Common to all programs is the provision of therapy in the home and its availability to all family members. Although the modality may differ, the move of the clinician from the traditional office or institutional setting to the family's setting has lessened resistance to treatment for parents and children. In addition, it has enabled the clinician to observe the family in its own environment and to identify more clearly and more quickly the range of the family's needs. Given the particular characteristics of multiproblem families and their historic rate of treatment failures, the ability to intervene clinically in the home environment appears to offer significant promise for improved outcomes.

As a result, most have viewed these programs optimistically. Some researchers ( [Frankel, 1988; Gelles, 1996; Wald, 1988](#)), have raised questions as to whether these programs actually are helping the children and families they are designed to serve. [Wald \(1988\)](#) argues that "family preservation cannot be an end in and of itself" and that "family preservation is appropriate only when it serves to protect and promote" the goal of the child's well-being. This is consistent with the point of view that the focus of family preservation programs should be the best interests of the child ( [Goldstein et al., 1973](#)). One model program with such a focus is the Family Support Service (FSS) developed by the authors.

## FAMILY SUPPORT SERVICE

The FSS is a time-limited, 24-hour, in-home, voluntary program for families in which a child is at imminent risk of placement outside the home. The program has been operated continuously since 1985 by the Yale Child Study Center in collaboration with the Connecticut Department of Children and Families. The genesis of the FSS came through the support of the Edna McConnell Clark Foundation, which has played a significant role in the family preservation movement nationally. The program is staffed by master's-level social workers or psychologists who join with trained, crisis-oriented lay family support workers to form a therapeutic team around each family in the program.

Six operating assumptions inform the work of the FSS. First, there are existing strengths in families that, once identified, can be used effectively by the therapeutic team. This assumption is significant because it represents a reversal of the traditional position of state child protection agencies, which by virtue of their legal mandate

seek to identify family weakness, parenting failure, and degree of risk.

Second, use of a therapeutic team is a particularly effective means of serving multiproblem families because it addresses the environmental and intrapsychic needs of the family simultaneously, while also providing mutual support for the team itself. Third, the work of the family support worker is central to the program's ability to effect change in the family. Families who often are socially isolated members of society's underclass, with previous histories of health, mental health, addiction, domestic violence, and other problems, and a historic inability to use the service system effectively, have significant difficulties in accepting and relating to traditional service providers. The FSS worker, a role model with whom they can identify, works with the families in their own homes, uses their own language, and understands their lifestyle. He or she is a critical and essential resource in breaking the cycle of isolation and reducing the distrust and distance that frequently have characterized families' past relationships with other helpers.

Fourth, team members play flexible roles. They may function as psychotherapist, diagnostician, evaluator, case manager, service broker, or advocate; they may meet with family members individually, as couples, or in family therapy; the specific clinical role is determined by the needs of each individual case. The intervention takes place in the home and in the community; the working relationships may be developed while driving to appointments, shopping for necessities, or attending a school visit.

Fifth, collaboration with other agencies is essential to the success of the intervention. FSS intervention is brief (no longer than 16 weeks); it does not replace existing services and programs. Work with community agencies from the time of referral to the time of discharge is expected and necessary if the goals for the family are to be attained during this brief period and maintained over time. Sixth and last, planning for discharge must begin at time of intake. Well planned, intensive, and brief involvement is a potent vehicle for change. This final operating assumption is embodied in the first question asked when a case is accepted into the program: What are the criteria for discharge? The brevity of the intervention is intended to minimize the intrusion into the family. It also capitalizes on the crisis that commonly exists at the time of referral by bringing the problem areas into sharp focus and stabilizing the family so that it can go on to use other, less intrusive and more familiar means of support.

The FSS program categorizes a family's risk for having a child removed in the following ways:

1. The referring child protection agency has decided that a child will be placed outside of the home within 5 days unless the family's resources can be rallied immediately.
2. A child already has been removed as a crisis response, but immediate return home may be indicated if additional internal or external support can be put into place quickly.
3. A referring worker's persistent discomfort about a child's health or safety propels a move toward placement, even though there may be significant clinical uncertainty about the benefit to the child of removal from the biological family. These often are cases in which significant parental dysfunction coexists with evidence of a positive child–parent attachment.

The FSS process begins with an intake assessment in the family home. At this time, the parent(s), the FSS worker, the clinician, and the referring worker jointly establish the goals for the intervention. Initial visiting schedules are established, task assignments are made, and roles are clarified.

Family support workers visit with their family clients twice weekly at a minimum, often more. The worker attempts to begin to establish a nonjudgmental, empathic relationship with the parent (or parents) and to arrange for concrete services for the family.

The social work clinicians screen and assess children and families for their service needs, keeping in mind the multiple environmental and intrafamily issues with which these families present. The family plays a central role in identifying the goals of the intervention and prioritizing the order in which the identified problems will be addressed. Ongoing communication between the family and the team is essential. Team members are available by beeper 24 hours a day, 7 days a week. Because consistent relationships are highly valued, team members are expected to respond to after-hours crises themselves, and do not rely on outside crisis responders. In actuality, once a working relationship is established with a family, workers receive minimal numbers of after-hours calls.

The FSS also has available a contingency fund for items such as security deposits, refrigerators, furniture, diapers, and food. The therapeutic team requests funding for its clients when such expenditures are thought to be important to maintaining a child in a family. For some families, this fund is used as a non–interest-bearing loan; for others it is given as a grant.

### **The Roles of the Family Support Team**

All of the work of the FSS team is clinically informed and theory based. The role of the social worker or psychologist is to apply clinical knowledge to the tasks of understanding underlying family dynamics and identifying both the parental behaviors and the environmental stressors on which the intervention can be focused. The clinician may be involved directly with the parent in brief individual treatment, parent guidance, or family therapy. A task of the family support worker may be to relate empathically to the parent/client as a nonjudgmental, caring role model and to assist the parent to obtain concrete services and negotiate appropriate service systems. The social, cultural, and personal background of the FSS worker may resemble that of the client. This similarity can enhance the client's acceptance of the FSS worker as a trusted and knowledgeable person. However, as stated previously, the roles of the team members are flexible, rather than fixed, depending on the individual case and the relationships that develop between the family and the members of the team. The pairing of the family support worker with the clinician focuses the work in a specific and goal-directed way that is based in the family's reality. Attention is first directed toward identifying and meeting the immediate basic needs of the family. After issues of food, clothing, and shelter are addressed, other issues can be seen more clearly and the intrafamilial work begun. In the process of assisting the family to obtain the necessary concrete services, the worker and parent begin to establish the relationship out of which real change can come. This approach assists families to cope with the immediate crisis, avoid further deterioration, work to gain some insight into the family situation, and achieve reorganization at a higher level.

In addition to assessing the immediate needs of the family, the team also assesses its needs beyond the time of intensive intervention. Together the family and the team identify other voluntary community resources that are appropriate, accessible, and available to address the family's longer-term needs. The professional status of the clinician facilitates access to certain services and makes possible more efficient case planning and transition. The FSS worker, with a working knowledge of grassroots resources, often is able to cut through bureaucratic red tape to obtain services by using contacts with line staff. The ability of the therapeutic team to relate to systems and services at two levels simultaneously makes possible more effective negotiation of the boundaries between agencies and systems than could be accomplished by either individual working alone. The use of the team thus seems to address both the concrete and psychodynamic needs of families in a way that mobilizes difficult-to-reach multiproblem families effectively.

Because threatened placement is a primary consideration for referral to programs such as FSS, it can be assumed that there is an existing concern for a child's safety. The active and continuous involvement of the FSS team with the state child protection agency is designed to encourage careful consideration and decision making focused on maintaining the child in the home when appropriate and possible, taking enlightened risks in the interests of continuity and consistency, and unraveling and ameliorating the complex web of environmental and intrafamilial problems that beset these families. The team provides a level of support and knowledge that enables the child protection worker to weigh alternatives carefully and to feel less anxious about leaving the child in the home. All those involved with the family thus can attend better to the real needs of the child and family. At the same time, the active involvement of the team ensures a watchful presence in the home and provides an assurance that concrete efforts will be made to improve the environment.

In summary, the team-structured FSS model holds promise for being an effective intervention for multiproblem families with children at high risk for poor developmental outcomes. The relationship-based, family-focused, child-centered approach central to FSS has been successful in maintaining these children in their homes and communities and addressing the constellation of needs identified by their families or revealed through careful clinical assessment of each child's health, mental health, or educational status.

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#### CASE ILLUSTRATION

This case illustration offers an example of an in-home, time-limited, team-structured intervention with a family whose children were at risk because of the multiple problems that affected the mother's ability to care for them appropriately. This example should be relevant for child psychiatrists or mental health professionals who are asked to evaluate, assess, or treat a child where the locus of the actual problem may be in the immediate family or family environment.

Mrs. M., a 32-year-old Hispanic mother of six children who ranged in age from 2 to 10 years, was referred for in-home family service 2 weeks after the sudden death of her husband. Mr. M. had negotiated all of the systems for her and for her family during his



lifetime. Mrs. M. spoke only Spanish, was illiterate, and was reported by the Association of Retarded Citizens as being mentally retarded and probably unable to manage the affairs of her family.

At the time of the initial home visit, Mrs. M. was in a deep state of mourning. Her children were reported to be delayed developmentally, to lack age-appropriate social skills, and to have little expressive language. In addition, the 7-year-old daughter, Maria, had difficulty controlling her impulsivity and aggression, and 2-year-old Juan appeared developmentally delayed, although further evaluation was needed to confirm the diagnosis. The primary supports for the family were Mrs. M.'s minister and some paternal family members. However, the minister, who was a key influence in this family, strongly doubted that Mrs. M. could provide for the children adequately enough for them to remain at home.

Working together with Mrs. M. to identify the strengths of the family, the clinician and the family support worker made an initial assessment that Mrs. M. might possess the ability to function adequately as a parent if she had assistance in negotiating the various provider systems that would be essential to her family's health, welfare, and well-being. She would need help in such basic areas as managing money, making the home safe for her children, making and keeping appointments, and advocating for the special educational needs of her children. This initial impression was derived from Mrs. M.'s clear devotion to her children and their apparent attachment to her, her insistence that she was able to protect them in a dangerous neighborhood, and the clinician's awareness of the fear and isolation Mrs. M. suffered because of her illegal alien status.

Because Mrs. M. already was in psychotherapy at a community clinic, the FSS clinician assumed the role of case manager and not of therapist. The family support worker began a twice-weekly schedule of visitation. Both the clinician and the family support worker offered assistance in obtaining concrete services. The family support worker transported her to the appropriate state agencies to help her qualify for survivor's benefits. She was accompanied to the housing project administration to report a problem toilet and sink and to the Department of Income Maintenance for the budget verification required to clarify her rental status. The clinician visited Maria's school to discuss the appropriateness of her school placement with the principal. With the school's support, the clinician gained Mrs. M.'s permission to refer Maria to a child guidance clinic for a full psychiatric evaluation.

During the first month of FSS involvement with this family, Mrs. M. felt increasingly overwhelmed by the problems stressing her family. Mrs. M. believed that she was approaching a "nervous breakdown." Shortly afterward, while Mrs. M. and the family support worker waited together for the children at the bus stop, Mrs. M. confided that she would like to place all her children in foster care and arrange to see them only on the weekends. Although the aide was surprised and discouraged, she told Mrs. M. that she would bring her information that would help her to begin the placement process. At the next visit, the family support worker suggested the alternative possibility of a day care program for the children and a homemaker for Mrs. M. Mrs. M. was interested in exploring these possibilities because she had realized that, in fact, she did not want her children to be placed away from her. She recognized that when she had spoken about placement she had been overwhelmed by the thought of caring for them by herself and had not been able to envision any other alternative. She also recognized that she needed help to keep her children with her.

With the goal of maintaining the children in the home now established, both the clinician and the family worker began to negotiate with other systems to find an appropriate homemaker as well as an affordable day care program. The family's food stamps, which were suspended temporarily because of their illegal immigrant status, had to be restored. The team visited the neighborhood health clinic to coordinate the family's health care and simplify the family's access to the health care system. Termination took place after the successful completion of the goals of the intervention. The home was considered to be safe, the children's relationships with their mother appeared to be stable, a homemaker provided Mrs. M. with assistance in managing her domestic responsibilities, and the children were in appropriate day care and school settings. At a discharge planning meeting attended by all of the service providers working with the family, Maria's therapist agreed to act as ongoing case manager. With these supports in place, the work of the FSS team was concluded.

#### **Comment**

There should be no illusion that this 12-week intervention provided a "cure." Mrs. M.'s limited intellectual functioning, her depression, and her continued mourning suggest that her functioning over time will be impaired. However, she had taken responsibility when in the past she had taken none, and she had begun to play a more active role in caring for the multiple needs of her children and herself. A support system had been developed and activated for Mrs. M. and her children that required her to relate to only two primary caregivers: the pediatrician and the child guidance clinic. Six children were then enabled to remain together with their biological parent. The mother's potential dependency and inability to function were modified, and multiple problem areas were addressed with positive results. The alternatives for the M. family might have more than doubled the sense of loss and abandonment that the death of their father already had occasioned and left them even more vulnerable.

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## **CONCLUSION**

Multiproblem families present a challenge to service providers because of their difficulties across multiple spheres of functioning. The families often are in a state of chronic dependency on public health, welfare, and social service systems that contributes to the current burgeoning costs of health and mental health care. These families also may exhibit a persistent failure to respond to help when it is offered, adding to the frustration of the service providers, who may already have a lack of confidence in the family's ability to institute change and in the service system's capacity to intervene effectively.

Because of the complex web of problems with which these families present, traditional treatment approaches are likely to be inadequate. Unidimensional interventions, whether psychotherapeutic, social service, or educational, designed to address individual problems in isolated domains, have not been effective in ameliorating the multidimensional problems of these families. A family-focused, child-centered frame of reference structures the intervention, supports the measurement of goal setting and goal attainment, and contains the scope of the work. Without such focus, the work can be diffused, less purposive, overgeneralized, and difficult to assess.

Many families experience problems in more than one domain. Some are able to use services and mobilize their inherent strengths on their own behalf; others are unable to recognize and draw on their own strengths or make use of other resources that might be helpful to them. Although we can map the combination of factors leading to either effective, adaptive functioning or chronic dysfunction with increased skill, we are not expert at making available preventive and interventive approaches that will promote healthy development and ameliorate the effects of negative familial and societal influences. Models such as FSS potentially offer to both family and service systems significant hope of breaking chronic patterns of maladaptive family interactions and coping.

The development of a therapeutic alliance also has proven to be a central element in effective intervention with multiproblem families. The relationship between the parents and the clinical team, similar in concept to the psychoanalytic use of the auxiliary ego, lends support to the parent as he or she moves toward a higher level of functioning ([Lewis and Stark, 1966](#)). The willingness to move the site of clinical treatment from more traditional clinic and examining room settings of to the family's home has been found to reduce resistance to engagement and reduce treatment disruptions.

Working with multiproblem families requires new, flexible approaches to treatment and reallocation of clinical resources. Implementation of these approaches has important implications for the training and practice of psychiatrists and other mental health professionals. The FSS program at the Yale Child Study Center has intervened successfully with these families since 1985. This success is reflected in the program's ability to prevent out-of-home placement of children in over 85% of the families served ([Adnopolz, 1994a](#)). Other program models appear to be equally effective ([Pecora et al., 1990](#)); however, [Dore \(1993\)](#) argues that extremely poor families may need more and continuing service. [Berry \(1992, 1993\)](#) has shown that these types of programs may not be as successful with chronic, long-term neglecting families. It is the authors' belief that although there still is much to be learned about these programs, investment in families previously believed to be unavailable for treatment offers our best hope of preserving and assisting families in crisis.

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# 123 THE RUNAWAY ADOLESCENT

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Approximately 1 million adolescents in the United States run away from home and live on the streets for at least part of each year. Perhaps 10% of all teenagers run sometime ([Edelback, 1980](#)). Many of these youths suffer significant and long-lasting physical and psychological damage; yet receive little or no care from any public or private agency ([Robertson, 1988](#)).

Our ability to anticipate and prevent running, and to remedy the effects of this experience, has been limited. In part this reflects a scarcity of careful empirical studies. However, it also reflects the impact of the runaway experience: Life on the street is so traumatic that when we study runaways it becomes difficult to distinguish which clinical features were antecedent and which were consequent to the runaway ([Justice and Duncan, 1976](#)). In spite of this, there is a growing appreciation of who these children are, what motivates them, what interventions will help, and, to a lesser degree, what the outcome of running away during adolescence is likely to be.

## PREVALENCE

Runaway teenagers come from all social groups and geographical regions but most are urban youths from lower socioeconomic status (SES) levels. Fewer than half live with both parents, and those who do are likely to come from very large homes ([Johnson and Peck, 1978](#)). More than one-quarter comes from single-parent homes, whereas another quarter comes from foster or group homes. Although the statistics are in dispute, girls predominate by a slim margin in most categories of runaways ([Garbarino et al., 1985](#)). Ninety percent of youths run fewer than 50 miles from home. Seventy percent of runaway episodes are brief (lasting 1 to 6 days), and follow a triggering incident (usually an argument with the parents); the child's focus remains on the home. However, as many as 15% of runaway adolescents are absent from home for many weeks or months: They develop an alternative identity that is streetwise and independent of their families. These youths may never return home. An additional group of runaway adolescents come and go from home numerous times over their teenage years; a dozen or more runaway episodes are not unusual. This group is more likely to come from an abusive environment and have their instability complicated by drug and alcohol abuse.

Such a variable pattern of running away causes the figure of 1 million runaway adolescents in the United States each year to be very approximate. Definition is crucial. Fewer than 400,000 youths are absent from home for months or years. On the other hand, many adolescents who leave home briefly (usually to the house of a friend or relative) are never reported. To include them might swell the number of runaways into the millions. Also, those children who run numerous times further confuse the statistics. Perhaps the key point is that running away is a substantial problem badly in need of interventions, particularly considering the negative consequences of even a brief period of time spent on the streets.

## RUNAWAY TYPOLOGY

Several detailed taxonomies are available that, with mixed success, attempt to categorize the complex phenomena of adolescent runaway ([Adams et al., 1985](#); [Dunford and Brennan, 1976](#); [Homer, 1973](#); [Jones, 1988](#); [Miller et al., 1990](#); [Roberts, 1982a](#)). Each incorporates one or more of the following organizing principles: the putative cause of running away, family dynamics, individual dynamics and/or psychopathology, sociocultural variables, age of the adolescent, and length of the runaway period.

The recent use of "runaway" as a diagnosis has been confused. Although DSM-II included the category of runaway reaction of childhood or adolescence, this was subsumed within conduct disorder, undersocialized nonaggressive type, in DSM-III. This latter diagnosis then disappeared completely in DSM-III-R and remains absent from DSM-IV, except as one of many possible criteria for conduct disorder. Runaway adolescents currently are to receive diagnoses for any specific conditions from which they suffer but not for their runaway behavior itself. At the very least, in most cases, this requires the "V" code of Parent-Child Relational Problem.

Although no single classification scheme has emerged as ideal for all purposes ([Hartman et al., 1987](#)), a typology based on the youth's reasons for running may be the most clinically useful, particularly when enriched by correlations with other important variables. Such a scheme must have at least two major categories: adolescents who "run from" home (runaway) and adolescents who are "pushed from" home (throwaway). It is essential, for clinical purposes, to know whether a child has chosen to leave home voluntarily or has been forced to leave by abusing or rejecting parents, even though this distinction frequently blurs in individual cases.

### Runaway Youth

Most adolescents on the street have run from home rather than been driven out ([Sharlin and Mor-Barak, 1992](#)). Moreover, most of these youths have come from turbulent homes with fractured family relations and are actively escaping a situation perceived as intolerable ([Council on Scientific Affairs, 1993](#)). A smaller percentage are adventure-seeking teenagers who are actually "running to" the exciting life on the streets. In some cases there is intergenerational transmission of running away, as sequential generations of children from one family "hit the streets" ([Plass and Hotaling, 1995](#)). Although both groups deserve clinical attention, the former may be more in need of, and responsive to, intervention.

### "RUNNING FROM" YOUTH

Many different situational and personal characteristics can predispose to runaway. Moreover, what a youth judges to be an acceptable reason for running differs with the culture and expectations of the youth's immediate peer group.

The most common reason for running away is a difficult home situation. A few adolescents are in homes that are truly intolerable by any standard; to escape from them may be both rational and healthy. These youths may be fleeing chronic physical or sexual abuse ([Famularo et al., 1990](#); [Farber et al., 1984](#)), often at the hands of antisocial, alcoholic, or drug-abusing parents. They may suffer equally severe psychological abuse by rejecting, punitive, or chronically neglectful parents. A few homes are so disorganized that the children are essentially without a history; they watch a parade of adults pass through their lives and do not know in some cases even who their natural parents are. Such families are frequently well known to community agencies and the children are often removed before they have a chance to run. Occasionally, however, the children must escape by their own means and thus end on the streets as runaways. They may find more warmth and caring on the street than they ever encountered at home, and if they become part of a "substitute family," their run may become permanent. In recent years, the substitute family willing and eager to adopt such disaffiliated youth has been one of the antisocial gangs that grace the midsize and major cities in the United States. In fact, the majority of gang members seem to have come from such dysfunctional families.

More commonly, the homes of runaways are characterized by a lesser degree of turmoil and by verbal abuse and poor communication. Typically the children and parents battle over one or more issues that neither seems able or willing to resolve. Parents identify problems with the teenager's behavior that range from the mundane (dinner table deportment, jewelry selection, hair style) through the expected (school performance, dating habits, curfew) to the profound (drug and alcohol abuse, illegal activities). The adolescents, on the other hand, feel themselves unheard, controlled and powerless, and without respect. Common to all these settings,

however, is reliance by both parties on the child's leaving home to resolve interpersonal conflicts. Girls predominate, perhaps because parents are more controlling and demanding of them. Family ties are usually intact, although loose, and reconciliation typically remains possible.

Alternatively, a crisis in the home can precipitate a runaway. Divorce, infidelity, financial disaster, or tension among other family members can cause an adolescent to flee. This may occur if the youth senses a loss of support when most family resources are directed elsewhere, but it also may represent the adolescent's need to call attention to the family's distress in an effort to get help. With otherwise stable parent-child relations, such running away is usually brief but may recur if the crisis persists or returns. However, if the child personally experiences the crisis (pregnancy, sudden trouble with the law, homosexuality revealed), the likelihood that he or she will run depends primarily on the reaction of the parents or expectation of an unacceptable reaction—and this depends in large part on the quality of the communications between parents and child.

Finally, some teenagers run from home as a manipulation. They may be seeking attention, trying to avoid punishment for a minor infraction, or trying to "let their parents know who is boss" (and have some fun while they are at it). This may occur with the adolescent whose parents have finally decided to crack down on increasingly unacceptable behavior. Such an episode is usually brief and done safely (e.g., run to a friend's house); it may be repeated if felt to be successful.

## **"RUNNING TO" YOUTH**

A significant minority of youths run away from home initially to exercise their independence, to escape boredom and seek excitement, and because of the allure of being "on the street." Home life for most of these adolescents is not supportive and as likely to be characterized by minimal control and parental indifference as by over-control and abuse, although some of these youths appear to have normal relationships with their parents and come from homes that seem relatively stable. Moreover, some report adequate (and even exceptional) school performance and uneventful peer relations. This diverse group of teenagers range from naive youngsters who have been inordinately influenced by pressure from their peers (and who often quickly return home after a brief adventure) to frankly antisocial adolescents for whom life on the streets differs little from the life they led before leaving home.

### **Throwaway Youth**

Approximately one-quarter of runaway children are actually throwaways (abandoned, castaways, homeless, pushouts) ([Gullotta, 1978](#)). These difficult, traumatized adolescents have either been encouraged to leave or been forcibly ejected from their homes. They are slightly more likely to be girls, to be older than more typical runaways, and to display high levels of conduct disorder, school failure, and drug and alcohol use. Typically their homes are very disturbed and marked by abuse, violence, and neglect. However, infrequently the homes are relatively stable, but the child has been scapegoated: identified as the "bad seed," whose removal from the home will solve the family's problems. In either case, relations between parents and child are chronically impaired, and neither party sees hope. Efforts to repair the family are usually unsuccessful.

## **CLINICAL DESCRIPTION**

Although several efforts to develop a psychological profile of adolescent runaways have been made ([Adams and Munro, 1979](#); [Jenkins, 1971](#); [Pietropinto, 1985](#); [Roberts, 1982a](#)), no single description can encompass the diversity of runaway youths. However, those large groups of runaway/throwaway youths who come from abusive and emotionally impoverished homes share some common psychological features.

Most studies find the majority of runaways to be demoralized and to have a poor sense of self. They are insecure and frightened in spite of a spirit of bravado and defiance. They frequently see themselves as inadequate, not only academically and occupationally, but also socially and interpersonally. This often reflects reality because many of these youngsters have a long history of failures: Almost all have been poor students or had significant school difficulties, and many have received limited occupational training. They typically are uneasy around and avoid adults. They are likely to be ostracized as "losers" by their more successful peers and are most comfortable with an equally impaired peer group. Compared to their peers, they typically choose maladaptive ways of coping with stress, including running away ([Roberts, 1982b](#)). Finally, in spite of the apparent independence required to run away, many such youth are passive and dependent when given the chance ([Benalcazar, 1982](#)).

Coincident with this self-image of incompetence is a sense that they have little impact on their environment. They frequently feel powerless to influence their parents, and this also may have a basis in reality because many of their parents are controlling and abusive or, alternatively, neglectful and disinterested. Nor do they feel that other adults in their environment, such as schoolteachers, respect them. As a result, leaving home becomes in part an effort to establish a sphere of self-esteem.

As might be expected, dysthymia and depression occur with increased frequency ([Feitel et al., 1992](#)). Estimates of depression among this population range from 20% to 80% ([Hersch, 1988](#)). Suicide attempts may occur in over one-third of this population ([Rotheram-Borus, 1993](#)). A key contributing factor to this pattern may well be the high incidence of previous and concurrent sexual abuse among runaways of both sexes ([McCormack et al., 1986](#)). As many as two-thirds or more of runaway females and one-third or more of runaway males ([Janus et al., 1987](#)) have been abused. Such experience is known to bring in its wake significant depression but also is associated with symptoms such as anxiety, dissociative episodes, sexual dysfunction and sexual acting out ([Seng, 1989](#)), substance abuse, conduct disorder, fear of adult men (by both males and females), and difficulty with close interpersonal relationships. These symptoms are frequently observed among runaways; past sexual exploitation may have contributed to the current clinical presentation of such a youngster.

Not only do they experience broad-based failure, but also they actively adopt, or are forced to adopt, self-destructive behaviors likely to worsen that course ([Greenblatt and Robertson, 1993](#)). They frequently abuse alcohol and a variety of drugs even before they run from home, further compounding their problems. In fact, substance abuse may occasionally dominate the clinical picture, leading to daily disruptions and generalized failures and resulting in expulsion from the home. Chronic truancy and status offenses are also common; the typical runaway is more likely to be a petty rather than a serious offender. The increase in conduct disorder among runaways, although real, may partly reflect the runaway experience itself. Adolescents for whom the fundamental problem is conduct disorder are more likely to have run at an earlier age, stay away longer, and get into more serious trouble.

Although by far the most common psychiatric conditions to be found among runaways are depression, substance abuse, and conduct disorders, other psychopathology also occurs with increased frequency. Some youths are markedly anxious and deserve an anxiety disorder diagnosis. These adolescents also seem to be more likely to suffer from attention deficit hyperactivity disorder and learning disabilities, yet good evidence is currently not available. Certainly, though, studies have found the runaway adolescent to be restless, demanding, and impulsive ([Goldberg, 1972](#)). Deeply disturbed or psychotic children living on their own in the community are present with a modestly increased frequency. That they are not even more common probably reflects both their likelihood of being institutionalized once recognized and the difficulty such youngsters with significant psychological disorganization have surviving on the street.

Although the majority of runaways can be contained within the preceding description, a sizable subpopulation of adolescents better fits another general description entirely. These are the youths who envision running as an adventure (the "running to" group) and have a value system consonant with life on the streets. They frequently are excitement-seeking, moderately antisocial adolescents who are likely to come and go from home as the mood suits them or to stay on the street for months or years if they find a niche of their own there. These are the teenagers most likely to exploit those around them and slip into a more permanent antisocial lifestyle.

Finally, some runaway adolescents are indistinguishable from normal. They have a generally good relationship with their parents and maintain close ties throughout one or more runaway episodes. They have strong ties to their peers (who may be confused by, or participants in, the running behavior) and perform well in school. Psychopathology is not evident, and the behavior produces few psychological repercussions because the running episodes are brief, infrequent, and safely performed.

### **Parental Patterns**

To understand the runaway teenager, it is necessary to recognize that the parents' role in generating disturbed family interpersonal relations is usually at least as important as their child's. A poor parent-child relationship is the most consistent finding in the home of a runaway. Although some homes and parents are psychologically unremarkable, often the parents of the more persistent runaway adolescents have features in common.

Most of these parents are exceptionally critical of and dissatisfied with their children. Parent-child communications typically are negative; the parents provide little in the way of compliments or support (and the child reciprocates in kind). They establish a standard for behavior (often poorly defined) that the teenager often finds



unattainable but then provides little help or counseling when the child fails. Their frustration and demands typically take the form of verbal abuse as well as occasional shoves or blows.

The communication of perpetual dissatisfaction with their child is usually accompanied by inconstant discipline. They are quite capable of being restrictive at times, particularly around specific issues such as dating and curfew, and lax at other times. The teenager interprets the restriction as parental rigidity and control and then rebels (or runs away). However, these efforts by the parents to make demands on their children typically are overlaid on a more fundamental pattern of disinterest and lack of supervision. Runaway adolescents are as likely to report that their parents have no time for or interest in them, as they are to report that their parents are too demanding. Many of these parents are absorbed in their own problems and desires and give little time or energy to their children (latch key children). They are not there to help with schoolwork, intercede with teachers, or discuss problems. The children rightfully see themselves as incidental to the parents and are less willing to adapt themselves to their parents' wishes. There is little real comfort to be had in these families; consequently, the child looks elsewhere.

A significant minority of these parents displays real psychopathology as well. Antisocial personality disorder and drug and alcohol abuse are common. Parental disorganization and instability may also result from depression, psychosis, or low intelligence. In any case, the parents may need intervention separate from work with the family.

## THE CONSEQUENCES OF RUNNING AWAY

Most teenagers leave home with a plan in mind. Unless their departure is precipitous, they usually are running to a friend's house, a distant relative, or a known location or town. Alternatively, they may be "hitting the road," but usually with one or more friends with whom they have planned their runaway. Girls frequently run to or with their boyfriends, who often provide them with money and a place to stay. Episodes of running often end when the money runs out. Many teens are quickly returned home by acquaintances or the police, or they end up in shelters as a way station to home or alternative residential placements. These well-contained runaway episodes generally do little long-lasting damage, occasionally provide the teenager with enhanced self-esteem, and may be beneficial by mobilizing the family to get help. (Unfortunately, many first-time runaway youngsters return to angry parents who decide to "crack down," producing a worsening spiral.)

Prolonged runaway attempts are less benign ([Young et al., 1987](#)). Few adolescents survive on the street for lengthy periods without trauma; the street is a great leveler. Unless friends or family provides for a youth, the dominant issue of living on the street quickly becomes survival. One estimate suggests that a youth on the street without resources will have to turn to crime within 1 month to survive ([Hersch, 1988](#)). This means turning to theft and street hustles, prostitution, pornography, drug selling, and the exploitation of others. Among chronic street adolescents, three-quarters have been involved in prostitution, three-quarters with selling drugs, and three-quarters have been incarcerated. The longer adolescents are on the street, the more likely they are to find their way into trouble and the courts.

More important, however, is the sheer physical danger of being on the streets. Violence (assault, battery, rape) and its associated injuries are the most serious problems a youth faces. Most street youths will be beaten or raped at some point. Some will become pregnant; some will receive permanent injury; some will be killed. In addition, their physical health will deteriorate. Skin infections, parasitic infections, respiratory and gastrointestinal diseases, dental problems, nutritional deficiencies, and medical conditions associated with drug abuse are common ([Council on Scientific Affairs, 1989](#)). They risk exposure to the elements, exhaustion, and musculoskeletal problems because of their hand-to-mouth existence. To make matters worse, hunger, fatigue, and drug-related cognitive impairments all compromise the teenager's ability to cope with routine stresses, let alone with the major challenges they face on the street.

All of these physical disabilities occur during a time when they have limited access to health care and limited ability to pay for it. Although some care can be provided to minors without parental consent in some states for certain conditions (e.g., pregnancy, venereal disease), most nonemergent care is unavailable unless the individual is an emancipated minor. Nevertheless, even if emancipated, they have the same difficulty in obtaining health care, as does anyone who is indigent.

Besides violence and drug abuse, the other major risk to the physical health of the runaway is sexually transmitted disease ([Rotheram-Borus et al., 1992a](#)). Runaway youths are perhaps the most sexually active of all teenagers, and their rates of syphilis and gonorrhea are the highest of any group ([Rotheram-Borus et al., 1992b](#); [Zylke, 1989](#)). This sexual activity, coupled with high rates of drug abuse, places runaways at high risk of exposure to acquired immunodeficiency syndrome (AIDS). Although an epidemic of AIDS among runaway teenagers is unlikely owing to the long latency of the disease, there is an epidemic of exposure to AIDS in progress ([Hersch, 1988](#)). In some inner city populations of runaway adolescents, the human immunodeficiency virus (HIV) seropositive rate may approach 10% with much of the exposure occurring through heterosexual contacts ([Hein, 1987, 1989](#)).

Prostitution (male and female) and pregnancy present other risks to runaways ([Brown, 1979](#)). As many as 900,000 adolescents are involved in prostitution each year; many, but not all of them, are runaways. Adolescent male prostitution is common, but female activity predominates. The average age of first intercourse for these girls is approximately 12 to 13, whereas the average age of beginning prostitution is 14 to 15. Prior traumatic sexual experiences are the norm: Many of these adolescent girls have been molested; half have been raped. Although some hard-core runaways prostitute for the money and glamour, most are trying to survive. With this group of runaways in particular, street drug use is extensive and other physical risks excessive ([Yates et al., 1991a](#)).

Teenagers pay a severe psychological price for life on the street; those who successfully adapt are usually the worse for it. Many of these youths are intensely lonely, fearful, and unhappy ([Kufeldt and Nimmo, 1987](#)). The longer they are on the street, the greater the number of demoralizing experiences they will have had. In fact, life on the streets itself can produce all of the symptoms usually associated with runaway adolescents. Running away often has forced them into a life they did not expect and do not want; however, they frequently have few options. Only 50% of chronic runaway teenagers have any possibility of returning home. As time goes by the consequences of running become long lasting; they have effectively excluded themselves from an education and a career. Determination to succeed flags, and many of these youths become permanent members of the underclass.

## TREATMENT

Runaway adolescents can be both difficult to treat as well as desperately in need of treatment. Perhaps the most formidable obstacle to treating them is that they rarely seek help ([Brennan, 1978](#)). Even those in medical need will frequently suffer rather than look for aid from adults, agencies, or shelters. In addition, they are frightened, defensive, hypersensitive, embittered, rebellious, and mobile—they do not stay in treatment long; they run away. Keeping them engaged requires tact, a nonjudgmental attitude, the development of a personal relationship (with someone), and, most frequently, patience. They are present oriented; because of the intensity of their feelings, they tolerate review of their past poorly. However, it is possible to have considerable impact if the child (and his or her family) is engaged skillfully at the right moment.

Most commonly runaways are reported missing by their parents and are brought to clinics or inpatient facilities by the parents, police, a relative, or a mental health worker. Occasionally, their absence from the home goes unreported, and their status only comes to attention when they appear at a shelter or on the street. These "forgotten" adolescents are typically the most impaired. The Juvenile Justice and Delinquency Prevention Act of 1974 prevents the long-term incarceration of these youths, yet their treatment in the eyes of the law reflects state statute: Some states consider runaways to be juvenile delinquents and send them through the courts, whereas other states consider them status offenders and send them home or to treatment facilities.

Once identified, the first order of business is to provide a safe environment. This usually involves a rapid assessment of the home and perhaps a return there, but may mean finding a shelter, group home, or other social service facility as an alternate placement until an appropriate long-term setting can be found. It helps to have an assessment unit as part of the clinic or shelter and to have ready access to medical care, because these youths may be in poor health and need prompt attention. Although their skittishness and lack of cooperation may interrupt treatment, immediate provision of welcome medical care can entice them into a treatment-receptive stance. Although some professionals worry about it, providing medical and psychotherapeutic aid to runaway adolescents in need without parental consent is generally acceptable in the eyes of the law ([Croxtton, 1988](#); [English, 1991](#)).

Appropriate treatment usually depends on the type of runaway and the length of time the teenager has been gone. Usually, however, the earlier the intervention, the better, although simply returning the child home rarely works. An effort at family therapy is essential for the "running from" youth ([Ostensen, 1981](#)). Such therapy is often effective, particularly if the running episode has been brief and if the family is basically stable but in acute crisis. More severely disabled families may respond to intervention; the focus needs to be on family stress rather than on the child's problems, education in parenting skills, and interpersonal skills training for all members. The parents need to learn to listen, show affection, develop consistency and parenting effectiveness, and care. They need to be alerted to and correct interpersonal distortions when they occur, and avoid scapegoating the child in service of their own marital disputes. They often need to develop more realistic expectations for their child, and this typically requires a careful assessment of the child's capabilities.

Like the teenagers, the parents are also often resistant to treatment. They may consider their problems so serious that a bad result is inevitable or too confidential to talk about. They may be defensive or demoralized and without confidence. It is essential to engage them in therapy before they become hardened to their youngster's problems or disenchanted by any treatment efforts. Some families will not follow through in spite of the best efforts.

In general, the more chronic and complex the family's problems, the more broad-based the interventions needed. If the child has been out of the home for a long period, however, the family ties may be so weakened that returning home is unrealistic and formal family therapy of little value. Family therapy is generally not useful for chronic street kids.

"Running to" adolescents, particularly if they are moderately antisocial and enjoying their time away from home, are resistant to treatment. Often the most that can be accomplished is to help them find acceptable, legal, and even constructive outlets for their energies and enthusiasms. Throwaway adolescents typically are the most traumatized of all and need support and stable placement, if they will accept such help. They also occasionally respond to intensive individual psychotherapy, but owing to their sensitivity and lack of trust, group therapy with peers is often a necessary and acceptable alternative.

Runaway adolescents are often difficult to engage in individual therapy, but when a therapeutic alliance develops, such treatment can be very effective. Many of these youngsters want to feel better and do better. Many want to return to school and be reintegrated into their community, only the hurdles seem overwhelming to them. The therapist must recognize their heightened defensiveness, anxiety, and impulsivity—it is impossible to be too gentle and supportive to many of these youths. If they are abusing drugs, every effort must be made to detoxify them. Issues around sexual abuse need to be addressed; nonetheless, the patient may respond with withdrawal, denial, cockiness, anger, or verbal abuse if the pace is too rapid. At least initially, the child needs to feel in control of the interviews. Many (most) of these youths can benefit from training in specific coping skills, well received often through role-playing exercises with peers. Of course, identifying and treating specific psychiatric disorders such as depression, substance abuse, and psychosis is essential, as is the recognition of specific deficiencies such as learning disabilities. Perhaps even more than in most psychotherapy, however, success depends on developing a personal relationship with the youngster that will weather the inevitable storms to follow.

Public and private resources of all types are inadequate for runaway adolescents. Some agencies avoid dealing with these teenagers in part because they can be difficult (Kufeldt, 1991). Comprehensive services with a continuum of care are needed but unavailable in most communities. This continuum should include not only medical care and individual psychotherapy but also runaway shelters for brief but safe stays, small group homes for longer stays, hostels and supervised but independent living apartments for selected older adolescents, carefully chosen foster homes, and secure facilities for very disturbed youths. However, some youths have trouble in any structured setting and may need to be treated for a while on the street (Kufeldt et al., 1992; Smart, 1991; Yates et al., 1991b). Emergency hotlines are available and advertised in some locations, and there are at least three runaway hotlines that serve a national or regional audience: National Runaway Switchboard (1-800-621-4000 [HIT-HOME]), Covenant House (New York) hotline (1-800-999-9999 ["nine-line"]), and National Center for Missing and Exploited Children (1-800-843-5678).

## OUTCOME

Careful outcome studies are rare (Olson, 1980; Robins, 1958), based on few patients, and dated. Only limited conclusions can be drawn. Runaways, as a group, are failures in later life. They never make up for lost schooling or missed occupational opportunities. They tend to slip to the lowest SES group and frequently become dependent on others (ironically, often their families) for support. Adult interpersonal relations are typically poor, and their sense of self remains low. The more significant their runaway activities as an adolescent (i.e., chronic or repeated versus brief and nonrepetitive), the more likely they are to fail as an adult. On the other hand, teenagers with brief, one-time runaway episodes may be indistinguishable from a normal group later in life.

There is insufficient information at this time to either go beyond these tentative conclusions or explain them. It is not clear, for example, how much of runaway behavior or outcome is owing to innate temperament and abilities and how much to parent-child conflicts and experience. It is clear, however, that the runaway adolescent is an individual deserving clinical and research attention.

## RESEARCH DIRECTIONS

The research base with this population is inadequate. Even fundamental clinical questions go unanswered: How diversified or uniform are the personality characteristics of these children? Is drug use etiologic or secondary? What are the features that determine a priori whether a child will engage in a brief or a sustained runaway? What personal characteristics allow a child to succeed as a runaway? Which interventions are effective, if any, and with which populations (Podschun, 1993)? Numerous interwoven factors have made this a difficult group to study; they need to be specifically identified and teased apart.

Any research must be broad-based, or at least must recognize its limitations. Results differ depending on the cultural and socioeconomic group studied, location of the study, and time of the study. Inner city runaways differ from suburban runaways; summer runaways may differ from those gone year-round. Outcome may vary with family structure, drug use, degree of criminality, and interventions available. All results may vary with the generation studied; studies during the restless 1960s do not necessarily apply today. Serious hope of influencing the runaway problem awaits clarification of many of these basic issues and questions.

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# 124 HOME-BASED CHILD AND FAMILY TREATMENT

Jean Adnopo, M.P.H.

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The functioning of any individual child is conditioned by the interaction between the child's innate, structural capacities, his physiology, cognition, and developmental status, and the ecologic systems in which he functions. The family system is the earliest and arguably the most significant influence affecting the child; its functional capacity is central to the child's ability to adapt to and cope with the vicissitudes of his or her life. Although family-focused interventions have been used to augment individual mental health treatment for disturbed children for some time, it was not until the final decades of the 20th century that the child's home and family became an essential focus of interventions designed to prevent negative parent and child behaviors in at-risk populations, or to ameliorate problems of serious emotional disturbance, inadequate or abusive parenting, or delinquent behaviors that placed children at risk for placement outside of their own homes ( [Lindblad-Goldberg et al., 1998](#)). The realization that many families in need of service were unable or unwilling to access traditional treatment programs, which, in turn, placed children at even greater risk for removal to more restrictive environments or prevented them from receiving assistance, made the home an important alternative service delivery site. Although scientifically derived evidence of their efficacy and effectiveness has been slow to accumulate, home-based services based on principles first articulated in by the Child and Adolescent Service System Program (CASSP) and further codified in Public Law 94-142, The Education for All Handicapped Children Act, and Public Law 96-272, The Family Preservation and Support Act ( [Solnit et al., 1997](#)), have proliferated across America. Home visiting does not represent a single treatment modality; rather, it describes a systematic mechanism for the delivery of preventive or interventive services ( [Olds et al., 2000](#)), even though the rhetoric of home-based services is similar across a broad range of programs.

This chapter describes the events leading to the current interest of health, mental health, juvenile justice, and child welfare systems in supporting the delivery of services in the home; reviews some of the home-based programs that have been reported in the literature, ranging from preventive, educational, and support models to intensive, focused interventions with delinquent adolescents and dysfunctional families; and presents an example of a program for children and adolescents with serious emotional disturbances.

## THE CHILD AND THE FAMILY

Although foster care or other residential treatment facilities are likely to be available in most communities to provide 24-hour care for those children for whom it is required, few resources are able to replace the family as the most effective, long-term institution for raising children. Adequately functioning families provide safe and secure environments in which the normal developmental processes of childhood and adolescence can unfold predictably. In such environments, children are likely to feel nurtured, cherished, protected, and secure. Consistent relationships with their adult caregivers support the positive use of curiosity and imagination and empower children to explore their world and move toward eventual independence and self-sufficiency. [Solnit \(1976\)](#) has described the family as "the bridge from the past to the future" that provides continuity and a sense of being rooted in time, place, history, and culture. Families socialize children, transmit intergenerational values and beliefs, and provide a place of respite from the challenges of the outside world.

Although most children in the United States live in families able to offer adequate care, society remains challenged by the substantial numbers of families unable to provide stable, consistent, and caring relationships for their offspring. As the United States moves into the 21st century, the country is faced by a crisis in the care of its children, much of which can be associated with inconsistent, inadequate family functioning. Harrison and others found that risk and protective factors associated with child and adolescent problems were determined primarily by parenting and family functioning ( [Harrison et al., 1999](#); [Najman, 1997](#)). Harrison found that children in unstable families with high levels of partner conflict demonstrate higher rates of both internalizing and externalizing behaviors; negative family functioning also has been highly correlated with adolescent substance abuse ( [Harrison et al., 1999](#); [Rhodes and Jason, 1990](#)).

Although the federal government has made serious attempts to assist states to provide services and community supports to preserve families for children ( [Solnit et al., 1997](#)), foster and institutional placements remain overused treatment options for troubled children and families in communities where there are few less restrictive alternatives. In 1998, approximately 500,000 children in the United States were in out-of-home, nonrelative placements ( [Nelson, 1996](#)); estimates of children in the United States living with relatives range from 4.3 million children living with mothers in the homes of other relatives ( [U.S. Bureau of the Census, 1992](#)) to an estimated one-half million children in kinship care arrangements ( [Scannapieco and Jackson, 1996](#)). Even though the effects of multiple caregiving disruptions on child development have been well documented ( [Goldstein et al., 1996](#); [Rosenfeld et al., 1998](#); [Solnit, 1980](#)), the incidence of out-of-home placements has more than doubled since the mid-1980s. This marked change in removals and placements may be associated with an increase in both the severity of symptomatology and the numbers of children being referred for intervention and child protection ( [Child Welfare League of America, 1994](#)). However, moving children in and out of placement in an attempt to address their needs for treatment or safety and protection may, in fact, foster even more severe disturbances by heightening their anxieties and fixing their self-destructive, negative internal representations. Children who are denied consistent, long-term relationships may be compromised developmentally and intellectually and be unable to form positive, intimate attachments as adults ( [Goldstein et al., 1996](#)). The zeal of society to protect and treat children by removing them from their families and placing them in other caregiving arrangements, including institutions and foster homes, in reality may be putting them in harm's way.

## ROOTS OF OUT-OF-HOME CHILD CARE

Community responsibility for the care of inadequately parented children and identification of alternative caregivers has been recognized for centuries. In the Judeo-Christian tradition, childless foster caregivers, extended family members as well as nonrelatives, were among the first adults willing to take on the care of children who were orphaned, abandoned, or beyond the control of their parents ( [Youcha, 1995](#)). As opportunities for work outside of the home developed, uncared-for children were enlisted as apprentices to tradesmen with whose families they lived, providing cheap labor in return for basic necessities. Historically, attempts to provide care for the ever-increasing numbers of destitute or insufficiently cared-for children were guided by altruism and religiosity, but children did not always fare well. Reports of exploitation, cruel treatment, and substandard living conditions challenged the widespread assumption that the family was the ideal place in which to raise children. For a time, orphanages replaced the family as the option of choice; institutional care was thought to be safer, more reliable, and more easily regulated. However, by the end of the 19th century it became evident that institutions were not effective as parental substitutes. Children in institutions evidenced signs of increased physical illness, loneliness, and apathy to their surroundings, reactions attributed by many to the loss of consistent adult relationships and their forced separation from extended families and familiar environments ( [Adnopo, 1998](#)).

One of the best-known interventions for the street children of the rapidly growing urban centers were the Orphan Trains conceived by the Minister Charles Loring Brace in the second half of the 19th century ( [Youcha, 1995](#)). Between 1854 and 1929, approximately 150,000 children were transported by train from the burgeoning cities of the East Coast to the Midwest ( [Rosenfeld et al., 1998](#)). Brace believed earnestly that by placing children with hard-working farm families he would rescue them from their poor, urban conditions and provide them with a more healthy lifestyle. In return, they would provide an important source of farm labor. However, the yearning to maintain their relationships with their families of origin made it difficult for some children to adjust to life thousands of miles away from parents and



siblings. As a consequence, many children made their way back to the communities from which they had come.

The antidote to the failure of the widely publicized Orphan Train movement was the idealization of the family. In 1909, a White House Conference on the Care of Dependent Children ended with the strong endorsement of foster families as the most promising alternative for children whose parents were unable to care for them. The conference did little to settle the controversy. The question of who should care for children when parents appear unable to do so has continued to be a subject of intense debate to the present time, even though for more than 30 years federal child welfare policy has been designed to reduce the numbers of children placed outside of their homes by encouraging states to provide a range of community-based services to maintain children with their families.

In the 1970s, the adoption of state and national child abuse and neglect statutes codified child protection policies and practices concerning the removal of abused, neglected, and at-risk children in all 50 states. The passage of the Adoption Assistance and Child Welfare Act of 1980 (Public Law 96-272) ([Hegar, 1993](#)) was designed to assist families to improve their parenting capacity. Although the act made permanence a central goal for all children entering the child welfare system ([Wells and Tracy, 1996](#)), policies that address the most appropriate means of ensuring permanency for vulnerable children continue to evolve. The Adoption Assistance and Safe Families Act of 1997 provided financial incentives to states to discourage foster care drift and ensure stability, safety, and permanence for high-risk children who often were without long-term plans. During the 1990s, several pronounced and disturbing trends emerged concerning children involved in the foster care system that raised questions about its efficacy as an intervention and treatment option for high-risk children. Over this period, the age of children in out-of-home care has decreased; one-fourth of all children entering the foster care system in the five largest states were younger than 6 years of age ([Berrick et al., 1997](#)). In addition, children entering foster care were more likely to be victims of trauma and to have chronic medical conditions and mental health disorders (Brereton, 2001, unpublished Master's thesis), and they often failed to receive the health care necessary to mitigate these problems while they were in care. Children placed in foster care also were likely to experience more than one placement; for example, in Connecticut, the average number of placements for every child who enters the system is 3.5 (Martin S, personal communication, 1999). Child Welfare data released in 1998 found that 66% of the children in the child welfare system who were placed outside of their homes were eventually reunited with a family member; however, their families may not have received any services while they were in placement or been provided any assistance in preparing for the child's return (Adnopoz and Ezepchick). Disturbingly, the care that was available while the child was out of the home was likely to be fragmented and episodic. As children moved from place to place, their social and medical histories often were left behind, leaving the new caregivers unprepared, uninformed, and at a loss to cope with behavioral or medical problems as they arose.

At the same time that children have been placed out of their homes, only to be returned to an unchanged home environment, many states have been faced with both a shortage of institutional beds and an inadequate supply of licensed foster homes into which children could be placed. The combined weight of these factors increased the interest in and support of child-centered, family-focused interventions as important alternatives for children for whom out-of-home placement was seriously considered. Traditional outpatient mental health resources, such as office- or clinic-based treatment, did not prove to be a good fit for work with chaotic, distrustful, multiproblem families in which children and parents have multiple, competing problems, including serious mental health disorders, many of which are reactive and cannot be relieved without concurrent changes in the environment.

With the support of advocates such as The Edna McConnell Clark Foundation, states were encouraged and funded to implement programs that could maintain children in their own homes and communities, promote permanency for the child, reduce the probability of placement outside of the home, and prevent the inevitable problems associated with foster care drift ([Nelson, 1996](#)). Homebuilders, a model-intensive, home-based intervention developed in Tacoma, Washington, was prized and endorsed by funders and policy makers because the program developers attempted to address the complexity of the interactions between the child, family, and environment and, as a result, demonstrated the ability to reduce placements. The belief that children who were maintained in their own homes would represent cost savings to taxpayers also supported the popularization of family preservation services.

## INTERVENTIONS THAT SERVE THE CHILD AND FAMILY IN THE HOME

Home-based services had their origins in the 19th century, when friendly visitors representing charitable organizations called on families to determine their needs and to work with them to increase their self-sufficiency. Many of these methods were adapted by social workers in the early years of the 20th century who recognized the advantages of entering and observing the family's environment at first hand and mobilizing natural, community-based networks ([Wells, 1995](#); [Woodford, 1999](#)). When clinicians enter the diagnostically rich, textured, and highly personal environment of the home, they are able to step directly into the family's own ecosystem. With their observations, they can piece together a tapestry of knowledge about the real-world experiences of the child and family that not only may facilitate treatment, but is likely to be unavailable to those working in traditional examining rooms (Adnopoz and Culler). The process of working in the home enhances the clinician's ability to understand and address the complexity of the dynamic reality that constitutes the everyday world of both child and parents.

Although the families in which children are at high risk for out-of-home care may be difficult to engage, distrustful of the traditional, clinic-based mental health system, and often unable to maintain regular appointment schedules, clinicians have found that they are more likely to become supportive of and consistently involved in their child's treatment and recovery when clinically informed and appropriate mental health services are delivered directly in the home and community (Adnopoz and Ezepchick, 2000; [Woodford, 1999](#)). Evidence supports in-home, family-focused intervention as a necessary component of comprehensive services developed for children and youth with problems of mental health, substance abuse, and delinquency ([Fraser et al., 1988](#); [Harrison et al., 1999](#); [Henggeler and Borduin, 1990](#); [Kaufman and Kaufman, 1992](#)). Many of the programs that have been studied offer sustained, relationship-based services that usually address three domains: parent–child, and child functioning ([Heinicke and Ponce, 1999](#)). Their success has enabled in-home intervention and treatment to emerge as an effective means of engaging difficult-to-reach families and preparing them to make better use of more traditional treatment modalities.

## IN-HOME PREVENTION AND EARLY INTERVENTION PROGRAMS

Provence ([Provence et al., 1983](#)), a pioneer in the use of home visitation as part of a comprehensive intervention for single, poor, inner-city mothers, was among the first researchers to find that mothers and children who received sustained services from a consistent group of providers through the child's first 30 months of life had positive, long-term outcomes in several domains compared with nonintervention control subjects. At the end of the intervention, children in the experimental group scored higher in language development; at the time of the 5-year follow-up, these children demonstrated higher school achievement and better school attendance, and were more task oriented. At the 5-year follow-up, mothers had fewer additional pregnancies, were more likely to be employed, had improved their socioeconomic status, and had made better use of community support resources. After 7½ years, mothers had completed more years of education, were more likely to be self-supporting, had more satisfying personal relationships, and had waited longer to have a second child. Mothers who received the intervention were more responsive to the needs of their children and reported a more pleasing relationship with them. ([Provence et al., 1983](#))

David Olds ([Olds et al., 1999a](#)), who began his work in Elmira, New York, continues to test his model of nurse-delivered in-home visitation for pregnant and parenting, single, poor, primiparous mothers with few social supports in multiple settings throughout the United States. Designed as a preventive public health intervention to affect the quality of parenting, prevent abuse, and improve maternal and infant health, Olds' initial randomized study in Elmira found that after 2 years of a structured, curriculum-driven home visitation program that included parent education, mothers had fewer preterm deliveries, smoked less, and had fewer kidney infections compared with community control subjects. Infants had higher birth weights and a 4.86-point increase in IQ at 3 and 4 years of age. The quality of the mother–child relationship was enhanced, and mothers provided more play material and used punishment less. In addition, the mothers in the Olds study were also able to make better use of their own partners as well as community supports. Children also were seen in the emergency department less frequently and had fewer accidents than the control group. These findings were sustained at the time of follow-up when the children were 15 years of age. Reduced incidences of child abuse and neglect also were found at 2 and 15 years of age. At the time of the 15-year follow-up, children in the experimental group were considerably less likely to have been arrested, use alcohol, smoke cigarettes, or have multiple sex partners ([Olds et al., 1999b](#)). When the children were 15 years of age, mothers in the intervention group were reported as less impaired by drug and alcohol since the birth of the index child ([Olds and Kitzman, 2000](#)). The initial study was replicated in Memphis, Tennessee with a primarily urban, African-American population, where many of the effects of the Elmira study were replicated, although the effect size was somewhat smaller. The number of health care encounters for injuries and injections was 23% lower than in the control group, and the number of hospital days required for serious injuries was significantly less. Olds reports that there were no differences in parents' reports of child behavior problems in either study. An economic analysis conducted by the Rand Corporation found that real economic benefits were accrued by the fourth year of life for low-income children; this finding did not hold for families of higher socioeconomic status and married women ([Olds and Kitzman, 1993](#)).

[Heinicke and Ponce \(1999\)](#), in a careful review of numerous early intervention studies, find considerable evidence to support the effectiveness of home visitors in improving maternal self-concept and satisfaction and enhancing the responsiveness of the mother to the needs of her infant ([Cicchetti and Toth, 1998](#)). One comprehensive program studied by [Lally et al. \(1988\)](#) used a home visitor, child development centers, and parent associations to address parent–child and partner relationships and child development. The intervention began in the prenatal period and continued until the child's fifth birthday. Children in the program scored higher on the Binet IQ at 36 months, although there were no cognitive or intellectual differences at 60 months. At a 10-year follow-up, program parents had a more positive capacity for relationships and more positive self-development. They reported feeling proud of their children's accomplishments and encouraged by their autonomy and

achievements. The girls in the program were more successful in school; boys and girls had lower rates and less severe forms of delinquency. Parents of premature infants in two separate studies by [Nurcome et al. \(1984\)](#) and [Minde et al. \(2001\)](#) received in-home and in-hospital services targeting parent–child interaction and mastery of parental tasks. They found that mothers demonstrated better relationships with their infants, were more responsive to them, and fostered the child's autonomy at 1-year follow-up.

The UCLA Family Development Project ([Heinicke et al., 1999](#)) demonstrated that a randomized home visiting relationship–based intervention for pregnant women in their third trimester who were classified as at high risk for inadequate parenting was able to effect experienced partner and family support by infant age 12 months compared with control subjects. In addition to home visiting, the experimental group participated in a weekly mother–infant group; the control subjects received regular pediatric follow-up. Children in the intervention group were more securely attached and more autonomous at 1 year and were encouraged by their mothers to be more task oriented. The intervention did not affect maternal depression or anxiety, although continued follow-up at year 2 may reveal statistically significant between-group differences. Heinicke and colleagues point out that the ability to achieve sustained effects with inadequately functioning families depends on the capacity of the intervention to address the adaptation needs of the parents ([Heinicke et al., 1999](#)). Although several programs cited by Heinicke and Ponce (1998) found that positive early cognitive differences between experimental and control groups tend to disappear by 36 months, other child differences remain over time. Studies of an intervention in Houston found that program boys had better peer relationships as preschoolers and were less impulsive, obstinate, and restless as elementary school students. An intervention for pregnant African-American teenagers that lasted until the infant's third birthday found that the children were in better health and were more socially adept and self-confident. Mothers used more appropriate discipline and means of control and in general were more responsive to the needs of their children ([Black et al., 1994](#)).

These studies demonstrate that sustained, relationship-based, in-home visitation can be effective in improving maternal competence and enhancing maternal capacity to enter into positive relationships, use partner and community supports, and attend to issues of self-development. In addition, preventive in-home services encourage more effective maternal responses to limit setting and the use of appropriate controls, as well as to the promotion of the child's autonomy, capacity for exploration, task orientation, and cognition. These studies also have demonstrated the centrality of the sustained relationship between the mother and the intervener in achieving the desired outcomes. The development of a trusting, accepting alliance provides the means through which behavioral changes can occur. The process variables associated with positive outcomes are the duration of the contact between mother and home visitor, the extent of the focus on issues of parenting, the mother's attitude, her willingness to work with the visitor, and her view of the visitor as helpful ([Korfmacher et al., 1998](#)).

## TARGETED IN-HOME INTERVENTIONS FOR HIGH-RISK CHILDREN

### Family Preservation

Family preservation and support services for families in which children are at high risk of out-of-home placement for reasons of abuse or neglect have been available for more than a decade. The Homebuilders program serves as a well replicated prototype, although variations on the model have proliferated across the United States ([Lindblad-Goldberg et al., 1998](#)). Family preservation programs are time limited (approximately 12 to 16 weeks), relationship-based, family-focused, child-centered, small-caseload, flexible services that are available to families 24 hours a day, 7 days a week. They are designed to prevent unnecessary out-of-home placements or promote family reunification by assisting parents to address their own needs and those of their children (see [Chapter 122](#)). Although most studies have shown that placement is prevented for 75% to 90% of the children receiving services, attempts to determine the effectiveness of family preservation programs have been limited by small effect sizes, the lack of randomized, controlled studies, and inadequate program standardization, all of which call into question the adequacy of both the programs and the research methodologies that have been used to evaluate them ([Burns et al., 2000](#)).

### Multisystemic Therapy

Since the early 1990s, family-focused, in-home services have been specifically tailored to meet the needs of children and adolescents with problems of mental health, delinquency, or substance abuse. Several randomized clinical trials have tested these models and found them to be effective in decreasing problem behaviors, improving family functioning, promoting the recovery of the index child, and reducing the need for more costly out-of-home placements, either in hospitals or residential programs. [Henggeler and Borduin \(1990\)](#) have developed a curriculum-driven, home-based therapeutic approach they call *multisystemic therapy* (MST), which has demonstrated its effectiveness with chronic juvenile offenders, adolescent sex offenders, and substance-abusing delinquents. MST directly addresses the interpersonal and systemic factors that are associated with adolescent antisocial behavior ([Henggeler and Borduin, 1990](#)) and considers the child's view of his or her world as well as the direct and persistent influence of family, peer, and school environments. Sessions are held frequently in the child's home and in the community; services are time limited and are designed to empower parents to understand and manage behavioral crises as they may arise after the intervention. The Missouri Delinquency Project examined the long-term effects of MST on the prevention of criminal activity in a sample of predominantly serious juvenile offenders by comparing MST with individual therapy, and demonstrated positive effects on perceived family relations, family interactions, parental symptomology, interfamilial conflict, and the youths' behavioral problems. The intervention also produced long-term changes in the youths' criminal behaviors. MST's focus on the child's ecology differentiates its approach from more traditional interventions with children in the juvenile justice system. Central to the success of Henggeler's programs has been an understanding of the familial and social contexts in which children function and by which they are strongly influenced, and a willingness to address the contextual issues in a structured curriculum. [Borduin et al. \(1995\)](#) suggest that improved family functioning, a result of in-home and community services, was the primary influence on the reduction of criminal behavior in the Missouri study.

[Henggeler and colleagues \(1999\)](#) have reported on a study to determine whether MST could be modified effectively for use with children presenting with psychiatric emergencies and used as an alternative to psychiatric hospitalization. Based on the hypothesis that the child's family plays a central role in predicting and preventing the need for hospitalization, and that behaviors are socially and ecologically influenced, Henggeler and colleagues designed and tested an intervention for children 10 to 17 years of age who were approved for emergency psychiatric hospitalization at the Medical University of South Carolina. These youths were randomly assigned to either an MST experimental condition or to hospitalization and aftercare. To serve this population more effectively, one child and adolescent psychiatry resident and several crisis caseworkers were added to the usual MST treatment team of master's-level clinicians and a single supervisor to provide coverage 24 hours a day, 7 days a week. Services were provided in the homes of family, relatives, or friends, and in community shelters, respite beds, or hospital. Caseloads were reduced from the MST standard of five families per clinician to three. In the beginning of the project, all cases were supervised daily by a child and adolescent psychiatrist; this was reduced later to three times weekly. Children in the experimental group had judicious and controlled access to community resources, including hospitalization and therapeutic foster care. Children in the control group received treatment as usual, often using some of the same resources.

[Henggeler et al. \(1999\)](#) report that MST was at least as effective, and in some cases more effective than emergency psychiatric hospitalization at decreasing child symptomology. Rates of decreased internalizing problems were similar across the two conditions; MST was more effective in decreasing rates of externalizing symptoms. Youths in the control condition reported increased self-esteem, whereas families in the experimental condition showed improved cohesion and increased structure. The researchers note that youth with serious psychiatric problems and their families presented with greater complexity and problem severity than they had expected based on their previous work. Further study, some of which already is in progress, is needed to address the issues raised by this evaluation ([Henggeler et al., 1999](#)).

### Wraparound

The concept of wraparound services that place the child in the context of the family as well as his or her broader social ecology emerged from the CASSP, which was dedicated to the creation of interagency collaborations, community-based, advocacy-oriented systems of care, and the expansion of parental decision making and involvement to meet the multisystemic needs of children with serious emotional disturbances ([Woolston and Berkowitz, 1998](#)). Theoretically based in environmental ecology, wraparound stresses unconditional care and the belief that changes in the environment will foster changes that persist over time for children, families, and communities ([Burns et al., 2000](#)). Wraparound is a strength-based process of intervention that values parental empowerment, culturally competent providers, and the use of natural supports to augment professional involvement. Outcomes are measured against goals established by the family. Unlike MST, wraparound teams are led by a bachelor's-level resource coordinator who does not provide clinical care; the quality of clinical care depends on the resources of the local system of care. Leadership in the MST model is provided by a master's- or doctoral-level clinician who acts as a family therapist. Wraparound services have no time limit, are available 24 hours a day, 7 days a week, and are provided in home, school, clinic, and community. In comparing the effects of MST and wraparound, [Burns et al. \(2000\)](#) found a need for randomized effectiveness trials to demonstrate the effectiveness of wraparound in light of the speed of its national replication, the observable variations in its implementation, its potential for cost savings, and the increasing availability of standards and measures of fidelity.

## IN-HOME SERVICES FOR CHILDREN WITH PSYCHIATRIC DISTURBANCES



Although research on the effectiveness of home-based intensive clinical services for children with serious emotional disturbances is limited, there is some indication that these services can enhance child and family functioning and prevent crisis-driven hospitalization of children. ( [Lindblad-Goldberg et al., 1998](#) ). As a result, programs incorporating elements of family preservation, MST, and wraparound have attracted interest as potentially useful alternatives to more restrictive and expensive out-of-home treatment facilities. Many state agencies, managed care companies, and private payers have endorsed home-based psychiatric services as effective both clinically and fiscally, and have added them to the existing local continuum of care.

## THE YALE IN-HOME CHILD AND ADOLESCENT PSYCHIATRIC SERVICE

The Yale In-Home Child and Adolescent Psychiatric Service (YICAPS), an intensive, in-home, goal-oriented intervention for children with serious emotional disturbance, represents an integration of theory and clinical practice that is guided by principles of developmental psychopathology, attachment, object relations, cognitive-behavioral, and family systems theory, and the process of wraparound. Children referred to YICAPS may be in the process of discharge from hospitals or residential treatment facilities, or their admission to such institutions may be preventable as a result of the intervention. It is funded both by managed care, as a strategy for reducing the need for more costly treatments, and by the state agency responsible for children's mental health services. The basic tenets of a medical model are essential to the program's design. Services are provided by teams consisting of a master's-level clinician (social worker, nurse, or psychologist) and a mental health counselor, who may have a nonclinical degree, both of whom work under the direct, weekly supervision of a child and adolescent psychiatrist or experienced clinician. A child psychiatrist assumes medical responsibility for the care of all patients and presides at weekly rounds. Treatment is focused on specific, defined problems that are viewed as amenable to measurable amelioration.

### Principles of Intervention

The unfolding interaction between the child and his or her psychosocial environment is seen as comparable in importance with any of the child's individual characteristics. As a result, all evaluations and assessments are considered simultaneously at the level of (a) individual child and family functioning, (b) the child's home and community environment, and (c) the child's and family's social networks. Human relationships are viewed as the mediating agents for behavioral and environmental change. Critical relationships exist on multiple levels: between the child and the family, between the team and the child and family, between the family and its physical environment, and between the child, the family, and the community. The ability to understand and address the complexity of the interactions among these levels is central to the success of the child's treatment.

The approach to the work is child centered and home and family based, relying on the development of a therapeutic alliance between the family members and the team to ensure progress toward the established goals. Goals common to all cases include stabilization, improved child and family functioning, and preparation for entry into more traditional, less intensive forms of community-based outpatient treatment. Each treatment plan respects the individuality of the child and family and is developed in partnership with them. The team works with the family to achieve the lowest level of change necessary to improve and sustain the child's more adaptive functioning and ensure his ability to remain in his or her home and community.

With the engagement and support of the managed care coordinator or the state agency staff member, each of whom are enlisted as collaborators in the work, the program is sufficiently flexible to allow for a titration of intensity from 3 to 20 contacts per week and to maintain involvement with the child and family for as long as clinically appropriate. In some rare instances, contact with the family has continued for upward of 18 months. However, the overarching YICAPS aim is to move the child and family to self-sufficiency with deliberate speed so that their need for intervention is as short and at the lowest level of intensity possible ( [Woolston and Berkowitz, 1998](#) ).

The Yale Children's Psychiatric Inpatient Service (YCPIS) has been an important collaborator with and contributor to the development of YICAPS, and the principles that guide the work on the unit have been applied successfully to the work in the child's and family's home. Because the child's behavior and long-term developmental course are recognized as context dependent, understanding and addressing the "goodness of fit" between the child's needs and his or her environment is a central task of staff in both institutional and home settings. While in hospital, the child can be helped to shift to a more functional developmental trajectory through the engagement of his or her family, school, and other treatment providers. During the period of the child's hospitalization, the family also is engaged in therapy and presented with opportunities to alter dysfunctional behavioral interactions with the patient and other family members. In addition, school personnel and outpatient providers are asked to participate in treatment planning so that they can be informed of the insights and information acquired by unit staff during the child's hospitalization. Similar strategies are used by YICAPS either after discharge or while the child remains in the home. The result is a program that integrates a well defined medical model with an ecologically oriented and family-focused approach to meeting the needs of seriously emotionally disturbed children and adolescents.

### Program Elements

The YICAPS program is characterized by six basic elements that can be augmented by others services as deemed necessary in each child's and family's individualized plan of care:

1. The core services, including home-based evaluation, individual and family psychotherapy, parent guidance, case management, crisis intervention, and medication management, are provided consistently by the two-person clinical team with the guidance and support of a child and adolescent psychiatrist.
2. In partnership with the family, the team assesses the strengths and the risk factors present in the home and in the environment to bring to light the factors that may influence the treatment.
3. The tasks of the team include developing collaborations and linkages with the resources and systems in the community with which the child is involved, including but not limited to the extended family system, health and mental health care providers, schools, and faith-based organizations, and assisting the family to advocate for their own interests with these systems and institutions.
4. The team joins with the child and family to develop and implement a treatment plan that meets with the approval of the managed care company or state funding agency and addresses the therapeutic, environmental, and concrete needs identified in the initial assessment.
5. Services are titrated in both intensity and duration to be responsive to the needs of the child and family.
6. The team is available 24 hours a day, 7 days a week to respond to child and family crises. Response may range from telephone consultation to in-home services.

### Who Benefits

The primary beneficiaries of the YICAPS approach are three distinct groups of children. The first group consists of children who are being discharged from psychiatric hospitalization and may require temporary but intensive services to sustain the behavioral and emotional gains they have made as inpatients. These children may have successfully negotiated the crisis that necessitated their hospitalization, but are returning to unaltered environments that may not promote their continued recovery. In these cases, the primary goal of the in-home treatment is to continue and expand the treatment begun on the hospital unit and to engage the child's school and other community resources more intensively. The longer-term goal is the prevention of rehospitalization or placement in a residential treatment facility. A second group of children are those whose behavior is so seriously out of control that they become candidates for acute hospitalization. These children may be referred to YICAPS from home, school, clinic, or emergency department. The immediate goal of the home-based team is to join with the family and the source of the referral to assess the family's capacity to work with the team and ensure the safety of the child in the home. If the family is able and willing to maintain the child at home, the team and the family develop a crisis intervention plan that will lead to the establishment of more distal goals as the therapeutic alliance deepens and the underlying psychodynamic and individual issues come into view. The third group of children for whom home-based services are a particularly viable option are those with serious emotional or behavioral disorders for whom traditional outpatient treatment or partial hospitalization has been insufficient or unsuccessful. Services delivered in the home to both the child and family augment these out-of-home services rather than supplant them, and, in fact, may enhance their use and effectiveness. By entering the family's psychosocial environment, the in-home team is presented with an invaluable opportunity to identify and address the issues and concerns of the child and the adults that serve as latent barriers to the appropriate use of community resources. When these obstacles are overcome, the child and family can return to more traditional treatment modalities.

### Outcomes

Outcome data from a sample of 138 successive cases indicate that the program has been particularly effective for youth who present with diagnostic comorbidity, severe impairment, and extensive psychosocial adversity and are in greatest need of acute psychiatric stabilization. YICAPS intervention for a mean of 19.25 weeks resulted in successful diversion from psychiatric hospitalization for 42% of the children served, and a reduced length of stay among the 26% of children who required

rehospitalization. Clinical improvement over the course of home-based treatment was evident in statistically significant changes in psychosocial functioning. Most children attained stability during the course of treatment. A small proportion experienced decreased levels of functioning, whereas a somewhat larger minority experience functional improvements. The most common admitting diagnoses among the 108 children served in fiscal year 1999 to 2000 included disruptive disorders such as attention deficit/hyperactivity disorder and oppositional defiant disorder (ODD), depressive disorders, parent-child relational problems, and posttraumatic stress disorder. Children also have been referred to YICAPS with diagnoses ranging from anxiety to pervasive developmental spectrum disorders. Patients were more likely to be male (58.3% to 41.7%), live in a one-parent families (64.8%), and be white (59.2%). African-American children comprised 16.6% of the 108 children served in 1999 to 2000, whereas 21.2% were Latino. Forty-eight percent of the children were between 6 and 11 years of age; 38% were between 12 and 19 years, and 3.8% ranged from 0 to 5 years of age. A study of 97 cases in which data were available revealed that 69% of the cases were significant for history of abuse, and 65% for parental psychiatric history. Parental substance abuse and domestic violence were present in 44% and 37% of the cases, respectively.

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#### CASE ILLUSTRATION: JORGE

*History:* At the time of his referral to YICAPS, Jorge was a 15-year-old Hispanic boy who lived with his adoptive mother, her biological daughter, age 10 years, and a brother, age 22 years, also adopted. His biological mother, a woman with a psychotic disorder and known to abuse substances, placed him at the age of 6 years with a family friend who became his adoptive mother. Jorge's primary symptoms were paranoid and aggressive behaviors, particularly directed toward his adoptive mother. Before the YICAPS referral, Jorge been had hospitalized nine times in a period of 3 years, often after acts of violence toward his adoptive mother. In addition, he had been in several partial hospital programs and had been enrolled in a therapeutic school for the past 18 months. His hospitalizations always were preceded by a period of noncompliance with his medication regimen. Without his medication, Jorge evidenced a mixed manic state characterized by out-of-control behavior. Jorge entered the mental health system at 7 years of age, shortly after his mother had placed him out of her home. At 12 years, he was diagnosed with bipolar disorder after a psychotic episode. At that time, Jorge accused his older brother of sexually molesting him and a restraining order subsequently was put into place. As a result, the brother moved to a nearby apartment; although the two saw each other daily, they did not speak.

*Assessment and Goals:* A YICAPS assessment found that although Jorge was being seen in weekly supportive therapy with his adoptive mother, the sessions were focused on her descriptions of Jorge's bad behaviors. Jorge was intermittently compliant with a medication regimen that consisted of 600 mg of valproate orally twice a day and risperidone, 2 mg orally twice a day. He did not have a treating psychiatrist at that time. The YICAPS team learned that Jorge was not taking his medication, which appeared to be linked to both his mother's anxiety and her controlling and intrusive behavior. In addition, he was attending school only intermittently, no more than three times weekly. The clinical formulation recognized that Jorge was becoming increasingly frustrated and angered by his mother's intrusiveness and retaliated by not taking his medication and not attending school regularly.

With Jorge and his mother, the YICAPS team set the following goals for the intervention: (a) establish consistent psychiatric care, (b) ensure compliance with medication, and (c) decrease truancy. The development of the therapeutic alliance that would be of singular importance to the success of the work required that the team join individually with Jorge and his mother. The team agreed with Jorge that he could give up his valproate if he would continue to take his risperidone and would agree to see a child psychiatrist consistently for medication monitoring. The team agreed with Jorge's mother that a meeting between Jorge and his brother, when the patient was ready to do so, might be a long-term family goal.

*Treatment and Outcome:* In recognition of the treatment needs of both the patient and his mother, one member of the YICAPS team was assigned to provide individual psychotherapy for Jorge, while another provided both individual treatment and parental guidance for his mother. Although it was difficult for his mother to give up control, she was able to accept Jorge's age-appropriate need for independence and autonomy and agreed to help him to find less self-destructive pathways for the discharge of his anger and feelings of inadequacy, powerlessness, and victimization. At the time of discharge from YICAPS, all of the goals established with the family had been met. Jorge had not been rehospitalized, he was meeting with his treating psychiatrist regularly, was compliant with his medication regimen and was attending school daily. At a 3-month follow-up, he had not been hospitalized and was still attending school daily. His mother had joined a long-term parent support group for parents of adopted children and was attending a NAMI (National Alliance for the Mentally Ill) group. Jorge and his brother had not yet spoken to each other.

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## SUMMARY

The YICAPS treatment model derives from a strongly held belief that the most promising approach to children with serious emotional disturbances lies in illuminating, understanding, and addressing the complex set of internal, familial, peer, and social interactions that characterize each child's ecology. The YICAPS approach promotes the development of a supportive relationship with the child and family and recognizes that behavioral changes are most likely to be mediated through the ensuing therapeutic alliance. Although YICAPS has begun to demonstrate that good treatment outcomes are associated with the capacity to sustain the intervention as long as clinically necessary, randomized studies of interventions such as YICAPS are required if their value to children with serious emotional disturbances is to be understood. There is a developing literature that provides evidence of the benefits of home- and community-based interventions for dealing with high-risk, vulnerable children and that speaks to the importance of providing carefully constructed and standardized programs that are monitored for treatment fidelity. The future support of effective, home-based mental health services such as YICAPS depends on the ability of program planners to develop and test interventions that demand specific, quantifiable standards while allowing for the clinical flexibility that defines the work.

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# 125 CONSULTATION WITH FOSTER CARE HOMES, GROUP HOMES, YOUTH SHELTERS, DOMESTIC VIOLENCE SHELTERS, AND BIG BROTHERS AND BIG SISTERS PROGRAMS

R. Kevin Grigsby, D.S.W.

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The continuum of services offered by child welfare programs presents a challenge to mental health professionals. Services have been developed to serve a diverse group of children and adolescents with multiple and varied needs. The sheer numbers of children needing assistance and the needs of those children at times overwhelm the system that is designed to serve and protect children. The number of children in foster care peaked in the 1970s, declined through the early 1980s, and rose sharply from the late 1980s until leveling off at about half a million children in out-of-home care in the mid-1990s. This represents growth in federal foster care expenditures from \$309 million to \$3.13 billion during the same period. By 1999, federal foster care expenditures were expected to have increased by 1,300% from the 1981 level ([Courtney, 1997](#)).

Children who present with problems that require more than minimal services contribute to the problem of too few services for too many children in the present child welfare system ([Lifting the Veil, 2000](#)). Child-serving agencies often turn to child mental health professionals for consultations or technical assistance as they try to serve children presenting with severe emotional problems. This chapter offers practical advice to the mental health clinician who is called on to provide consultation to child-serving agencies such as foster care homes, residential group homes, emergency youth shelters, domestic violence shelters, and Big Brothers and Big Sisters programs.

## THE CONTINUUM OF SERVICES IN CHILD WELFARE

The child welfare field has evolved into a complex, multidimensional matrix of services and programs since the early 20th century. A continuum of services has developed that vary in level of intensity although these programs and services may vary from community to community and state to state ([McCroskey and Meezan, 1998](#)). In the least intense mode, child advocates lobby for and support policy initiatives that seek to improve or maintain children's rights. Services include community-based emotionally supportive programs offered to children and their families, such as family-centered childcare facilities, school-based health clinics, and support groups. In a moderately intense mode, home-based intensive family preservation services are offered to at-risk families in need of specialized assistance such as respite childcare, services for developmentally disabled children, or adolescent pregnancy/parenting services. Families in crisis or at risk of dissolution may be in need of family-centered services designed to preserve families through intensive home- and community-based intervention. More intensive services may use out-of-home placement of children to provide for the care of children whose parents cannot or have not provided those children with an adequate level of care ([Berrick, 1998](#)). Out-of-home placement varies in intensity in that children may be placed into a foster care setting with relatives (kinship care), licensed foster parents, an emergency youth shelter, a group home, or a more institutional setting such as a residential treatment center.

Youth shelters, group homes, and foster homes are representative of a portion of the child welfare service continuum. Various providers of these services may call on mental health professionals for assistance, because these programs typically do not include mental health professionals as staff members. Even so, many of the children and youth that they serve present with a wide range of problems, including behavior commonly dealt with by mental health providers in more intensive programs, such as community mental health centers, residential treatment centers, and hospitals. Children and youth rarely present themselves for emergency psychiatric services ([Halamandaris and Anderson, 1999](#)). It is very likely that child welfare agencies are the referral source of children and youth in need of emergency psychiatric services. There is empiric evidence suggesting that the prevalence of psychopathology among children in family foster care is higher than would be expected in a typical population of children or children from deprived backgrounds ([Pilowsky, 1995](#)). Leon and associates (2000) found that residential treatment centers serving children and youth frequently refer low-risk individuals for psychiatric hospitalization, suggesting that the residences are struggling to meet clients' needs. The findings of a recent study in Britain revealed particularly high levels of serious psychiatric disorder, comorbidity, and a significant number of undetected, potentially treatable psychiatric disorders in 13- to 17-year-old adolescents living in residential or foster care ([McCann et al., 1996](#)). The demand for psychiatric consultation with this population is significant. Continuing trends suggest that demand will not decline in the near future.

## WHAT IS CONSULTATION?

[Kadushin \(1977\)](#) offers a list of representative definitions of *consultation* from the human services literature. For the purpose of this discussion, Boehm's definition appears to be the most useful:

Consultation is an educational process whereby expertness in knowledge or skill is made available for the purposes of help with the solution of a problem by the provider of the consultation to the recipient of consultation, the latter assuming the responsibility for seeking the consultation and the use, nonuse or partial use of the fruits of the consultation ([Boehm, 1956](#), p. 241).

This chapter concerns itself with the "expertness in knowledge and skill" that relates specifically to the area of child and adolescent psychiatry. This is an important distinction, as many times consultation is requested related to agency functions or problems that are not specifically related to the area of knowledge and skill in child psychiatry. For example, the consultant may be asked to offer help regarding dealing with employee absenteeism. Although the consultant may wish to explore the area further in order to clarify clinical issues (are employees not coming to work because they are feeling too frustrated with the clients?), it is important to remember that employee absenteeism may be a problem that requires "expertness in knowledge and skill" that is outside of the realm of child psychiatry. Consequently, the consultant may find himself or herself acting to refer agency personnel to other "experts" (e.g., employee assistance programs) for help. The consultant may spend a great deal of time helping agency personnel refine questions so that the core problems or issues can be addressed.

## TYPES OF CONSULTATION

It is unlikely that the consultant will meet with a specific problem that can be identified easily. If this were the case, the consultant's help probably would not have been requested in the first place. Rather, the consultant may be met by a situation that is uncertain and a group of workers who are very frustrated because their attempts to intervene may have failed. In fact, unsuccessful intervention may have worsened the situation. The first task of the consultant is to ascertain the type of help that is needed; it can be categorized into one of two types: (a) *clinical* consultation and (b) *programmatic* consultation.

Clinical consultation is usually related to an individual person, family, or group. By and large, the consultant is asked to "reveal" information about the person, family, or group that will somehow improve the agency's ability to serve that particular "unit of intervention." Quite often, clinical consultation takes place on the "turf" of the consultant. For example, a foster mother may be having a difficult time dealing with the behavior of a child and may ask the child welfare worker to arrange for a meeting with the consultant to discuss alternatives for dealing with the child's behavior. The foster mother then meets with the psychiatric consultant at the local child guidance clinic for several sessions in order to make use of the clinician's insight and recommendations related to the foster child.

Programmatic consultation usually relates to groups or classes of persons who are served by the provider. This type of consultation may be offered on the consultant's "turf," but it is usually offered on-site through actual observation or participation by the consultant. Both types of consultation are directed toward



improving services, but the goals may differ significantly in that clinical consultation is directed toward smaller, specially identified units of intervention. Programmatic consultation is directed toward broader organizational change with the goal of improving the care provided or services rendered.

Types of consultation closely related to programmatic consultation are training, education, and supervision of agency volunteers and employees. These types of consultation are covered in more depth elsewhere in the literature but are discussed briefly because they pertain to the programs in the child welfare continuum.

## COMMON THEMES IN CONSULTATION

The mental health consultant should be able to address five closely related themes in most situations. The themes of separation, loss, identity, continuity, and crisis are common to all children who are placed in youth shelters, foster homes, and group homes ([Siu and Hogan, 1989](#)). Children involved in support programs like Big Brothers and Big Sisters programs may have had to deal with some of these issues, although the degree of intensity may be less as they have not necessarily had an out-of-home placement ([Big Brothers and Big Sisters, 2000](#)). A common quirk in the referral process is that the consultant never may be asked directly about any of these themes by the agency or persons asking for help. It is imperative, however, that the consultant be cognizant of the importance of these issues to the children involved in the programs. Further, the consultant should use his or her status as an expert to raise the consciousness of the agency or program staff to the need for consideration of these factors in the day-to-day operation of the program.

A common area of consultation is related to the understanding and management of "acting-out behavior." The consultant should recognize that agency and program staff might often use this term incorrectly; however, that is usually of little consequence. Rather, it is important to recognize that when program personnel describe behavior as "acting out," they may be indicating that they do not have an understanding of this child, or of the child's behavior. This is a cue that the program personnel may be asking the consultant to make use of his or her "expertness" in order to help them understand and deal with the problem child or behavior on both the individual and programmatic level. Helping the program personnel know which behaviors are beyond their scope of intervention also is important. For example, a youth shelter program may not be able to offer shelter to a child who is actively setting fires. Most shelters do not offer the necessary close behavioral monitoring that is imperative in serving children with histories of setting fires. A referral to a more intensive program may be necessary. Shelter personnel may need to be supported in their refusal to admit the child who sets fires. Advocacy for the child may be necessary to assure placement in a program equipped to serve children with histories of fire setting.

In general, the common themes addressed in dealing with children who are being served by these programs are not so different from those themes that are dealt with in evaluating or treating children and youth in the child guidance clinic. One difference is that the consultant is one step removed from the child as the agency or program personnel are assisted through consultation. Another difference is that collateral information, although indispensable, may come from a multitude of sources ([Halamandaris and Anderson, 1999](#)). Deciphering this morass of information may require an inordinate amount of time when scarcely enough time is available to actually examine the child.

## CONSULTATION WITH FOSTER HOMES

Children in out-of-home care have been placed into a variety of settings, including youth shelters, group homes, or foster homes. When children can not be placed with a parent, foster homes are the "placement of choice" whenever possible, because it is felt that they offer the most home-like environment for children who have often experienced significant turmoil in their lives and required protective service intervention. Foster homes offer latency-age and younger children an environment that is temporary in nature and that offers the child the care and nurturance that is needed at the time of placement. Persons who become foster parents must go through a procedure of licensing, but they usually do not have any special training in dealing with children, except for their own life experiences. In recent years, therapeutic foster homes and "special" foster homes (e.g., homes specifically designed for HIV-infected children) have been developed, which make use of foster parents who have a high degree of training. The exact number of licensed foster homes in the United States is difficult to determine, because there is no uniform data set collected by states. The Child Welfare League of America (2000) estimates that there were approximately 300,000 children in foster homes in 1996. This figure is the latest year of a complete data set and does not include children living in residential group care.

Foster parents often are in need of help in order to understand and manage the behavior of children placed into care in the foster home. For example, foster parents may approach the mental health consultant for help with a specific problem they are experiencing with a child, such as nocturnal enuresis. Usually, the foster family has attempted to deal with the problem by making use of their own resources and understanding. If this has failed, they may then approach the consultant for help. It is important, however, that the consultant recognize that the foster parents may also need help with feelings of frustration or failure that they may have because they were unable to deal with the problem on their own. The consultant must consider this as he or she helps the foster family to understand the problem through the use of the knowledge base of child psychiatry. In a sense, the consultant must move through a series of roles as educator, clinician, and "coach" as he or she helps the foster family to deal with the problem at hand.

Child welfare workers may need assistance in understanding the nature of parent-child, sibling-sibling, and grandparent-child attachment relationships and the importance of supporting attachment relationships. Too often, workers underestimate the need for visitation and are not "cognizant of the importance of encouraging and maintaining attachments between children in foster care and persons to whom they are attached" ([Grigsby, 1994](#)). The mental health consultant must remember to advocate for the child's best interest, even if arranging visitation requires a great deal of effort on the part of the child welfare worker.

In some cases, programmatic consultation is requested with regard to the operation of foster homes. The mental health consultant may be asked to train a group of foster parents around some issue or issues pertinent to caring for foster children. For example, a group of foster parents who are providing foster care for adolescents may ask for help with trying to deal with the behavior of these foster children. A time-limited, group-oriented training module for these foster parents on understanding adolescents is one possible option. Information should be presented in such a way that it is specifically related to the children that the foster parents have in their homes. Child psychiatric knowledge should be applied to both the generic and unique situations encountered by these parents. Additionally, the consultant should be prepared to offer consultation to foster parents that is child specific. This may need to be done outside of the group situation, however.

## CONSULTATION WITH GROUP HOMES

Community-based living facilities for youth have come into wide use over the past several years. The Child Welfare League of America (2000) estimates that approximately 48,000 children were living in residential group care in 1996. This figure does not include children in therapeutic settings.

Although group homes have been used for various categories of persons (mentally ill, physically handicapped, recovering substance abusers), this discussion is limited to group homes for clients who are dependent youths, emotionally disturbed youths, and/or socially maladjusted youths ([Shostak, 1987](#)). Group homes of these types usually serve children who have reached adolescence. These facilities are different from residential treatment centers. Their small size, reliance on other community resources such as public schools, and lack of treatment plans indicates that they are more accurately defined as a congregate living facility rather than a therapeutic environment. Between 1965 and 1981 there was a sizable increase in the number of residential group care facilities of this type. Even so, there was a "decrease in the relative proportion of residential group care for dependent, neglected, or abused children and youth" ([Dore et al., 1984](#), p. 493). This was accompanied by a reduction in facility size ([Dore et al., 1984](#)), which suggests that a change in the orientation of services may have taken place during the period of 1966 to 1981. [Dore and Kennedy \(1981\)](#) argued that three major principles affected the delivery of services between 1960 and 1980. The movement toward deinstitutionalization, normalization, and the right to treatment has caused changes to take place within the child welfare system that have greatly affected the clientele and programs at group homes. In a sense, there has been an impetus to improve the services offered in order to meet more effectively the needs of the population served at these facilities. Group home administrators often request consultation to help them:

1. Obtain, retain, and train childcare personnel.
2. Prevent premature discharge or termination.
3. Evaluate and briefly intervene with problem residents.
4. Develop treatment plans and strategies for dealing with seriously disturbed or maladjusted youth placed in the group homes.

The mental health consultant can be most helpful to these programs by focusing on the unique situation of each facility while promoting the generic knowledge offered by the theory base of child psychiatry. In other words, the application of clinical knowledge acts as a guide to help the group home personnel understand the needs and behavior of residents and, to some degree, of staff themselves. The consultant can then guide the program personnel to better serve the group home residents.

The mental health consultant also can be helpful in advocating for these programs. Too often, community members presume that locating a group home in their

neighborhood will have a negative effect. Research indicates that the expectations of negative effects are much greater than what is experienced by neighbors ( [Cook, 1997](#)). Advocating for the establishment of group homes in residential neighborhoods allows for creating a living situation that is more typical of family life and less like life in institutional care.

## CONSULTATION WITH YOUTH SHELTERS

Youth shelters traditionally have been called on to provide safe haven for runaway and homeless youth. The problems of this population group are complex and include severe aggression, conduct problems, and use of alcohol and other drugs at rates substantially higher than nonrunaway and nonhomeless youth ( [Booth and Zhang, 1996](#); [Green et al., 1997](#)). The complexity of presenting problems has created greater pressure on both shelters and the state child welfare agencies to adequately serve youth. Programs designed to shelter children and youth should be equipped to serve children with serious problems, including suicidal behavior and histories of sexual and physical abuse ( [Molnar et al., 1998](#)).

Another complexity in serving runaway and homeless youth in shelters is the difficulty in tracking them to provide follow-up care ( [Pollio et al., 2000](#)). This is typical of children who are commonly referred to as “street youth” (USDHHS, 1998). Many of these children and youth become involved in the sex industry as they struggle to survive on the streets. High-risk sexual behaviors ( [Forst, 1994](#)), substance abuse ( [Greene et al., 1997](#)), and episodic primary health care, if any, are chronic problems of many homeless and runaway youth. Typically, resolution of these problems requires an extended period of contact. Many children and youth will be suspicious of any offer of support or attempt to help. Adults offering help and support to many of these children and youth are likely to have been pimps or pedophiles they met on the streets. Although the initial involvement with those adults may have been positive, it is likely that exploitation was quick to follow because sex and prostitution were expected as “payback.”

Although many of the youth entering shelters have lived with a parent or relative within the past year ( [General Accounting Office, 1989](#)), family life is likely to have been marked by sexual exploitation, mutual aggression, and violence. Simply returning children and youth to parental custody is a naive solution to a terribly complex situation ( [Whitbeck et al., 1997](#)). Many youth entering shelters may have left foster or substitute care situations. A Canadian study found that a significant portion of youth living “on the street” (46%) had left a foster home or other form of government sponsored care ( [Kufeldt et al., 1992](#)).

In spite of the high level of need, service delivery to this population historically has been difficult, and services that are available often have been fragmented or distant. Although some shelters provided basic mental health services long ago ( [Gordon, 1978](#)), many are not equipped to provide these services on site. Mental health consultants may be asked to provide service either on or off site. A model for mental health consultation at shelters for homeless and runaway youth has been developed that makes use of naturalistic methods to provide mental health consultation that is both clinical and programmatic in nature ( [Grigsby, 1993](#)). This model assumes that there is not only a need to understand the individuals who are placed in the shelter, but also a need to understand the “culture” of the shelter environment, if the consultant is to be successful in both the clinical and programmatic senses. This model allows the consultant to enter the “culture” of the shelter in a way similar to that of the field researcher or ethnographer entering a foreign culture and attempting to study that culture as a participant observer. Schein explicates the “clinical perspective” as being different from that of ethnography in that “clinicians enter an organization if they are requested to do so by someone in the organization or someone acting on the organization’s behalf seeking some kind of help” ( [Schein, 1987](#), p. 24). An understanding of the shelter is necessary before any meaningful programmatic consultation can take place. The behaviors of both residents and staff may be better understood as the consultant begins to understand the shelter. Shelter staff begins to accept the consultant as someone who “has been there” and who has an understanding of the day-to-day operations of the shelter. On-site involvement also leads to the formation of relationships between the consultant and residents and between the consultant and shelter staff. The staff and residents will begin to approach the consultant for help as the consultant is accepted as a person who has an understanding of the situation. In order to get to this point, however, many weeks of on-site participant observation may be necessary. Eventually, the consultant will be able to gain enough trust that he or she will be able to offer consultation about residents without waiting to be solicited for advice. As clinical consultation regarding individual residents becomes routine, programmatic consultation can then begin to take place. In one consultation effort at a shelter in New Haven, CT, the rate of unplanned discharge was reduced from 43.5% to 18% within a 6-month period through the use of this model ( [Grigsby, 1993](#)). By taking the clinician out of the clinic and placing him or her in the community, the work of both the mental health professional and the community staff can be enhanced as trust is built. The gulf between the mental health professional and the community staff is reduced, and a statement of the value of the shelter program and its staff is made.

This consultation is not treatment. Rather, it is the use of clinical skills, insight, and knowledge to support those who work on the front line with homeless youth. The consultation helps them to understand the strengths as well as pathology of the individual clients and helps them to gain insight into the group dynamics within the shelter facility itself.

In providing programmatic consultation to emergency youth shelters, the limitations of the facilities and programs may require that the consultant advocate for major change in programming so that the facilities are better able to meet the needs of the children who are referred. In a less sophisticated sense, a consultant may become aware that modifying or altering the present program may still fall far short of what is needed. In these cases, the consultant may need to work toward developing a model or prototype program that will better serve the needs of clients. Advocacy is needed for the creation of shelter care programs for homeless, at-risk youth that are designed to accommodate those with histories of psychiatric problems in need of mental health evaluation and intervention. In order to insure that shelter programs serve the best interests of children, shelters should promote a nonjudgmental philosophy that encourages the admission of children in need while considering the limitations of the emergency youth shelter. Specific recommendations about the types of clients who *cannot* be appropriately served in a shelter care setting may need to be made.

## CONSULTATION WITH DOMESTIC VIOLENCE SHELTERS

Domestic violence is a frequent theme in the families of children being served in the child welfare continuum ( [Findlater and Kelly, 1999](#)). Mothers with children may seek refuge from abuse by entering domestic violence shelters. Since the early 1970s, community-based domestic violence services have grown in size and scope, with the total number of shelters exceeding 1,300 by 1997 ( [National Coalition Against Domestic Violence, 1997](#)). Approximately one-half of the residents in battered women’s shelters are children.

Dealing with “crisis” is common when working with children referred by domestic violence shelters, especially those witnessing interpersonal violence between adult caregivers. Intrafamilial domestic violence can have serious negative effects on children who witness it ( [Edelson, 1999](#); [Fantuzzo and Mohr, 1999](#)). Public awareness of the negative effects of domestic violence on women is growing, and mental health professionals have begun to recognize the needs of children witnessing domestic violence ( [Groves, 1999](#)). Mental health consultants may be asked to evaluate children referred by domestic violence shelter personnel. The impetus for referral may result from to a mother’s request or shelter staff’s expressions of concern about a child’s behavior. Although many children are legitimately traumatized by domestic violence and in need of mental health intervention, shelter staff may attribute children’s behaviors to witnessing violence when the behaviors were present long before any abuse was witnessed. For example, children may lack social skills and appear to be withdrawn as a result. The behavior may be attributed to the child experience of domestic violence when a lack of social skills is the real issue. Consultants can assist shelter staff and, at times, parents in understanding the child’s behavior and intervening when appropriate.

Mental health consultants can be helpful to the shelter staff and the family by providing clinical consultation that allows children to appropriately express their feelings related to witnessing intrafamilial violence and subsequent separation from familiar persons, places (especially school), and pets. Because shelter placement is typically short-term, the mental health consultant can assist with planning for long-term mental health intervention, if necessary, after the family leaves shelter care.

Programmatic consultation helps shelter staff to learn about interventions with children accompanying their mothers. Many domestic violence shelters are part of community-based agencies serving battered women. Some programs may be staffed entirely by volunteers, whereas others may employ social services professionals. Typically, caseloads are high and the focus of the services is on the battered woman, leaving little time to offer child-specific services. Mental health consultants can be helpful in linking these programs with other community-based services for children, including programs oriented toward preventing victimization of children ( [Wolfe and Jaffe, 1999](#)). Advocating for school-based services and other developmentally appropriate intervention strategies can help community-based agencies to better serve children.

## CONSULTATION WITH BIG BROTHERS AND BIG SISTERS PROGRAMS

Big Brothers and Big Sisters of America is the nation’s oldest youth mentoring organization, with programs in all 50 states that match kids with mentors. Founded in 1904, adult volunteer mentors help children “to reach their full potential and fulfill their dreams” ( [Big Brothers and Big Sisters, 2000](#)). Typically, adult mentors are matched with a child from a single-parent home. Mentors and children are usually of the same gender. The programs make use of adult volunteers who have limited



training in working with children but who receive supervision and support from professionally trained social workers employed by the local Big Brothers and Big Sisters programs. These programs may request consultation related to helping volunteers have a better understanding of the needs and behavior of the children that they serve. At times, these programs may request training workshops that are intended to help volunteers have a better understanding of the children who are involved. Training related to the five common themes of separation, loss, identity, continuity, and crisis ( [Siu and Hogan, 1989](#)) may be of value to volunteer mentors as many of these children may have life experiences related to one or more of these themes, especially themes of separation and loss. Consultation also may be requested on a specific child or a specific problem area, such as thumb sucking or fighting with siblings. It is important to remember that the consultant is asked to bring "expertise" on the subject, and that a better understanding of the subject area can be reached if the consultant is able to present the material in a manner that is relevant. In the case of Big Brothers and Big Sisters programs, the presentation should be case specific if possible, as volunteers are likely to be more accepting of the consultation if they feel that the issues are personally relevant. Although a discussion of theory may be fruitful when working with group home or youth shelter personnel, it may not be as useful when consulting with volunteer foster parents or Big Brothers and Big Sisters. Behaviorally explicit, personally relevant, and practical information is most helpful.

## THE LIMITS OF CONSULTATION

As Boehm relates in his definition of consultation, the recipient of the consultation is responsible for the "use, nonuse or partial use of the fruits of the consultation" ([Boehm, 1956](#), p. 241). It is the nature of the consultant's role to have little control over the operation of the consultee's program. In the hospital or clinic setting, the mental health professional has a great deal of responsibility, and to a degree, the concomitant control. The lack of control that is inherent in the consultant's role is at times frustrating to mental health professionals. Even though the consultant may offer concrete advice relating to a situation in a program, the program personnel may choose to disregard the consultant's advice, or to make only partial use of the consultant's advice. A situation may get worse by not using the consultant's advice. The consultant may be sought out for further help at that point, and it may be difficult for the consultant to refrain from expressing his or her displeasure that the initial advice was disregarded or modified. This is the nature of consultation. The consultant may experience a great deal of frustration and at times may be tempted to "take over" as the agency director, supervisor, or foster parent. It is obvious that this is not helpful. Therefore, the consultant must be cognizant of and agree to the limitations of the consultant role from the outset of the consultant–consultee relationship.

## CONCLUSIONS

Child-serving agencies must deal with many children whose needs are varied and broad. Although these agencies are charged with meeting the needs of children, they are at times ill-equipped to do this. When these agencies are overwhelmed or faced with situations of crisis, they may call on mental health professionals for help. Quite often, these agencies have difficulty in articulating the type or degree of problem that they are having. First, the mental health consultant must help the requesting person or persons to define the problem and refine the questions that they wish to have addressed. Second, the consultant must be clear about his or her role as a helper in the problem-solving process. The consultant must recognize the limits of his or her knowledge and power and must be ready to assist in locating an appropriate source of help if the problem or problems encountered fall outside of the scope of the consultant's knowledge base in child psychiatry. Consultants should recognize that issues related to insight, unconscious motivation, and acting out may be encountered and dealt with in the consultation process. Even though these issues are well within the scope of the consultant's knowledge base in child psychiatry, "the consultant's function is essentially educational, that learning develops out of a relationship, but that this relationship need not be therapeutic" ( [Stein, 1956](#), p. 249). Finally, by taking the time to understand the culture of the consultation site through a naturalistic or ethnographic intervention; the consultant is better able to understand the point of view of the client. This allows the mental health consultant's knowledge of child psychiatry to be most useful to agency personnel, volunteers, and the children who are served by these programs.

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[The Pediatric Symptom Checklist](#)  
[Use of Stimulants in Pediatric Primary Care Practice](#)  
[Use of Antidepressants in Pediatric Primary Care Practice](#)

Collaboration between pediatricians and child psychiatrists not only makes intuitive sense, but is a necessity in providing children with responsible, comprehensive health care. Nevertheless, numerous obstacles have interfered with meeting this goal. Differences in training philosophies and experiences, time pressures, managed care policies that discourage the integration of mental health services, and limited access to services all have contributed to difficulties in implementing effective collaboration.

Haggerty and colleagues first described psychosocial concerns as the “new morbidity” of pediatrics more than 25 years ago ([Haggerty et al., 1975](#)). Today, psychosocial problems are the leading cause of disability in childhood ([Costello and Pantino, 1987](#); [Costello et al., 1988a, 1988b](#)). Conservative epidemiologic data indicate that 12% to 20% of children have a psychiatric disorder ([Bernal et al., 2000](#); [Brandenburg et al., 1990](#); [Institute of Medicine, 1989](#); [U.S. Congress, Office of Technological Assessment, 1986](#)), with prevalence rates rising ([Achenbach and Howell, 1993](#)). The rates of childhood psychosocial problems recognized by primary care clinicians more than doubled between 1979 and 1996 ([Kelleher et al., 2000](#)); still, less than half of children with psychiatric disorders are identified in primary care settings, and only a fraction of these children are referred for mental health services ([Bernal et al., 2000](#); [Brandenburg et al., 1990](#); [Costello, 1986](#); [Costello et al., 1996](#); [Lavigne et al., 1993](#)). Pediatricians are being asked by families to deal with psychiatric disorders and are being encouraged if not required to limit their use of mental health, especially child psychiatric, referrals. The frequency with which primary care clinicians are prescribing psychotropic medications for children, including preschoolers, has increased significantly in recent years ([Kelleher et al., 2000](#); [Pincus et al., 1998](#); [Zito et al., 2000](#)). These statistics highlight not only the need for skilled assessment and treatment of psychiatric difficulties in children, but the need for early recognition and intervention in an effort to prevent more lasting suffering and impairment.

In 1996 there were over 53,000 practicing pediatricians in the United States ([American Medical Association 1997–1998](#)). In 1990, there were only 4,212 child psychiatrists, with 5 states having less than 2 child psychiatrists/100,000 youth. Regions with the highest rates of poverty (and thus the highest prevalence of psychosocial concerns) also had the fewest number of child psychiatrists ([Thomas and Holzer, 1999](#)). Even with the overwhelming need identified, a perception of competition between behavioral pediatrics and child psychiatry also has been a barrier to care.

The relationship between pediatrics and child psychiatry has been marked by both tension and cooperation. Although some psychiatrists have voiced concerns that the growing field of behavioral pediatrics may encroach on their “turf,” others have been actively involved in collaborative projects with pediatrics, such as development of the *Diagnostic and Statistical Manual for Primary Care* (DSM-PC). DSM-PC was published in 1996 as a joint effort between the American Academy of Pediatrics and the American Psychiatric Association ([Wolraich et al., 1996](#)) to improve the classification, recognition, and diagnosis of childhood mental disorders in pediatric primary care. DSM-PC provides a descriptive classification system for a continuum of psychosocial situations and symptoms that range from normal developmental variations, to problems, to psychiatric disorders as defined by the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV). Specific codes were added to encourage appropriate billing for time devoted to psychosocial concerns. By recognizing that primary care clinicians often are faced with assessing and managing psychosocial issues that may or may not meet full criteria for a specific disorder, it is hoped that DSM-PC will contribute to better identification, treatment, and reimbursement of a spectrum of psychosocial concerns. [Table 126.1](#) is a modified sample page from DSM-PC highlighting its structure and congruence with pediatric primary care practice.



**Table 126.1. Sample Page from the Diagnostic and Statistical Manual for Primary Care.**

The *Bright Futures* initiative, sponsored by the Maternal and Child Health Bureau, has been another valuable tool in addressing psychosocial issues in primary care. *Bright Futures in Practice: Mental Health* ([Jellinek et al., 2002](#)) was recently published as a guide that will help implement primary care goals set forth in *Bright Futures: Guidelines for Health Supervision of Infants, Children, and Adolescents* ([Green and Palfrey, 2000](#)). *Bright Futures in Practice: Mental Health* focuses on mental health promotion and early recognition of emotional difficulties by providing primary care clinicians with developmental information, tips for addressing areas of concern, and guidelines for assessment of and interventions for emotional problems and disorders. Recognizing that no minimum standards currently exist for the provision of mental health services in primary care, *Bright Futures* serves as a guide for Medicaid programs and a potential quality assurance standard for all insurers and providers. A compendium of teaching cases for pediatric residents on psychosocial issues also has been developed by [Emans et al. \(2000\)](#), as a teaching initiative consistent with the goals of *Bright Futures* and DSM-PC.

Additional efforts to identify and treat emotional and behavioral problems in children include the development of specialty board certification requirements for behavioral pediatrics by the American Board of Pediatrics, as well as increased training requirements for ambulatory and behavioral pediatrics for pediatric residency programs. The American Academy of Pediatrics (2000) also published practice guidelines for the diagnosis and evaluation of children with attention deficit/hyperactivity disorder (ADHD), for use by primary care practitioners.

Although these initiatives have a well accepted structure to support the mental health of children and adolescents in primary care settings, difficulties remain. This chapter discusses these difficulties and reviews strategies to address them. Topics that present particular challenges to the pediatric clinician, such as psychosomatic disorders, Munchausen syndrome by proxy, and the use of psychotropic medications, also are covered.

### SCREENING

Primary care providers are under increasing pressure today to see a higher volume of patients in shorter amounts of time. Given this productivity expectation, many

pediatricians feel not only that they do not have enough time to ask about psychosocial issues, but overwhelmed and at a loss to address the problems that they might discover. Patients and families are aware of these pressures. In a survey of mothers coming to private pediatric offices, less than a third reported that they were planning or able to discuss significant psychosocial concerns with their child's physician, even when these issues were the main questions on their mind ( [Hickson et al., 1983](#)). Focused interviewing and screening questionnaires can aid primary care clinicians in gathering important psychosocial information in an efficient manner.

The information gathered during a pediatric visit can be highly variable depending on who is providing the information, the pediatrician's receptivity, and the environmental context ( [Hack and Jellinek, 1998b](#)). [Wisow et al. \(1994\)](#) described three simple communication skills used by pediatricians that were associated with disclosure of sensitive information during pediatric visits: asking about psychosocial issues, making supportive statements, and listening attentively. A framework for asking psychosocial questions throughout development is available in the [American Academy of Pediatrics Guidelines for Health Supervisor \(1997\)](#), [Bright Futures: Guidelines for Health Supervision of Infants, Children, and Adolescents \(Green and Palfrey, 2000\)](#), and [Bright Futures in Practice: Mental Health \(Jellinek et al., 2002\)](#). [Hack and Jellinek \(1998b\)](#) have suggested specific questions, based on a review of the literature, that are most likely to provide pediatricians with enough information to identify children and families in need of further evaluation (see [Appendix 1](#)).

Screening questionnaires that can be completed by parents in the waiting room are another useful approach in identifying potential areas of concern. Any problem areas noted on screening can then be followed by more focused interviewing to assess the nature and severity of the problem. To be useful in a primary care setting, a screening questionnaire needs to be easy to administer and interpret, and needs to be well validated in differentiating children who are most likely to be symptomatic. The Pediatric Symptom Checklist (PSC) is one instrument that has been designed specifically for use in a pediatric waiting room (see [Appendix 2](#)). The PSC has been validated for use in children from 4 to 16 years of age, in a variety of settings and among various populations. It is a 35-item questionnaire, focused on assessing a child's major areas of functioning, that most parents can complete in under 5 minutes without assistance. A single score is calculated by the addition of numeric responses to each question, ranging from 0 to 2 as a symptom is rated as being "never," "sometimes," or "often" present. If the score is 28 or higher (24 or higher for preschoolers), there is a 70% likelihood that the child is experiencing significant dysfunction. For children who score below this cutoff, there is a 95% likelihood that they do not have serious difficulties. In previous validity studies, the PSC matched national estimates of psychosocial problems in children and performed comparably with the longer Children's Behavior Checklist (CBCL) ( [Cassidy and Jellinek, 1998](#); [Hack and Jellinek, 1998a](#); [Jellinek, 1998](#); [Jellinek et al., 1999](#)). If a child's PSC score is above the cutoff score, the primary care physician can then spend more time reviewing specific symptoms and assessing severity to determine the next course of action. Although the CBCL ( [Achenbach, 1991](#)) is the most validated psychiatric screening instrument for children, its length and more complex interpretation make it better suited to specialty psychiatric practice and research, or as a follow-up assessment measure of children identified as being at risk ( [Hack and Jellinek, 1998a](#)).

Screening questionnaires that focus on specific symptoms also can be useful in primary care practice when a particular area of concern has been identified. For ADHD, the Conners Parent and Teacher Rating Scales are helpful in assessing the presence and severity of symptoms, and in providing objective measures of treatment response ( [Cassidy and Jellinek, 1998](#)). DSM-IV criteria for ADHD also have been formatted into a checklist questionnaire that can be used for assessing and tracking symptoms.

For mood disorders, children are the best source of information regarding their own emotional states ( [Costello et al., 1988a](#)). Several self-report measures to evaluate depressive symptoms in children and adolescents are available, including the Beck Depression Inventory (BDI), the Children's Depression Inventory (CDI), and the Center for Epidemiological Studies Depression Scale (CES-D). Of these measures, the BDI has been the most widely used and tested. It is written at a fifth grade reading level, and therefore may be more appropriate for use with adolescents ( [Roberts et al., 1991](#)). The CDI was derived from the BDI, and can be used for children 7 to 17 years of age, although it is written at a first grade reading level. All of these measures are brief and easy to use and interpret ( [Hack and Jellinek, 1998a](#)). However, screening for depressive symptoms is a complex matter, with most available rating scales having a relatively low specificity ( [Eisert et al., 1991](#)) and many youth falling within the "at risk" or mild/moderate category. Rating scales for depression in children and adolescents cannot take the place of a careful clinical interview; they may be most useful in helping clinicians assess severity of symptoms and in tracking the progress of symptoms over time.

Focused interviewing and screening measures can help primary care clinicians with the first step of recognizing psychosocial problems. After recognition and assessment of severity of symptoms, clinicians are faced with the even more complex task of determining a course of action. Options for interventions are guided by the nature and severity of impairment. The range of potential interventions may include any of the following (see DSM-PC, pp. 23–27):

- For a concern that appears to be a normal developmental variation or is resolving: routine follow-up or monitoring of symptoms
- For a problem: further evaluation at the primary care level, regular follow-up visits, consideration of mental health consultation or referral, comanagement with mental health clinician(s)
- For a disorder: mental health consultation, mental health referral for a comprehensive evaluation, emergency referral, comanagement and ongoing collaboration with mental health clinician(s) ( [Jellinek, 1998](#))

## COLLABORATION

Many factors affect how effectively pediatricians and child psychiatrists can work together in meeting the needs of children and families. Differences in training, philosophy, approaches, and skill sets offer opportunities for each field to complement the other's efforts, yet also can lead to differing expectations and goals. Pediatricians typically see hundreds of children for short visits, whereas child psychiatrists see fewer children more intensively. Pediatricians gain a broad view of the variations in normal development, whereas child psychiatrists have the opportunity to understand more fully the depth of an individual child's experience or distress. Without open communication, stereotyped perceptions can interfere with providing comprehensive care. Psychiatrists may view pediatricians as more concrete, action oriented, and less able to tolerate slow progress or appreciate subtle dynamics. Pediatricians expect that psychiatrists will be less available and communicative than other specialists, more likely to see pathology instead of strengths, working at too slow a pace, and unhelpful in providing practical and useful interventions ( [Granger and Stone, 1996](#); [Lewis, 1994](#)).

These stereotypes do each field a disservice by depriving both of essential information that the other can provide, and by failing to recognize that within specialties there are wide variations in personalities, approaches, and skills. Collaboration is most effective when it based on a foundation of mutual respect. Primary care providers often have known a child or family over a period of time, and can offer insights into medical, developmental, and family history that are invaluable in treating emotional and behavioral difficulties. Psychiatrists can support medical care in a variety of ways, including providing a fuller picture of how a child is functioning in important areas (e.g., school, family), understanding the impact of physical symptoms or illness on a child's emotional development, and appreciating dynamic factors that may affect compliance with medical care.

Even when primary care providers and psychiatrists approach each other with respect and appreciation for the other's skills, other obstacles may interfere with collaboration. Pediatricians may be reluctant to refer a child or family for mental health services because of worries about stigma or how the referral will be viewed by the family and if it will jeopardize their alliance. Psychiatrists may limit communication with primary care providers because of concerns about maintaining patient confidentiality. Time pressures also interfere with efforts to maintain ongoing communication between providers. Financial considerations may decrease the number of referrals made when managed care plans require that mental health visits be paid for out of the general pediatric budget or offer incentives for limiting referrals; when preferred mental health colleagues are not in the panel of or credentialed (accepted) under a particular insurance plan; and when utilization review decisions are made by managers, not the clinicians involved. Last, in many parts of the country, primary care providers face a real shortage of child psychiatric colleagues ( [Thomas and Holzer, 1999](#)).

Various approaches can be used to address these obstacles. Pediatricians can decrease the stigma that families may associate with mental health services by establishing a practice in which psychosocial issues are routinely assessed and discussed. Families are more likely to follow through with a mental health referral if they understand the reason for the referral (e.g., patterns such as a child with too few friends or intense sibling rivalry) and if their pediatrician conveys that he or she knows and has confidence in the mental health clinician. Pediatricians also can clarify with both families and psychiatric colleagues what their goals are for the referral (e.g., assessment of depressive symptoms in a child with a chronic illness; parent guidance for managing separation anxiety) and how they plan to remain involved in the child's care. In situations where a pediatrician is unsure of how to broach a mental health referral with a child or family, discussing possible approaches with a psychiatric colleague often is helpful.

Child psychiatrists may receive referrals directly from a pediatrician, or from families or outside agencies with or without the knowledge of the child's pediatrician. In all of these situations, care is optimized by the psychiatrist conveying that he or she views the pediatrician as providing a crucial role in supporting the child's well-being. When a psychiatrist meets with a child or family for the first time, discussing how information will be shared with the pediatrician and obtaining signed releases are important in addressing any potential concerns about confidentiality. This process also can underscore to families that the child psychiatrist is committed to taking a comprehensive view of their child's health. For families that may be reluctant initially to allow the psychiatrist to communicate with their pediatrician, clarifying the type



of information that will be shared and the why this information is important to the child's care usually is sufficient to reassure most families.

Other obstacles, such as time pressures and limited access to child psychiatric services, necessitate more creative and flexible approaches. Each clinician needs to develop his or her own system of maintaining communication with other care providers, through scheduled phone calls, letters, e-mail, or regular meetings. When feasible, the inclusion of a mental health clinician in a pediatric practice provides a streamlined and built-in mechanism for meeting the mental health needs of children in the practice. Pediatricians may consider teaming with a mental health colleague in providing educational services for patients and families such as newsletters, parent groups, or informational Web sites. Economic factors and limited access to psychiatric services have led to the development of innovative models of collaboration. Child psychiatrists may consider approaching pediatric colleagues to endorse their application to insurance panels. Pediatric practices can develop an ongoing relationship with a child psychiatrist in the community for informal "curb-side" consultations, or more formalized meetings in which the psychiatrist may meet with a group of pediatricians for case discussions. Technologies such as telemedicine also have made it possible for primary care providers in more remote areas to have access to specialized consultation.

## PSYCHOSOMATIC DISORDERS

The assessment and treatment of psychosomatic disorders in childhood is an area that truly reflects the interface between pediatrics and child psychiatry. Traditionally, the term *psychosomatic* has been used to describe illnesses in which a loss or change in physical functioning appears to be due to psychological factors ([Graham, 1985](#)). Psychosomatic symptoms may range from frequent somatic complaints to more dramatic conversion disorders. Pediatricians often feel frustrated by children who have no organic findings or spurious findings, yet continue to be symptomatic despite their best treatment efforts. Despite this frustration, pediatricians may be reluctant to make a psychiatric referral because of concerns that the child or family will be angered at the suggestion of an emotional component to the physical symptoms. In many of these families, the child's symptoms are presented as the "problem" when in fact they are a defensive solution that avoids confronting emotionally charged family issues. Parents also may communicate their resistance to psychological interventions or they may "doctor shop" to avoid psychiatric referral. As a result, these children typically have been referred to multiple specialists in ongoing efforts to find a specific cause for their symptoms.

[Jellinek and Herzog \(1991\)](#) have suggested a framework that child psychiatrists can use to guide pediatric colleagues in broaching the need for psychiatric consultation in psychosomatic cases:

1. Avoid coming to a final etiologic diagnosis. Suggest that both physical and psychological avenues require further exploration.
2. Have the pediatrician gather information about psychosocial history, family psychiatric history, sources of stress, and emotional consequences of the child's dysfunction. This assessment may identify psychological needs that are quite separate from the child's physical symptoms.
3. Suggest that a psychiatric evaluation is needed to address the child's dysfunction rather than only to pursue a diagnosis.
4. Recommend that the pediatrician be patient with the family, allowing enough time to foster a sufficient alliance with the family.
5. Recommend an "if . . . , then" approach. The pediatrician should discuss with a reluctant or resistant family that if the last round of appropriate diagnostic tests is negative, then a psychiatric consult is warranted.
6. If pediatric hospitalization is necessary, initiate the child psychiatric consult on the first day. This conveys to the family that the psychiatric evaluation is an important part of the child's work-up.
7. Some families will continue to refuse a psychiatric referral. Support the pediatrician in recognizing this refusal as a family problem and not as a reflection of the pediatrician's competency.
8. If the family continues to pursue high-risk, invasive procedures, alert the pediatrician that the child may be at risk for abuse. In extreme cases, referral to social service agencies may be required to protect the child.

Once the family agrees to accept a psychiatric referral, the child psychiatrist can proceed with careful evaluation. This evaluation often is done in the midst of tension and concern on the family's part that they will be told that the problem is "all in your child's head." By adopting a gentle, nonconfrontational approach, the psychiatric consultant can reduce the family's anxiety. Framing the absence of findings in a positive manner is helpful ("The good news is that your child does not appear to have a long-term, irreversible illness"). Explaining that pain, physical disability, or depressed mood in childhood can interfere with developmental tasks encourages the family to focus on ways to increase the child's functioning. Acknowledging the family's frustration at not having a definite etiology for their child's symptoms is important. Providing specific interventions, such as nutritional plans and physical therapy, helps the child and family feel more in control and may gradually allow the child to give up the physical symptoms in a dignified manner.

As the child psychiatrist earns the family's trust, a more comprehensive understanding of the child's and family's psychosocial functioning can be developed. In addition to physical symptomatology, does the child have evidence of further psychiatric dysfunction, such as anxiety or depressive symptoms ([Livingston, 1993](#))? Is there a family history of somatic presentations? Is this a child with a history of traumatic life events, school problems, or poor social competence ([Walker et al., 1994](#))? Are there family stressors such as marital tension, divorce, or parental substance abuse? Is the child unable to tolerate any awareness of unpleasant, angry affects, or internal conflicts ([Campo and Fritsch, 1994](#)), or is the child a symptom bearer for the family? Each of these areas potentially may become a focus for treatment independent of the presenting physical symptoms ([Campo et al., 1999](#); [Fritz et al., 1997](#)).

## MUNCHAUSEN SYNDROME BY PROXY

Munchausen syndrome by proxy [factitious disorder by proxy (FDP)] is an extreme form of child abuse in which a parent's psychopathology is manifested in the pediatric arena. Its key feature is the repeated, intentional production or feigning of symptoms in a child by a caretaker. The motivation for this behavior is thought to be a complex psychological need by the caretaker to assume the sick role by proxy. This syndrome is particularly difficult to diagnose and treat. Because of its potentially lethal outcome and high risk of long-term physical and psychological morbidity, recognition and close collaboration between pediatrics and child psychiatry is critical. The following features may initially alert the pediatrician to suspect a case of FDP ([Schreier and Libow, 1993, 1994](#)) (see also [Chapter 102](#)):

- The child most commonly is of preschool age.
- The perpetrator is usually the child's mother, and often has had some degree of medical training.
- Most symptoms involve the gastrointestinal, the genitourinary, or the central nervous system.
- The child's signs and symptoms may be incongruous with the presented history or usual clinical course of illness.
- Signs and symptoms may occur only when the caretaker is present, or do not seem to respond to prescribed treatments.
- The caretaker may report that the child is allergic to a wide variety of foods or drugs.
- Laboratory results often are inconsistent.
- The caretaker may report that commonly accepted treatments have not been effective or have been poorly tolerated.
- The caretaker may remain constantly at the child's bedside, but may not appear appropriately worried about the child's illness.

Because of the difficulty in diagnosing and addressing this disorder, a supportive team approach between medical and psychiatric, nursing, and social work staff is essential. If a case of FDP is suspected, the following interventions should be pursued ([Sugar, 1990](#)):

- Ensure the child's safety while a confirmatory work-up is being completed. If the child is hospitalized, he or she should be under constant observation by medical staff. If the child is not hospitalized, or if the caretaker insists on taking the child home before the work-up is complete, medical staff need to decide if the child is in sufficient danger to seek legal custody through the courts or child protective services.
- Inform hospital attorneys that a case of FDP is suspected.
- Attempt to form an alliance with family members while obtaining as much medical and psychosocial history from all available sources.
- Initiate child psychiatric consultation for assessment, treatment planning, and support for the child, family, and staff.
- Encourage all staff involved to communicate openly about all aspects of the child's care, to reduce the possibility of the perpetrator "splitting" members of the team.
- Carefully discuss with all staff involved how to present the conclusion to the family, once enough evidence has been obtained to make the diagnosis. Follow-up with needed services (child protective agencies, mental health services for the child and family) should be in place when presenting diagnosis and treatment options to the family.
- Carefully document all treatment and interventions, as repeat offense is common and definitive diagnosis is often difficult to make.

Adapted from *Bright Futures in Practice: Mental Health* ([Jellinek et al., 2002](#)).

## PRESCRIPTION OF PSYCHOTROPIC MEDICATIONS

The use of psychotropic medication in the treatment of children and adolescents has increased significantly since the mid-1980s. Primary care providers, with varying levels of training in and comfort with psychopharmacology, are prescribing a large proportion of these medications ( [Kelleher et al., 2000](#); [Pincus et al., 1998](#); [Zito et al., 2000](#)). Stimulants have long been used in pediatrics for the treatment of ADHD, and the use of selective serotonin reuptake inhibitors (SSRIs) in primary care pediatrics is now commonplace. [Rushton et al. \(2000\)](#) reported that in a survey of over 1,100 pediatricians and family physicians in North Carolina, 72% had prescribed an SSRI for a child or adolescent, yet only 8% of these physicians felt that they had adequate training in the treatment of childhood depression. Recognizing the widespread prescriptions of stimulants and SSRIs by pediatricians, *Bright Futures in Practice: Mental Health* ([Jellinek et al., 2002](#)) has suggested a framework for the use of these medications in primary care (see [Appendix 3](#) and [Appendix 4](#)). The American Academy of Pediatrics has developed its own set of guidelines for the treatment of ADHD in primary care practice. Primary care providers who do prescribe psychotropic medications to children often require guidance from psychiatric colleagues to clarify diagnosis and offer input regarding medication dosage and combination, and duration of treatment.

## OTHER AREAS OF COLLABORATION

The previous sections describe a few of the common areas of interaction between pediatrics and child psychiatry. Other areas include supporting children with chronic illness, children whose parents are coping with medical illness or other difficulties, children who have experienced physical trauma, and children who are coping with more common stressors (e.g., parental divorce). Although these topics are not covered here (see also [Chapter 33](#), [Chapter 103](#), [Chapter 109](#)), they also offer rich opportunities for collaboration.

## ECONOMIC CONCERNS

In the face of increasing psychosocial dysfunction in children and adolescents, pediatricians and child psychiatrists grapple with economic and market forces that have affected provision of high-quality care and have created barriers to effective collaboration between pediatrics and child psychiatry. Managed care practices have resulted in carve-outs of mental health benefits from general pediatric care, lower reimbursement rates for mental health services, and cost shifting for mental health services to general pediatrics and the public sector. Findings released by the federal Substance Abuse and Mental Health Services Administration in July 2000 reported that two-thirds of substance abuse treatment and over half of mental health treatment is paid for by the public sector ( [Jellinek and Little, 1998](#); [Substance Abuse and Mental Health Services Administration, 1997](#)).

Data are emerging that untreated mental health problems in children lead to chronic psychosocial morbidity, and that children with psychosocial dysfunction have higher costs of health care use ( [Bernal et al., 2000](#); [Kelleher and Starfield, 1990](#)). Noncompliance with medical care also increases health care costs. Unfortunately, these data have not yet significantly affected medical practice or market forces because the financial "payback" for the early detection and treatment of psychiatric problems in children is not evident in the time frame that matters to the carve-out company.

Addressing these barriers will require ongoing efforts in many arenas. Pediatricians and child psychiatrists both need to play critical roles in advocating for the inclusion of mental health care benefits as an integral part of general pediatric care, in negotiating contracts that allow for quality services, and in appealing denials of needed care from insurers. By implementing quality assurance efforts such as screening and tracking measures for children at risk, both disciplines also can contribute to databases with the potential to shape health care policy and managed care practices.

## CONCLUSIONS

This chapter has focused on the interactions between child psychiatrists and pediatricians in managing and treating psychosocial problems in children and adolescents. Pediatricians see many more children than child psychiatrists; they often are the first point of contact for psychiatric concerns ranging from common disorders such as ADHD, learning disorders, and substance abuse, to rarer problems such as psychosis. Currently, the system as a whole functions short of the sum of its parts. Children's needs are viewed in a narrow context, depending on the arena in which a child is seen. Medical care is separated from academic services, which in turn often are segregated from a child's family and community experiences. In a system this fragmented, thoughtful assessment and treatment of children's psychiatric needs is a daunting task.

Models do exist for the provision of more seamless and comprehensive care for children. State agencies, school-based health clinics, and community partnerships with schools can be integrated to provide high-quality care. In an ideal system, the roles of pediatricians and child psychiatrists would be established within this network of care. As a first step toward this goal, both fields need to work together in identifying children at risk, determining when and how to refer, understanding the obstacles to effective collaboration, and advocating for their patients' care.

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## Appendix 126.1 / Psychosocial Questions for Use in Clinical Pediatric Office Practice

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### FAMILY

1. Who is in your family and who lives with you in your home?

### Household Stability

2. How many times has your family moved?
3. Have there been any changes in your family or life style since the last time we met?
4. How are you and your child coping with this change?

### Marital Conflict and Divorce

5. Are you and your spouse able to work together in raising your child?
6. How do you handle disagreements in raising your children or between yourselves?
7. When things get out of hand, how far do they go?

### Crisis

8. Has your family ever been through a major crisis?
9. How did you all deal with that crisis?

### Parental Mental Health

10. Have you or any of the members of your family ever suffered from a mental illness or substance abuse problem?
11. Are you aware of any effects that this problem may have had on your child or his or her care?

### SCHOOL

12. How does your child like school and his or her teachers? *How do you like school and your teachers?*
13. How did your child do in first grade?

#### Academic Achievement

14. What grades does your child get on his or her report card? *What grades do you get on your report card?*
15. Has your child ever stayed back a grade?
16. Has your child ever been in special education classes?

#### School Attendance

17. Have you ever had difficulty getting your child to go to preschool or school?
18. Has your child ever missed more than 10 days of school in 1 year? What was the reason?
19. Has your child ever cut school? How often?  
*Have you ever cut school? How often?*

#### PEER RELATIONSHIPS

20. How does your child get along with peers?

#### Friends

21. Does your child have a best friend? (In adolescence . . . a group of good friends?)  
*Do you have a best friend?*

#### Bullies and Victims

22. Does your child seem to enjoy picking on, bothering, or bullying weaker children?  
*Did you ever like to pick on or bother another kid?*
23. Does your child always seem to get picked on?  
*Are you a kid who always gets picked on?*

#### ACTIVITY

24. What does your child like to do?  
*What do you like to do?*
25. Is there something your child is really good at doing?  
*Is there something you are really good at doing?*

#### EMOTIONAL HEALTH

26. What emotions do you see in your child these days?  
*Everyone feels sad or angry at times. How about you?*
27. Has your child suffered the loss of someone important to him or her?
28. Has your child ever been treated for an emotional or school problem?

#### MISCELLANEOUS TOPICS

#### Poverty

29. Have you ever been on welfare or been unable financially to support your family?

#### Injury

30. Has your child ever had to go to the emergency room for treatment of an injury? How many times?

#### Substance Abuse

31. Do you ever drink alcohol? If yes, then . . .
  - C Have you ever felt the need to **cut down** on your drinking?
  - A Have people ever **annoyed** you by criticism of your drinking?
  - G Have you ever felt **guilty** about your drinking?
  - E Have you ever taken a morning **eye opener** to steady your nerves or get rid of a hangover?
32. Do you think your child is drinking alcohol or using drugs?  
*Have you ever tasted beer, wine, or alcohol?*  
*How old were you when you began to drink alcohol?*

#### Risk-Taking Behavior

33. Is there anything that your adolescent is doing or has done to himself or herself that has you really concerned?

#### Suicide

34. Has your teenager ever tried to intentionally hurt himself or herself?  
*Did you ever feel so upset that you wished you were not alive or that you wanted to die?*  
*Did you ever do something that you knew was so dangerous that you could have gotten hurt or killed?*  
*Did you ever intentionally try to kill yourself?*

Reproduced from Hack S, Jellinek MS: Historical clues to the diagnosis of the dysfunctional child and other psychiatric disorders in children. *Pediatr Clin North Am*45:25–48, 1998. W.B. Saunders, with permission.

\*Questions in regular type are questions for parents. Questions in italics are questions for children.

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### Appendix 126.2 / The Pediatric Symptom Checklist

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Please mark under the heading that best fits your child



	Never	Sometimes	Often
1. Complain of aches or pains	___	___	___
2. Spends more time alone	___	___	___
3. Tire easily, little energy	___	___	___
4. Fidgety, unable to sit still	___	___	___
5. Has trouble with teacher	___	___	___
6. Less interested in school.	___	___	___
7. Acts as if driven by a motor	___	___	___
8. Daydreams too much	___	___	___
9. Distracted easily	___	___	___
10. Is afraid of new situations	___	___	___
11. Feels sad, unhappy	___	___	___
12. Is irritable, angry	___	___	___
13. Feels hopeless	___	___	___
14. Has trouble concentrating	___	___	___
15. Less interested in friends	___	___	___
16. Fights with other children	___	___	___
17. Absent from school	___	___	___
18. School grades dropping	___	___	___
19. Is down on himself or herself	___	___	___
20. Visits doctor with doctor finding nothing wrong	___	___	___
21. Has trouble sleeping	___	___	___
22. Worries a lot	___	___	___
23. Wants to be with parent more than before	___	___	___
24. Feels that he or she is bad	___	___	___
25. Takes unnecessary risks	___	___	___
26. Gets hurt frequently	___	___	___
27. Seems to be having less fun	___	___	___
28. Acts younger than children his or her age	___	___	___
29. Does not listen to rules	___	___	___
30. Does not show feelings	___	___	___
31. Does not understand other people's feelings	___	___	___
32. Teases others	___	___	___
33. Blames others for his or her troubles	___	___	___
34. Takes things that do not belong to him or her	___	___	___
35. Refuses to share	___	___	___

Courtesy of Michael S. Jellinek, M.D (available on the web: [psc.partners.org](http://psc.partners.org)).

### Appendix 126.3 / Use of Stimulants in Pediatric Primary Care Practice

Children and adolescents with attention deficit/hyperactivity disorder (ADHD) usually require multiple interventions to address their difficulties. In addition to behavioral treatment and collaboration with the family and school, pharmacologic interventions have been shown to be effective in improving functioning in children and adolescents with ADHD. Guidelines for considering a medication trial are offered in the following.

- Given the prevalence of ADHD and its responsiveness to stimulant medication, many primary care clinicians may feel comfortable considering a medication trial in children and adolescents who have good family support and no significant coexisting disorders. Ongoing communication with the child's family and school (following guidelines for confidentiality) through mechanisms such as rating scales is essential in monitoring a child's response to medications.
- Children and adolescents who do not respond to a stimulant trial, who experience adverse effects, or who show evidence of depressive or anxiety symptoms, substance abuse, developmental delays, history of tic disorders, or significant family stress may be more complicated to treat. Although some primary care clinicians have experience in treating ADHD symptoms along with these associated problems, other providers may wish to obtain psychiatric consultation.

Adapted from Jellinek M, Patel B, Froehle M: *Bright Futures in Practice: Mental Health*. Arlington, VA, National Center for Education in Maternal and Child Health, 2002.

### Appendix 126.4 / Use of Antidepressants in Pediatric Primary Care Practice

In addition to interventions such as individual therapy and work with the child's family, school, and peers, medications may be helpful to some children and adolescents with depressive symptoms. In prepubertal children with depression, the diagnostic process, treatment planning, and medication management issues are sufficiently complex to warrant a referral to a child psychiatrist. For older adolescents, primary care clinicians may choose to treat moderate depressive symptoms with medication. In these cases, periodic consultation with a child psychiatrist colleague regarding medication selection, dosing, duration of treatment, and management of adverse effects is highly recommended. Guidelines for considering antidepressant treatment for childhood and adolescent depression are offered in the following.

- Clinicians should be aware that bipolar disorder will develop in 20% to 40% of children and adolescents with a major depressive episode. Therefore, any child or adolescent who undergoes a trial of an antidepressant should be closely monitored for signs of increased agitation or irritability.
- Children and adolescents with coexisting difficulties such as suicidal ideation, significant irritability or impulsivity, anxiety, attention deficit/hyperactivity disorder, substance abuse, or significant conduct problems are likely to present diagnostic and treatment challenges that ideally are managed by a child psychiatrist.
- Although the safety and efficacy of selective serotonin reuptake inhibitor (SSRI) antidepressants has not been as well established for children and adolescents as for adults, available data indicate that the short-term use of SSRIs appears to be safe and potentially useful in the treatment of childhood and adolescent depression.
- In moderately depressed older adolescents with good family support, primary care providers may consider using an SSRI in certain situations:
  - In adolescents with a clear family history of depressive disorders that have responded well to medication treatment
  - In adolescents who had previously been functioning well, with acute impairment due to depressive symptoms
  - In adolescents whose depressive symptoms have continued despite individual, group, or family therapy
- An adolescent who does not respond to an initial trial of an SSRI or who experiences adverse effects with a medication trial should be referred for further psychiatric evaluation.

Adapted from Jellinek M, Patel B, Froehle M: *Bright Futures in Practice: Mental Health*. Arlington, VA, National Center for Education in Maternal and Child Health, 2002.

# 127 POLICE–MENTAL HEALTH RESPONSES TO CHILDREN EXPOSED TO VIOLENCE

## The Child Development-Community Policing Program

Steven Marans, Ph.D., Robert A. Murphy, Ph.D., and Steven J. Berkowitz, M.D.

- [The Scope of Violence](#)
- [Community Policing and Effective Partnerships](#)
- [Police Officers, Mental Health, and Specialized Training](#)
- [Changing Mental Health and Policing Systems of Care](#)
- [The Child Development Community Policing Program](#)
- [The Child Development-Community Policing Program Principles of Collaboration](#)
- [Seminars in Child Development](#)
- [Changes in Police Responses](#)
- [Collaborative Responses to Trauma: The Child Development-Community Policing Program Acute Response](#)
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### THE SCOPE OF VIOLENCE

It is well established that exposure to interpersonal violence disrupts the basic preconditions for optimal child development. Children may be traumatized in the face of stabbings, beatings, and shootings, because they are unable to contain the stimulation within existing mental and neurophysiologic structures that enable accommodation and assimilation. When systems are immobilized, a cascade of psychological and neurophysiologic processes ensues. The disruption of psychological and biological systems may lead to symptoms of hyperarousal, withdrawal, and re-experiencing of traumatic events; other children may develop specific, circumscribed symptoms, such as disruptions in sleeping, eating, and toileting ( [Boney-McCoy and Finkelhor, 1996](#); [Foy and Goguen, 1998](#); [Gorman-Smith and Tolan, 1998](#); [Marans and Adelman, 1997](#); [McCloskey and Walker, 2000](#); [Pfefferbaum, 1997](#); [Pynoos, 1993](#); [Terr, 1991](#)). As a result, they may become distracted, unable to concentrate or pay attention in school or at home. Transient oppositional behavior also may emerge and represent both an attempt to reassert power in the face of feeling vulnerable and an effort to cope with the anxiety and hyperarousal caused by the dysregulation of the stress response systems. When children are exposed to the dangers of violence on a chronic basis, however, symptoms may no longer serve the function of restitution and recovery. Rather, symptoms become chronic and maladaptive such that physiologic, cognitive, and emotional capacities are severely impaired. Exposure to chronic violence, which is often punctuated by further acute episodes, is associated with increased depression and anxiety, alcohol use, and lower school achievement ( [Garbarino et al., 1992](#); [Martinez and Richters, 1993](#); [Pfefferbaum, 1997](#); [Widom, 1999](#)). A summary of children's responses to traumatic events is presented in [Table 127.1](#).

Type of Reaction	Clinical Manifestations
Affective distress	Irritability, aggressive behavior, angry outbursts, lethargy, difficulty being soothed, tearfulness, sadness, crying, fearfulness
Cognitive changes	Difficulty concentrating, inattention
Reexperiencing	Reoccupation with frightening thoughts and feelings, increased day dreaming, sudden changes in quality of play, repetitive play related to violence and other traumatic experiences, decreased creativity and spontaneity of play
Regression	Loss of previously obtained developmental skills, acting immature or "babyish"
Regulatory disruption	Hyperarousal, exaggerated startle response, distress in response to traumatic cues or reminders (direct and indirect), lability of mood and behavior
Separation anxiety	Substantial difficulty with separations, complaints of loneliness, increased dependence on caregivers
Sleep disturbance	Nightmares, difficulty falling asleep, waking during the night, enuresis
Somatic complaints	Headaches, stomachaches, aches and pains without medical etiology
Withdrawal	Loss of interest in games, school, or pleasurable activities, social withdrawal, reduced spontaneity, display of range of affect, lack of appropriate affect, dissociation, avoidance of traumatic cues or reminders

**Table 127.1. Summary of Children's Responses to Traumatic Events.**

Most tragically for children and society, another long-term adaptation to ongoing violence is the perpetration of violence. There may be a common progression from witnessing to being the victim of and then to engaging in violence in densely populated, impoverished urban areas where the prevalence of violence is especially high (Marans et al., [1996a,b](#)). The children who shoot and stab other children all too often have been exposed to the abusive relationships of family members, and have themselves been at the receiving end of beatings and neglect ( [Kaufman and Henrich, 1999](#)). Frightened victims become frightening assailants. A longitudinal study by [Thornberry and colleagues \(1994\)](#) demonstrated this pathway. Children who had been victims of violence within their families were 24% more likely to report engaging in violent behavior as adolescents than those who had not been maltreated in childhood. Adolescents who were not directly victimized, but who had grown up in families in which partner violence occurred, were 21% more likely to report violent delinquency than those not so exposed. Finally, children exposed to multiple forms of family violence reported twice the rate of youth violence relative to those from nonviolent families.

In spite of the significant decreases in juvenile crime rates since the early 1990s ( [Snyder and Sickmund, 1999](#)), far too many children living in the United States experience acts of violence that leave them vulnerable to the psychological trauma associated with their exposure:

- In 1998 more than 2.7 million youth between the ages of 12 and 19 years were victims of violent crime, including simple and aggravated assault, rape and robbery ( [Rennison, 1999](#)).
- Children and adolescents are 4.5 times more likely to be the victims of serious crime than are adults ( [Centers for Disease Control and Prevention, 1997](#)).
- In a given year, 1 million children are substantiated victims of abuse or neglect. At least 130,000 children are sexually abused, and 2,000 children die as a result of abuse or neglect ( [Bash, 1997](#)).
- Each year more than 10 million American children witness a physical assault between their parents. There is repeated violence between the parents in two-thirds of these cases ( [Straus and Gelles, 1990](#)).
- Child abuse occurs in 50% of families where domestic violence also occurs ( [Centers for Disease Control and Prevention, 1999](#)).

In light of our current knowledge about the complex interplay of psychological and neurobiologic responses to overwhelming events, ample reason exists for concern about the enduring consequences for children's maturation and development when potentially traumatic responses to violence exposure go unrecognized. Although essential, primary prevention programs alone are unlikely to avert the acute and chronic symptomatic sequelae of children's exposure to violence. Early intervention immediately following a serious violent or traumatic event is both possible and desirable. Psychological and pharmacologic interventions may prevent the acute stress response from becoming permanently dysregulated. By intervening immediately after the event, children and families may be more able to integrate their experiences in a manner that ameliorates the fear and stress that in turn exacerbates and perpetuates acute biologic and maladaptive coping responses.

Children and families who are most vulnerable to the deleterious effects of violence may also lack access to sufficient mental health care. In addition to constraints on overall resources, families who simultaneously contend with the chronic and acute effects of violence, poverty, social disadvantage, and individual and family psychopathology may be unable to access traditional clinic- or office-based services when they are available. In essence, those with the greatest need may receive the least care. In response to these concerns, there has been a proliferation of community-based services where mental health care is provided in homes, schools, and community settings, with an emphasis on acute stabilization of individual and family psychopathology and collaboration with other community providers ( [Aber et](#)



al., 1998; Woolston et al., 1998).

### Community Policing and Effective Partnerships

Beginning in the 1970s and gathering momentum in the latter 1980s, community leaders began to recognize that the academic, social, and emotional health of youth could not be separated from their feelings of safety and security in their schools and neighborhoods. Leaders in law enforcement recognized the need for a change in traditional policing as youth violence peaked. Many departments began a critical transition from a crisis-oriented and response-driven system to one reflecting the principles of problem-oriented community policing (Thurman, 1995), and many of these departments adopted a specific emphasis on the concerns of children and youth. In contrast to a reactive, crime-fighting response, community policing adopts a “proactive, solution-based, and community driven” perspective on policing in which the need to arrest offenders is integrated with a preventive approach based on relationships and problem-solving efforts with a range of community constituencies.

Despite variation across communities, departments that have embraced community policing emphasize accessibility of police to the citizens they serve through problem solving and preventive approaches to policing, proactive order maintenance designed to diffuse situations prior to an emergency complaint, collaboration with community agencies, neighborhood involvement in policing, and sponsorship of community-based crime prevention initiatives (Goldstein, 1987; Thurman, 1995). Community-based policing integrates police officers within the community, where they are known as individuals rather than only by role, and they come to know the people they serve as individuals. Because the model brings police officers into regular, ongoing contact with children and families within a given neighborhood, it requires a new type of police officer with specialized training, supervision, and support.

In a comprehensive review of crime prevention programs, the Office of Justice Programs (Sherman et al., 1998) outlined a range of effective crime and violence prevention programs, yet few feature direct collaboration and integration of psychiatric and law enforcement services. Nonetheless, police can be effective in their efforts to decrease criminal and violent activity among youth. For example, intensified monitoring of repeat offenders by community police and probation officers, as well as additional patrols in areas with high rates of youth crime can ameliorate recidivism. Presumably, these approaches reduce anonymity through supervision and the development of relationships with youth, enhance a sense of security among residents, and foster proactive intervention by police officers.

### Police Officers, Mental Health, and Specialized Training

Police routinely confront the psychological sequelae of violence and psychiatric illness during their patrols. Despite decades of practical experience in responding to psychiatric crises, few police departments provide extensive training in the identification and management of acute mental health problems. Traditionally, their options have been limited to an evaluation of an individual's danger to self or others with resultant alternatives of involuntary psychiatric commitment or arrest (Teplin, 2000). In her recent review, Teplin (2000) noted that the likelihood of arrest was 67% greater for criminal suspects with signs of mental disorder relative to other nondisordered suspects. Two explanations for these findings arise, although neither is exclusive of the other. Police may lack sufficient training in effective responses to psychiatrically disturbed citizens and may rely on arrest in the absence of effective training and awareness of alternative responses. Alternately, they may resort to arrest to secure services in the context of diminished access to behavioral health care in the public mental health sector. Officers may recognize the complex risks and needs among this population that has not been adequately served by mental health or law enforcement alone. From this latter perspective, arrest may serve a therapeutic function through the application of police authority toward the goal of securing necessary mental health care.

The training needs of police departments were highlighted in a survey of mental health crisis responses among 174 urban police departments. More than half (55%) lacked specialized response procedures for mental health crises, despite such incidents comprising a significant proportion of all service calls (Deane et al., 1999). Within the remaining 45% of departments, 3% (six departments) relied on specific officers with crisis intervention training to act as liaisons with mental health providers. Another 12% (20 departments) retained civilians as mental health consultants or providers. The remaining 30% of departments (52) relied on referral to non-police-affiliated mental health professionals. Although most departments, including those lacking a specialized response, perceived their efforts on behalf of psychologically impaired citizens as effective, none were able to point to specific outcomes or benefits that accrued to the individuals in question.

No department specifically addressed the mental health concerns of children, suggesting that the relative paucity of organized police responses to psychiatric concerns is more widespread in regard to children whose difficulties may go unrecognized by officers who lack training and resources for responding to the psychiatric needs of children and adolescents. Training officers to identify psychiatric disorders and symptoms, conduct mental status evaluations, recognize medical syndromes that may cause psychiatric symptoms, understand legal and ethical aspects of involuntary commitment, and utilize interpersonal and communication strategies for interacting with seriously disturbed individuals has improved officers' ability to respond to psychiatric disturbances (Borum, 1999). The effectiveness of police responses to children has not been specifically addressed, despite the ubiquity of children at many of the scenes to which officers respond.

In one of the few evaluations to directly address police and mental health collaboration, the conjoint identification of youth psychopathology by mental health clinicians and police detectives resulted in enhanced treatment access and intervention within an urban European police department (Scholte, 1992). After training juvenile detectives to recognize risk factors for psychopathology among adolescents, detectives began daily rounds with clinicians to discuss juvenile cases from the previous day and develop appropriate treatment recommendations. Programmatic analysis revealed a 74% concordance between youth identified as at risk for serious psychopathology by police and a standardized clinical screening instrument. Although subsequent interventions were not well operationalized, post hoc analyses suggested that a greater number of youth about whom detectives and clinicians consulted engaged in treatment more readily and experienced fewer psychiatric symptoms in the 6 months following referral.

The limited research to date suggests that community policing may be particularly effective at improving residents' satisfaction with police services and subjective feelings of neighborhood safety and security (Catalano et al., 1999; Zhao and Thurman, 1997). In a study of two New York City police precincts that experienced a large drop in civilian complaints, police officers' responsiveness toward citizenry resulted in more positive views of police, as well as fewer complaints about their conduct (Davis and Mateu-Gelabert, 2000). Residents cited a respectful approach by police officers, as well as involvement of patrol and supervisory officers in community initiatives as central to their positive relationship with local police.

As one might expect, adolescents tend to be more ambivalent than children or adults in their views of police. Beyond the expectable reactions of adolescents to figures of authority, a history of criminal victimization, arrest, or questioning by police officers has been associated with negative attitudes toward police among youth (Hurst and Frank, 2000). These findings of ambivalent views toward police should come as no surprise to the developmentally informed officer or clinician who recognizes the significance of adults in positions of authority as figures to be both emulated and defied in the service of identity formation. Comparisons based on types of policing strategies—for example, community policing versus response driven approaches—have not been conducted, but it appears that the attitudes of adolescents toward police vary, depending on individual factors and their previous experiences of victimization, neighborhood crime, and contact with other officers.

### Changing Mental Health and Policing Systems of Care

Despite progress in the development and implementation of community-based mental health interventions, as well as increased attention to the mental health needs of youth in the juvenile justice system, the potential for police officers to exert a specific influence on the mental health of acutely traumatized children and adolescents has been neglected. Unlike the majority of mental health and juvenile justice providers, police officers are, by definition, constantly available to intervene in instances of acute childhood distress and traumatization. Police officers are the first to respond to scenes in which children may be in acute distress, yet they typically lack the resources, in terms of their own training in child-oriented policing and the availability of adequately responsive mental health colleagues, to effect change at the crucial time of their response. Although mental health providers, particularly those working in trauma-related fields, have extolled the importance of acute intervention and its catalytic effects, few have taken advantage of the reality that police officers respond at precisely the moment when previous defenses and ways of coping have faltered, and perhaps, children and families are both more needy and more responsive to intervention.

### The Child Development Community Policing Program

The Child Development-Community Policing Program (CDCP) (Marans et al., 1995; Marans and Cohen, 1993) has capitalized on the shared expertise of police officers and mental health professionals in intervening with children and their families who have been exposed to violence and may be experiencing its posttraumatic effects. The development of the program has been closely allied with research related to children's mental health systems of care (Bickman et al., 1995; Friedman and Burns, 1996), where multiple systems converge in an integrated manner to maximize quality and continuity of care. The Child Development-Community Policing Program extends work related to mental health care systems that have been focused on psychiatric and community support services to encompass law enforcement,

juvenile justice, and domestic violence personnel in an effort to correct the historical isolation of mental health providers from law enforcement and justice personnel.

The Child Development-Community Policing Program was initiated by the Yale Child Study Center and the New Haven Connecticut Department of Police Service in response to shared concerns about the paucity of policing and clinical responses to children and families at greatest psychological risk because of involvement with violence in their homes, neighborhoods, and schools ([Marans and Cohen, 1993](#)). It was recognized that although police officers come in daily contact with children who are victims, witnesses, and perpetrators of violence, they generally did not have the professional expertise, the time, or the other resources necessary to meet these children's psychological needs. Conversely, clinic-based mental health professionals might have been professionally equipped to respond to children's psychological distress following episodes of violence, but these acutely traumatized children were rarely seen in existing outpatient facilities until months or years later, when chronic symptoms or maladaptive behaviors brought them to the attention of parents, teachers, or juvenile court personnel. When there were no collaborative responses immediately following violent incidents, valuable opportunities to intervene were lost at precisely the moment when concerted police and mental health contact could provide both immediate stabilization and bridges to a variety of ongoing services to address their trauma and maladaptive symptomatology. The CDCP program placed mental health professionals in the community, with the police, to develop collaborative strategies for intervening early when violence occurs. The program alters the ways in which policing, mental health, and now other related services are delivered to children and families who are regularly exposed to community violence ([Marans and Adelman, 1997](#); Marans et al., [1995, 1998](#); [Marans and Schaefer, 1998](#)).

The CDCP program involves five central components:

1. **Seminars in Child Development:** The course constitutes approximately 24 hours of seminars and observations in clinical settings (e.g., a child psychiatric inpatient unit, a pediatric emergency room, and a psychiatric clinic in a juvenile detention center) for officers, new clinicians, juvenile probation officers, domestic violence advocates, and child protective service workers. Reflecting the collaborative nature of the CDCP program, the seminars are co-led by a team consisting of a senior/supervisory police officer and an experienced clinician. Seminars focus on principles of child development, human behavior, and trauma as they apply to community policing.
2. **Clinical Fellowships for Police Officers:** Officers who have completed the seminars continue to participate in the program by attending weekly CDCP case conferences, supervising rank and file officers who respond to calls of acute episodes of violence that involve children and families, and coordinating follow-up services for victims, witnesses, and perpetrators. These officers become local and national leaders in the CDCP program and become centrally involved in program dissemination and replication through teaching, presenting, and developing educational curricula.
3. **Police Fellowships for Clinicians:** As police become familiar with aspects of child development, clinicians learn about basic police practices through their attendance at CDCP case conferences, completion of 20 hours of seminars in police practices and protocols, participation in a minimum of 40 hours accompanying officers on patrol, and involvement as an on-call clinician for the acute consultation service.
4. **The Acute Consultation Service:** Clinicians are available by pager 24-hours a day, 7 days a week to the police department for consultation involving children, adolescents, and families who have been involved in violence or other situations that arouse officers' concerns about their psychological/psychiatric status and well-being. Clinicians respond acutely to incidents in which children are affected by violence as witnesses, victims, and perpetrators. Clinicians meet with children in police stations and substations, schools, homes, and emergency rooms and arrange necessary clinic or hospital-based outpatient and inpatient care in the immediate aftermath of violence and trauma.
5. **Program Case Conferences:** Law enforcement officers, clinicians, juvenile probation officers, domestic violence advocates, and child protective service providers meet weekly to discuss and plan coordinated interventions for cases that have been generated through the acute consultation service, as well as through subacute referrals from patrol officers, specialty police investigative units, child protective services, schools, and juvenile probation officers. Discussion at the case conference might involve the potential for clinical engagement and the role of a patrol officer in assuring future safety. For example, clinicians and officers may arrange for a joint follow-up visit to the home of a child who was recently the victim of a neighborhood assault, as the combined police and clinical presence communicates a shared commitment to security and psychiatric intervention.

### **The Child Development-Community Policing Program Principles of Collaboration**

The CDCP program relies on a process of learning about the perspectives and experiences of the other professional as a basis for considering the developmental implications of their respective observations and actions. Clinicians have learned that to be useful to police officers they first must see the clinical phenomena from the officer's point of view. This has been accomplished through the development of fellowships for clinicians and supervisory officers. In the Police Fellowship, clinicians learn about the tasks, demands, and professional needs of officers and move into police settings through spending time with officers during routine patrol, collaborating with police at crime scenes, and sitting in on discussions of case investigations. At the same time, through the Clinical Fellowship, officers become familiar with mental health settings and psychodynamic and developmental perspectives on psychopathology and treatment through observations of clinical activities and consultations.

Although the coordinated police-mental health response remains at the core of the program, the range of additional partners has expanded to include domestic violence advocates, juvenile probation officers, child protective workers, school personnel, clergy, and community members. Wherever the CDCP program has been implemented, innovations have been developed to meet the specific needs and characteristics of each of the local communities. For example, in Charlotte, North Carolina, the early involvement of the county child welfare agency in the CDCP program originated from a preexisting, informal relationship between the police department and child protective services based on their history of collaboration around childhood abuse and neglect cases. In Baltimore's Eastern District, CDCP was implemented with community members and clergy as full partners who are designated as Community Fellows. They are involved in cross-training, acute response, and follow-up activities of the Baltimore program. In Nashville, police department interests in developing new ways of responding to children and adolescents converged with the efforts of the Mental Health Center for Children and Families to better address the psychological needs of battered women. The Nashville CDCP program forged new partnerships that coordinated and expanded services available to all of those affected by domestic violence and introduced acute and longer-term interventions for children and families affected by other forms of violence and related trauma. In Newark, the CDCP collaboration between the Newark police department and the University of Medicine and Dentistry of New Jersey worked closely with the Catholic church and public housing authority to develop additional services in an area of the city with a dense population of families exposed to high rates of poverty and violent crime. Whatever innovations have developed with regard to additional partners or areas of emphasis, the five central program components serve as a basis for the continued development of the Child Development partnerships.

### **Seminars in Child Development**

Child development seminars provide an initial opportunity for participants with diverse backgrounds and professional experiences to develop a common language from which to consider the needs of children and families ([Berkowitz and Murphy, 2000](#); [Marans et al., 1998](#)). The central task of the seminars is to engage officers and other partners in examining how an understanding of emotional, interpersonal, cognitive, and motor development can enhance law enforcement responses to children and families. Particular attention is paid to the ways in which traumatization may be experienced and demonstrated by children at different phases of development, who invariably present with varied histories, family circumstances, and ecologic disadvantage. (See [Table 127.1](#) for a summary of children's responses to trauma and violence.) Exploring the range of psychological and physiologic responses to overwhelming, traumatic events prepares officers to recognize those situations that may place children at the greatest risk for acute and long-term consequences were their exposure to violence goes unnoticed.

Proceeding along a developmental sequence, the seminars highlight the ways in which phenomena originating in an earlier phase of development may be observed in various forms throughout the life cycle. Seminar leaders rely on scenarios encountered in police work, films, and videotapes about children, and cases histories from the acute consultation service to demonstrate that a greater understanding of human functioning need not portend inaction or decreased vigilance with regard to personal safety. Rather, the goal of the seminars is to help officers and others (e.g., juvenile probation officers, child protective workers, educators) discover new ways of observing and formulating responses to children that are informed by an understanding of their development. In addition, officers have the opportunity to establish a more realistic appreciation of the impact they can have on the lives of children and families with whom they interact.

### **INFANCY**

Discussions begin about early development following a first meeting in which seminar members introduce themselves and talk about their expectations of the course. The supervisory officer, who coleads the seminar with a clinician, introduces the topic of infancy by describing the following scene:

You have responded to a complaint of breach of the peace and arrive at an apartment where music is blaring. An angry young mother greets you. The apartment is disheveled and dirty, and three children, ranging from several months to four years of age, are in similar disarray. Diaper changes for two of the children appear long overdue. What is your reaction?

The officers often begin the discussion by expressing their feelings of despair and anger about a scene that is all too familiar. As the instructors probe the nature of



these reactions, the class begins to identify concerns about the babies who are unable to fend for themselves, about the children's physical discomfort, and about the notion that the mother doesn't care enough about them. What emerges from the discussion is the group's awareness of an infant's physical and emotional needs and the role of the mother in mediating distress and providing crucial care and affection. The seminar leaders inquire, "What happens to the infant if those basic needs aren't met?" The answers suggest that the baby will be overwhelmed with pain, discomfort, and despair because the baby is not yet equipped to feed, clothe, comfort itself, or satisfy the demands of its feelings on its own. The leaders ask for more details, and the class responds by identifying developmental capacities and vulnerabilities—the absence of verbal language, motor maturation and coordination, cognitive processes for problem solving, and finally, the utter reliance of the infant on the mother for the experience of physical and emotional well being.

Attention is then focused on the young mother. "How," the leaders ask, "Do we understand this mother's apparent insensitivity or incompetence?" The discussion must first address the mother's surly response to the officers and their subsequent indignation. Seminar leaders introduce clinical phenomena that underlie presenting behavior, and participants begin to discuss symptoms that they quickly recognize but have not necessarily associated with depression and stress. Invariably the discussion turns to examples of irritability and substance abuse, which becomes an opportunity to discuss the complex biological and psychological relationship among stress, anxiety, depression, and substance abuse.

In addition, the concept of transference is introduced. The seminar leaders expand the discussion of a young woman apparently unable to look after her children, let alone herself. They ask, "How might she feel about herself?" The answers vary. "Like a failure? . . . Maybe she just doesn't care!" The seminar leaders ask, "Given either of those possibilities, how might she feel when two police officers come to her door?" The officers respond, "Like we're going to tell her off, tell her what she should be doing, how she should behave." "Who are you to her at that moment? Who tells you you're not getting it right, messing up? Parents? Teachers? A critical boss or colleague?" In one session, an officer interjected, "Right, and then when she feels criticized, she takes on an obnoxious attitude and treats us like dirt." Another officer added, "As though she already knows who you are."

In this particular discussion, the clinical coleader suggested that perhaps from the moment of their arrival, the officers represented something very familiar to the young woman. The clinician added, "Before you open your mouth, you may be the critical voice, the presentation of authority that agrees with her own self-criticism and assessment of personal incompetence. What is it like to feel inadequate and to have someone who, by their very presence on your doorstep, points out your own shortcomings, real and imagined? Is it possible that her surly and combative response serves a defensive function that is triggered by you but is not about you personally?" The discussion culminates with greater appreciation for the complexity of the scene, its interactions, and potential points for response.

#### ATTACHMENT

The Robertsons' film *John* ([Robertson and Robertson, 1969](#)) is shown in the following session. In the discussion that follows, seminar members describe their responses to viewing this film of a 17-month-old child's efforts to soothe himself in the midst of a 9-day separation from his parents. They note John's attempts to reach out to childcare nurses, cuddly toys, and the observer, and his utter despair when these efforts fail. The discussion compares John with the other children who have spent their entire lives in the residential nursery. Seminar members observe that although seeming unfazed by the limited attention and multiple changes of nursing staff, in contrast to John, these children appear dominated by aggressive, driven, and need-satisfying behavior. In an attempt to understand why John may be so vulnerable to the disruption in care, the participants begin to consider individual differences and the transaction among genetics, temperament, and environment. Who John is as a synthesis of his biology and psychology becomes a central topic for conversation. Slowly and often painfully, as the discussion continues, the simple solution of removal from care when parenting seems inadequate fades along with increased recognition of the central importance of a child's attachment to parents or other primary caregivers. The idea that removal always represents rescue is replaced by a growing appreciation for the complexity of the child-parent relationship. Officers recognize the importance of maintaining consistent and continuous relationships between parents and children and the damage that can occur when that relationship is disrupted. In addition, seminar members have a fuller understanding of the balance between the child's physical and emotional needs, the developing capacities to respond to them and the distress that follows when children's needs are persistently unmet.

#### TODDLERHOOD

The link between psychological processes, overt behavior, and the observers' responses is pursued as the seminar moves into the next session in which the hallmarks of the toddler phase are introduced. Focusing on issues of power, size, and capacities, officers discuss the ways in which the challenges and conflicts of early childhood are repeated later in life. This is most evident in situations where police authority is required to contain the impulses and actions of people who are not able to contain them. When they intervene in fights, civil disorders, drug raids, traffic stops, and the like, officers may often feel like parents attempting to set limits on behavior and the intensity of toddlers' demands. It is often at these moments that, in their positions of authority, the police may easily become the lightning rod for the explosive feelings individuals may have when they feel that their power and capacities are challenged or curtailed. Officers report that appreciating the dynamics and earliest origins of these encounters helps them to respond with greater emotional distance and calm. Like parents of young children, the ability of police to maintain professional distance may spell the difference between a short-lived tantrum and a protracted and dangerous battle.

#### PRESCHOOL

As officers participating in the seminars view videotapes of 4- to 6-year-old children engaged in imaginative play, they are able to consider the preschool child's use of fantasy, identifications, and burgeoning cognitive and physical resources to master new demands. In the process, officers appreciate the fact that children's perception of, and communications about, real events often reflect the interplay of fact and fantasy. For example, in one sequence a 5-year-old boy is seen introducing a play scenario in which puppets designated as a mother and father, kiss, fall down covered in blood, and are then taken to the hospital by ambulance. Appearing in the context of other play sequences that depict family life, the officers in the seminar often assume that the child has been exposed to domestic violence or has been abused himself. Officers then learn about the boy's stable home life and overall developmental health and that he had recently been in a minor traffic accident with his mother, who sustained a small cut that required only a single bandage. The boy's "factual" account of the accident was far more elaborate, involving a description of his mother's copious bleeding and serious damage to the car, when neither actually had occurred. From this example, seminar participants reflect on the interplay between the boy's experience of the accident and developmentally expectable concerns about aggression, bodily damage, and intimacy between parents. As a result, they acquire greater appreciation for the multiple ways in which young children tell their stories about life events. From this perspective, previous assumptions about children's testimony during police interviews and courtroom proceedings can be evaluated from an informed and critical developmental perspective, as officers recognize that children may invest their versions of events with more than the simple facts needed for police investigations ( [Portwood et al., 2000](#); [Wells et al., 2000](#) ).

#### LATENCY AND ADOLESCENCE

Subsequent seminars provide a similar level of detail in examining the developmental tasks of the latency and adolescent stages of development. Failures in negotiating oedipal conflicts over competition, envy, love, and hate and the often unstable, overstimulating home situations in which children reside are explored in discussions of latency age children who come to the attention of police because of their antisocial activities. Similarly, puberty is discussed in terms of the intensification of struggles over sexual and aggressive urges. Discussion of each phase of development is used as another opportunity to consider the impact of violent traumatization and the ways in which individual history and normative tasks and challenges shape the child's experience of events and the extent of symptomatic responses to them.

Seminar leaders introduce phases of development by asking officers to describe the most salient aspects either observed or assumed to play a part in a given period of life. As the discussion evolves, officers invoke their own memories as a vehicle for understanding the behaviors they observe and encounter on the street and as a way of becoming conscious of the complicated identifications that these interactions may evoke. For example, when discussing puberty and early adolescence, officers may describe their concerns about provocative, tough, drug-involved, or pregnant teens and the frustration they experience when logic and warnings about consequences seem to have no impact on behavior. As they begin to talk about their own experiences of this phase of development, however, their frustration and angry dismissal are substantially altered. Officers may describe memories from their own lives or about particular children whom they have met over the course of their work and have been unable to forget. Stories of fighting, social isolation, school difficulties, and losses alternate with the ones about best friends, romantic relationships, team sports, and the like. The discussions inevitably focus on concern about body image, group acceptance, struggles with parents, losses, and the overarching experience of embarrassment and urgency in the competing wishes for competent, independent functioning and continued reliance on parents for direction and security. All of the accounts speak to the vulnerability, anxiety, and loneliness so common in this period of development and the various means used to defend against these feelings.

John Singleton's film, *Boyz n' the Hood* ([Singleton, 1991](#)), and Michael King's award-winning documentary film, *Bangin* ([King, 1999](#)), are used as the "texts" for seminars that address adolescence. Each of the films is used to initiate discussion about development in an especially familiar territory: the challenges, hopes, and

dilemmas inherent in adolescence. The discussion of *Boyz n' the Hood* moves from issues of race and inner city socioeconomics to a focus on the internal and external contributions to the fate of the two brothers in the film. One becomes a gun-toting drug dealer and the other a high-school football star bound for college, until he is shot dead by gang members. *Bangin'* follows two cases involving the death of adolescents who were victims of violence and uses interviews with their peers and other adolescents who have been incarcerated because of their involvement in violent crimes. Both films provide an opportunity to focus on the interplay between early development, trauma, the presence or absence of intervention, and the positive and tragic outcomes seen in adolescence ( [Marans and Adelman, 1997](#)).

As the seminars conclude, officers increasingly refer to their reactions to the scenes of violence and suffering they confront on a daily basis. From the perspective of their internal experience they may speak of sealing over and minimizing their emotions, "getting used to it," and distancing themselves as best they can from their troubling experiences. Others describe displacing their frustration onto citizens with whom they interact or family members, which may engender a dichotomous world view (i.e., an "us versus them" approach to their most difficult assignments), and a heightened sense of vigilance to real and imagined danger. The range of officers' responses are discussed in terms of the defensive functions they may serve in warding off unwanted feelings of fear, inadequacy, sadness, despair, and anger, as well as the potential interference they may pose in achieving the desired goals of their interventions.

### Changes in Police Responses

Regardless of the setting, the aim of the seminar discussions for both officers and clinicians is to adopt, to the extent possible, the unique perspectives of individual children, to place themselves in the position of children of different ages, developmental phases, and backgrounds ( [Goldstein, Solnit et al., 1996](#)). For the officers, the opportunity to reflect on what they observe, have a framework for ordering what might have otherwise been too overwhelming to notice, and have colleagues with whom to share the burden of responding at any hour has led to dramatic changes in police practices regarding children. These changes are reflected in officers regularly referring children who have witnessed and experienced violence and, increasingly, children who have committed serious violent offenses, for clinical evaluation and treatment.

Similarly, officers have become more attentive to the potential for dangerous confrontation when they deal with adolescents, especially juvenile offenders, in a harsh manner that may evoke feelings of humiliation and undermine nascent feelings of independence and autonomy. As officers have become regular fixtures in neighborhoods, they have replaced anonymous responses to the groups of youth who congregate on street corners with interactions that are informed by familiarity and individual relationships. From the seminars to the streets, this contact is enhanced by officers' increased appreciation of normative adolescent upheaval that is so often compounded by the despair and impotence associated with severe social adversity. As a result, wholesale condemnation, frustration, and anger are not the only responses to the provocative or illegal behavior with which adolescents confront them on a daily basis. Officers recognize that by dint of their authority and uniformed presence, they will often bear the brunt of powerful emotions that are displaced onto them by the people with whom they find it most difficult to work. Knowing that the origins of such an intense, emotional response is not personal, even when it feels that way, has led to a more judicious and strategic use of police authority. This has been especially true with regard to police-adolescent interactions on the street. In New Haven for example, police report that the sometimes necessary imposition of authority (e.g., clearing a street corner known for drug activity, keeping public noise down, picking up truant students) is more frequently met with compliance rather than an immediate escalation to violent confrontation and arrest because of their ability to assert their authority in a manner that discourages an escalation of conflict and resistance to police authority by the youth who are involved.

Since the inception of the CDCP program, each police officer in New Haven, from line officer to senior supervisory officer, has been trained in the principles of child development and acute traumatic responses. The police have welcomed participation in the program and have believed that it has enhanced their professional functioning as police officers. Collaboration with their mental health colleagues has also helped them deal with the alienation and burnout of their ceaseless confrontation with senseless pain and injury. Working alongside mental health professionals, police officers have been able to respond more humanely and effectively to thousands of children in New Haven and other CDCP sites who have witnessed and experienced violence, as well as hundreds of children who have committed violent crimes ( [Child Development Community Policing Program, 1998](#)).

Many of these children and adolescents have received psychological intervention within minutes of being exposed to murders, shootings, stabbings, beatings, fires, drownings, and other forms of violence. A clinical team composed of child and adolescent psychiatrists, psychologists, and social workers provides immediate and extended clinical assessment and intervention to children who come to the attention and concern of police officers. Together with pediatricians, primary care providers, and other nonmental health colleagues, the clinical team addresses neurophysiologic processes and psychiatric, psychosocial, and environmental concerns, and then attempts to develop the most effective and useful range and type of interventions. Children have been cared for employing many different modalities and have been treated individually and as part of larger groups in their homes, at police substations, within schools, in their neighborhoods, and in traditional clinic settings. A major result of the police-mental health collaboration has been the expansion of the clinical field of observation. Officers and mental health professionals now initiate therapeutic contact on the scene. Whereas the application of developmental principles has affected police approaches to routine interactions with youth on the streets and in the schools, it also has led to interventions that are anything but standard in the traditional approach to law enforcement.

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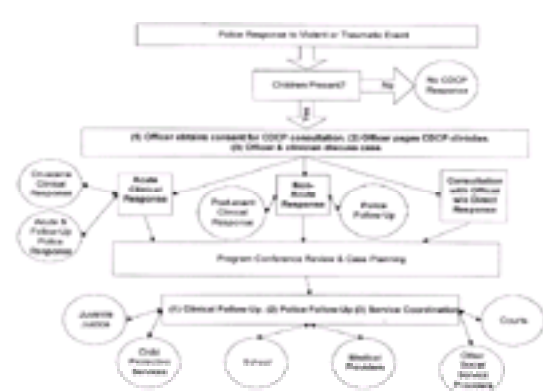
#### CASE ILLUSTRATION

Following the shooting death of a 17-year-old gang member, there was ample reason for concern about retaliation and further bloodshed. In the days that followed the death, grieving gang members congregated on the corner where the shooting had taken place. The effort at increased presence and removal and containment of violence was in the form of police, neighborhood-based probation officers, and clinicians spending time on the corner listening to gang members express their grief. As one senior police officer put it, "We could show our concern for their trauma by being with them, lending an adult ear to their misery. Alternatively, we could put more officers on the street, show them who's boss and, with a show of force, sweep them off the corner as often as necessary. We could then offer them an additional enemy and wait for them to explode." At the crucial moment, however, the police did not assume the role of enemy. They did not serve as the target for displaced rage or, in confrontation, offer an easy antidote to sadness and helplessness. Rather than exacting payback in blood, the common gang pathway for turning grief into action, the gang discretely assisted the police in making a swift arrest in the shooting. As the brother of the victim put it to a neighborhood cop, "You were there for us; that helped . . . and we were there for you."

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### Collaborative Responses to Trauma: The Child Development-Community Policing Program Acute Response

Police requests for mental health services on behalf of children whom they have identified as "at risk" results in the provision of a collaborative response for hundreds of cases each year; in calendar year 2000, CDCP clinicians were consulted on 250 cases involving more than 440 children. The largest proportion of police referrals, approaching 50%, results in an immediate clinical response, usually within minutes of the occurrence of a violent event (CDCP, 1998). Clinicians contact the remaining cases within several days of the referral, typically within 24 hours. Although an immediate response is often preferable from a clinical standpoint, initial contact may be delayed for a range of reasons, including concerns about the safety or placement of children, the status of ongoing criminal investigations, or the availability of caregivers to avail themselves to a clinical meeting. The CDCP response ( [Fig. 127.1](#)) ranges from a simple request for consultation from a clinician to a police officer about a child with whom they have had contact to an acute response involving a child and their family who have witnessed or been victimized by violence within the home or community.



**Figure 127.1.** Child Development-Community Policing Program acute response sequences.



## THE CHILD DEVELOPMENT-COMMUNITY POLICING PROGRAM ACUTE RESPONSE

The acute police–mental health response to children and families exposed to violence represents the foundation of the CDCP intervention for potentially traumatized children. The CDCP acute response is initiated by police officers. (See [Table 127.2](#) for a summary.) As they develop concerns about children and families following a violent event, they may contact the clinician by pager directly at any time of day or night. Alternately, they may choose to consult with their immediate supervisor, who typically has received more extensive training through the child development seminars and fellowship, about the utility of a CDCP referral. As a police initiated referral, it is essential that all officers in a department are oriented to the CDCP program and know how access the clinical team. After consultation with the parent or guardian about their concerns for the child and discussing the availability of clinicians, the police officer seeks verbal consent for an acute clinical response. When the officers contact clinicians, they discuss the nature of the event that has led to referral, including who is present, their involvement and relationship to one another, and their specific concerns about the children. Clinicians are informed of any logistical details, as well as any specific issues related to the safety and security of the proposed meeting site. Together the officer and clinician decide if an immediate clinical response is required or if a next day follow-up would be a more appropriate and useful response, although those children and families who receive an immediate response appear more likely to maintain a therapeutic relationship beyond the acute event (CDCP, 1998).

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Officer reports violent or potential traumatic incident involving children or victims whose presence is requested.  
Officer discusses concerns about welfare of children with adult caregiver(s).  
Parent/caregiver acceptable to contact CDCP clinician.  
Officer shares CDCP clinician's name.  
Officer and clinician discuss case circumstances, history, and location of incident response site if police case or neighborhood, and safety concerns.  
Officer provides name and contact information to supervisor/officer.  
Officer provides name of service and location of incident (location of incident to children's location).  
Officer meets with adult caregiver to collect history and discuss concerns.  
Officer adopts a child-centered approach using drawing and conversation to focus on child's predictability and comfort response to trauma.  
Officer provides psychoeducational information to parent/caregiver about response to traumatic events.  
Officer offers additional services to caregiver for child.  
Officer and clinician meet case to review CDCP form at weekly program conference.  
CDCP form develops and implements conditions for a history police and mental health.

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**Table 127.2. Summary of Child Development Community Policing Program (CDCP) Acute Response Sequence.**

The primary goals of the acute clinical intervention are to initiate a process of psychological stabilization following a violent event and to allow the child to gain a sense of control in the face of a potentially traumatizing experience. In the face of the ongoing realistic concerns about danger that may result from interpersonal violence, a psychotherapeutic intervention alone will be both naive and counterproductive. Therefore, it is essential that the lead officer and clinician discuss all pertinent aspects of the case together on the clinician's arrival and decide on the best intervention process together. For instance, the timing of a clinical response may be dependent on issues of safety that require leaving the crime scene and relocating the children to another home or a police substation. In other instances, the involvement of child protective services becomes a prerequisite to any clinical intervention, as children whose safety cannot be assured will not benefit from a therapeutic encounter. These questions and decisions may be made prior to the clinician's arrival during the telephone consultation, but should be revisited when the clinician arrives at the incident location. Without attention to concerns about physical and psychological safety, the potential therapeutic effects of the police–mental health response are fatally compromised. Conversely, attention to these matters advances the initial clinical goal of restoring a fledgling sense of safety and predictability in the wake of violence or trauma.

After the child and family's safety is discussed and assessed, the officer serves as the conduit in establishing the initial relationship between the clinician and the family. This allows for the police and clinician to be viewed as a team who together promote both psychological and physical security and expands the role of police beyond their traditional law enforcement response to include recognition of children's mental health needs in the wake of violence. Often, the children ask for the officer to remain present during the clinical consultation, and this request is best honored whenever possible. It sends a message that their physical safety is assured, allowing them the opportunity to feel and express what may be dangerous or overwhelming emotions.

Clinicians may meet with children individually or in small groups depending on the wishes of the family, availability of additional clinicians, and developmental and psychological characteristics of the children themselves (e.g., age range, cognitive development, status as victim, witness, or offender). Multiple clinicians allow in depth assessment of individual children, although when groups of similarly aged children share a common experience, small group meetings may foster expression and mutual support. Concurrent preliminary discussions with adult caregivers focus on gathering relevant history about prior child and family psychosocial functioning and trauma history, as well as providing developmentally informed education and guidance about children's responses to trauma and violence.

In order to help the child gain a sense of control in the face of potentially traumatic violence, the clinician typically follows the child's lead as expressed in words, play, or drawing and through these media helps the child elaborate his or her concerns. Associations to other events or internal psychological dangers are frequently more pressing or prominent than those related to the event that precipitated the intervention. The clinician's ability to listen quietly and empathetically, without telling or cuing the child about what the clinician might wish to hear, permits the child to assume control in metabolizing the event into an understanding consonant with their needs. Rather than becoming a passive recipient of generic strategies for coping with potentially traumatic events or following a proscribed sequence for responding to traumatic experiences, children are encouraged to adopt an active role in communicating their unique concerns, which may or may not resemble the factual events to which they have been witness. Although child-centered, the clinician becomes an active listener, asking for pertinent clarification, and by the meeting's conclusion, providing the child and the family with their knowledge and expertise about a range of potential interventions and expectable responses to violence and trauma.

Clinicians provide psychoeducational information in verbal and written forms about possible traumatic responses and possible mechanisms to cope with them. The former facilitates the burgeoning clinical relationship; the latter provides a concrete reference that can be returned to at times of heightened concern or distress. At the conclusion of the meeting, the clinician provides the family with contact information, asks to follow-up with a visit or phone call (at the family's discretion), and provides information about longer-term consultation and treatment. Specific information, along with the connection to a clinical expert, normalizes the child and family's response to their experience, provides them with support and the security in the knowledge that help is available and accessible, and facilitates a clinical relationship at a time when defenses are more malleable and receptiveness to intervention is accentuated.

The CDCP Acute Response is the clearest manifestation of the police–mental health partnership and the clearest departure from the traditional activities of both police officers and mental health clinicians: the former as purveyors of punishment and reproach and the latter as office-based theorists with scant awareness of the daily adversity with which their youngest patients must contend. The CDCP Acute Response not only provides intervention to children and families sorely in need, but also changes the paradigm by which police and mental health professionals respond to these needs and concerns. Whereas officers have an opportunity to expand their knowledge and repertoire of interventions within a model of youth-oriented community policing, the collaboration with the police has allowed child therapists a new setting in which to increase their understanding of the impact of violence and trauma and provide clinical care to youth prior to the point at which symptoms crystallize into serious psychiatric disorders.

Collaborative discussions about consultation service referrals focus on the extent to which children describe the violent events they have witnessed in terms of the developmental phase-specific anxieties that are aroused. By following the unfolding stories of children exposed to violence, child therapists come to understand the specific features of a violent event and its sequelae that individual children experience as exceptional, overwhelming, and therefore traumatizing. All too often, clinical assumptions and notions about the nature of a child's traumatization seem to be determined by observable features of the violence as it occurred, yet these assumptions may have little to do with the child's experience of violence or the attributions generated in its aftermath. In turn, there may be little attention paid to learning about the child and his or her previous psychological functioning as a basis for understanding his or her unique responses to an experience of violence, which can only be understood in this context of life history, temperament, family constellation, phase development, and defense configuration. Without awareness of basic historical material and child specific response to violence, the clinician has scant data from which to develop recommendations for police and psychiatric intervention. For clinicians and officers alike, closer attention to the details of the individual child's experience has prompted changes in their responses and allowed them to move beyond proscribed responses, which may not adequately address the child's distress.

## CASE ILLUSTRATION

Sergeant G described an incident in which a 7-year-old girl witnessed a beloved neighbor bleed to death after being fatally stabbed by another neighbor. Believing he was protecting her from the gore of the crime scene, Sergeant G had the child wait on the porch outside while officers conducted their investigation. He was haunted by the intense gaze, a mixture of despair and rage, that the girl fixed on him when he finally invited her back into the apartment as the officers were leaving. The next day the sergeant went back to the house and spoke with the girl and her grandmother and understood that his attempt at being helpful had been without the benefit of considering what this girl was experiencing, what was needed, and from whom.

As he explained in the case conference, "Especially in the midst of so much blood and terror, what she needed was to be close to her grandmother, the most stable figure in her life, not to be stranded alone with images of the scene." Both the girl and her grandmother eagerly accepted his offer of a referral for clinical services. As it emerged in her treatment, being alone with frightening ideas and fantasies involving extremes of love and hate was common for this young girl. Her heroin-addicted mother capriciously appeared in and disappeared from her life, and she was additionally burdened by her realistic uncertainty about her grandmother's health and longevity. Both internalized and external conflicts were boldly underlined by her confusion of loyalties in the stabbing. Although she mourned the death of one beloved and idealized maternal substitute, she anxiously told her therapist about the love letters she was writing to the assailant, now in jail on murder charges, but whom she viewed as safer and more available than her inconsistent and absent mother.

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## CASE ILLUSTRATION

The therapist's focus on helping to integrate a teen's detailed description of violent events with phase-appropriate concerns and past conflicts is illustrated again in this second case.

Mark, 15 years of age, was robbed at gunpoint on a Friday evening. He'd been walking with friends when two men put a reportedly large-caliber semiautomatic weapon in his face and demanded all of his money and gold jewelry. Mark had been walking behind several friends and they were unaware of what was occurring in an alleyway off of the main sidewalk. Mark later reported that the men repeatedly shoved the weapon in his face and told him they would shoot him. After taking his valuables the assailants fled, and Mark ran home. He ran to his room crying uncontrollably, hid on the floor of his closet, and, in spite of his mother's urging, refused to come out. After a while, through sobs Mark told his mother what had occurred, and she phoned the police.

Each of the three officers who responded had been trained through the child development seminars; the supervisory officer had completed the intensive fellowship. As one of the officers approached the bedroom, Mark began to scream. The officer told him that he had heard what had happened to him and realized the hold-up was a terrifying experience. Mark would not look at the officer and yelled at him to leave the room. The officer was about to leave when the supervisor pointed to his gun and utility belt. With this the officer removed his holster and weapon, explaining to Mark that he would leave them outside of the room because he understood how frightening guns might be to him. Mark continued to sob and shake uncontrollably but allowed the officer to help him out of the room and accepted the suggestion that he go to the emergency room for treatment. The acute consultation service clinician was called and met Mark at the hospital.

During the course of the interview, Mark was able to look at the clinician only after a comment was made about "how feeling very frightened could make a guy feel small and helpless," a particularly undesirable feeling for a 15-year-old boy. Mark began to talk about the events, repeating the same scene and assailants' commands to him over and over. The repetition gradually began to include some slight alterations in the facts, and Mark protested that he should have "grabbed the gun and kicked each of the [attackers] in the balls." He described the size of the gun muzzle as huge and insisted that he thought they would kill him with this huge weapon. As his shaking, hyperventilation, and sobbing subsided, Mark began to talk about the earlier part of the evening. He explained that before being robbed he had been "hanging back from his homeboys because they were with their ladies" and he wanted to "give them space." He shyly told the clinician that he didn't have a girlfriend and then quickly exploded with rage and then tears. He wanted to get a gun and kill the guys who "messed with him." He protested that he didn't deserve what had happened to him—he was a good student in school and had just completed an important history paper. He explained that he'd bought all of the thin gold chains he wore for himself, clarifying that he was not to be lumped together with "low-life drug dealers." Mark began to cry again, as he swore revenge. The clinician commented that it felt humiliating to have to feel so terrified and that Mark was wishing that he could undo his experience. Mark replied that if he had a gun or had disarmed his attackers, he wouldn't have to feel as though he'd "wimped out." The clinician agreed that feeling powerful would certainly be the opposite of what he had experienced with a gun in his face. Mark brightened and looked up suddenly, exclaiming that he now remembered the gun more clearly—it wasn't a 9-mm semiautomatic; it was a BB gun. As the acute terror diminished, he was also able to remember the make of the car the assailants took off in and provide clear descriptions of the two men. His restitution fantasies of revenge began to take another form as Mark talked about helping the police make an arrest. On his request, Mark spoke with the detective involved in the case to offer the information he had recovered in the course of the interview. Two hours after the admission to the emergency room, Mark was discharged.

Mark was seen in outpatient sessions during which he continued to go over the events of that Friday evening as well as exploring equally disturbing lifetime events. The fantasies of what he should have done in both domains were intermingled with talk of the mortification of feeling helpless and the increasing recognition that, in fact, there was nothing he could have done to alter what had occurred. In addition to his hard work in psychotherapy and his enrollment in a special academic program, another ingredient played a crucial role in Mark's improved adaptation. In the months that followed the robbery, the officer who had unhitched his utility belt began stopping in on him regularly for brief chats during the course of his usual beat patrol. The officer's friendship and authoritative monitoring played an essential role in deterring Mark from what emerged as his beginning involvement with drug dealing and engaged him in an after-school activities group. Eighteen months after the incident, Mark has still not "scored" a gun, and instead of continuing to recite numerous violent revenge fantasies, he spoke of the latest academic demands at school and his friendships with the cops on his beat. Although he has not forgotten the terror or rage associated with his experience, Mark knew that his good memory had been instrumental in the arrest and later conviction of the two men who had attacked him. And, as he said, "That feels really good."

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## Extending the Model

Recognizing the ubiquity of domestic violence in the lives of children, CDCP officers, clinicians, outreach workers, and domestic violence court advocates have implemented a specialized protocol designed to reduce the incidence and severity of domestic violence. In two pilot districts, domestic 911 calls to police result in a CDCP referral and subsequent coordinated intervention involving police, clinical, and court personnel. Initial strategies focus on the safety of mothers and their affected children. Neighborhood police officers and clinical outreach workers conduct follow-up visits to victims' homes where they address pressing concerns about safety. This outreach team ensures appropriate linkage to advocates based in a specialized domestic violence court and assists with communication about protective orders. Mothers who may have difficulty engaging law enforcement or clinical services for their own benefit may respond more readily to these professionals' concerns about the welfare of their children. In the best of circumstances, the neighborhood officer and clinical outreach worker become increasingly familiar to the family, heightening the likelihood of an earlier response should future instances of violence occur and forging an improved sense of safety and security through benign relationships with police authorities and community-based clinical providers that are based on mutual concern for the effects of witnessing domestic violence on children's development.

Preliminary impressions suggest a decrease in recidivism for those domestic violence cases that have received intensive police and clinical follow-up ( [Akiyama and Nolan, 1999](#); [New Haven Department of Police Service, 2001](#) ). In some instances, police follow-up visits have resulted in additional charges being filed against the perpetrator, primarily for violations of court orders. Increased referrals to the CDCP clinical team have provided additional support and resources to domestic violence victims, including intensive clinical services for children who have been exposed to repeated violence and victims whose sense of safety has been compromised. Officers have reported an increased sense of effectiveness in dealing with domestic violence cases, and victims report satisfaction with the attention and support they have received from officers.

Police, juvenile probation officers, child protective service workers, and mental health professionals have also expanded their collaboration to address the inefficiency and limitations of the juvenile justice system in addressing the needs of children and adolescents involved in violent and other criminal activities. Multiple professionals have known various children arrested for delinquent acts prior to their involvement in the juvenile justice system, yet often they have not benefited from efforts at engagement in mental health treatment. Therefore, arrest can represent an opportunity for collaboration and interdisciplinary problem solving as an alternative to traditional approaches that emphasize the independence of the court and associated agencies and often result in duplication of efforts or court-ordered stipulations that are mismatched to the child's needs.

In the current collaboration police and probation officers have begun to share supervision and monitoring of juvenile offenders by relocating probation officers to community substations and by engaging neighborhood officers in supervising community service projects for youth on probation. Comprehensive clinical evaluations are combined with information contributed by probation, police, child protection, mental health, and education professionals so that interventions can be developed, coordinated, and monitored from multiple dimensions of the child's life. These services include the integration of individual, family, and group psychotherapies; mandated community service; curfews; shared probation and police monitoring and supervision; and special education interventions; some or all of which might comprise the individual treatment package for a specific youth.

## Summary

Coordinating responses through The Child Development-Community Policing Program has led to multiple changes in the delivery of clinical and police services. Mental health clinicians and police officers have developed a common language for assessing and responding to the needs of children and families who have been exposed to or involved in violence. Learning from each other, these unlikely partners have established close working relationships that improve and expand the range of interventions they are able to provide while preserving the areas of expertise and responsibilities of each professional group. The immediate access to witnesses, victims, and perpetrators of violent crimes through the acute consultation service provides a unique opportunity to expand the understanding of clinical phenomena from the acute traumatic moment to longer-term adaptation, symptom formation, and recovery. In turn, the initiative introduces the systematic study of basic psychological and neurobiologic functions involved in traumatization as well as the investigation of psychotherapeutic and pharmacologic therapies. Similarly, program involvement with juvenile offenders has led to a coordinated response from the police, mental health, and juvenile justice systems. This project provides an opportunity to develop detailed psychological profiles of antisocial behavior as well as to determine the characteristics that might predict with whom community-based interventions might be most successful. A recent survey of New Haven public school students has yielded promising evidence that youth-oriented community policing and the CDCP model are having a positive impact on the quality of life through promoting feelings of safety and security among youth, decreasing levels of exposure to domestic and community violence, and enhancing perceptions of police as responsive and benevolent authority figures ( [New Haven Public Schools and Yale University, 2000](#); [Schwab-Stone et al., 1999](#) ).

No single group of professionals is able to address the multiple needs of the children and families subject to massive environmental stress. When professionals attempt to intervene in isolation of one another, the multiple and awesome needs of these profoundly stressed children and families become overwhelming. The collaboration among police, probation officers, child protective service workers, and mental health professionals in New Haven provides a model of interdisciplinary action on behalf of children at great developmental risk owing to their exposure to violence and who would be unlikely to receive effective intervention through traditional models of social service delivery.



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# 128 CHILD PSYCHIATRY AND THE JUVENILE COURT

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Our Humanity shall be judged by how we treat our criminals, our children and our insane.

Oliver Wendell Holmes

The first juvenile court was established approximately 100 years ago. Following in close proximity, the Juvenile Psychopathic Institute, directed by Dr. William Healy, was created to investigate the etiology of delinquency. Early child welfare reformers had the prescience to know that child psychiatrists and child psychologists can contribute to the success of juvenile courts ( [Fox, 1996](#)). Although through the years faith has wavered in the court's ability to reform delinquents and psychiatrists' ability to "solve" delinquency, overall the amiable relationship among the juvenile courts, child psychiatry, and community has remained steadfast.

In the following chapter, we discuss the child psychiatrists' role in delinquency cases. In particular, we focus on the role of psychopathology in the formation and treatment of youthful offenders. After reviewing a brief history of the juvenile court system and the contribution of the child psychiatrist to the juvenile court, we discuss the potential developmental pathways of delinquency. Next, we provide an examination of relevant research findings from our own work and the work of others. Finally, we conclude with a discussion of future research and clinical directions for the child psychiatrist involved in the juvenile justice system.

## A BRIEF HISTORY OF THE JUVENILE COURT

In the late 19th century, a separate justice system was created for juveniles because it was apparent that the system for adults was not suitable for children. Developmental reasons (e.g., immaturity) were now for the first time acknowledged to be sufficient grounds for not holding a youth completely responsible for his or her actions and crimes, standing alongside reason of insanity. In addition, a group, often dubbed "child savers," argued that, in comparison to adult offenders, child offenders are more amenable to rehabilitation, less likely to benefit from punishment, and should be held less responsible for their actions ( [Moon et al., 2000](#)). One major outcome of the child savers' arguments was the creation of the first juvenile justice system in Chicago in 1899.

The legal concept of *parens patriae* served as the foundational basis for the establishment of the juvenile justice system. That is, the State would serve as a surrogate parent for those individuals (i.e., juveniles) who could not care for themselves properly. The original juvenile justice philosophy encompassed five central ideas:

1. A special court was needed for neglected, dependent, or delinquent children under the age of 16.
2. Rehabilitation, not punishment, was the purpose.
3. All records and proceedings were to be confidential.
4. When incarcerated, children were to be separated from the corrupting influence of adult criminals.
5. All proceedings were to be informal ( [Crosby and Reppucci, 1993](#)). For the first half of the 20th century, the unoperationalized term "in the child's best interest" guided the juvenile justice system ( [Grisso et al., 1988](#)).

Because of the juvenile court's intrinsic interest in the welfare of children, psychiatrists and their theories on the development and prevention of delinquency generally have been welcomed. Nearly 50 years ago, Markey and Langsam wrote:

The [juvenile] court's legal responsibility has been influenced by basic psychiatric attitudes without becoming subservient to them. The court is thereby stimulated to learn more about the underlying causes of delinquency. This, in turn, prompts an increasing demand for better trained professionals. The psychiatrist acts as an auxiliary to the judge and does not attempt to point out specifically how the judge shall pass sentence or make plans. It is reasonable to suggest that psychiatry has been accepted because it is helpful and applicable, rather than because it adds prestige to the court program ( [Markey and Langsam, 1957](#), p. 799).

Today, the field of forensic psychiatry continues to grow. For the most part, there appears to be a mutual level of appreciation and respect between the courts and the science of psychiatry. Given our newly acquired knowledge of the prevalence of treatable psychiatric disorders in this population, and the fact that we have at our disposal more efficient and well tested treatments for specific disorders, we are in the position to offer new and helpful interventions that are likely to have an immediate and distal impact on the lives of these children.

## DEFINITIONS OF DELINQUENCY

Any clinician working with delinquent or conduct disordered juveniles needs to differentiate among antisocial acts, delinquency, criminality, conduct disorder, antisocial personality disorder, and psychopathy ( [Steiner and AACAP, 1997](#); [Steiner and Feldman, 1999](#); Steiner et al., 1997b). We have discussed these distinctions repeatedly and refer the reader to these publications for a fuller discussion ( [Steiner, 1999](#); [Steiner and Cauffman 1998](#); [Steiner and Wilson, 1999](#)). Suffice it to say that we believe that only a portion of delinquents exhibit an association between their antisocial acts and psychopathology. Certain forms of psychopathology (e.g., conduct disorder and attention deficit hyperactivity disorder [ADHD]) are overrepresented. It is the task of the psychiatrist to help subcategorize the extremely heterogeneous population of delinquents and see to it that those with diagnosable disorders are appropriately treated. We hope that such treatment will reduce antisocial acts, such as has been shown in the case of treatment of ADHD with stimulants across many different studies ( [Hinshaw et al., 1989](#); [Klein and Abikoff, 1997](#)).

## THE CONTRIBUTION OF THE CHILD PSYCHIATRIST TO THE DEVELOPMENT OF DELINQUENCY THEORIES

Several excellent population-based studies of youth at high risk have shed light on the origins of delinquency ( [Loeber et al., 1999](#)). It has become clear that the model that accounts best for the origins of delinquency is a cumulative risk factor model ( [Loeber, 1990](#)). Each risk factor seems to act in a cumulative fashion ( [Rutter et al., 1999](#)). In most cases of delinquency and conduct disorder, there appears to be a gradual accumulation, during the course of childhood and adolescence, of multiple risk factors that are not offset sufficiently by protective factors and that ultimately result in chronic conduct problems ( [Loeber et al., 1993](#); [Steiner and AACAP, 1997](#)). Delinquency arises from risks that accumulate from birth on, including ecological factors, constitutional factors, parenting and parent-related factors, as well as

internal factors such as personality functions, academic performance, and relationship factors, all of which grow in importance as development unfolds. Some of these risks are genetic or constitutional, others environmental or situational. We have newer studies that seek to estimate the relative contributions of genetic and environmental factors to the problem showing a more balanced contribution of environment and internal factors ([Raine, 1993](#)) when comparing these factors in adult criminals. In their data from the Minnesota Family Twin study, [Taylor and colleagues \(2000\)](#) were able to estimate the respective contributions of genes and shared environmental characteristics between children (e.g., parenting), shared environmental characteristics between parents and children (e.g., socioeconomic status), when accounting for similarities between monozygotic and dizygotic twin pairs. Additionally, they measured and estimated the contributions of nonshared environmental characteristics (e.g., life events, specific treatment by parents, and peer influences). In their model, nonshared environment accounted for 56% of the variance in self-reported mild to moderate delinquent acts; genetic influences accounted for 18%, and shared environmental characteristics for 26% in this sample of 17-year-old boys and girls. It is most likely that many of these factors do not exert their influence in a linear and solitary fashion. An interaction among a significant number of such factors ultimately leads to criminality, as has been shown by [Raine and associates \(1994\)](#). In this study, the constitutional risk of birth complications exerted their maximum risk for future violent behavior in the presence of maternal rejection at age 1 year.

It has become clear that delinquency describes a large and heterogeneous population ([Sholevar, 1995](#); [Steiner and AACAP, 1997](#)). Mapping psychopathology onto these samples may facilitate defining the subcategories and help them to receive appropriate intervention. The utility of the diagnosis of conduct disorder by itself appears to be a highly comorbid condition that only sometimes appears in isolation ([Steiner, 1999](#); [Steiner and AACAP, 1997](#)). Another way of putting this is that in the adolescent age range where antisocial and even delinquent behavior is extremely common, most other psychopathological conditions seem to associate with such behaviors. It is an open question as to whether comorbid conduct problems, especially those that are antedated by the onset of other psychiatric disorders (e.g., depression) show a differential outcome from singly diagnosed conduct disorders. From a criminologic viewpoint, it still remains to be seen as to whether a diagnosis such as conduct disorder offers any additional explanatory force beyond the mere criminologic characterization of an individual. Is it true that the label "conduct disorder" rather than that of "delinquent" helps to better explain his or her current or future status (i.e., somebody whose antisocial acts have led to legal conviction and punishment)? One of the main advantages of this label is the independence of legal status. It thus calls for attention to a problem before in a legal sense; it exists, making it amenable to prevention and intervention. By perhaps looking at conduct disorder from the viewpoint of a clustered disturbance of the aggression system, we might add to the understanding why certain youths persist in criminality even after given chance upon chance to break from a lifetime pattern of crime, whereas others desist from such activities easily and quickly even with the most minor intervention. It will be our task in the near future to assess the differential importance of psychopathologic conditions in this cohort, especially in response to well-designed and delivered multimodal interventions. Such studies currently are not available, because we have just begun to map prevalence rates and understand the importance of psychopathology in the context of delinquency.

## THE CONTRIBUTION OF THE CHILD PSYCHIATRIST DURING ADJUDICATION OF JUVENILE DELINQUENTS

The youthful offender may encounter a child psychiatrist or other mental health expert at one or more phases of the legal process: prior to adjudication, after the youth has been dispositioned to help with treatment options, during probation or incarceration, and while on parole. The child psychiatrist can function as a clinical consultant, an expert, and as a part of the juvenile justice mental health system. In each of these roles, a high diversity of training, command of a wide range of interventions, and ability to shift between multiple roles are assets. Our responsibilities and tasks change, as do our relationships with the patients across the various appointments.

During the adjudication phase of a delinquency case, the child psychiatrist may be asked to comment on a number of issues, such as culpability, future conduct, and extenuating circumstances involved in the commission of a crime. In this role, we often have to abandon our usual privileged relationship with the patient. All of these procedures require a public report, which becomes part of the official crime record and is accessible to staff and judiciary as well as the youth. We function as agents of the legal system. We need to take great pains to make sure that the youth and his or her family understands this situation, because it has important implications for confidentiality. In essence, no confidentiality can be granted; information on new or unreported crimes may lead to further prosecution. In California there is a specific legal requirement that new crimes be reported to authorities so they may decide on the necessity of further prosecution. The psychiatrist cannot protect the youth from this reporting requirement, especially when details of events are given. Any psychiatrist should be well aware of the legal statutes in the state of his or her practice governing this set of circumstances.

Child psychiatrists may also be called on to determine the ability of a minor to waive his or her *Miranda* rights. Like adults, juveniles must be read their *Miranda* rights before being interrogated. The *Miranda* warning is read to suspects to ensure that defendants are aware of their rights to not self-incriminate and have a lawyer present when interrogated. It is sometimes questioned whether juveniles have the ability to understand and appreciate, and therefore competently waive their *Miranda* rights. A competent waiver of *Miranda* rights must be done voluntarily, knowingly, and intelligently. [Grisso \(1980, 1981\)](#) found that juvenile offenders aged 14 years and younger were significantly less likely to be considered competent to waive their *Miranda* rights than 15- and 16-year-old and adult offenders. Moreover, 15- and 16-year-old children of low intelligence were significantly less likely to be considered competent to waive their *Miranda* rights than adults of similar intelligence. And, importantly, Grisso found that over 90% of juveniles waived their rights (all of the participants 14 years and younger did so), compared to about 40% to 60% of adults. [Grisso \(1981\)](#) concluded that juveniles should not be allowed to waive their rights to counsel, and recommended that an attorney's presence be required during interrogation. Although this recommendation was made nearly two decades ago, it is only now that some states (e.g., Illinois) are considering legislation to require the presence of an attorney when a child is questioned and during the videotaping of child suspect interrogations.

Another situation in which a forensic psychiatrist may be called on is to evaluate the suitability of a juvenile for transfer of prosecution to adult criminal court. In the recent past, this has become increasingly important because all but five states have made the transfer process easier ([Bilchik, 1999](#)). The two general standards for juvenile transfers are that: (1) the juvenile is not amenable to treatment; and (2) the community needs protection from the juvenile ([Grisso et al., 1988](#); [Poulos and Orchowsky, 1994](#)). In regard to both standards, the following considerations must be taken into account when deciding to transfer cases: whether the juvenile is a recidivistic offender, whether the juvenile has received and/or responded to prior treatment, and the nature of the present charge (e.g., felony, murder) ([Kruh and Brodsky, 1997](#)). [Poulos and Orchowsky \(1994\)](#) found that prior mental health treatment history was negatively associated with transfer decisions, such that those juveniles who had mental health treatment in the past were less likely to be tried in adult court. Finally, some states require the accused juvenile to demonstrate that he or she will benefit from rehabilitation.

Another major task of the child forensic psychiatrist during the adjudication phase is the evaluation of competency. The questions raised regarding the child's competency have become increasingly important, as juvenile court reforms have resulted in making juvenile court more like adult proceedings and as juveniles are being waived to adult court to stand trial. Much of the research conducted on juveniles' legal competency has focused on decision making in the medical arena, specifically on informed consent and the decision to seek medical treatment. Briefly, earlier studies demonstrated that adolescents did not differ from adults in their cognitive decision-making processes ([Grisso and Vierling, 1978](#); [Mann et al., 1989](#)). However, in terms of competency to stand trial, more recent research has revealed that young offenders typically do not meet legal standards and are not comparable to adults ([Grisso, 1997](#)). [Cowden and McKee \(1995\)](#) examined contributing factors to assessments of incompetence, marginal competence, and competence in a population of juveniles. Among the factors to correlate with group assessment was age. Only 16.7% of juveniles 12 years and younger were considered competent. Similarly, [Cooper \(1997\)](#) examined juvenile offenders' competency to stand trial. Before training, only two out of 112 children aged 13 to 16 years were considered competent. After training (i.e., an educational competency training videotape), only 12 juveniles reached a score indicating competence. These results suggest that juveniles need substantial training to be considered competent, that is, to understand and play a meaningful role in their defense.

To overcome the problems associated with juvenile's lack of knowledge, [Grisso and associates \(1987\)](#) recommended that a hearing to determine a juvenile's competency to partake in delinquency proceedings be required when any *one* of the following criteria is met: (a) the juvenile is 12 years or younger; (b) there is an indication of major mental illness or mental retardation; (c) there is an indication of depressed intellectual functioning (borderline IQ or lower), or learning disability; or (d) there is an indication of deficits in memory, attention, or interpretation of reality. Although a majority of juvenile offenders have low intelligence, and many have diagnosable disorders, only one state (Virginia) requires competency hearings for transferred juveniles ([Redding, 1997](#)).

## THE CONTRIBUTION OF THE CHILD PSYCHIATRIST DURING INCARCERATION OF JUVENILE DELINQUENTS

Child psychiatrists are usually employed by the probation or prison system or the judiciary when functioning as consultants when juveniles are incarcerated. The juvenile justice system is charged with the appropriate rehabilitation of the juvenile offender; hence, the questions asked usually involve planning of interventions for the patients and recommendations about appropriate settings in which to carry them out (the assessment of "treatment needs"). On an emergent basis, we are often asked to provide assistance in crisis situations that arise during confinement, or to address issues regarding good or bad conduct in the program. Questions arise regarding the appropriateness of release and future potential for violence as confinement time comes to a close. In California, there are two specific requirements that psychiatrists are often asked to comment on: (a) that the youth develop insight into his condition; and (b) that the youth assume full responsibility for his or her actions. In some special cases, when confinement time is expiring, psychiatrists are asked to assist in the determination of whether confinement time should be extended because of further treatment needs of the patient, usually because the child has responded insufficiently. Very often, as part of such an assessment, we are



asked to assess the risk the patient poses to a specific community or person, if released in his or her essentially unrepentant state.

The situation is somewhat different when child psychiatrists function as employees of juvenile justice systems and provide diagnostic and therapeutic services. In such cases, notes need not reflect the treatment process in as great detail as in a legal record, which may be the object of close cross-examination. Information can be somewhat protected, provided that one defines these parameters with the referring staff. The record still needs to be sufficient to support our treatment decisions. However, ultimately even one's treatment record can be made part of the criminal record by order of the parole board or the court, at which point the material is treated as any other criminal record. Again, the youth needs to be made aware of this possibility, because it might have implications on his or her ability to disclose facts in treatment. Generally, we find it sufficient to issue a warning statement at the beginning of treatment. Such warnings frequently make most youths circumspect in their disclosures. We usually find that this way of proceeding does not impede the delicate and complicated work of exploratory and/or supportive psychotherapy indicted in the treatment of these cases.

Surprisingly, it is only in the recent past that researchers have investigated the mental health of youthful offenders. The research that has been conducted has indicated that the frequency of mental health disorders is higher for delinquents than for comparable nondelinquent adolescents. For example, [Cocozza \(1992\)](#) estimated that every year 150,000 juveniles who come into contact with the juvenile justice system meet the diagnostic criteria for at least one mental disorder. Moreover, certain disorders are extremely common, such as alcohol and substance abuse and dependence; estimates have ranged from 30% to 80% in juvenile delinquents ([Steiner and Cauffman, 1998](#)). Another disproportionately high diagnosis among juvenile offenders is posttraumatic stress disorder (PTSD). Steiner and colleagues (1997a) and [Cauffman and coworkers \(1998\)](#) discovered that in a population of juveniles incarcerated in secure facilities, approximately 50% of the females and 30% of the males fulfilled all criteria for current PTSD. Similarly, [Carrion and Steiner \(2000\)](#) found that in their sample of juvenile delinquents, 28% met criteria for a dissociative disorder and nearly all (97%) self-reported a history of traumatic events. Other disorders, such as affective, psychotic, learning, attention, and anxiety have been found with increased frequencies in other states as well ([Atkins et al., 1999](#); [Pumarieqa et al., 1999](#)), at levels comparable to or exceeding those found in youths from community mental health centers and inpatient units. Clearly, this population is psychiatrically highly compromised, justifying the child psychiatrist's involvement and attention.

Another intriguing line of research involves the frequency and type of personality trait disturbances and even personality disorders among youthful offenders. As we know from the path breaking research by [Robins \(1966\)](#) and others ([Rutter et al., 1999](#); [Zoccolillo et al., 1992](#)), such problems deserve special attention, because they predict in many cases transition to antisocial personality disorder and perhaps even psychopathy as defined by [Hare and colleagues \(1991\)](#). These outcomes portend poorly for the future of the child ([Harrington et al., 1991](#)): Lifetime persistent criminality is likely and, to date, interventions have not been successful, and may even worsen the outcome ([Hare, 1999](#)). A cautionary note should be added: (a) only about half of conduct disordered youth show such progressions; and (b) to date there are no studies with biological interventions in this cohort, and we have some reason to think that at least some psychopharmacologic agents might be effective in this population. Still, the presence of these characteristics in a youth must be considered as serious. Not surprisingly, adolescents adjudicated delinquent often meet the criteria for conduct disorder (CD), oppositional defiant disorder (ODD), and ADHD ([Atkins et al., 1999](#); [Steiner, 1999](#)). In some of our earlier research, we have found that delinquents may not have pure conduct disorders, but rather can be diagnosed with conduct disorder and another one or more diagnoses. In a sample of clinically referred delinquents in custody, 38% were found to have conduct disorder and at least one other internalizing diagnosis (e.g., depression, PTSD), 26% had CD and at least one other externalizing diagnosis (e.g., ODD, ADHD), and 22% had CD by itself (Steiner, 1999).

An examination of stable personality traits (i.e. nonsyndromally defined characteristics) of delinquents also offers an interesting portrayal, and these traits may have predictive value in regard to recidivism and lifetime persistent criminality. One research tool that we find very promising in terms of depicting delinquents is the Weinberger Adjustment Inventory (WAI) ([Weinberger and Schwartz, 1990](#)). The WAI assesses broad personality functioning along two major dimensions: distress (e.g., anxiety, depression) and self-restraint (e.g., suppression of aggression, consideration of others). Intersecting the two dimensions at age-appropriate means creates a four-quadrant typology. The four types are: (a) reactive (high distress, low restraint); (b) suppressor (high distress, high restraint); (c) nonreactive (low distress, low restraint); and (d) repressor (low distress, high restraint). [Steiner, Coffman, and Durburg \(1999\)](#) used this typology to predict recidivism rates among serious institutionalized juvenile offenders. Interestingly, nearly 90% of the nonreactives were rearrested in the 4½ years follow-up. In contrast, less than 50% of the suppressors were rearrested. Findings from this study highlight the importance of examining personality traits and the implications for delinquency prevention and treatment.

## A MODEL OF PERSISTENT DELINQUENCY BASED ON PSYCHOPATHOLOGY

Defining psychopathology as a "harmful dysfunction," [Wakefield \(1992\)](#) states that: (a) a condition causes harm or deprivation of benefit as judged by social norms (and evolutionary purpose); (b) results from the failure of some internal mechanism to perform its natural function (an effect that is part of the evolutionary explanation of the mechanism); (c) It results in adaptive failure-lack of progression in stage-salient tasks and competencies. We would like to add to that definition the notion that the condition has become context independent (i.e., mostly internally driven) in two specific ways: It is independent of social context (i.e., it occurs in all different social settings, as benign and supportive as one can imagine). And it is also independent of the developmental phase of the person who is so affected—temporal context no longer defines the person's conduct and thought. Some forms of delinquency, and hopefully most of the ones associated with the label of conduct disorder, may be viewed from this perspective. Such a model, which is compatible with developmental principles, will assist us in identifying causal processes involved and get us beyond the currently descriptive phenomenology, which may or may not offer advantages over simple criminologic classifications. The model should additionally and increasingly provide us with the needed sophistication to discern those who are very likely to persist in patterns of crime despite all kinds of escapes provided, and ultimately lead them to intensive and new forms of treatment, which has a higher chance of being successful than our current rationalistic appeal to their consciences, sense of responsibility, and ability to think things through.

Despite recent advances in reducing crime and especially juvenile crime ([OJJDP, 1999](#)), juvenile delinquency is still a significant problem in the United States. Whether data are obtained from youth self report or from official crime statistics, the same pattern emerges: There is a sharp rise of antisocial activity between early puberty and late adolescence for both genders ([Snyder, 1994](#)). Some studies ([Caspi et al., 1990](#); [Moffitt and Silva, 1988](#)) report that up to 80% of juveniles are involved in delinquent acts. Although the majority of cases involve property-related offenses, it is also true that juveniles are involved in the perpetration of crimes against persons. Most of all of these crimes appear to be perpetrated by a relatively small number of juveniles: Approximately two-thirds of all violent crimes are committed by juveniles with four or more arrests ([Snyder, 1994](#)). On the other hand, it is also true that although antisocial behavior and delinquency are extremely common in the adolescent age range ([Steiner and Feldman, 1999](#)), the predictive validity of such labels is not very strong for lifetime persistent problems: In fact, the majority of delinquents exit from a lifetime career of crime ([Rutter et al., 1999](#)).

The challenge is to introduce new models of taxonomy and observation to more reliably identify those who are at high risk for reoffending ([Steiner, 1999](#); [Steiner and AACAP, 1997](#)). One such model may be based on the study of the prevalence of psychopathology in delinquent youths. Such a model is distinct from delinquency (a criminologic construct) in that the youth's diagnosis does not depend on their legal status ([Steiner and Cauffman, 1998](#)); it is distinct from the study of traits, such as impulsivity and temperament, in that it seeks to establish qualitative taxa that relate to specific and cost-efficient treatments (e.g., depression, mania, and PTSD).

The model we are proposing attempts to do the following in the prescribed sequence.

### Step One: Diagnose Antisocial Acts That Occur as a Function of Psychopathology

See the modified Wakefield proposition given in the preceding. This step delineates coincidental, situational, and ecologically driven antisocial demeanor, which has a high chance of terminating with change of environment, punishment, education about crime and victims, and so on (i.e., the antisocial action that is generally the purview of classic criminology) from antisocial acts that have a high chance of persisting because of an internal harmful dysfunction that makes the behavior context independent and less amenable to simple criminologic interventions.

### Step Two: Establish the Psychopathologic Process That Associates with the Antisocial Behavior

A primary dysfunction of the aggression system in the youth (e.g., in oppositional-defiant disorder, CD, intermittent explosive disorder), or a dysfunction in the aggression system induced by other forms of psychopathology (e.g., disorders of depression, anxiety, or attention), or more serious forms (e.g., psychosis, pervasive developmental disorders) and macroanatomic disorders of the central nervous system (e.g., seizures, chronic head trauma, or frontal lobe dysfunctions). Also consider whether the disturbance reflects a state (a relatively short-lived disturbance, extending from weeks to months), a trait (extending months to years or lifelong) or a combination of both.

### Step Three: Assign a Causal Sequence to the Disturbance

Did oppositional defiant disorder lead to conduct disorder and substance abuse? Oppositional aggression would then be the primary driving force and primary target for intervention. Or did mood problems lead to substance abuse, conduct disorder, and exposure to traumatogenic incidents, later leading to PTSD and further problems with aggression? Such a developmental sequencing should have therapeutic and prognostic implications and preserve a necessary parsimony in designing treatment plans, a highly desirable feature when operating in juvenile justice settings.

The impetus for introducing this model into the study of delinquency derives from two lines of empiric evidence. First is the relative lack of success of purely criminologic approaches to improve outcomes in delinquent youths (Steiner et al., 1999). Usually, juvenile delinquency has been handled by placements outside of parental homes, removal of parental rights, counseling sessions, and supervision of schooling. During confinement, juveniles receive similar services in a more intensive fashion. Such interventions, however, produced few positive results: Several reviews from the past decades end on a very pessimistic note (Bartol and Bartol, 1989). In the state of California alone, the population of incarcerated juveniles has doubled in the last 10 years (Tinklenberg et al., 1996), whereas recidivism rates of 67% within 4.5 years of their release have been reported (Steiner et al., 1999). From our perspective, such lack of response to selective incapacitation, as incarceration is referred to often in the criminologic literature (Haapanen, 1990), or generic treatment suggests the presence of a substantial psychopathologic component in delinquent youths. In many cases, their behavior has become independent of social context—one of our defining criteria for psychopathology (Steiner and Hayward, 2000).

The second impetus for our model is the fact that there is an accumulating body of evidence that prevalence rates of psychopathology, as traditionally defined within psychiatric nomenclatures, are exceedingly high in delinquent populations either on parole or within confinement. Coccozza (1992) first called attention to this fact in the early 1990s. As mentioned, studies of specific diagnoses yielded very high prevalence rates of PTSD (Cauffman et al., 1998; Foy et al., 1996; Steiner, Garua, Matthews, 1997), dissociative disorder (Carrion and Steiner, 2000), and other diverse psychopathology, especially in comparison to nonclinical populations and on par with prevalence rates found in hospitalized clinical populations (Atkins et al., 1999; Pumariega et al., 1999).

In the first results of our own screening study carried out in the California Youth Authority, we found that using standardized measures of psychopathology (Youth Self Report) (Achenbach, 1991) in approximately 3,638 youths (mean age = 16 years, 92% boys), 20% of boys and girls were in the clinical range on internalizing disorders, and 19% of boys and 30% of girls were in the clinical range on externalizing disorders (Table 128.1). More important, these scales correlate very highly with some personality measures, which have been shown to contribute to criminal recidivism, when age at first offending, number of previous convictions, and severity of current offense were held constant (Steiner et al., 1999; Tinklenberg et al., 1996). The variable of restraint, an obverse measure of impulsivity, relates significantly with the total psychopathology score on the Youth Self Report ( $R = -0.64$ ), the Externalizing subscale ( $R = -0.81$ ), and the Internalizing subscale ( $R = -0.33$ ). This suggests that as the youth identifies internalizing problems (e.g., anxiety and depression) or externalizing problems (e.g., aggression and other disturbances of conduct), their self-reported restraint level (i.e., ability to abstain from impulsive action) decreases. This confers on these morbid youth higher likelihood of recidivism 4.5 and 10 years out, as shown in our previous prospective studies (Steiner et al., 1999; Tinklenberg et al., 1996). The relationship between externalizing problem and restraint is much stronger than with internalizing ones, as one would expect, but the correlation with internalizing problems is not inconsequential, indicating that both types of problems can put youths at increased risk for lifetime persistent patterns of crime. Conversely, we would expect that treating some of these underlying conditions successfully will restore or improve restraint, as we have shown in a small randomized controlled clinical trial of an antikindling agent, divalproex sodium, in a sample of severe and violent conduct disorders (Steiner et al., 2000).

	Males		Females	
	Borderline (%)	Clinical (%)	Borderline (%)	Clinical (%)
Total score	14	23	16	22
Internalizing	12	19	9	20
Withdrawn	8	3	6	2
Anxious/depressed	11	7	11	5
Somatic complaints	4	4	6	2
Externalizing	12	19	7	30
Delinquent behavior	27	21	25	24
Aggressive behavior	5	2	6	8
Attention problems	7	5	10	3
Social problems	5	2	4	4
Thought problems	9	8	18	7

**Table 128.1. Percentage of California Youth Authority Youths Who Fall into Clinical and Borderline Ranges on the Youth Self Report by Gender**

These numbers are all the more impressive given the fact that this self report instrument tends to underestimate the rate of psychopathology in this population, when compared to structured interviews, such as the Diagnostic Interview Schedule for Children (DISC) (Atkins et al., 1999) and compared to other screening instruments, such as the Massachusetts Youth Screening Instrument (MAYSI) (Grisso et al., 1996), which are specifically constructed for the delinquent population (Haapanen and Ingram, 2000). We are currently in the process of concluding our own structured interview study in this population, and preliminary results show that these youths suffer from high levels of psychopathology, between one-third and twice as high as estimated by the Youth Self Report (YSR) results.

The presence of such high levels of psychopathology calls for increased child psychiatric involvement in the systems that handle delinquent youths. No longer is it sufficient to provide the occasional consultation, but is necessary to help the juvenile justice system identify unmet needs, existing and lacking resources, and provide this population with the necessary interventions and services to improve their outcomes more dramatically than we have been able to do to date. It is true that conduct problems are extremely common complications and accompaniments of psychopathology in adolescents. One could argue that such a combination should result in the assignment of youths to treatment facilities rather than incarceration. One also will need to examine the failure of the mental health system in providing care sooner prior to the advent of severe conduct complications. However, at the present time it is also clear that the climate of the country is not such that we are looking to exculpate perpetrators; quite the contrary, there are strong voices calling for increased punishment and the death penalty for juveniles. Against such resistance it is unlikely that we will be successful to assign perpetrators to treatment alone. As the debate continues as to how best handle these cases, we need to be prepared to help with an intermediate step of progress: the introduction of modern continua of care into systems of juvenile detention.

Ultimately, we probably need to help the juvenile justice system rethink where the lines of responsibility and culpability should be drawn. At the present time, it is clear that the McNaughton rule applies to only a small fraction of delinquents: Most of them are not *non compos mentis*, as the rule requires. However, as discussed, it is clear that delinquents are encumbered by factors that are not necessarily operative in the general adolescent population. Most likely we will have to make allowances for these special risks in the legal sense. The child psychiatrist must be prepared to lend a helping hand in this debate. The standard of insanity may not be the right one in these cases. To provide an illustrative metaphor, suffering from depression, PTSD, or disruptive behavior disorders and impulsivity may be more akin to having to drive on the wrong side of the road: Although most of us can perform such a feat for prolonged periods of time when visiting Australia, for instance, certain conditions may change our ability to do it well temporarily: anger, intoxication, fatigue, stress, worry, fear, and general distress, to name a few. Given what we know about the lives of children who end up being delinquent, these emotions and states are extremely common occurrences. Such interference may then manifest itself in at least two ways: (a) resorting to poor reflexive action when rapid decision making is called for (i.e., making a left turn in the car and ending up on the wrong side of the road); or (b) significant drifts in time of antisocial behavior as vigilance relaxes, firm guidelines for prosocial behavior are missed, and influences calling for antisocial action increase (i.e., ending up on the wrong side of the road while driving a deserted country lane, only to recognize that a truck is headed straight for us, while we of course think that he is on the wrong side of the road).

Finally, practitioners in this system need to be aware of the fact that delinquent youths often suffer from a wide range of other health problems, such as tuberculosis, HIV, nutritional deficits, seizure disorders, and head trauma, which can contribute to their psychiatric morbidity and possibly their delinquent behavior (Lewis et al., 1994; Needleman et al., 1996; Thompson and Farrow, 1993). Appropriate consultation should be obtained, especially in cases that present with surprising findings and do not respond to standard treatment. Usually, such consultation is readily available in the systems of juvenile justice, although the quality of services tends to vary considerably.



## SETTINGS FOR INTERVENTION

One of the main problems in the successful rehabilitation and treatment of delinquents is the discontinuity of services these youths experience as they become older and their legal status changes. In general, diversion programs are the domain of police services, social services, and sometimes mental health services. Counties run probation programs, whereas youth authorities are state institutions. During parole, the juvenile returns to county jurisdiction as a rule. All of these services are supported by separate tax dollars; each has its own structure and administrative pyramid. Needless to say, from the perspective of the juvenile offender, such a mosaic can be an absolute nightmare and directly stand in conflict with his or her needs. Because the systems are distinct, they do not provide continuity of care. Because they are administered by different professions, they reflect the parochialism of these professions. It is not unusual for a youth to receive shock incarceration, grief counseling, and victims awareness programming while on probation, vocational counseling and special education while incarcerated, and haloperidol while on parole. Coordination of treatment planning and extension of services needed beyond specific settings are the exception, not the rule. Some notable program models serve as exceptions (e.g., Milwaukee Wrap-Around) (Kamradt, 2000). Yet, if we are to be successful with this population, such conjoint management and consultative arrangements should be the rule and not the exception.

### Juvenile Hall

This is an acute detention center, a pretrial detention facility, or a short-term jail. Programs that are provided usually address schooling and mental health evaluation needs. Casework services, and sometimes day treatment facilities, are available. Lengths of stay are usually short (days to weeks).

### Group Homes and Other Residential Settings

These are long-term placements once the court has found that a youth cannot return home. Stays are extensive, and there is usually an attempt to provide suitable outpatient mental health services in conjunction with the programs offered in the placement setting.

Juvenile detention camps are one such form of extended placement (length of stay is several months). Typically county-run, they are similar in character and concept to incarceration. Placements here are a last resort before referring the youth to a state-run youth authority.

### Youth Authorities

These are state-run systems for the rehabilitation of youths. They are usually operated as part of the department of corrections, which confine delinquent youths who have failed all other alternatives or who have committed few but particularly serious crimes. Although these systems vary in character from state to state, most bear a resemblance to prisons. Programs are administered by peace officers, including treatment and parole staff. Psychologists are on staff and help with program planning and evaluations. Schooling is provided, which may even include junior college level courses. In California, the youth can be confined until the end of his or her sentence or until age 25, whichever comes first.

Programs use behavioral principles to reward good behavior and punish bad behavior. Performance in such programs is monitored daily to weekly, and different privilege levels signal the success or failure of the juveniles or wards of the state, as they are known. A variety of specialized programs are offered (alcohol and drug abuse, victim awareness, specialized sex offender needs, and anger and aggression management). These programs differ widely in their quality and quantity.

Periodically, cases are reviewed by the youthful offender parole board, which makes decisions regarding youths' treatment needs and remaining confinement time, in consultation with the staff and mental health professionals involved in each case. Ultimately, referral to parole is made, at which point the youth is transferred back to the county of origin. Any reoffense generally results in further confinement at the youth authority prior to the age of 18. Any later reoffending is usually dealt with by confinement in adult correctional facilities. Following release from the youth authority and successful completion of parole, the youth's record is permanently destroyed, and need never be mentioned, for example, in future job applications.

### Parole

The last part of a youthful offender's formal rehabilitation is usually spent in the community, with weekly or monthly parole supervision provided by a parole agent. This is a system that is severely burdened by extensive caseloads, and realistically can only provide minimal support, supervision, and structure.

## INTERVENTIONS AND OUTCOMES

Older reviews of the success rates of rehabilitation programs for juvenile delinquents usually concluded that nothing worked (Bartol and Bartol, 1989). The most spectacular failure was that of the Cambridge-Summerville program, often cited as a clinical trial of psychotherapy. Delinquents who were randomly assigned to counseling showed worse or no different outcomes than those who received no counseling (McCord and McCord, 1969). By modern standards this would not be considered a clinical trial for a variety of methodologic reasons, the most important of which is that the delivered treatment was not at all standardized. Lipsey's metaanalysis of more than 400 programs in the early 1990s offered a more positive picture of intervention programs (Lipsey, 1992). He found an overall improvement rate of 10%, which, although modest, is still impressive given the high cost of crime and incarceration and the very diverse nature of the programs reviewed. Furthermore, a significant stratification in the programs was effective in this population; successful programs tended to be privately run, were based in the community, provided interventions with a high level of intensity and duration, and used multiple modes of intervention and a great deal of structure. The most successful variant of this type of program is Henggeler and Borduin's Multisystemic Treatment (MST), which has been tested extensively and shown to be superior to many other alternatives (most notably incarceration), in terms of both efficacy and cost (Borduin et al., 1995; Henggeler, 1997). These principles reflect what is currently known of any successful psychotherapeutic intervention, delivered across all ages and in a wide range of diagnoses (Lock et al., 2001; March, 1995).

The literature is quite deficient in controlled clinical trials of psychopharmacology in this population. Table 128.2 provides a tally of all currently available psychopharmacologic agents tested in proper randomized controlled clinical trials. As of this date, we have a total of 12 studies available, with a grand total of 483 subjects involved (Mean age = 9 years). These studies are predominantly with males, are of very short duration average of about 8 weeks. Six agents were tested, in descending order of number of trials in which these medications were employed: lithium carbonate, divalproex sodium, methylphenidate, haloperidol, carbamazepine, and risperidol. Depending on how we count the study by Klein and Abikoff (1997), which produced equivocal results for one, but not the other agent, eight (or seven studies) favored the medication, four (or five) were equivocal. The studies are difficult to compare: Uniform instrumentation measuring aggression that shows age-appropriate sensitivity and specificity does not exist. All studies enrolled very small samples. Very few compared more than one agent; however, a preliminary recommendation can be given: lithium carbonate, methylphenidate, divalproex sodium, and haloperidol as well as Risperidone seem to be effective in the treatment of conduct disorders. It is highly likely that different comorbidities determine responsiveness to intervention: Concomitant attention problems would signal the use of methylphenidate, affective mood and irritability problems call for mood stabilizers, and cognitive deficits and neurologic impairment would most likely support the use of haloperidol and Risperdal. None of these studies tested a comparison behavioral or other psychotherapeutic intervention, and we can say nothing at this point about the augmenting or detrimental effects of such combination treatments. None of these studies extend over sufficient lengths of times to be able to say with confidence that such treatments continue to show effects after the initial period. We cannot comment on the long-term tolerability of and adherence to such regimens in this difficult-to-treat population. All of these questions deserve an answer. Suffice it to say that these initial studies certainly support continued investigation of these compounds in these youths, regardless of the systemic and other impediments we might encounter.

Study	n	Age	Di	Agents	Setting	Measure	Outcomes
Geier et al., 1992	21	8-11	CD	Haloperidol	4 weeks	Aggression	Drug + placebo
Campbell et al., 1994	51	7	Opp	CCO, Hal	1 month	ORE	Hal + CCO
Werner et al., 1997	22	8	Opp	CCO	2 weeks	ORE	Drug + placebo
Lee et al., 1997	22	11	CD + ODD	CCO, MPH	2 weeks	Aggression	Drug + placebo
Seidman et al., 1991	1	15	CD + ODD	CCO	6 months	ORE	CCO + placebo
Campbell et al., 1992	21	7	Opp	CCO	1 month	ORE	CCO + placebo
Carroll et al., 1994	22	7	CD	CCO	6 weeks	Aggression	CCO + placebo
Werner et al., 1997	22	8	CD	MPH	2 weeks	Oppositional/Defiant	MPH + placebo
McCauley et al., 1994	22	11	CD	CCO	1	PA Aggression Index	Drug + placebo
Henggeler et al., 1999	22	11-12	CD	MS	12 weeks	Self-Reported Conduct	Drug + placebo
Werner et al., 2002	22	7	CD	MPH	2 weeks	CD, ODD	High-Dose + low-dose
Shaw et al., 2002	22	11-12	CD	MPH	2 weeks	CD, ODD, IQ	Drug + placebo

Notes: Studies are coded as CD = conduct disorder, ODD = oppositional defiant disorder, Opp = oppositional, CD + ODD = conduct disorder and oppositional defiant disorder. Agents: Hal = haloperidol, CCO = carbamazepine, MPH = methylphenidate, CC = carbamazepine. Aggression: ODD = oppositional defiant disorder, ORE = oppositional defiant disorder and aggression.

**Table 128.2. Randomized, Placebo-Controlled Clinical Trials of Medication for the Treatment of Conduct Disorder (CD).**

## AIMING FOR CONTINUA OF CARE

We are able to distill several salient treatment principles that have promise for success, although our current knowledge base is by no means complete ( [Steiner and AACAP, 1997](#)). The nature and degree of psychopathology that associates with delinquency calls for several program characteristics that need to be implemented to increase our chances of success.

1. Because juvenile delinquents are a highly heterogeneous group, with differing needs and levels of accompanying psychopathology, it is unrealistic to expect that any one intervention or even any one program will be equally effective for all members of such a diverse population. Great care therefore must be taken to profile the youths and address their multiple problems and match interventions to the primary problem. Primacy of problem in a highly comorbid picture can be difficult to establish, but an investigation along developmental principles usually is helpful ( [Steiner and Hayward, 2000](#)). If a condition precedes another in time, it is reasonable to assume that there is a causal connection between them. If a youth is depressed at age 9, substance abusing at age 12, and conduct disordered at age 14, then it would be indicated to see his depression as the pathogenetic center for his condition. His antisocial behavior should respond to treatment of his mood and substance problems.
2. There is little room for complacency or therapeutic nihilism. Loeber and Farrington have most succinctly stated the general message of recent investigations of program efficacy in a recent summary of the accumulated wisdom of an expert panel on the issue: It is never too early and it is never too late (Loeber and Farrington, in press). Early intervention is effective and prevention is preferable to treatment if at all possible. It has a higher chance of success when fewer risks have accumulated ([GAP, 1999](#); [Mrazek and Haggerty, 1994](#)). Conversely, it is also important to continue interventions throughout adolescence because there is accumulating evidence that this approach also can be effective ( [Borduin et al., 1995](#)).
3. Multiple treatment targets should be selected, because most of these youths are deficient in many domains of functioning. Their deficits compound each other: Problems with learning lead to lack of academic success, assortative mating, and social isolation.
4. Most experts agree that there is little chance that isolated single interventions will be effective against all forms of delinquency. Interventions need to be multimodal, they need to be applied over sufficient lengths of time (i.e., over the course of months, not weeks). As much as possible, they need to be delivered in settings that retain the child in the social context to which he or she will return.
5. Simple inoculation approaches and interventions based on single-event hypotheses—boot camps, wilderness programs, and shock incarceration—are not successful ([Grizenko et al., 1993](#); [Mulvey et al., 1993](#); [Sholevar, 1995](#)). All of them, whether biologically or psychosocially based, are at best ineffective and at worst injurious, especially when used in isolation ( [Cowles et al., 1995](#); [Kazdin, 1992](#); [Mendel, 1995](#)).

## FUTURE DIRECTIONS

In this chapter we hoped to establish that juveniles in the legal system are in great need of psychiatric assistance. In order to be more effective, we need to tally the areas of research that we will need to pursue to maximize our impact onto this system. Combining our expertise in development and psychopathology we are in a unique position to offer new tools and interventions that should lead to improved diagnosis, management, and treatment of youths in the system. The following are some areas that deserve particular attention in our opinion.

1. Developmental and psychopathological information should inform the routine procedures of the system. [Steinberg and Cauffman \(2000\)](#) delineated four reasons why an understanding of adolescent development is crucial for an understanding of adolescent criminal behavior. First, adolescence is a period of rapid change, changes in physical, intellectual, emotional, and social appearances, as well as actual capabilities. Second, adolescence is a period of great flexibility in which family, peers, school, and the like can all influence development. Third, adolescence is a formative period in which negative behaviors and attitudes become harder to be modified. Finally, adolescence is a period of great variability within and across individuals. Efforts to gather empiric information on the developmental (cognitive and social) differences between adolescents and adults should be continued and ultimately used to shape public policy and legal practices for adolescents involved in our justice systems. Moreover, developmental information should be considered in conjunction with information on psychopathology.
2. Although we are just beginning to view delinquency through the lenses of development and psychopathology, we already have gained a great deal of insight as to why monochrome approaches to these youths fail. A plotting of the developmental trajectories of these illnesses and disorders so prevalent among these youths, their overlap, our ability to identify them well and quickly, and our ability to establish appropriate services for them should be a high priority given the high levels of disturbance found. We anticipate that the establishment of suitable treatment programs will conflict with criminologic necessities, and there will have to be an active discussion on how best to integrate programs. A top priority also should be the establishment of continua of care that provide appropriate services to youths across funding streams. New models of administration will be necessary to accommodate these.
3. We are becoming increasingly aware of the diverse needs of boys and girls in this system ( [Zoccolillo and Rogers, 1991](#)). It is becoming evident that girls have treatment needs that are several orders of magnitude higher than boys. It is not clear that our current systems are suitable to provide appropriate care. This is an issue that needs to be intensively investigated.
4. It is quite evident that we need sophisticated treatment outcome studies to properly evaluate the short- and long-term efficacy of multimodal treatment programs. These youths have disturbances in multiple domains that overlap and augment each other. We have to bring to bear all that is necessary and that we can muster in a rationally integrated fashion. Particularly missing are studies of modern psychopharmacologic agents that hold considerable promise for cost-effective and efficacious treatment. At the present time, youths in these systems receive low doses of medications over short periods of time. Agents chosen are usually prescribed off label, and there are few if any systematic attempts to evaluate their efficacy. This must change, especially as we have some preliminary evidence accumulating that medications are potentially helpful. Clinics need to be established and evaluated, as should sophisticated randomized controlled clinical trials. Legal impediments to these studies will need to be evaluated and reconsidered.
5. Finally, the study of youths who are in the early phases of going on to life time persistent crime patterns needs to be intensified. The unfortunate youngsters who show the early signs and symptoms of psychopathy should be a high priority. Our ignorance about them has not led us to effective interventions. We should learn considerably more about what might aid them in changing their dismal life trajectories through appropriate delineation and study. The focus to do that has been on their deficient arousal system and possibly dysfunctional frontal lobe functioning. These studies need to be repeated in young subjects, along with a clear mapping of their developmental trajectories and psychopathologic disturbances. It is only with better knowledge that we can hope to help them, and such knowledge will have to include the biological underpinnings of their conditions.

## CONCLUSIONS

Juvenile delinquency is likely to remain a serious problem in the United States for the foreseeable future. Our recent insights into the improved delineation of delinquency from psychopathologically driven antisocial behavior provide us with new opportunities to provide useful psychiatric assistance to the juvenile justice system. Delinquency is often accompanied and/or driven by high rates of coincidental and/or causal comorbidities. We have to be ready to import tested methods of intervention into the system, and retest them in cohorts of these youths. Effective treatment programs for delinquency must use multimodal approaches tailored to each youth's particular set of psychopathologies. Child psychiatrists are uniquely qualified to provide assistance and leadership in the treatment of delinquent youths, because a developmental psychopathology model best captures their problems. Successful involvement in the treatment of delinquent youth requires that psychiatrists maintain a diverse set of skills, high level of flexibility in treatment approaches, and special awareness of the legal parameters governing the rehabilitation of these youngsters. We have suggested that the extensive comorbidity observed in cases of delinquent children may provide a convenient subclassification scheme, because many of the comorbid conditions have immediate management and treatment implications ( [Steiner, 1999](#)). However, it is quite likely also that the sheer accumulation of comorbid diagnoses will have prognostic implications, as it becomes more and more difficult to address each illness in an appropriate manner. Highly compounded psychopathology seems likely to affect multiple domains of functioning and make treatment more difficult, but this hypothesis has yet to be appropriately tested.

We hope that this chapter provides an overview for the child psychiatrist to become more effective and helpful in addressing the needs of youthful offenders. In the past, we have repeatedly called for the increased involvement of our profession ( [Steiner and Cauffman, 1998](#); [Steiner and Stone, 1999](#)), yet only a small minority of child psychiatrists are involved in the diagnosis and treatment of delinquent youths ( [Steiner and Stone, 1999](#)). Most intervention programs are planned and implemented by allied health professions, such as social work, psychology, and criminology. It is our hope that we can improve this state of affairs by providing a state-of-the-art summary of relevant psychiatric research and identifying areas in which this research can have a beneficial impact on the effective treatment of



juvenile offenders.

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# 129 TELEPSYCHIATRY: AN EMERGING SERVICE DELIVERY MODEL IN CHILD AND ADOLESCENT PSYCHIATRY

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The explosion of advanced telecommunications technology has changed the day-to-day lives of countless people. A range of technological innovations, from the Internet to intercontinental two-way audiovisual teleconferencing, has helped to minimize the barriers that space and time once placed in the process of human communication and interaction. Telemedicine is most generally described as the use of advanced telecommunications technology to deliver health care at a distance. Because it is not within the scope of this article to discuss all of the various nuances of telemedicine, readers are referred to [Bashshur \(1995\)](#) for a more definitive discussion of telemedicine. On the other hand, common sense suggests that innovations on such a large scale will lead to multiple applications in health care and in psychiatry, in particular. In fact, telepsychiatry has been in use for over 40 years ([Baer et al., 1997](#)).

## BACKGROUND

The use of telemedicine first documented in professional literature is the use of closed-circuit television at the Nebraska Psychiatric Institute in the early 1950s ([Wittson and Benschoter, 1972](#)). The first reference to *telepsychiatry* is found in a description of psychiatric consultation at Massachusetts General Hospital that was completed through the use of interactive television ([Dwyer, 1973](#)). Since then, numerous telepsychiatry initiatives have occurred and are documented in the literature ([Baer et al., 1997](#)). However, most efforts in telepsychiatry have not been specific to child and adolescent patients and have been alternatively described as “psychiatry” or as “mental health” in general ([Allen and Wheeler, 1998](#)).

Early references to the use of audiovisual communication technology specific to child and adolescent psychiatry are descriptions of videotaped monologues created by adolescents with drug problems ([Wilmer 1968, 1970](#)). This technique created tapes that could be reviewed by clinicians and patients *ex post facto*. Real-time, interactive audiovisual communication over distance was not yet feasible owing to exorbitant costs of the technology available at the time. In large part, the advances in technology often described as the *digital revolution* provided the impetus for calling the present period the *Information Age*. Technological advances now make it possible for real-time, face-to-face, interactive audiovisual communication to occur at a fraction of the cost of just a few years ago. This has given rise to the creation of “telemedicine” networks allowing the delivery of health care in rural and isolated areas. Whether these networks are created as statewide systems offering a full range of medical specialties ([Adams and Grigsby, 1995](#)), as networks dedicated to state mental health systems ([Graham, 1996](#)), or personal computer-based video conferencing using standard telephone lines (also known as POTS—“plain old telephone system”) ([Ermer, 1997, 1999](#)), they have the common goal of connecting patients and clinicians. Readers with an interest in the technological and operational aspects of telemedicine in general may find additional information in a book by [Rashid Bashshur and colleagues \(1997\)](#), the definitive work on the subject to date.

## TELEPSYCHIATRY WITH CHILDREN AND ADOLESCENTS

The first documented telepsychiatry program specific to children and adolescents involved the establishment of bidirectional video linkage of Mount Sinai School of Medicine in New York with a child health clinic in east Harlem ([Straker et al., 1976](#)). This project preceded the development of digital technology. At the time of the project's inception, analog signals were sent through coaxial cable, which is roughly the equivalent of closed-circuit TV. The authors concluded that the use of this technology was successful as a means of service delivery to inner-city children.

Over the next two decades, child and adolescent telepsychiatry went through a period of slow or no growth. In the early 1990s, improvements in technology allowed for innovative projects in all areas of telemedicine to proliferate. These programs typically were oriented to delivering services in rural and isolated areas. Child telepsychiatry programs were developed in Newfoundland and Labrador, Canada ([Elford, 1998](#)), Australia ([Gelber, 1998](#); [Gelber and Alexander, 1999](#)), and rural Kansas ([Ermer, 1997, 1999](#); [Sargent et al., 1999](#)) and Kentucky ([Blackmon et al., 1997](#)). Descriptions of these programs offer the common feature of linking child and adolescent psychiatrists to patients at a distance. Although rigorous evaluation of these programs only now is beginning to occur, the authors concluded that these programs offered clinical interactions that were comparable with live, face-to-face interaction. Typically, these projects were small in scope and saw limited numbers of patients. Only recently have randomized, controlled trials of child and adolescent telepsychiatric services been published in the professional literature ([Elford et al., 2000](#); [Gelber and Alexander, 1999](#)). Gelber and Alexander conclude that there are “significant benefits in using the technology,” but add, “it is important to keep monitoring the technology's effect to ensure best practice standards are maintained” (p. 22). The authors argue that videoconferencing is not a substitute for face-to-face contact, but is a tool that complements live interaction. [Elford et al. \(2000\)](#) conclude that child psychiatric assessments can be conducted successfully using personal computer-based videoconferencing technology. These findings are consistent with the findings of [Gelber and Alexander \(1999\)](#). The authors argue that no difference was found in the satisfaction levels of patients or parents, mirroring the findings of [Rohland et al. \(2000\)](#) that telepsychiatry is acceptable to rural populations. A high level of consumer satisfaction with telemedicine child psychiatry consultation has been documented by [Blackmon et al. \(1997\)](#) in rural Kentucky. In fact, most of the parents participating in the [Elford et al. \(2000\)](#) project preferred videoconferencing over traveling long distances. [Karp et al. \(2000\)](#) demonstrated that delivery of services by telemedicine not only was acceptable to families of children with special health care needs, but resulted in decreased out-of-pocket patient travel costs. [Ermer \(1997, 1999\)](#) argues that the quality of clinical interactions by telepsychiatry appears to be comparable with live, face-to-face meetings with children in need of psychiatric services.

## AREAS OF EXPANSION

Child and adolescent telepsychiatry is likely to grow in the coming years. Programs specific to child psychiatry as a stand-alone service will continue to offer services. The integration of child and adolescent psychiatry into other areas is beginning to be documented in the literature. School-based mental health services ([Sargent et al., 1999](#)) and integrated school-based mental health and learning evaluations by telepsychiatry have been documented ([Sargent J., 2000](#)). Likewise, psychology and learning evaluations in schools by telepsychiatry demonstrate that other mental health professionals also have begun to use this means of communication ([Sargent M., 2000](#)). Although school-based telemedicine clinics are not typically established for the purpose of dealing with child behavioral/emotional issues, [Whitten and Cook \(1999\)](#) document a number of cases of children seen for this specific purpose in a school-based telemedicine program.

Integration of telepsychiatry into programs for children with other health care needs is a trend that is likely to continue. Integration of mental health services in school-based telemedicine programs has been discussed, but there are many other examples of programs where telepsychiatry may be useful. [Wheeler \(1998\)](#) describes a pediatric telemedicine network for children with special needs in Texas where technologically dependent children can receive a range of services (p. 17). Homeless children are seen through a telemedicine link in Tampa, Florida (p. 18). The only child psychiatrist in South Carolina fluent in American Sign Language uses telemedicine to see deaf clients from the state School for the Deaf and Blind (p. 20).

Other programs incorporating telepsychiatry include a multidisciplinary team consisting of a child psychiatrist, pediatric neurologist, and other health professionals who see children and adolescents by telemedicine in a pediatric multispecialty clinic for children with significant neurologic, psychological, and social problems ([Vought et al., 2000](#)). [Hilty et al. \(2000\)](#) offer a case example of telepsychiatric consultation in a primary care setting where consultation specific to attention deficit/hyperactivity disorder is offered by telemedicine.

## OTHER USES

Use of telemedicine systems for clinical evaluation in child and adolescent psychiatry is not the only way that networks are being used. A telemedicine network used in South Australia and the Northern Territory is used to deliver educational services to child and adolescent mental health workers. [Mitchell et al. \(2000\)](#) document the

benefits of networking and peer support, reduced travel costs, and improved efficiency of health services. At the same time, the costs of the program, the lack of technical support, and the need for staff training (on using the technology) are all problems encountered in using the network. Using telepsychiatry technology for delivering education and training is likely to be an area of growth in coming years.

## PROBLEMS WITH TELEPSYCHIATRY

Adoption of a new idea, even when it has advantages over the status quo, is not easy. [Everett M. Rogers \(1995\)](#) describes the process as “diffusion of innovations” in a classic work of that title. In essence, Rogers offers a model process through which all innovations move, as they become part of the mainstream thinking and practice. Telepsychiatry is no different from any other innovation. As with the adoption of the telephone, it will take some time before telemedicine is part of the mainstream of medical practice. Likewise, the adoption of telepsychiatry will take time and will encounter obstacles on the way. Although patients may be blamed for the lack of acceptance of telepsychiatry, adoption of the technology by child and adolescent psychiatrists may be more of an obstacle. Even successful programs may have unanticipated consequences or problems because of the novelty of telepsychiatry ([Zaylor, 1999](#)).

In July 1998, the American Psychiatric Association Board of Trustees approved a “Resources Document on Telepsychiatry Via Videoconferencing” describing telepsychiatry applications and other issues. Issues related to privacy, confidentiality, informed consent, medical records, and training/licensure/liability are discussed. These areas represent potential problem areas in the adoption and implementation of telepsychiatric programs. For space reasons, it is not possible to provide a thorough description of these problem areas; suffice it to say that the same problems exist in most live, face-to-face practice situations, especially in the evaluation and treatment of persons at a distance. The unique problems of child and adolescent telepsychiatry are those problems that come with the creation of a widely dispersed caseload where communication depends on the telecommunications infrastructure. As such, the variables affecting telecommunications infrastructure (e.g., weather, integrity of the technology, network support, financial support) may directly or indirectly affect the psychiatric intervention.

## SUMMARY

Telepsychiatry is only one of many major changes underway in the delivery of child and adolescent psychiatric services. The proliferation of telemedicine programs and concomitant telepsychiatry programs suggests that service delivery through advanced telecommunications technology is becoming a mainstream activity in both adult and child and adolescent psychiatry. The Committee on Psychotherapy Work Group on Training and Education of the [American Academy of Child and Adolescent Psychiatry \(1998\)](#) recently issued “Recommendations for Psychotherapy Training,” which specifically refers to telemedicine as a means of linking programs strong in psychotherapy training with other training programs. Although this cannot be taken as a full-scale endorsement of telepsychiatry practice, it is an indication that the advantages of telepsychiatry are being recognized and adoption of this innovation is in process. It probably is most accurate to describe the adoption of telepsychiatry as in an early stage. At this point, there are a number of “early adopters” ([Rogers, 1995](#)) using telemedicine, but most child and adolescent psychiatrists are just beginning to learn about its availability and advantages. The adoption or rejection of child and adolescent telepsychiatry really is not in question. What remains to be seen is the degree to which telepsychiatry will be adopted as a part of mainstream child and adolescent psychiatric practice.

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### CASE ILLUSTRATION

Tina, a 15-year-old, mildly retarded girl, was referred to her primary care physician because of concerns about her angry outbursts at home and school. Tina recently attempted to run away from home but was picked up by the local police and spent the next 24 hours in juvenile detention. Her behavioral changes began after starting special education classes in a new high school 4 months ago. Her parents and teachers described Tina as a polite teenager who previously never got into trouble.

The nearest child psychiatry services were 80 miles away. The nearest child psychiatrist in practice was 30 miles away and did not provide care to Medicaid patients. The Community Health Center where Tina's physician practiced recently had become part of the statewide telemedicine system by initiating a child telepsychiatry clinic. The telepsychiatry system included a T-1 connection with television monitors projecting video images at real time. This allowed the psychiatrist to interview Tina alone, meet with her parents, and consult with her special education teacher, school social worker, and referring physician. An initial diagnosis of major depression was made without evidence of suicidal, delusional thinking or hallucinations. Recommendations included individual therapy with Tina, parent counseling, school consultation, and a trial of medication. Tina's parents refused consideration of medication but were eager to continue seeing the psychiatrist through the telepsychiatry clinic. Although therapy with an adolescent had not been attempted, the psychiatrist agreed to see Tina weekly, as well as provide consultation to her family, school, and local physician. Over the next 6 months there was marked improvement in her mood, as well as adjustment to high school.

The advantages to telepsychiatry in this case were many. Tina herself indicated that she liked talking with the doctor on the television. Communication between all persons involved in her care, including her father, was possible because the father could attend sessions without missing work. The family did not have to travel long distances, the school was able to participate directly in both assessment and treatment, and the local physician was able to receive consultation from the specialist.

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### CASE ILLUSTRATION

The second case illustrates a different model of child telepsychiatry. Terry is a 24-month-old girl referred to a multidisciplinary neuropsychiatric team at a major medical center in Georgia, 200 miles north of the family's home. Terry presented with delays in speech, social adaptation, and gross motor skills. Her mother was concerned that her daughter was autistic. At the time of the on-site evaluation, the child was fussy and noncompliant, the young mother was uncomfortable, and there was little understanding of the resources available in the home community. Medical records were incomplete, making a definitive diagnosis and treatment recommendations difficult.

The same multidisciplinary team conducted ongoing telepsychiatry consultations at a health clinic not far from Terry's home in south Georgia. Terry was seen within the next month. The local Early Intervention team, a state and federally supported program providing multidisciplinary services to children from birth to age 3 years presenting with developmental problems, was contacted and able to conduct a preliminary developmental assessment of Terry in the home. Terry was seen by telepsychiatry by the specialty team at the medical center. The technology allowed the child neurologist to complete a neurologic examination, the occupational therapist to assess Terry's gross and fine motor movements, and the other specialists to complete their respective diagnostic evaluations. The assessment was facilitated by the information provided by the local team. The diagnosis supported the mother's impression of autism. Local care was able to provide speech therapy, occupational therapy, a social skills group, and in-home parent teaching to help Terry's mother. The team was able to follow Terry through the telepsychiatry clinic every 6 months for reassessment and treatment changes.

The advantage of telepsychiatry in this situation included the ability of multiple disciplines to work as a team providing specialty care. The child and single mother did not have to travel long distances for a comprehensive evaluation and follow-up consultation. The mother expressed satisfaction with telepsychiatry not only because of the decreased need for travel but because of the simplicity of seeing the specialists in one room, thus avoiding the difficulties of moving between different specialty clinics in large medical centers. Local health care providers were enabled to provide care at a critically important age for Terry with the support of distant specialists.

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# 130 RECRUITMENT, TRAINING, AND CERTIFICATION IN CHILD AND ADOLESCENT PSYCHIATRY IN THE UNITED STATES

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As we begin the 21st century, there are differing opinions about the number of recruits into child and adolescent psychiatry that is necessary to fulfill the research, educational, and service needs of the field and of society. Needs assessment studies routinely show that 5% or 6% of children have a severe mental disorder, and another 10% to 15% have a diagnosable disorder ([Costello, 1989](#); [U.S. Department of Health and Human Services, 1999](#)). This indicates that there should be two or three more times the current number of practitioners and researchers, which is approximately 7,000.

## THE HISTORY OF TRAINING

Medicine began by being passed down as an apprentice-taught skill, not as a university-taught education. Even after the advent of schools of medicine, and up until the middle of the 20th century, the science of medicine, learned at the university, was considered far less important than the art of medicine, learned from a more experienced colleague. It is because of this tradition that we still speak of residency training rather than residency education.

Child psychiatry in this country began during the first decade of the 20th century. The state of Illinois established in 1899 the Juvenile Court of Cook County, the first of its kind in the United States. As a means to study and treat delinquents, Dr. William Healy in 1909 formed a child guidance clinic, the Juvenile Psychopathic Institute in Chicago. This became the prototype for future clinics and employed a multidisciplinary staff that consisted of the so-called holy trinity: psychiatry, psychology, and social work. As child guidance clinics grew in numbers and in size during the second quarter of the century, it was increasingly necessary to train personnel for this new type of work. The clinics typically were headed by a psychiatrist, but the tasks accomplished by the staff of the three disciplines, with the exception of prescription writing and psychological testing, became increasingly interchangeable. It soon became clear that psychiatrists who worked with children must have training that was more extensive and specific than that obtained in their general psychiatry residency. Under the auspices of the Commonwealth Fund, a major conference was held in 1944 that provided an agreed-on set of skill areas that should be mastered by psychiatrists who treat children and their families ([Cohen, 1987](#)). These skill areas included growth and development, psychodynamics, work with parents, administration, and community organization. The psychoanalytic viewpoint initially was by far the most prevalent nationally, but the emphasis of any particular clinic's training was unique to the beliefs of the director, the makeup of the staff, and the clinic's setting.

The year 1946 was a watershed one for child psychiatry training. World War II was over, psychiatry had proven itself as a useful medical specialty, and many psychiatrists returned to civilian life. There was a national thrust to do all that could be done to raise children to live in a better world tomorrow. The baby boom generation was born, and an optimistic American society wanted to be assured that this new postwar generation would grow up right. Included in the Mental Health Act of 1946 were monies for the training of child psychiatrists. Also in 1946, the child guidance clinics banded together in a confederation called the American Association of Psychiatric Clinics for Children (AAPCC). The AAPCC set up a training committee. Clinics that wanted to offer approved training had to apply to the committee and be surveyed ([Krug, 1969](#)). This was a rigorous process, and only approximately one-half of the founding clinics were initially approved for training.

Scientific and training articles in the field of child psychiatry during this time tended to be published either in the *American Journal of Psychiatry*, the *American Journal of Orthopsychiatry*, *The Psychoanalytic Study of the Child*, or in pediatric journals. It became clear that child psychiatry needed its own forum, and in 1953 the American Academy of Child Psychiatry was established. This was a by-invitation-only organization for those who had made a "significant" contribution to the field. For the most part, membership in the Academy was synonymous with being a child psychiatry educator. One of the first tasks of the Academy, therefore, was to transfer training accreditation away from the clinic-based and multidisciplinary AAPCC and to an organization that was more medically oriented.

The American Board of Psychiatry and Neurology was established in 1934 and the American Board of Pediatrics in 1933. It was clear that recognition of child psychiatry as a medical subspecialty would better legitimize its status. Many of the early and influential Academy members were pediatricians originally, and there was vigorous debate whether child psychiatry would more appropriately be a subspecialty of pediatrics or of psychiatry. A mail poll was conducted of leading child psychiatrists, and the choice was made for psychiatry. However, the American Board of Pediatrics demanded, and received, the right in perpetuity to appoint a pediatrician member who would be part of the Committee on Certification in Child Psychiatry. With this stipulation, the American Board of Medical Specialties approved the petition, and the first subspecialty candidates in child psychiatry were examined in 1959.

Inextricably linked to subspecialty certification are standardized training criteria formulated through the Accreditation Council of Graduate Medical Education (ACGME). The ACGME Residency Review Committee (RRC) in Psychiatry oversees periodic surveys and decides whether to accredit and reaccredit each program for training ([Schowalter, 1987](#)). This approach was much more medically oriented than the AAPCC reviews. The ACGME demanded that child psychiatry training programs be linked to accredited general psychiatry residency programs and to medical centers approved by the Joint Commission on the Accreditation of Hospitals. These requirements forced the child guidance clinics interested in training to leave, at least somewhat, their community roots and to become more attached to medicine. It also stimulated the development of new child psychiatry training programs that were situated in medical centers rather than freestanding in the community. The relative equality between the "holy trinity" disciplines also shifted because ACGME subspecialty training requirements with each new revision increasingly emphasized the necessity for child psychiatrists to receive the bulk of their training from child psychiatrists.

In 1969, the Academy opened its doors to all child psychiatrists who graduated from or who were in training in ACGME-approved programs. In this same year, the Society of Professors of Child Psychiatry was established. The membership of this organization was limited to chiefs of academic divisions of child psychiatry. Although not every division was accredited for training, the vast majority were, and the Society's relatively small membership made its meetings ideal forums for the discussion of training issues.

The American Association of Directors of Psychiatric Residency Training (AADPRT) and the Association for Academic Psychiatry are more recently formed organizations devoted to education and training. Although they began with an almost exclusive focus on general psychiatry, both organizations in the 1980s deliberately expanded their involvement with child psychiatry training and training directors.

## PROGRAM ACCREDITATION

The ACGME consists of representatives from the American Medical Association (AMA), the American Board of Medical Specialties, the Association of American Medical Colleges, the American Hospital Association, and the Council of Medical Specialty Societies and is responsible for setting training requirements for all specialties and subspecialties approved by the American Board of Medical Specialties. There are two sets of requirements.

The General Requirements are generic and the same regardless of the specialty being reviewed. They are concerned less with the particular training area than with the overall support and surveillance provided by the medical center in which the training program is embedded. These issues include requirements for the selection of trainees and assurance that there are procedures for evaluation, evaluation feedback, grievance reporting, and due process. There also must be adequate compensation, an emphasis on education rather than on service, and acculturation help for those trainees who need it. Until the 1980s, the General Requirements were not stressed nearly as much as the Special Requirements. The ACGME site visitors, who usually survey a program every 3 to 5 years, have recently begun very seriously to query hospital officials about their involvement in and support of the individual program being reviewed. An individual program can be put on probation if the General Requirements are not fulfilled. More often, however, the threat of probation causes the medical center administration to become more forthcoming in its interest in and support of each of its residency training programs. It was the realization that many individual training programs falter because of supervisory or financial neglect by the central administration that caused the ACGME to place increasing emphasis on the General Requirements. Because hospitals and medical



schools do not like to have their programs disaccredited, most program directors have found the General Requirements to be a useful lever whereby to obtain greater institutional support.

The Special Requirements are specific to a particular specialty or subspecialty. They are revised approximately every decade, although discrete changes may be made between revisions. The revisions of the Special Requirements and the evaluation of the ACGME surveys are the responsibility of the Residency Review Committee (RRC). There are RRCs for all accredited specialties, and the RRCs report to the ACGME. RRC members for psychiatry are nominated by three organizations: the AMA, the American Board of Psychiatry and Neurology (ABPN), and the American Psychiatric Association (APA).

According to RRC meeting minutes, the RRC for Psychiatry and Neurology was organized in Chicago in October 1954. Members were chosen by the ABPN and by the AMA Council on Medical Education and Hospitals. At its October 1959 meeting, the RRC noted that the ABPN had developed a "sub-board in child psychiatry," and the question arose whether to review child psychiatry training programs separately or at the same time as the survey of a general psychiatry residency. A decision was made in favor of the former. At the March 1960 meeting, the *Essentials for Child Psychiatry* was reviewed and subsequently approved. At the October 1960 meeting, a child psychiatrist, Dr. William Langford, was invited as a guest. He reviewed 17 programs and approved 11. Dr. Langford's recommendations needed an RRC vote to become official. This format of child psychiatry program review continued until in 1967, the Committee on Certification in Child Psychiatry of the ABPN petitioned for the position of a voting child psychiatrist member on the RRC. The RRC denied this petition at its April meeting, but for the October 1967 meeting listed the child psychiatrist as an "ex-officio member without vote," rather than "guest," as had been done before. This protocol continued unchallenged until the October 1978 meeting, when the RRC expressed concern that child psychiatry program evaluation was in fact decided, if not voted on, by someone not part of the RRC. This seemed suspect functionally and legally, and the RRC requested that an official child psychiatry member be appointed by each of its two parent organizations. The ABPN approved, but the AMA Council on Medical Education refused. At the March 1979 meeting, the lone ex officio child psychiatrist reviewed 28 programs, whereas 6 general psychiatrist members reviewed a total of 30 programs, and 6 neurologist members reviewed only 15 programs. Because it was clear that more child psychiatry reviewers were needed, the RRC repeated even more strongly its request that two child psychiatrists be appointed as full Committee members. This second appeal was approved by both parent organizations, and at the March 1980 meeting two child psychiatrists attended as full Committee members. Two years later, the RRC was restructured so that the neurology and psychiatry components met on separate occasions. It also was decided to include the APA as a third parent organization for the RRC, and as part of its contribution the APA supported a third child psychiatrist member in 1983. Traditionally, the RRC Chair was from the ABPN and the Vice Chair was from the AMA Council on Medical Education. In 1985, the Committee appointed a child psychiatrist as Vice Chair, and this practice has been continued since. In 1989, it was decided to have each program's survey reviewed by two, rather than one, Committee member. This change required enlarging the RRC membership. In 1990, a fourth child psychiatrist was added, and in 1995 a fifth and sixth. In 2002, there were 115 approved residency programs, and 874 approved residency positions in child and adolescent psychiatry.

Although the initial draft of a Special Requirements revision for child and adolescent psychiatry is written by the RRC members, the draft is sent to the program directors of the accredited training sites and to the specialty's foremost national organizations for review. If major changes are made, the second draft is again sent for comment to program directors and to organizations. The revision process usually takes 2 to 3 years. The final approval of Special Requirements comes from the ACGME board. The most recent child and adolescent psychiatry Special Requirements revision went into effect in 2000. Although there are with each revision a greater number of requirements and a stricter enforcement of the requirements, most child and adolescent training directors have historically believed that the quality and quantity of requirements are fair and possible to do in the 2 years allotted ( [Schowalter, 1989](#)). There has not been a comprehensive survey since the late 1980s. At that time, approximately one-fourth of program directors believed that general psychiatry should be reduced to a single year of training, and approximately one-third believed that the child and adolescent psychiatry residency would have to be extended to 3 years before the end of the century. Although this deadline has passed, there is no ground swell for expanding to 3 years. This lack of pressure seems based less on disbelief that expanded knowledge deserves expanded training time, but on practical issues such as resident debt, lack of institutional funding, and fear of a reduction of our applicant pool.

Although there is a continuous debate as to whether child and adolescent psychiatry should, as did pediatrics from internal medicine, split from general psychiatry, the pressure for this is more through rhetoric than action. Most child and adolescent psychiatrists want the ability also to treat adults. And in practical terms, there are very few child and adolescent psychiatry training programs that have a faculty and financial base broad and deep enough to enable them to function as an autonomous department. One commonly raised proposal is to begin subspecialty training in the third postgraduate year and concentrate the next 3 years of residency on work with children and adolescents. This approach has found no support from either Directors of the ABPN or members of the general psychiatry RRC. These two bodies, crucial in the determination of any policy change, believe that less than 2 years of residency with adults is insufficient training to make one board-eligible in general psychiatry.

An innovative 5-year integrated training sequence in pediatrics, general psychiatry, and child and adolescent psychiatry, better known as the Triple Board, began as an experiment in 1985 and was approved nationwide as a combined residency in 1992 ( [Schowalter, 1993](#)). This track is sponsored by the ABPN, the ABPN Committee on Certification in Child and Adolescent Psychiatry, and the American Board of Pediatrics. For trainees in approved programs, the sponsoring bodies waive the usual training periods required for eligibility for their certifying examinations.

## CERTIFICATION OF INDIVIDUALS

Although it is the RRC's responsibility to accredit training programs, the ABPN certifies individuals as competent to practice as specialists. The ABPN determines the accuracy of the applicant's credentials in regard to schooling and residency. To be a candidate for certification in child and adolescent psychiatry, one must have completed at least 3 postgraduate years of ACGME-approved residency in general psychiatry and a 2-year approved residency in child and adolescent psychiatry. One also must have passed the written and oral ABPN examinations in general psychiatry. In 1987, the ABPN approved the expansion of the subspecialty of child psychiatry to child and adolescent psychiatry. This decision was based on the fact that the RRC Special Requirements for child psychiatry already mandated training with adolescents and that more than one-fourth of the content of the written and oral certifying examinations in child psychiatry already were devoted to the adolescent age range. An important impetus for the expansion was administrative. In the late 1980s, there was much interest in forming psychiatric subspecialties. This interest was based both on an expansion of the knowledge base in such areas as geriatric, forensic, and addiction psychiatry, and on a wish for subspecialty recognition for credentialing and differential remuneration. There was at this time also a move to form a separate subspecialty of adolescent psychiatry. By granting this age group to child psychiatry, where it was placed traditionally, the possibility of having a subspecialty of adolescent psychiatry disappeared.

Another major decision in the late 1980s for the ABPN and the Committee on Certification in Child and Adolescent Psychiatry was to move toward time-limited certification. By this is meant that every 10 years the diplomate must meet a set of criteria to remain certified for the next decade. Not only must reassessment be meaningful, but it is important to keep its cost and annoyance to a minimum. In November 1989, the ABPN decided to begin time-limited certification in both general psychiatry and in child and adolescent psychiatry beginning with examinations held after October 1, 1994. There have been many iterations during the past 10 years as to how continuing certification can be best established. The ABPN made a decision in 2000 that beginning in 2002 all recertification examinations would be taken in a secure setting. Although the ABPN originally planned to allow take-home recertification examinations, pressure from the public and from other Boards led to the final decision.

Although there have been increasing numbers of psychiatrists who take the certification examinations, both in general and in child and adolescent psychiatry, certification is technically voluntary. In point of fact, reimbursement and hospital credentialing are increasingly tied to specialty certification. Also, a number of state medical licensing boards have proposed legislation that ties medical relicensure with either the periodic passing of a state examination in general medicine or evidence within the time period of passage of a specialty board licensure or relicensure examination. Therefore, although in psychiatry and in child and adolescent psychiatry there is a "grandfather" clause that exempts anyone certified before the onset of the time-limited certificate to be recertified, hospital credentials boards and state medical license boards may nonetheless require recertification as part of their requirements.

An ongoing challenge for all examining boards is how to provide a truly competence-based examination. It is relatively easy to obtain reliability with a multiple-choice written examination. To show validity (i.e., that the test performance is linked to actual clinical practice) is much more difficult. With any oral examination, the candidate always has the variability of hard or easy graders in the examiners he or she draws. To avoid these variables, the American Board of Internal Medicine dropped its oral examination in 1977, and the American Board of Pediatrics dropped its in 1989. The ABPN is the only board still to use patients with an oral examination. In the child and adolescent psychiatry examination, there is an adolescent patient. With a patient interview, reliability always suffers because the candidate has the luck of the draw for both patient and examiners. Although fairness and reliability are much surer for written examinations, especially with multiple-choice questions, these questions cannot show a candidate's skill level in interacting with a patient. Videotapes of patients, written vignettes, and actors who simulate patients are all possible substitutes for actual patients. Videotapes of patients are used by many boards, but some candidates seem unable to extract material well from a relatively lifeless tape. It seems unfair to fail candidates on video-watching rather than on clinical psychiatry. Written vignettes usually are brief and used more as a stimulus for discussion than as a substitute for a clinical interview. Written patient management problems help keep the focus of the examination on process rather than product ( [Langsley, 1986](#)), but there is little evidence to prove that the ability to answer didactic questions about psychiatry is highly correlated

to practicing well. In the future, there undoubtedly will be an increased use of an examination format that uses computers—for example, with a CD-ROM format. In 1999, the ABPN voted to build a computer center at its headquarters in Deerfield, Illinois. Other Boards have similar facilities in other parts of the country. In time, these centers will allow each other's candidates to take computer examinations in a facility that is geographically close. Although CD-ROMs can present extended video clips of patients and extensive historic, imaging, and laboratory information, they are not able to assess professional demeanor. To measure this most essential psychiatric skill is the main argument for the use of patients or patient simulators. In public opinion surveys, attitudinal qualities are judged to be equal or close to equal to a practitioner's knowledge skills ([Langsley and Yager, 1988](#)). This is even true for much less patient-oriented specialties, such as radiology ([Tarico et al., 1986](#)). In 1999, the ABPN began exploring the use of standardized patients (SP). An SP is an upgraded simulated patient. Psychiatrist experts on various disorders write scripts for what the SP will say and how he or she will act. The SPs are programmed to give the same history and the same responses with every candidate, based on the candidate's ability to elicit them. Standardization eliminates the reliability problems associated with actual patients' vast diversity in severity and in their willingness and ability to cooperate with the candidate. SPs might be belligerent, seductive, or quiet, but it will be enacted the same for every candidate. Although standardization can be presented on a CD-ROM, with the SP, the candidate's personal empathy and professionalism can be measured directly by the SP on a standardized scale. Given the SP, examiners also are standardized to a much greater degree in their question-asking and less able to ride personal hobby-horses. SPs have been used extensively for medical school and licensing examinations ([Hodges et al., 1998, 1999](#)), but never in as high-stakes an examination as Board certification. Some ABPN Directors did not believe SPs could portray mental illness realistically for trained clinicians, but all were convinced after observing SPs in a mock examination. It will be decided in 2003 whether to begin planning to substitute SPs for actual patients in future examinations, including the adolescent section of the child and adolescent examination.

It is clear that a continuum of evaluation is most accurate—a motion picture rather than a snapshot of a candidate's functioning. Program directors can be sources of information to the ABPN about a candidate's clinical and ethical behavior during residency, but the training director's understandable bias often renders such judgments unrealistically positive. There is pressure on the ABPN from the field to have residents take and pass the Part I written examination in the third or fourth postgraduate year. This probably will not much affect child and adolescent psychiatry residencies because many residents take this examination now during their subspecialty years.

Recertification provides an ideal opportunity to create a prospective, longitudinal research component to follow diplomates' careers and to test results over time. Perhaps in this way it can be discovered how to understand better and test for competence as demonstrated in actual clinical performance. The concept of maintenance of certification has become popular with some other Boards. This includes chart audits, as well as periodic examinations. Because of the necessary confidentiality of psychiatric charts, this approach is unlikely to be adopted by the ABPN.

## RECRUITMENT

Child and adolescent psychiatry has a chronic recruitment problem. The Graduate Medical Education National Advisory Committee (GMENAC) Report of 1980 declared child psychiatry the least sufficiently staffed medical specialty and recommended that it triple its numbers from 3,000 to 9,000 in 10 years. The number was not even doubled in the time allotted. Surveys have shown a shortage of child and adolescent psychiatry trainees, faculty, and, especially, researchers ([Beresin and Borus, 1989](#); [Enzer, 1989](#); [Institute of Medicine, 1989](#)).

The last national conference on recruitment for child and adolescent psychiatry was held in 1989. There was consensus that the greatest need was for more researchers and faculty. It also was acknowledged that improvement of the quantity and quality of residents was the best approach to enhance faculty recruitment. Deterrents to recruitment include a relatively long training period coupled with relatively low income. The latter is due to the high-ambulatory, low-procedure nature of work with patients whose families have not yet reached their earning capacity. In addition, researchers' training is extended, and their salaries tend to be lower than those of practitioners and clinically based faculty. The conference participants proposed a great number of suggestions, the foci of which extended from undergraduates and medical students to postfellowship researcher support ([American Academy of Child and Adolescent Psychiatry, 1990](#)). Personal contact is crucial for recruitment. This can be provided through the teaching of child development in college and medical schools, the organization of student child psychiatry clubs, and the provision of individual mentors for interested medical students and general psychiatry residents. Good communication must be established and maintained between child psychiatry and general psychiatry training directors. It is essential that child and adolescent psychiatry rotations be provided to general psychiatry residents before the time at which they must apply for subspecialty training. A greater emphasis on research should be added to the Special Requirements, and the RRC should be more vigorous and eliminate programs that cannot provide comprehensive training. More money is necessary from government, foundation, proprietary hospital, endowment, and other sources to better support child and adolescent researchers through their postresidency fellowships and early faculty years. Without more full-time researchers, there is little likelihood that investigative productivity can be substantially enhanced.

The rise of behavioral managed care organizations and the plummet of the percentage of health care dollars going into psychiatric services ([National Association of Psychiatric Health Systems, 1999](#)) and the federal bias toward primary care training in the 1990s have made a career in psychiatry relatively less appealing. Child psychiatry has suffered proportionally less because for an extra 1 or 2 years of training, residents can be a dual specialist for the rest of their professional careers. Being able to treat, teach about, and supervise for adult, child, and adolescent patients make the subspecialist sought after. In 2000, the American Academy of Child and Adolescent Psychiatry appointed a Task Force on Work Force Needs to update the country's needs for child and adolescent practitioners.

Recruitment into child and adolescent psychiatry varies greatly throughout the world. There are tremendous national differences in the pathways into child psychiatry and in the formal organization of child psychiatry. Therefore, issues for recruitment must be understood in the context of the status of child psychiatry as a profession, the availability of training programs, and the nation's commitments to child and family welfare. In many of the less developed nations, there are few formally trained child psychiatrists. There are nations in which there are fewer than half a dozen individuals who would call themselves child psychiatrists and in which there is not one person who has had training equivalent to that in the United States. Often in such developing nations, child mental health is covered by pediatricians or psychiatrists with special interests in children who may have a rather informal program of practical experience and some specialized tutorials/seminars. The latter focus is on public health and severely disturbed children. In developing nations, including virtually all of Africa and most of the Middle East and Latin America, the field of child psychiatry as a whole is still in early stages of development. There are individuals in these nations who would like to become trained in child psychiatry/child mental health, but there are no or only very limited training opportunities. In these situations, the central issue is not recruitment, but national priorities and availability of academic resources.

In developed countries, the major pathway to child psychiatry usually is through pediatrics or through programs that lead directly to child psychiatry and include training in general psychiatry, pediatrics, and neurology along the way as part of the child psychiatry training. There are few nations such as the United States that require full and prior training in general psychiatry. In many of the developed nations, other than the United States, recruitment into child psychiatry is not a major issue. In some nations, a rate-limiting issue is the number of residency slots that are available (e.g., in the Netherlands, these are limited and individuals may not be able to find residency training in areas of interest). There are no special problems in recruitment in nations such as Germany, Sweden, Israel, the Netherlands, South Korea, or the United Kingdom. These are nations in which child psychiatry is secure as a medical profession. In these nations, child psychiatry usually is a distinct field and not just a subdivision of general psychiatry. There are definite differences in Europe in the nature of what is considered child psychiatry. In Italy, for example, there are literally thousands of child psychiatrists. They are divided into two distinct groups. Most function in community settings and are essentially social advocates/facilitators/family guides; a smaller number are more closely related to neurology; and some are devoted to psychoanalysis. On the other hand, child psychiatrists in Germany and the Netherlands are very similar in orientation and function to those in the United States, although they tend to serve much more as specialists and consultants. Those in Sweden usually are trained in pediatrics and function as pediatric subspecialists. In all of these nations, child psychiatrists usually are not trained in adult psychiatry. Because of the possibility of physicians moving across national borders in the European Union, the European nations have adopted a uniform set of recommendations for the scope of training in child psychiatry. In content, these are similar to those in the United States.

## CONCLUSIONS

Child and adolescent psychiatry is the first subspecialty of psychiatry, but subspecialty training continues to struggle with the field's level of interdependence with general psychiatry. This has been true for time allotment, faculty duties, standards, and recruitment. How best to train and certify child and adolescent psychiatrists is an ongoing task made more complicated by the burgeoning of research and clinical data, coupled during the 1990s with daunting cutbacks in training and clinical monies. Answers will arise only through a constant dialogue between the members of the American Academy of Child and Adolescent Psychiatry, the Society of Professors of Child and Adolescent Psychiatry, the Residency Review Committee, and the American Board of Psychiatry and Neurology. A permanent structure for this ongoing debate is necessary and should be developed.

*Editor's Note:* For discussion of antecedents of child and adolescent psychiatry found in historical accounts of the discovery of the child, see [Chapter 133](#). For further



discussion of the history of mental health delivery systems in the United States, see [Chapter 133](#).

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# 131 RESPECT FOR CHILDREN AS RESEARCH SUBJECTS

Robert J. Levine, M.D.

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“So act as to treat humanity, whether in thine own person or in that of any other, in every case as an end withal, never as a means only.” The German philosopher Immanuel Kant provided this formal statement of the ethical principle of respect for persons. Persons are to be regarded as ultimate values in and of themselves; they are not to be used merely as means to another’s goals.

Those who conduct research involving human subjects first define their goals and then identify persons whom they use as means to accomplish these goals. This is not unethical. What is proscribed is the use of persons merely as means—“as means only.” To avoid this, researchers are required both ethically and legally to secure the approval of persons to be used as research subjects through a process called informed consent. If this approval entails acceptance of the researcher’s goals, then the subject is not used merely as a means. Rather, the subject freely chooses to embrace the goals as his or her own and thus remains an end.

This chapter is concerned with informed consent and other issues related to the principle of respect for persons (e.g., privacy and confidentiality) as they relate to the involvement as research subjects of adolescents and children of various ages.

## ETHICAL PRINCIPLES

The basic ethical principles identified by the National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research ( [National Commission, 1978](#)) as those that should underlie the conduct of research involving human subjects are “respect for persons,” “beneficence,” and “justice.” These principles were endorsed subsequently by the President’s Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research ( [President’s Commission, 1983](#)) as “basic values” for medical practice as well as for biomedical and behavioral research, calling them by somewhat different names: “respect,” “well-being,” and “equity.” According to these authoritative commissions, research involving human subjects should be conducted in accord with norms or rules designed to uphold and embody these basic principles or values. These rules are assembled in federal regulations for the “protection of human research subjects”; most relevant to the present concerns are those of the Department of Health and Human Services (Code of Federal Regulations, Title 45, Part 46; hereafter abbreviated as 45 CFR 46) and the Food and Drug Administration (Code of Federal Regulations, Title 21, Parts 50 and 56; hereafter abbreviated as 21 CFR 50 and 21 CFR 56).

In this chapter there are frequent references to federal regulations. This is not intended to suggest that all ethical considerations are reflected adequately in the law. Rather, the regulations in this field generally represent a broad social consensus about what ought and ought not to be done. Even for research not covered by the regulations, they have come to be regarded as establishing a community standard, departures from which require justification ( [Levine, 1988](#)). The regulations include both substantive and procedural rules (norms). A substantive rule specifies behaviors that are required (or forbidden) because they are morally right (or wrong). Some procedural rules specify procedures that should be performed to determine the most appropriate behavior when the behavior has not been specified by a substantive rule. Other procedural rules are designed to provide documentary evidence that research has been conducted in accord with the relevant substantive rules ( [Levine, 1988](#)).

### An Illustration

The principle of respect for persons requires that human persons must be treated as autonomous agents. The substantive norm that requires informed consent is a specification of one way in which this principle is to be made operational in the conduct of research involving human subjects. The procedural norm that requires review and approval by an institutional review board affords a method for determining what specific bits of information must be divulged to prospective subjects in a particular research protocol. Another procedural norm that requires the signing of a consent form provides documentary evidence that the behavior required by the substantive norm has been accomplished.

According to the National Commission:

Respect for persons incorporates at least two basic ethical convictions: First, that individuals should be treated as autonomous agents, and second, that persons with diminished autonomy and thus in need of protection are entitled to such protections.

An autonomous person is . . . an individual capable of deliberation about personal goals and of acting under the direction of such deliberation ( [National Commission, 1978](#)).

To show respect for autonomous persons requires that we leave them alone, ven to the point of allowing them to choose activities that might be harmful (e.g., hang gliding), unless they agree or consent that we may do otherwise. We are not to touch them or encroach on their private spaces unless such touching or encroachment is in accord with their wishes. Our actions should be designed to affirm their authority and enhance their capacity to be self-determining; we are not to obstruct their actions unless they are clearly detrimental to others. We show disrespect for autonomous persons when we either repudiate their considered judgments or deny them the freedom to act on those judgments in the absence of compelling reasons to do so.

Clearly, not every human being is capable of self-determination. The capacity for self-determination matures during a person’s life; some lose this capacity partially or completely, owing to illness or mental disability or in situations that severely restrict liberty, such as prisons. Respect for the immature or incapacitated may require one to offer protection to them as they mature or while they are incapacitated.

Because the central focus of this chapter is on respect for persons, it is necessary to emphasize that the other two principles are of equal importance in the sense that they have equal moral force. Research involving human subjects can be considered ethically justified if, and only if, it is adequately responsive to each of the three basic ethical principles ( [Levine, 1988](#)). As we shall see, considerations of justice and beneficence place constraints on, for example, whom we can ask to serve as research subjects and how much risk we may ask them to accept in the interests of research.

## INFORMED CONSENT

Principle I of the [Nuremberg Code \(1949\)](#) provides the definition of consent from which definitions supplied in all subsequent codes and regulations are derived:



The *voluntary* consent of the human subject is absolutely essential.

This means that the person involved should have *legal capacity* to give consent; should be so situated as to be able to exercise *free power of choice*, without the intervention of any element of force, fraud, deceit, duress, over-reaching or other ulterior form of constraint or coercion; and should have sufficient *knowledge* and *comprehension* of the elements of the subject matter involved as to enable him to make an understanding and enlightened decision. (emphasis supplied)

Thus, consent is recognized as valid if it has each of these four essential attributes: It must be competent (legally), voluntary, informed, and comprehending (or understanding).

It is through informed consent that the investigator and subject enter into a relationship, defining mutual expectations and their limits. This relationship differs from ordinary commercial transactions in which each party is responsible for informing himself or herself of the terms and implications of any of their agreements. Professionals who intervene in the lives of others are held to higher standards. They are obligated to inform the layperson of the consequences of their mutual agreements.

It is worth noticing that the Nuremberg Code defines and requires “voluntary consent.” Since 1957 this term has been replaced by “informed consent,” a term that reflects an idealized vision of the person as a rational, self-determining agent ( [Katz, 1984](#)).

Federal regulations identify “elements” of information that must be transmitted during the negotiations for informed consent (45 CFR 46.116a); these are:

1. A statement that the study involves research, an explanation of the purposes of the research, and the expected duration of the subject's participation, a description of the procedures to be followed, and identification of any procedures that are experimental
2. A description of any reasonably foreseeable risks or discomforts to the subject
3. A description of any benefits to the subject or others that may reasonably be expected from the research
4. A disclosure of appropriate alternative procedures or courses of treatment, if any, which might be advantageous to the subject
5. A statement describing the extent, if any, to which confidentiality of records identifying the subject will be maintained
6. For research involving more than minimal risk, an explanation as to whether any compensation and an explanation as to whether any medical treatments are available if injury occurs and, if so, what they may consist of or where further information may be obtained
7. An explanation of whom to contact for answers to pertinent questions about the research and research subjects' rights, and whom to contact in the event of a research-related injury to the subject
8. A statement that participation is voluntary, refusal to participate will involve no penalty or loss of benefits to which the subject is otherwise entitled, and the subject may discontinue participation at any time without penalty or loss of benefits to which the subject is otherwise entitled

In addition, according to the regulations, the following elements must be provided “when appropriate”(45 CFR 46.116b):

1. A statement that the particular treatment or procedure may involve risks to the subject (or to the embryo or fetus, if the subject is or may become pregnant) that are currently unforeseeable
2. Anticipated circumstances under which the subject's participation may be terminated by the investigator without regard to the subject's consent
3. Any additional costs to the subject that may result from participation in research
4. The consequences of a subject's decision to withdraw from the research and procedures for orderly termination of participation by the subject
5. A statement that significant new findings developed during the course of the research that may relate to the subject's willingness to continue participation will be provided to the subject
6. The approximate number of subjects involved in the study

The regulations define only minimum standards for informed consent. In most cases it seems appropriate to supplement these basic requirements with additional elements of information ( [Levine, 1988](#)). For example, prospective subjects should be told why they have been selected as invitees to participate in the research; ordinarily, this consists of a statement of the major inclusion and exclusion criteria for the protocol. In addition to the statement of “additional costs to the subject” required by the regulations, there should also be accurate statements of any cash payments or other economic advantages associated with participation in the research as a subject.

How does one determine whether any particular fact (e.g., any particular risk of injury) must be disclosed? The legal criterion for disclosure in the context of medical practice is “material risk”; that is, any fact that is material to the patient's decision must be disclosed ( [Holder, 1978](#); [Levine, 1988](#)). The determination of which risks are material in that they must be disclosed may be accomplished according to three different standards or tests ( [Curran, 1974](#); [Levine, 1988](#)). Until recently, the prevailing standard was that of the “reasonable physician”; the determination of whether any particular risk or other fact should be disclosed was made on the basis of whether it was customary to do so in the community of practicing physicians.

The standard that is applied most commonly is the “reasonable person” or “prudent patient” test. In the case of [Canterbury v. Spence \(1972\)](#), the court held that the disclosure required was determined by the “patient's right of self-decision,” a right that can be effectively exercised only if the patient possesses enough information to enable an intelligent choice. A risk is thus material when a reasonable person, in what the physician knows or should know to be the patient's position, would be likely to attach significance to the risks or cluster of risks in deciding whether or not to forego the proposed therapy.

Some courts have adopted the rule that a risk is material if the particular patient making the choice or decision considers it material. Of the three standards, this rule, which some call the “idiosyncratic person” standard, is most responsive to the requirements of the ethical principle of respect for persons ( [Levine, 1988](#)). It is, however, a highly impractical standard.

In the author's view, the reasonable person standard should determine the minimum amount of information that should be imparted by the researcher to each and every prospective subject. Then, in the course of the consent discussions, the researcher should attempt to learn from each prospective subject what more he or she would like to know.

Federal regulations permit “a consent procedure which does not include, or which alters, some or all of the elements of informed consent” or, in some cases, waiver of the entire requirement for informed consent, if:

[a] The research involves no more than minimal risk to the subjects, [b] The waiver or alteration will not adversely affect the rights and welfare of the subjects, [c] The research could not practicably be carried out without the waiver or alteration, and [d] Whenever appropriate, the subjects will be provided with additional pertinent information after participation (45 CFR 46.11 6d).

Implicit in these conditions—particularly second condition—is recognition of the standard of materiality. One may not withhold any material information without adversely affecting the rights of subjects. Waivers and alterations are commonly used in research involving medical records, “leftover” specimens of tissues and body fluids from which personal identifying information has been removed, survey research, and so on. It is more problematic when researchers propose to alter information for purposes of deceiving prospective research subjects ( [Levine, 1988](#)).

The Department of Health and Human Services (DHHS) makes it clear that, “Nothing in these regulations is intended to limit the authority of a physician to provide emergency medical care, to the extent the physician is permitted to do so under applicable federal, state, or local law” (45 CFR 46.116f). Implicit in this rule is a recognition of two exceptions to the legal requirement for informed consent: the emergency exception and therapeutic privilege. <sup>1</sup>

The Food and Drug Administration (FDA) permits waiver of the consent requirement for the use of investigational new drugs (or other regulated “test articles”) in the treatment of individuals in “life-threatening situations” in which “informed consent cannot be obtained . . . because of an inability to communicate with, or obtain legally effective consent from, the subject” (21 CFR 50.23). There is also a provision in FDA regulations for an exception from informed consent requirements for emergency research; this exception is designed for research activities in which most or all of the prospective subject population will be unable to consent and it will not be feasible

(usually owing to lack of time) to get consent from a legally authorized representative (21 CFR 50.24).<sup>2</sup>

## Consent Forms

Thus far we have been considering informed consent, a process designed to show respect for subjects, fostering their interests by empowering them to pursue and protect their own interests. The consent form, by contrast, is an instrument designed to protect the interests of researchers and their institutions by defending them against civil or criminal liability. I believe that one of the reasons there has been so little successful litigation against investigators, as compared with practicing physicians, is the very formal and thorough documentation of informed consent on consent forms. Consent forms may be detrimental to the subject's interests not only in adversary proceedings; signed consent forms in institutional records may lead to violations of privacy and confidentiality ( [Levine, 1988](#)).

DHHS regulations require that:

A written consent document that embodies the elements of informed consent. . . . This form may be read to the subject or the subject's legally authorized representative, but, in any event, the investigator shall give either the subject or the representative adequate opportunity to read it before it is signed (45 CFR 46.117).<sup>3</sup>

Although the primary purpose of the consent form is to protect the interests of researchers and their institutions, it is forbidden by federal regulations to:

include any exculpatory language through which the subject or the representative is made to waive or appear to waive any of the subject's legal rights, or releases or appears to release the investigator, the sponsor, the institution or its agents from liability for negligence.<sup>4</sup>

DHHS requires that "[a] copy shall be given to the person signing the form" (45 CFR 46.117). The primary purpose of the form notwithstanding, it can and should be designed to be helpful to the subjects. Having a copy of the form will afford them an opportunity to continue to get more information as additional questions occur to them. It can also serve as a reminder of the plans they must follow in order to accomplish the purposes of research, of the symptoms they should watch for to protect their own safety, of the perils of omitting doses of drugs, and so on. It can serve as a guide to conversations they might choose to have with family, friends, personal doctors, and other trusted advisors about whether they should consent; in some cases such conversations should be recommended during the consent negotiations. In the use of these forms, however, the researcher should heed the words of the [President's Commission \(1982\)](#): "Ethically valid consent is a process of shared decision-making based upon mutual respect and participation, not a ritual to be equated with reciting the contents of a form that details the risks of particular treatments."

No consent form can be designed so as to anticipate all of any particular prospective subject's wishes to be informed. The consent form is most effective when it is viewed by the researcher as a guide to the negotiations with the prospective subject. The consent form should contain at least the minimum amount of information and advice that should be presented during the negotiations. If any substantive new understandings are developed in the process of negotiations that have any bearing on the prospective subject's willingness to participate, these should be added to the consent form signed by that individual.

DHHS regulations (45 CFR 46.117) permit waiver of the requirement for documentation of informed consent if:

1. The only record linking the subject and the research would be the consent document and the principal risk would be potential harm resulting from a breach of confidentiality.
2. The research presents no more than minimal risk of harm to subjects and involves no procedures for which written consent is normally required outside of the research context.

In some cases in which the regulations permit waiver of the requirements for documentation, it may be advisable to provide subjects with information sheets. These documents provide a written account of all information that could serve subjects' interests in ways suggested earlier. They differ from consent forms primarily in that they are not signed by subjects and retained by researchers. Thus, they afford limited protection to the researcher and the institution.

## JUSTIFICATION OF RESEARCH INVOLVING CHILDREN

Children, as a class of persons, lack the legal capacity to consent. Moreover, many of them, particularly the younger ones, are not only incapable of sufficient comprehension to meet the Nuremberg Code's standard but are also not "so situated as to be able to exercise free power of choice." It is necessary to rely on other devices to show respect for children because they cannot consent. Two of these devices are permission and assent.

Permission of one or both parents or of the legal guardian is closely related to what was formerly called "proxy consent." With few exceptions, federal regulators regard permission as a necessary condition for authorizing the involvement of a child as a research subject; for children who cannot assent, it is usually a sufficient condition as well. The transactions involved in negotiating a valid permission are in all respects identical to those of informed consent.

Assent by the child should be as close an approximation of consent as the child's capabilities permit.

Before proceeding with our discussion of permission and assent, it is necessary to return to a consideration of the basic ethical principles.

Respect for persons requires that we treat individuals as autonomous agents only to the extent that they are autonomous. As noted earlier, "[p]ersons with diminished autonomy and thus in need of protection are entitled to such protections" ( [National Commission, 1978](#)). In response to this ethical conviction, there is established in federal regulations a standard called "minimal risk," which "means that the probability and magnitude of harm or discomfort anticipated in the research are not greater in and of themselves than those ordinarily encountered in daily life or during the performance of routine physical or psychological examinations or tests" (45 CFR 46).<sup>5</sup> Minimal risk serves as a threshold standard in that plans to involve children in research that presents more than minimal risk require special justification and procedural protections.

But many therapeutic procedures present far more than minimal risk, and it is not customary to obstruct children's access to them by calling for special procedural protections. The regulations make it clear that the minimal risk standard is applicable only to procedures that do not "hold out the prospect of direct benefit for the individual subjects" (45 CFR 46.405). Therapeutic procedures, by contrast, are to be authorized and justified precisely as they are in the practice of medicine. That is to say, the risk of any procedure is justified in terms of the benefit expected for the individual child-subject who will bear that risk. Also, as in medical practice, the relationship of anticipated benefit to the risk presented by the procedure must be at least as advantageous to the subject as that presented by any available alternative, unless, of course, the subject (or his or her parents) has considered and refused to accept a superior alternative. These rules are responsive to the ethical principle of beneficence, which as articulated by the [National Commission \(1978\)](#) is expressed in the form of two general rules: " (1) Do no harm; and (2) maximize possible benefits and minimize possible harms."

Justice, as envisioned by the National Commission, requires a fair sharing of the burdens and benefits of society ( [Levine, 1988](#); [National Commission, 1978](#)). In the distribution of these burdens and benefits, special consideration is to be given to those who are vulnerable or disadvantaged. Children are considered vulnerable and are to be protected from exploitation because they lack the capacity to consent. They are not to be involved in research that is irrelevant to the class of person of which they are representative. When appropriate, research should be done first on adults and then on older children before involving younger children and infants ([Levine, 1988](#)).

In summary, children and their parents are not completely free to do their own thing. There are constraints grounded in ethical considerations and enforced by regulations on whom researchers may invite to serve as subjects and how much risk they may be asked to assume for research purposes. With these constraints in mind, we now turn to further consideration of assent and permission.

## Assent

Respect for children does not require that we leave them alone even to the point of allowing them to choose dangerous activities unless they agree that we may do otherwise. Young children have no such liberty rights. What they have instead is a right to custody ( [Freedman, 1975](#)). We show respect for them by fostering their



well being, protecting them from harm, and guiding them to become “the right kind of people.”

As we have already noticed, federal regulations reflect the obligation to protect children from harm and to secure their well being. Let us now consider the obligation to guide their moral and social development—an obligation not recognized explicitly in the regulations.

In the 1970s there was a spirited debate over the legitimacy of using persons who are incapable of consent (“unconsenting subjects”) as research subjects. [Paul Ramsey \(1976\)](#) argued that it is always morally wrong. [Richard McCormick \(1974\)](#), arguing the opposing viewpoint, pointed out that members of a moral community have certain obligations. One of these is to contribute to the general welfare when to do so requires little or no sacrifice. In the case of children, one may presume that they would consent if they could; he calls this a “correctly construed consent.” In his view, when supplemented with parental permission, correctly construed consent authorized the use of children as subjects in research that fulfilled an important social need and involved “no discernible risk.”

At this point, [Terrence Ackerman \(1979\)](#) entered the debate, arguing that we tend to fool ourselves with procedures designed to show respect for the child's very limited autonomy. He claims that the child tends to follow “the course of action that is recommended overtly or covertly by the adults who are responsible for the child's well-being.” He further contends that, in general, this is as it ought to be. “Once we recognize our duty to guide the child and his inclination to be guided the task becomes that of guiding him in ways which will involve his well-being and contribute to his becoming the right kind of person.”

[Willard Gaylin \(1982\)](#) tells the story of a man who acted in accord with Ackerman's position. After directing his 10-year-old son to cooperate with a venipuncture for research purposes, he explained that his direction arose from his perceived moral obligation to teach his child that there are certain things one does to serve the interests of others even if it does cause a bit of pain:

This is my child. I was less concerned with the research involved than with the kind of boy I was raising. I'll be damned if I was going to allow my child, because of some idiotic concept of children's rights, to assume that he was entitled to be a selfish, narcissistic little bastard.

Parenthetically, while it is appropriate to guide and persuade a 10-year-old boy to submit to a venipuncture for research purposes, it is not ethically defensible to command him to do so against his will; it is also contrary to the requirements of federal regulations. Guiding children to become the “right kind of persons” entails teaching them about and encouraging them to embrace the sense of obligation to the moral community discussed in the preceding. It further entails showing respect for their maturing capacities for self-determination; one hopes the child will learn to choose to do unto others as the child would wish them to do unto her or him.

At what age does a child become capable of assent? Federal regulations specify no age, leaving it to the discretion of the Institutional Review Board (IRB), taking into account not only the age but also the maturity and psychological state of the children involved.

As the assent regulation is written, it seems to reflect a presumption that the capability to assent is an all-or-none phenomenon; the child is either capable or incapable of assent. This presumption is incorrect ([Weithorn, 1983a, b](#)) and, the author believes, unintended by the regulation writers. In the author's view the regulations are intended to be interpreted to permit a determination that prospective child-subjects may be capable of understanding some but not all of the elements of informed consent. Thus, for example, it may be appropriate to provide some children with “a description of any reasonably foreseeable risks or discomforts,” without providing “an explanation as to whether any compensation [is] available if injury occurs.”

It is possible to make some general comments on the capabilities to assent of children having normal cognitive development in various age groups. According to [Lois Weithorn \(1983a\)](#), who relates her empirical findings to Piaget's concepts of cognitive development:

in general, developmental research suggests that most school-aged children are capable of meaningful assent for participation in most types of research studies. This means that the children probably are capable of comprehending the nature of the proposed procedures, the general purpose of the research, and of expressing a preference regarding participation. Research suggests that normal children ages 6 and older are quite capable of thoughtful and reasoned consideration of the types of information that investigators may provide.

Typically, at about age 11, children's cognitive development enters the “stage of formal operations,” during which they become increasingly sophisticated in their capacities to reason about “possibility” and other abstract concepts. From ages 7 to 11, in the “stage of concrete operations,” the child is more or less limited to thinking about matters that are not too far removed from concrete reality. Thus, there are those who argue that the “age of assent” should be around 6 or 7, and others who say it should be around 11 or 12.<sup>6</sup>

[Weithorn \(1983a\)](#) continues:

Early empirical findings also suggest that, although they may not be legally authorized to provide independent consent for treatment or research in most jurisdictions, normal adolescents age 14 and older may be as capable as adults of making competent decisions about such participation, according to the more stringent legal standards of competency.

The authority of mature and emancipated minors to consent is discussed subsequently in this chapter. (See [Chapter 113](#) for definitions of “mature minors” and “emancipated minors.”)

## Permission

Parental permission is envisioned by the National Commission as a reflection of the collective judgment of the family that an infant or child may participate in research ([Levine, 1988](#); [National Commission, 1977](#)). In most cases the permission of one parent is sufficient; one may assume that he or she will represent the family's wishes satisfactorily.

When more than minimal risk is presented by a nontherapeutic procedure, the permission of both parents is required unless one is “deceased, unknown, incompetent, or not reasonably available, or when only one person has legal responsibility for the care or custody of the child” (45 CFR 46.408).

There are three additional criteria for justification of nontherapeutic procedures that present more than minimal risk (45 CFR 46.406): First, the degree of risk is limited to “a minor increase over minimal risk.” Second, the procedure or intervention must be “likely to yield generalizable knowledge which is of vital importance for the understanding or amelioration of the subject's disorder or condition.”<sup>7</sup> Third, the procedure or intervention must present “experiences to subjects that are reasonably commensurate with those inherent in their actual or expected medical, dental, psychological, social or educational situations.” This means that the procedures must be ones that they or others with the specific disorder or condition under study will ordinarily experience by virtue of their having or being treated for that disorder or condition. Thus, it might be appropriate to invite a child with leukemia who has had several bone marrow examinations to consider having another for research purposes.

The requirement of commensurability reflects the National Commission's judgment that children who have had a procedure performed on them might be more capable than are those who are not so experienced of basing their assent on some familiarity with the procedure and its attendant discomforts; thus their decision to participate will be more knowledgeable.

Even though the parent gives permission, the child's refusal to assent to nontherapeutic interventions should be respected. Those who are incapable of assent may have some capacity to make their wishes known. The term *deliberate objection* is used to recognize that some children who are incapable of meaningful assent are able to communicate their disapproval or refusal of a proposed procedure. A 4-year-old may protest, “No, I don't want to be stuck with a needle.” However, an infant who might in certain circumstances cry or withdraw in response to almost any stimulus is not regarded as capable of deliberate objection. A child's deliberate objection usually should be regarded as a veto to his or her involvement in research ([Levine, 1988](#); [National Commission, 1977](#)).

In the case of therapeutic interventions or procedures, the situation is much different. Federal regulations state simply, the assent of children is not a necessary condition (45 CFR 46.408). Parents have both the right and responsibility to override the objection of school-age children to necessary therapy. With regard to teenagers, decisions regarding authorization of investigational therapies are about as complicated as they are in the practice of medicine. The law recognizes the authority of emancipated and some mature minors to consent to or refuse standard or accepted therapy; these rules are not recognized explicitly in federal regulations

regarding investigational therapies.

In the practical world of decision making about who can authorize a therapeutic procedure, whether it is investigational or accepted, it rarely suffices to point to the law and thereby identify the person who has the legal right to make the decision. Many factors must be taken into account in reaching judgments about the capability of various persons to participate in and, in the event of irreconcilable disputes, prevail in such choices. In general, these judgments become more complicated as the child gets older or the stakes get higher ( [Gaylin, 1982](#); [Thomasma and Mauer, 1982](#)).

IRBs have the authority to waive the requirement for permission when "it is not a reasonable requirement . . . provided an appropriate mechanism for protecting the children is substituted" (45 CFR 46.408). The regulations suggest as an example of research in which a requirement for parental permission might not be reasonable is that on "neglected or abused children." The [National Commission \(1977\)](#) specified several other examples:

Research designed to identify factors related to the incidence or treatment of certain conditions in adolescents for which, in certain jurisdictions, they may legally receive treatment without parental consent; research in which the subjects are "mature minors" and the procedures involved entail essentially no more than minimal risk that such individuals might reasonably assume on their own; research designed to meet the needs of children designated by their parents as "in need of supervision," and research involving children whose parents are legally or functionally incompetent.

The [National Commission \(1977\)](#) further elaborates:

There is no single mechanism that can be substituted for parental permission in every instance. In some cases the consent of mature minors should be sufficient. In other cases court approval may be required. The mechanism involved will vary with the research and the age, status and condition of the prospective subject. . . .

Assent of . . . mature minors should be considered sufficient with respect to research about conditions for which they have the legal authority to consent on their own to treatment. An appropriate mechanism for protecting such subjects might be to require that a clinic nurse or physician, unrelated to the research, explain the nature and the purpose of the research . . . emphasizing that participation is unrelated to provision of care.

Another alternative might be to appoint a social worker, pediatric nurse, or physician to act as surrogate parent when the research is designed, for example, to study neglected or battered children. Such surrogate parents would be expected to participate not only in the process of soliciting the children's cooperation but also in the conduct of the research, in order to provide reassurance for the subject and to intervene or support their desires to withdraw if participation becomes too stressful.

The recommendations of the National Commission reflected their assumption that the "normal" family is one in which the members stand in a loving relationship to one another and that the parents in such a family will strive to protect the interests and welfare of their children. This point notwithstanding, they were aware of the fact that there are exceptions. In obvious cases (e.g., neglected or abused children) they recommended that the IRB should have the authority to waive the requirement for parental permission. In the less obvious cases, of which there may be many, decisions as to whether the parents are loyal to their children and welfare require sophisticated professional judgment. The National Commission wisely refrained from recommending that IRBs engage in micromanagement of such cases. Implicitly, the responsibility for such judgments is assigned to the investigator. The National Commission provided a margin of safety by recommending special substantive and procedural protections in cases in which more than minimal risk is presented by interventions that do not hold out the prospect of direct benefit for the individual child-subject.

In recent years there has been increasing interest in giving mature minors independent authority to authorize their own participation in certain types of research without requiring permission of their parents or guardians. To some extent this interest reflects a general trend in public policy toward facilitating inclusion as research subjects members of groups who were previously excluded ([Levine, 1994](#)). This interest also represents a pragmatic response to problems presented by the AIDS pandemic; specifically, many, and perhaps most, adolescents will not enroll as research subjects if this necessarily entails allowing their parents or guardians to learn or even suspect certain details of their sexual or drug-taking experience ( [Rogers et al., 1994](#)).

Most of the current federal regulations for the protection of human subjects reflect the attitude that prevailed in the 1960s and 1970s when they were written, that investigational drugs and participation in research were dangerous and researchers were likely to exploit subjects. Since the mid-1980s, primarily as a consequence of the efforts of AIDS activists, this vision has been largely replaced by one that research participation and access to investigational drugs are both more beneficial than burdensome. As a result of this shift in perception, public policies that were designed to protect persons from harm and exploitation, particularly those persons who are considered vulnerable by reason of limitation in their capacity to give informed consent, are being reinterpreted or rewritten to assure the same classes of persons equitable access to the benefits of investigational drugs as well as to the benefits of participation in research ( [Levine, 1994](#)).

It has long been known that restrictive policies on involvement of children in research have resulted in a class injustice. Children as a class have been deprived of the benefits of the new knowledge that could result from carrying out research involving children as subjects. This deprivation is exemplified by the fact that approximately 80% of the drugs approved by the FDA for commercial distribution are not labeled for use in children, usually because sufficient studies have not been conducted in children ([American Academy of Pediatrics, 1995](#)). In recent years agencies of the federal government have begun to respond to this class injustice. The National Institutes of Health now require that applications for grants or contracts to conduct research involving human subjects must include plans to enroll children unless the applicant can justify their exclusion ( [National Institutes of Health, 1998](#) ). Similarly, the Food and Drug Administration requires inclusion of children in research done to support an application for a marketing permit unless the sponsor can justify their exclusion (Public Law 105-115; Center for Drug Evaluation and Research).

At the time of this writing, federal policies have not been revised to permit involvement of mature minors as research subjects on their own authority except as discussed earlier in this chapter. However, it seems reasonable to predict that in the near future there could be such changes in the relevant policies or their interpretation. [Rogers and associates \(1994\)](#) and [Levine \(1995\)](#) have published examples of specific proposals.

Inclusion of children who are wards (e.g., of the state) in research in which more than minimal risk is presented by nontherapeutic interventions requires special procedural protections that are beyond the scope of this discussion (45 CFR 46.409) ( [Levine, 1988](#) ). Many states and cities have regulations designed to protect the interests of foster children. Concern has been expressed recently that such regulations often create formidable bureaucratic barriers to involving such children in randomized clinical trials or gaining them access to investigational drugs. For two reasons this presents a special problem to children with AIDS: (a) A large percentage of such children are foster children; and (b) Many apparently effective therapies for AIDS and its complications are investigational drugs ( [Secretary's Work Group on Pediatric HIV Infection and Disease, 1988](#) ).

## DOCUMENTATION

The regulatory requirement for documentation of permission is exactly the same as it is for informed consent. In circumstances in which there is no requirement for permission because the minor is authorized to assent for himself or herself the same requirements for documentation obtain. Apart from this, when children are asked to sign forms, as they often and quite properly are, the principal purpose, in the author's view, is to enhance their sense of participation in the process.

### Privacy and Confidentiality

Privacy is, "the freedom of the individual to pick and choose for himself the time and circumstances under which, and most importantly, the extent to which, his attitudes, beliefs, behavior and opinions are to be shared with or withheld from others" ( [Kelman, 1977](#) ). Because this is the definition used in this chapter, some matters considered by the law to fall under the rubric of privacy are excluded (e.g., the right to abortion and contraception). In general, in clinical research, intrusions into individuals' privacy are permitted only with their informed consent. There is no invasion when an informed person allows a researcher into his or her private space.

*Confidentiality* is a term that is all too often used interchangeably with *privacy*. Confidentiality refers to a mode of management of private information; if a subject shares private information with (confides in) a researcher, the researcher is expected to refrain from sharing this information with others without either the subject's authorization or some other justification.

The ethical grounding for the requirement to respect the privacy of persons may be found in the principle of respect for persons. The ethical justification for confidentiality, according to [Sissela Bok \(1982\)](#), is grounded in four premises, three of which support confidentiality in general; the fourth supports professional



confidentiality in particular.

First and foremost, we must respect the individual's autonomy regarding personal information. To the extent they wish, and to the extent they are capable of doing so, they are entitled to have secrets. This facilitates their ability to live according to their own life plans.

Closely related is the second premise, which recognizes the legitimacy not only of having personal secrets but also of sharing them with whomever one chooses. This premise, which embodies an obligation to show respect for relationships among human beings and respect for intimacy, is exemplified by the marital privilege upheld in American law, according to which one spouse cannot be forced to testify against the other.

The third premise draws on the general requirement to keep promises. A pledge of confidentiality creates an obligation beyond the respect owing to persons and existing relationships. Once we are bound by a promise, we may no longer be fully impartial in our dealings with the promisee.

These three premises, taken together, provide strong prima facie reasons to support confidentiality. That is to say, they are binding on those who have accepted information in confidence unless there are sufficiently powerful reasons to do otherwise—as, for example, when maintaining confidentiality would cause serious harm to innocent third parties.

Bok's fourth premise adds strength to the pledge of silence given by professionals. The professional's duty to maintain confidentiality goes beyond ordinary loyalty, "because of its utility to persons and to society. . . . Individuals benefit from such confidentiality because it allows them to seek help they might otherwise fear to ask for" from doctors or others who can provide it.

Investigators, of course, are not necessarily professionals to whom individuals turn for professional help. Thus, only part of the fourth premise applies to investigators, the part that grounds the justification and requirement for confidentiality in its social utility. If researchers violated the confidence of their subjects, subjects would refuse to cooperate with them. This, in turn, would make it difficult, if not impossible, for researchers to contribute to the development of generalizable knowledge.

Over the millennia, the professions have viewed the obligation to maintain confidentiality as very important. The Hippocratic Oath requires, "What I may see or hear in the course of the treatment or even outside the treatment in regard to the life of men, which on no account one must spread abroad, I will keep to myself holding such things shameful to be spoken about."

[Thomas Percival's Code of Medical Ethics \(1803\)](#), on which was based the first code of ethics of the American Medical Association, incorporated the following exhortation: "Patients should be interrogated concerning their complaints in a tone of voice which cannot be overheard."

In all states, the law not only recognizes the obligation of physicians and many other professionals to maintain confidentiality, it requires it. In many states, there are statutes granting testimonial privilege to information secured by physicians from patients in the course of medical practice ( [Brennan, 1983](#)). Testimonial privilege means that physicians cannot be compelled to disclose such information even under subpoena. Although the United States Supreme Court refused to extend the constitutional protections of privacy to physician–patient communications, the *Federal Rules of Evidence*, promulgated by the Judicial Conference, defer to state law on physician–patient privilege ( [Brennan, 1983](#)).

Most state laws on physician–patient privilege contain various exceptions, including mandatory reporting of information regarding battered and abused children, various communicable diseases, gunshot wounds, and certain proceedings concerned with health issues, including workers' compensation and insurance claims ( [Brennan, 1983](#)).

In 1974, in the case of *Tarasoff v. Board of Regents*, the supreme court of California ruled that a psychiatrist has a duty to protect the intended victim of a patient's threat of violence, if it is likely that such a threat would be carried out ( [Holder, 1985](#)); this often but not always entails a duty to warn the intended victim. Subsequently, this duty has been recognized in many other states. The same principle is often invoked in debates over whether state laws should be changed to either permit or require doctors to warn lovers of persons infected with the HIV virus if they are unwilling to do so themselves ( [Gostin, 1989](#)).

It must be recognized that the right to confidentiality or privilege belongs to the patient. The patient may authorize sharing of private information for whatever purposes he or she chooses. In civil or criminal litigation where the patient makes the information in his or her medical record material to the support of his or her position, as so often happens in child custody cases or malpractice litigation, the court usually requires that the contents of the record be disclosed ( [Holder, 1985](#)).

## Confidentiality in Research

Most direct social injuries to research subjects result from breaches of confidentiality. An investigator may identify a subject as a drug or alcohol abuser, as a participant in various deviant sexual practices, as having any of a variety of diseases that may be deemed unacceptable by his or her family or social or political group, as having a higher or lower income than acquaintances might have predicted, as a felon, and so on. If certain individuals know such information, it might cost the subject his or her reputation, job, social standing, credit, or citizenship.

In recognition of these threats of social injury, federal regulations require the IRB to determine that "Where appropriate, there are adequate provisions to protect the privacy of subjects and to maintain the confidentiality of data" (45 CFR 46.111a).

How does one determine that provisions to maintain confidentiality are "adequate"? The first step is to become aware of the variety of factors that may pose threats to confidentiality in the research context. The second is to become aware of the various devices that are available to secure the confidentiality of research data. <sup>8</sup>

The [National Commission \(1978\)](#) offered some suggestions for safeguards of confidentiality: Depending on the degree of sensitivity of the data, appropriate methods may include coding or removal of individual identifiers as soon as possible, limitation of access to data, or the use of locked file cabinets. Researchers occasionally collect data that, if disclosed, would put subjects in legal jeopardy. Because research records are subject to subpoena, the National Commission suggests that when the identity of subjects who have committed crimes is to be recorded, the study should be conducted under assurances of confidentiality that are available from DHHS as well as the Department of Justice. For detailed information on these assurances of confidentiality, which provide immunity from subpoena, see [Office for Protection from Research Risks \(1993\)](#).

## CONSENT

As mentioned in the preceding, informed consent regulations require "a statement describing the extent, if any, to which confidentiality of records identifying the subject will be maintained." Statements about confidentiality of research records should not promise more than the researchers can guarantee. For most studies in which the private information to be collected is not especially sensitive, it suffices to state that the researchers intend to maintain confidentiality, that they will take precautions against violations, and that all reports of the research will be in the form of aggregated data and devoid of identifiers. When dealing with more sensitive information, it may be useful to specify some of the precautions; for example, videotapes will be destroyed within 60 days or, if the subject so requests, earlier; data will be kept in locked files; individuals will be identified by a code and only a small number of researchers will have access to the key that links code numbers to identifiers.

Plans to incorporate data in the subjects' medical record should be made explicit. In general, when these data are relevant to patient management, they should be incorporated unless the subject objects. Incorporation of the results of nonvalidated diagnostic tests may lead to false diagnostic inferences, with adverse consequences to the patient's medical care or insurability. Most people do not understand the full implications of signing forms that release their medical records to insurance companies ( [Siegler, 1982](#)).

It is essential to disclose all serious threats of confidentiality breaches that can be anticipated. One such disclosure was used in a study of devices designed to encourage young children who are thought to have been abused sexually to speak freely of their experiences. A portion of the permission form for the normal control subjects reads as follows ( [Levine, 1988](#)):

Because this study is not intended to be related to either diagnosis or therapy for you or your child, you are entitled to decide whether the information

obtained during this study should be entered into the medical record. If, however, during your child's interview, his or her behavior raises a concern of sexual abuse, a clinical evaluation of your child will be performed. . . . If after that evaluation the suspicion of sexual abuse persists, the case will be reported as mandated by law.

FDA regulations require (21 CFR 50.25a) "a statement describing the extent, if any, to which confidentiality of records identifying the subject will be maintained and notes the possibility that the [FDA] may inspect the records."

As noted earlier, federal regulations permit waiver of the informed consent requirement under certain conditions. These conditions are usually but not invariably met in studies of medical records (given adequate safeguards of confidentiality) or of "leftover" specimens obtained at autopsy, surgery, or collection for diagnostic purposes of body fluids such as blood or urine. In general, use of tissues or fluids without consent is justified if two conditions are satisfied ( [Levine, 1988](#)): (a) No more tissue or fluid is removed than the amount needed to accomplish the medically indicated purpose of removal; and (b) the specimens are obtained by the researcher under conditions of anonymity (i.e., the diagnostic laboratory removes all personal identifiers before giving the specimens to the researcher).<sup>9</sup>

The practices just described are invasions of privacy. In such cases, patients are said to have a right of notice, that is, a right to be notified that such practices occur in the institution. These notices, which partially mitigate the invasion, are commonly printed in patient information brochures and on permission forms for surgery or autopsy. Although they are designed to afford patients opportunities to object, objections are rare. For examples of such notices, see [Levine \(1988, pp. 179–180\)](#).

In some cases, with suitable justification, even the right of notice may be waived. Examples of such activities include collections of cord blood from neonates under conditions of anonymity in a nationwide study to determine the prevalence of HIV antibodies ( [Levine, 1988](#)); monitoring for compliance in some randomized clinical trials ( [Levine, 1988](#)); and covert observation or recording of public behavior ( [Levine, 1988](#)). Further discussion of these activities and their justification is beyond the scope of this chapter. It must be noted that covert observation is a mild form of deception. In some cases even stronger forms of deception may be justified ( [American Psychological Association, 1982](#); [Levine, 1988](#)).

Researchers commonly use medical or laboratory records to identify patients who might be suitable subjects for their studies. Having identified them, they then contact them by telephone or mail with invitations to participate in research. Those who are contacted usually do not recall having read notices describing such activities in hospital brochures. Some will wonder why a stranger who knows his or her diagnosis is calling. Careful plans must be made to avoid offending patients in such activities ( [Levine, 1988](#)).

## CHILDREN AND PRIVACY

The concerns of young children about privacy are different from those of older children and adolescents ( [Thompson, 1990](#)). Young children first develop territorial privacy ("This is my room") and possessional privacy ("This is my tricycle") and only later begin to develop informational privacy (concerns about others' knowledge of one's activities, associations, and interests). Thus young children may not know that what they say to others may be detrimental to the privacy interests of their families.

DHHS regulations are responsive to this concern. There are several classes of research that are generally considered so free of complicated ethical problems that they are exempted from coverage by the regulations (45 CFR 46. 101b). Two of these exemptions do not apply to research involving children: (a) research involving survey or interview procedures; (b) research involving observation of public behavior in which the researcher is a participant. These activities are not exempt in cases in which they "could reasonably place the subject at risk of criminal or civil liability or be damaging to the subject's financial standing or employability."

Researchers often must secure the approval of various custodians of children to involve them in research. Such custodians may be teachers, day care workers, camp counselors, and the like. At times it is important to withhold or disguise some of the purposes of the research in order to protect the family's privacy or to avoid prejudicial treatment of children. For example, one would generally avoid telling teachers that the children are selected because they are offspring of parents with emotional disturbances.

As mentioned, informed consent requires "an explanation of the purpose of research." Does this mean that parents must always receive a full disclosure of purposes? Suppose the purpose is—as it was in one study—to determine whether little boys with XYY chromosome patterns are more likely than those with XY patterns to develop violent behavior. Disclosure of such a purpose could become a self-fulfilling prophecy. For further discussion of withholding the purpose of research and its justification, see [Levine \(1988, p. 117\)](#).

As mentioned earlier, the requirement for parental permission may be waived in "research designed to identify factors related to the incidence or treatment of certain conditions in adolescents for which . . . they may legally receive treatment without parental consent." In general, the types of activities contemplated by this rule are treatment of sexually transmitted diseases, provision of contraceptive advice, and other matters that teenagers consider highly sensitive. We recognize the teenager's right to privacy, but how far should we go to protect it? Some of the factors that require consideration are illustrated in the following exchange.

[Herceg-Baron \(1981\)](#) published a case study in which she detailed some of the special problems involved in research in the field of family planning involving minors as subjects. Many adolescents wish to seek advice about such matters as contraception without the awareness of such others as their parents. She details her institution's policy for protecting the minors' confidentiality. For instance, with regard to follow-up, investigators are required to offer various options, for example, telephone calls during certain hours when the minor knows she will be at home alone; contacts through school personnel such as nurses, teachers, or counselors; contacts by mail containing no agency letterhead or other identifying information; or leaving messages with friends.

In commenting on this case study, [Carol Levine \(1981\)](#) raises several concerns. First, she suggests that many adolescents are ambivalent about clandestine sex and would, with some encouragement, welcome open discussion with their parents. The great concern with privacy seems to undermine the possibility for what could be valuable communication within the family. Levine is further concerned about the fact that the institution not only approves deception, it collaborates with the adolescent in deceiving her parents. This, she argues, sets a very poor example for the adolescent.

This brings us back to a point discussed in the preceding. Parents are not the only adults having responsibility for guiding the child to become the "right kind of person." Professionals must be aware of the fact that children see them as models of proper behavior. Consciously or otherwise, they provide examples that children will emulate. Accordingly, they should be especially careful not to suggest by example that promises (e.g., of confidentiality), truthfulness, and other ethical matters discussed in this chapter are to be taken lightly. In short, they should help children understand the importance of showing respect for persons.

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<sup>1</sup>For an authoritative commentary on these two exceptions, the reader is referred to Appendix 1 of the [President's Commission's report, Making Health Care Decisions, 1982](#), pp. 199–201. A more concise discussion may be found in [Levine \(1988, pp. 149–152\)](#).

<sup>2</sup>There are further conditions specified by FDA in its regulations. For further discussion of 21 CFR 50.23, see [Levine \(1988, pp. 150–152\)](#). For 21 CFR 50.24 see [Brody \(2000\)](#).

<sup>3</sup>The Department of Health and Human Services regulations also permit use of a "short form." Use of this form of documentation seems even more complicated and cumbersome than use of the standard consent form. Moreover, its use requires a witness to the consent discussion ( [Levine, 1988, pp. 134–135](#)).

<sup>4</sup>Such language is also forbidden in the consent discussion (45 CFR 46.116).

<sup>5</sup>The term minimal risk presents many problems. As it is defined, it may be interpreted in several different ways. For a discussion of its deficiencies, see [Levine \(1988, p. 65\)](#) and [Kopelman \(1981, 1985\)](#). For an excellent discussion relating the concept of risk to the child's level of development, see [Thompson \(1990\)](#).

<sup>6</sup>According to the [National Commission \(1977\)](#), a child with normal cognitive development becomes capable of meaningful assent at about the age of 7 years, although some may be younger and some older. The Department of Health and Human Services (DHHS) did not accept this recommendation. Rather, at the time the proposed regulations were published, DHHS solicited public comment on which of three options it should adopt for nontherapeutic procedures: either age 7, age 12, or leaving the age to the discretion of the IRB. The final regulations reflect the third of these options.

<sup>7</sup>What constitutes "minor increase" and "vital importance" is not defined in the regulations. Responsibility for deciding such matters in relation to particular research proposals is assigned to the IRB. If the IRB cannot decide or if it does decide that the degree of risk is more than a minor increase over minimal risk, it must refer the judgment to the Secretary of The Department of Health and Human Services ( [Levine, 1988, pp. 247–250](#)).

<sup>8</sup>A full discussion of these threats and devices is beyond the scope of this chapter. A treatment of these topics adequate for the purposes of most clinical researchers is found in [Levine \(1988\)](#). [Boruch and Cecil \(1979\)](#) provide an excellent general overview directed primarily at social scientists, with particular concentration on the development of statistical methods for making the responses of any particular subject uninterpretable even if the research records are subpoenaed.



<sup>9</sup>In some cases it may be impossible to remove identifiers, as when the patient's physician is also the researcher. In such cases the requirement for consent may be waived if the research will not yield any information having diagnostic significance. For further discussion of this point, see [Levine \(1988\)](#), pp. 178–181).

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# 132 ETHICS IN THE PRACTICE OF CHILD AND ADOLESCENT PSYCHIATRY

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## OVERVIEW

Ethical codes in medicine date back to the fifth century BC, yet they received little attention in our medical literature until the 1990s. Greater awareness and interest in ethics can be attributed to (a) heightened consumerism with increased emphasis on patient rights, (b) high-technology medical developments that offer choices unheard of in the past and in turn introduce the need for health care rationing and decisions about prolonging life, and (c) changes in the delivery of health care that alter physician's autonomy, impose the role of gatekeeper, and challenge our traditional ethical codes.

Ethical codes are not laws but standards of conduct expected from a professional. They exist to help professionals to reconcile providing service while also earning a living from that service. In medicine, these codes define the norms, duties, and virtues expected in our professional work. As noted by [Reiser et al. \(1977\)](#), "Self-conscious reflection on standards of conduct is one of the defining characteristics of a profession." Ethical codes serve to protect the profession and benefit the patient and society as well. In maintaining the image and standard of conduct of the profession, they enable the patient to establish trust in the physician.

The Hippocratic Oath originated in the fifth century BC, but it was not widely applied until the 10th century. It stresses the physician's power to heal and the need to divest this power from killing. In doing so, as noted by Margaret Mead, the code clearly separates the physician from the sorcerer or shaman, who has the power both to harm and to cure ([Mead, 1972](#)). The Hippocratic Oath stresses the physician's obligation to the patient and the duty to keep confidences. It prohibits abortion, euthanasia, and sexual relationships with patients. [Dyer \(1988\)](#) notes that the hippocratic tradition has come under scrutiny by critics who contend that it is anachronistic. Critics argue that it does not deal with the technologic advances in medicine or with problems of cost containment. Many contend that it is too paternalistic and does not adequately address the rights of patients. [Dyer \(1988\)](#) recommends that we accept the oath "symbolically in terms of the intent and the concept of the profession it outlines."

Psychiatrists today follow the *Principles of Medical Ethics with Annotations Especially Applicable to Psychiatry* (1993). These guidelines provide us with a way of thinking about ethical dilemmas, but they do not necessarily solve them. Often competing ethical principles come in conflict, such as a woman's right to autonomy and to refuse a cesarean section versus her physician's concern for the welfare of her fetus. To understand these ethical guidelines, it is helpful to appreciate the ethical principles that underlie them. The four basic moral principles that guide us in medical research and health care are analyzed in great detail by [Beauchamp and Childress \(1989\)](#) and are briefly summarized here. *Autonomy* comes from the Greek words for self and rule and in medicine refers to the ability to make decisions for oneself without being controlled by others. Autonomy becomes the basis for informed consent and therapeutic privilege. *Nonmaleficence* is a concept derived from the Latin "primum non nocere," or first do no harm, and it stems from the Hippocratic Oath, which states "I will use treatment to help the sick according to my ability and judgment, but I will never use it to injure or wrong them." The principle of *beneficence* refers to the obligation to help others to further their legitimate interests and, more specifically, to promote the welfare of the patient. The principle of *justice* refers to offering fair treatment to all.

## NATURE OF THE DOCTOR–PATIENT RELATIONSHIP

A *fiduciary relationship* is one in which one person receives the trust or confidence of another and is under a duty to act for the benefit of that person. Examples include an attorney–client or broker–client relationship. Trust is the cornerstone of the fiduciary relationship that exists between physician and patient. The physician as fiduciary is expected to act for the benefit of the patient and not exploit that relationship for personal gain. [Simon \(1987\)](#) reminds us that the psychiatrist's main source of gratification should arise from the psychotherapeutic process, and his or her only material reward is payment for service.

*Trust* is essential to both evaluation and treatment. Without trust, patients would be reluctant to divulge the intimate details about their lives that are often necessary to arrive at diagnoses and embark on treatment. The fiduciary relationship is less clear-cut in regard to children. Issues of trust and confidentiality are more complex in child and adolescent psychiatry because we must deal with parents as well as the child. Parents' rights to or need to know certain information about their child need to be balanced with the child's interests. Further, the age and cognitive maturity of the child have a bearing on the child's ability to participate in decisions about treatment or medication, as well as disclosures to others.

Doctor–patient relationships are also defined by *boundaries* that keep us in our professional role and prevent us from exploiting patients. Boundaries provide a sense of security to both physician and patient. They help us to maintain objectivity and allow us to focus on the patient's best interest. Boundaries discourage acting out by both patient and physician and foster respect for the patient's autonomy and dignity. The forces of both transference and countertransference threaten the therapist's neutrality and, if not recognized and resolved, may erode boundaries and undermine therapy. Straying from one's usual practices may be a warning sign of boundary violations. Psychiatrists who begin to see patients at unusual locales or times, who waive usual billing procedures, or who start socializing with patients and their families may be on a slippery slope. They need to reflect on that behavior and to consider the consequences, because seemingly benign boundary violations often lead to more serious ethical violations.

## CONFIDENTIALITY

The terms *confidentiality* and *privilege* are often confused. Privilege, the narrower of the two terms, refers only to the patient's right to bar disclosure of information obtained in treatment in judicial or quasijudicial proceedings. Confidentiality, in contrast, refers to the disclosure of information learned in treatment to third parties. The physician's duty to maintain confidentiality is both a legal and an ethical one that derives from the right to privacy under common law and our ethical codes. [Appelbaum and Gutheil \(1991\)](#) note that the ethical foundations for confidentiality are twofold. First is the concern that without assurance of confidentiality patients would be reluctant to seek treatment. Second is the argument that, having implied that communications are confidential, mental health professionals must keep their implicit or explicit promise.

Privilege and confidentiality can be waived only by the patient, with certain exceptions. Generally, these exceptions include when the patient is in danger of harming himself or herself or others, such as a sexually active patient who is positive for human immunodeficiency virus who refuses to take precautions or to inform sexual partners and state laws mandating reporting of child abuse or impaired physicians. However, exceptions continue to grow and now include insurance company audits, which usually call for blanket rather than informed consent to release records, and insurance fraud investigations involving either the psychiatrist or the patient ([Schetky, 2000](#)).

The psychiatrist may be faced with a moral dilemma when a subpoena demands patient records and their release is not in the best interest of that patient. The



psychiatrist's conscience must guide him or her whether to defy the law or seek to quash the subpoena. For discussion of the legal ramifications of breach of confidentiality and consent issues, see [Chapter 112](#) and [Chapter 113](#).

## Consent

Minors, with some exceptions, are not competent to give *consent* to treatment, to medical research, or for release of medical information, but they may be asked to give their assent in accord with their developmental age. Consent must be obtained from parents or guardians unless state law allows adolescents to consent to treatment or unless they are emancipated. For consent to be informed, parties must know the nature of the condition treated, the risks and benefits of the proposed treatment and their choices, and the risks of no treatment, and they should be free, that is, without undue influence, to agree or disagree. It is advisable to obtain consent in writing for high-risk treatments and for release of records. If parents are separated or divorced, consent must be obtained from the custodial parent. If the child is in a shared custody arrangement, the psychiatrist should attempt to contact the other parent regarding treatment decisions, because joint decision making on medical matters is usually part of shared custody arrangements. If one is in doubt about the terms of parental rights, one can always ask a parent to bring in the divorce decree.

The child and adolescent psychiatrist, perhaps more so than the adult psychiatrist, faces many pressures to violate confidentiality owing to all the collateral contacts that arise during our work with children. These include parents, teachers, guidance counselors, other therapists involved with the family, day care providers, the child's physician, and sometimes personnel from protective services or other agencies. Even when there is consent for release of information, the psychiatrist must delicately balance how much information a school needs to know about a child's turbulent family life to help a child while respecting the family's wish for privacy. The child and adolescent psychiatrist may need to decide when it is necessary to override a child's plea not to disclose certain information such as speaking to a teacher when parents have authorized such communication. The following case illustrates the many levels at which we must weigh decisions about confidentiality.

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### CASE ILLUSTRATION

Sally Barnes, age 8 years, has been in treatment for an anxiety disorder that waxes and wanes. She lives with her recently widowed mother and younger sister. One day, she is brought to her weekly appointment by her grandmother and tells her therapist, Dr. Coles, that her mother is home sick with the flu. Sally then reveals that her mother was drinking excessively the night before at a friend's house and drove Sally and her sister home while under the influence of alcohol. Sally heard her mother vomiting during the night, and in the morning her mother was so sick that she asked Sally to stay home to care for her little sister. Dr. Coles is faced with the issues of (a) using Sally's disclosures to confront Mrs. Barnes, (b) whether to share concerns with Sally's grandmother, (c) whether to contact Mrs. Barnes' therapist, and (d) whether to involve protective services. She chooses to say nothing to the grandmother because she does not have the mother's permission to speak with her. Sally is eager for her therapist to talk to her mother. Dr. Coles calls Mrs. Barnes, who initially denies the allegations made by Sally but who then backs down and agrees to allow Dr. Coles to contact her therapist. Later, she admits that things are very out of control in her life, agrees to an inpatient admission, and approaches her mother to help care for the children. Dr. Coles does not feel the need to involve protective services at this juncture. The question of how much to tell the school and day care is discussed with Mrs. Barnes and is left to her discretion. Sally may have been afraid that Dr. Coles would speak to her mother about her drinking problem. In that case, she would have had to deal with Sally's fear around disclosing this secret and her rationale for overriding Sally's objections in taking the steps she did to ensure her welfare.

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## Limits of Confidentiality

Child psychiatrists need to define to both patient and parents the *limits of confidentiality* at the onset of evaluation and treatment. The extent to which communications from parent to therapist will be shared with the child should be discussed. If the psychiatrist needs to share the child's confidences with the parents, there are several options. The first is to urge the child to do so or to meet jointly and discuss the issues. If this fails, the psychiatrist may then tell the child why he or she needs to share the information with the parents and what will be told.

In small, underserved communities, the psychiatrist may, like it or not, have to medicate and sometimes treat more than one family member or friends of patients. This poses a challenge to the psychiatrist in terms of double bookkeeping, that is, remembering what information was heard from whom and storing away what may have been heard but cannot be used because it was shared in confidence. Additional problems may arise around advocacy and issues of countertransference. In general, dual agency such as this is best avoided. If no alternatives exist, the psychiatrist should at least make each patient from the same family aware that he or she is seeing the other.

## Double Agency

*Double agency* is a term that refers to serving two masters simultaneously. This is a potential problem that may arise from consultants when they are not clear about their roles. For instance, an adolescent may reveal to a consulting psychiatrist that he or she is dealing drugs in school. If the psychiatrist shares this information with the school, the student is likely to be expelled. If he or she conceals this information, other students are at risk. The consultant needs to be clear that his or her duty is to the school, which hired him or her. The dilemma could be minimized by informing the student at the onset of the evaluation about the limits of confidentiality.

Double agency may also arise when a therapist pursues the parents' agenda without regard to the child's best interests. For instance, Mr. and Mrs. Black seek help from Dr. White in regard to their 14-year-old son, Tom. They complain that he is defiant, questions his father, talks back, and refuses to attend services at their fundamentalist church. They hope that Dr. White will render Tom more compliant and will bring back their "good little boy." If Dr. White colludes with their agenda, he risks becoming their agent. Tom, conversely, does not see that he needs help other than using Dr. White's authority to get his parents off his back so he can gain more freedom. An overidentified therapist could be tempted to collude with Tom's agenda. Dr. White empathizes with Tom's plight but views his role as helping Tom to separate and individuate from his family and to develop responsible autonomy. Unless he spells out where he stands with the family, therapy is not likely to succeed.

## Media

Child and adolescent psychiatrists need to be on guard against violating confidences when giving press interviews. It is usually prudent to be circumspect and to limit comments to what is already public knowledge and to comment on issues rather than specifics of a case. Parents may give consent for a therapist to talk with the media about their children in high-profile custody and abuse cases. There is an unfortunate trend to try these cases in the media. Parents who are caught up in the heat of litigation are not always the best judges about whether media attention will be harmful to their children.

The psychiatrist needs to guard against exploiting high-profile cases for his or her own personal gain. Occasionally, psychiatrists may be tempted to go above the law and try to justify rash actions such as releasing confidential reports to the media as being in the child's best interests. Rarely can such actions be justified, and when closely examined they usually represent grandiosity, narcissism, and unchecked countertransference on the part of the psychiatrist.

## Professional Presentations and Publications

Confidentiality must be preserved when psychiatrists write about patients or present them or their artwork at conferences. One has the choice of disguising material sufficiently to preserve the patient's identity or seeking permission from the child and his or her parents to use the material. Therapists may be tempted to write books about their patients. It is difficult to reconcile this with keeping the patient's interests foremost, and such an agenda is likely to derail therapy. Literary exploitation of therapy in the mass media is unsettling to the public and does not promote trust in our profession. Even if this occurs with the patient's consent, questions may be raised about how informed the consent was, as in the case of author, Anne Sexton, whose psychiatrist released therapy tapes to her biographer after Sexton's death.

## Dual Relationships

*Dual relationships* pose a challenge to maintaining confidentiality. Anyone practicing near where they live or whose children attend the same school as their patients do is bound to encounter awkward situations. The child psychiatrist's children are unaware of who is or is not a patient of their parent. They may wish to invite patients to birthday parties, play at their homes, or share car pools. As the child psychiatrist's children grow older, attend larger schools, and engage in more extracurricular activities, it becomes increasingly difficult to screen patients for their potential ties to one's children. In small towns, one may end up evaluating or treating children of one's colleagues or one's children's teachers or future teachers. If there are no other resources, turning down requests for help in times of crises may be viewed as inhumane and does not help one's image. How well these dual relationships work often depends on the nature of the patient's disorder, the extent of family psychopathology, and the therapist's ability to maintain boundaries. Each new adult patient or parent of a child patient should be viewed as one less potential friend. The two-way give and take of friendships cannot exist in therapy, because the therapist may not use the relationship for his or her own personal needs. It may be necessary to explain this to certain families at the onset of one's professional involvement with them.

The longer one is in practice, the more likely one is to run into patients all over town. The psychiatrist may need to patronize parents of patients, be they shop owners, pharmacists, plumbers, or restaurant owners. Adult patients and patients' parents may be appointed to boards one sits on or may join one's organizations. The child and adolescent psychiatrist either learns to deal with these encounters or retreats to the high ground and becomes a recluse. There is much to be said for patients' seeing their psychiatrists as a real person, whether at the dump on a Saturday morning or at the local high school basketball game. Successfully negotiating these encounters requires that the psychiatrist to be comfortable with his or her public persona, process these encounters, and maintain boundaries and confidentiality. In assessing how to address patients in public, one learns to take cues from patients. Children may be unabashed about seeing their therapist in the supermarket, whereas their parents may be less comfortable. Teenagers may shirk from public contact or may surprise you by wanting to introduce their friends.

### Psychiatrist's Family

One learns to train family members (and friends) not to ask "How do you know so and so?" Some psychiatrists develop nonverbal cues with spouses for handling awkward social situations. Should the psychiatrist's child learn the identity of a patient, he or she must appreciate that it is the patient's choice whether to disclose the psychiatrist-patient relationship. In some communities, children may be quite comfortable telling another child that he or she is a patient of their parent. Conversely, I have had to curb my son's one-time enthusiasm for trying to refer classmates whom he thought were in need of help. Adult patients may deliberately attempt to become friends with their therapist's spouse, and this becomes awkward if the professional relationship is not known to the spouse. The therapist may have to intervene with the patient in such situations.

### FORENSIC ISSUES

*Forensic evaluations* differ from regular diagnostic evaluations in that their intent is not therapeutic. Forensic evaluations are intended to help the court to find the truth and address the legal question at hand. To do so, the forensic psychiatrist must strive for impartiality and must avoid cases in which prior ties, be they social or professional, could tinge objectivity or neutrality. Whenever possible, forensic examinations should be separated from treatment ( [Schetky, 2001](#); [Strasburger et al., 1997](#)). Therapists inevitably become advocates for their patients, and, in doing so, they may be less than objective. Furthermore, they rely on narrative rather than on historical truth and usually do not seek out the type of corroborative material relied on by the forensic psychiatrist. Another reason to avoid such a dual relationship is that confidentiality is compromised once the therapist has to testify in court. Some attorneys may try to draw therapists into child custody battles or may try to persuade them to change their opinions. These issues are discussed extensively by Gutheil and Simon (1999). The forensic psychiatrist needs to be clear from the start with families who have retained him or her and must discuss the limits of confidentiality. It is not an unusual ploy for a parent to attempt to suppress an unfavorable custody report by stating that she went to the psychiatrist for therapy and that the therapist is therefore violating her confidences. When court appointed, the child psychiatrist may operate with quasijudicial immunity, which protects the psychiatrist from liability, whereas protection from liability is less certain in other situations.

The forensic psychiatrist should request payment up front in the form of a retainer. This approach ensures payment, makes clear that one is being paid for one's time rather than for one's opinion, and lessens the possibility of bias. It is customary to charge more for forensic evaluations, because they require more expertise and can be very disruptive to one's practice. It is always unethical to accept a case on a contingency fee because this creates too much vested interest in the outcome of the case.

Psychiatrists who practice forensic psychiatry need to accept the limits of their experience, not inflate credentials, and avoid exceeding data bases and making unsubstantiated statements. For further discussion of the pitfalls in these cases, see Schetky ( [1998](#), [2001](#)).

Forensic psychiatrists who perform assessments out of their own state should be aware that many states now require licensure for this activity. This is a result of the AMA's decision that forensic psychiatry constitutes the practice of medicine. Failure to address local licensure requirements may result in not being allowed to testify, as well as possible civil or criminal liability ( [Simon and Shuman, 1999](#)).

### DEALING WITH THIRD-PARTY PAYERS

The disadvantage of private practice is having to deal with third-party payers and paperwork. With the advent of managed care, telephone trees and voice mail, and the need for prior authorizations and written treatment plans, the task has become even more time consuming and distasteful. Ethical dilemmas arise when the patient's best interests and the psychiatrist's wish to be paid conflict with insurance companies' interest in minimizing cash outflow. This creates temptations on all sides to engage in unethical behaviors.

Insurance companies may deliberately lose or destroy claims, may reject claims for spurious reasons, or may endlessly "research" disputed claims as a delaying tactic. Noncustodial parents may pocket insurance payments. Patients or parents may request that diagnoses or codes be altered out of concerns about confidentiality or so they can receive better reimbursement. The psychiatrist may be tempted to exaggerate the patient's condition to have needed services approved or to obtain higher reimbursement rates. There may be the temptation to alter dates of service, as when Medicaid will not pay for a parent visit on the same day the child is seen, regardless of how far they have traveled. A similar problem arises when only one psychiatric visit is allowed per day, even if the patient needs admission after an outpatient visit. Physicians may be tempted to exaggerate duration of visits to compensate for low rates of reimbursement. Regardless of one's motives, these practices are fraudulent and, as such, are subject to criminal prosecution. The psychiatrist who engages in fraud may also be subject to ethical investigations and sanctions.

With the advent of health care rationing and a profit motive for managed-care companies, serious concerns arise around who is responsible for the patient once a managed-care company decides that it will no longer pay for hospitalization or authorize further outpatient visits ( [Appelbaum, 1997](#)). Little regard is given to the impact on patients of forcing them to change therapists because their managed-care company changes as a result of a job change or a takeover of a managed-care organization. What are the ethics of managed-care companies that direct children of their subscribers to providers who lack adequate training in child therapy? Managed care further disrupts a practitioner's referral patterns if he or she is restricted to obtaining consultations from a list of providers in a particular managed-care company. Additional concerns arise over confidentiality as it becomes necessary to share more and more information about patients to justify ongoing treatment. Of particular concern are the blanket consent forms authorizing release of medical information that subscribers are expected to sign when they become insured by private health care companies, Medicaid, or Medicare. This sort of consent is not informed because the subscriber does not know or cannot anticipate what may be in the records being released. Physicians who refuse to release requested information may be penalized by the withholding of reimbursement or even recoupment notices regarding prior payments.

Managed care pressures physicians to become gatekeepers and to consider not only the patient's needs but society's needs as well when it comes to allocation of health resources. [Levinsky \(1984\)](#) reminds us that this is an untenable position, and "physicians are required to do everything that they believe may benefit each patient without regard to costs or other societal considerations."

### REPORTING ETHICAL VIOLATIONS

The AMA *Principles of Medical Ethics*, section 2, states "A physician shall deal honestly with patients and colleagues and strive to expose those physicians deficient in character or competence, or who engage in fraud or deception." Reporting a colleague is a most unpleasant experience yet necessary to maintain the welfare of patients and the credibility of our profession. Ethical complaints may be filed with the district branch of the American Psychiatric Association (APA), with local medical societies, or with state licensing boards. Complaints are handled confidentially. Where appropriate, the psychiatrist should urge patients to file a complaint. If an ethical violation is confirmed by the APA, there are four possible sanctions, ranging from admonishment or reprimand to suspension or expulsion from the APA. The defendant psychiatrist is entitled to appeal.

Reports of possible ethical violations may be based on our own observations, disclosures from patients, or extrinsic evidence. The psychiatrist may be reluctant to believe allegations by a patient, particularly if they involve sexual misconduct by a colleague known to the psychiatrist. The patient may feel protective of the abusing therapist or fearful of the investigation process and may not wish to disclose. The treating psychiatrist may be reluctant to act contrary to his patient's wishes. Anonymous complaints by therapist or physician usually cannot be investigated. The APA Council on Ethical and Judicial Affairs ( [American Psychiatric Association, 1991](#)) believes that physicians must report sexual misconduct to the appropriate authorities. One should also be aware of state laws regarding the reporting of an impaired physician or one involved in abusive behavior.



Reports based on extrinsic evidence pertain to information a psychiatrist may have read in a newspaper or an event based on a legal fact, such as when a psychiatrist adopts a patient. Filing a complaint allows the ethics committee to further look into the matter. Extrinsic evidence may also be used to bypass an ethics hearing if the facts speak for themselves.

## SUMMARY

Child and adolescent psychiatrists need to familiarize themselves with the ethical codes that govern their practices. They should not be pressured by patients or insurance companies into acting in ways contrary to the patient's interests or our code of ethics, nor should they exploit insurance companies for their own financial gain. If in doubt as to whether a certain behavior is ethical, child and adolescent psychiatrists may refer to AACAP: *Annotations to AACAP Ethics Code* (1995) or consult with the ethics committee of the American Association of Children and Adolescent Psychiatrists or their APA district branch ethics committee.

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## Appendix: Principles of Practice of Child Psychiatry

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A child or adolescent and the family may expect the child psychiatrist to

Have as primary concerns the welfare and the optimal development of the individual child or adolescent assessed in the context of the family, school, and community based on scientific knowledge and collective and personal experience;

Foster the unique and nurturing relationship among the child or adolescent and the parents or caretakers and the family;

Recognize the child's or adolescent's need for the support of adults;

Avoid all actions that may have a detrimental effect on the optimal development of the child;

Use his or her unique relationship with the child or adolescent and family to foster their well-being and optimal development;

Promote, by all appropriate means, the uniqueness of the individual;

Seek to develop with the child or adolescent as thorough an understanding as possible of the child psychiatrist's role, opinions, conclusions, and recommendations;

Protect specific confidences of the child or adolescent and the parents or guardians and others involved, unless this course would involve untenable risks or jeopardize caretaking responsibility;

Seek to develop with those involved in the care or treatment of the child or adolescent (parents or guardians, and, when appropriate, the family, teacher and school, court or correctional agency, physician, and others) as thorough an understanding as possible of the child psychiatrist's role, opinions, conclusions, and recommendations;

Help the child or adolescent to recognize the influence of his or her own relationship with family members and the consequences of his or her decisions;

Help family members to resolve differences in their views of professional judgments or recommendations;

Avoid acting solely as an agent of the parents, guardians, or agencies;

Maintain the integrity of professional judgments and behaviors independent of influence of the source of compensation.

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## 133 HISTORY OF CHILD PSYCHIATRY

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In order for the field of child psychiatry to exist, there first had to be the separation of childhood and adolescence from the remainder of the life span. Although infants and toddlers have usually been seen as separate from adults, the appreciation of childhood with characteristic stages emerging into adolescence and youth is a modern achievement. Especially in the 19th and early 20th centuries, the first score years of the life span took on great interest and stimulated extensive study.

Psychiatry itself is a recent profession. From superintendents of mental hospitals and the occasional alienist in private practice in the 19th century, a new medical specialty slowly emerged. In 1934, the American Board of Neurology and Psychiatry began certifying practitioners, and about this time, a few physicians in America and Europe started using the words “child psychiatry” to denote their specialty. Nevertheless, psychiatry remained a small profession through World War II.

Although child psychiatry did not flourish until after World War II, its roots are much more extensive. These fall into two broad categories, study of the normative development of children and investigation of various abnormalities that may affect children. Often, these two streams of research have paid little attention to one another, although in recent decades their topics of study have drawn closer together.

An example of systematic investigation of normal development is the work of G. Stanley Hall (1844–1924), an early psychologist who received his doctorate under William James (1878) and later helped to found the American Psychological Association (1891), for which he served as its first president. Hall's interest was in education and in the nature of the normal child. His leadership among educators led to his selection as the first president of Clark University, in Worcester, Massachusetts (1888). There he immediately created a center for the study of child psychology—said to be the first in the nation—gathered coworkers and instituted intensive surveys of child development. Much of his work was published in journals he had founded: the *American Journal of Psychology*, the *Pedagogical Seminary*, and the *Journal of Applied Psychology*. This whole process was termed the *child study movement* and progressed in part through questionnaires distributed to teachers, students, and parents. From them he sought to learn the characteristics of children at various ages, how they learned, and what they typically knew. The topics ranged from “the teaching instinct” to “anger” ( [Ross, 1972](#)). Out of such studies came ideas that prefigured more recent research such as that on the moral development of children. In 1894, Margaret E. Schallenberger concluded from questionnaires that the child shifts from a morality of consequences to a morality of intention at about age 9, a finding similar to much more recent investigations ( [Siegel and White, 1982](#)).

Perhaps because of Hall's close association with educators, the child study movement had its greatest impact on the teaching profession, not among academics and professional psychologists. The concept of child development with its stages and unfolding competencies enhanced the role of teachers as experts in instruction. Ironically, schools of education pay little attention today to the psychology of child development; its reintroduction through such innovations as the Comer School Project is seen as breaking new ground ( [Comer, 1993](#)).

Hall had what we could now call a romantic view of child development, because he believed that the progression from child to adult recapitulated the history of mankind. Adolescence, for example, represented the “heroic age.” Hall popularized the term *adolescence*, and his fascination with that stage of life is demonstrated by the hefty two-volume work *Adolescence*, published in 1904 ( [Hall, 1904](#)).

Why were the teenage years so important in the late 19th century? Certainly a major reason is that the need for an educated workforce pushed the common endpoint for education from the eighth to the 12th grade. With the proliferation of high schools came a need to direct the legions of nonworking youth into acceptable paths. Where better to turn for advice on rearing this crucial generation than to experts like Hall? Advice and guidance, however, were not aimed solely at adolescents; younger children also could benefit from scientific training. During the first two decades of the 20th century, the Boy Scout and Girl Scout movements took hold, stressing practical and moral education. The child study movement proclaimed an ideal path of development that merged with the “Americanization” campaign directed especially at the growing numbers of new immigrants. In the swirl of this development, the public school was the linchpin that connected the youth movements and the reeducation of the latest Americans.

Sigmund Freud (1856–1939), who influenced child psychiatry profoundly, came from a direction opposite to that of Hall. Unlike Hall, Freud was a physician who studied pathology to illuminate normality, as Virchow had done in the study of organic disease in mid-19th century. Freud postulated certain phases of development from the psychoanalysis of adults. His one case study of a child, *Little Hans* (1909), illustrated the oedipal complex but, because he treated Hans through the father, Freud did not have contact with the boy ( [Freud, 1955–1972](#)). Primary psychoanalytic contact with children rather than adults characterized the career of Freud's daughter Anna, who extended her father's theoretical work. She also established for children a clinic near London that trained child psychiatrists and child psychoanalysts ( [Young-Bruehl, 1988](#)).

The concepts of psychoanalysis reached the United States through several routes. Freud himself was invited to Clark University's 25th anniversary in 1909, when Hall conferred on him an honorary doctorate, the only one he was ever to receive. This brief visit helped to spur an interest in psychoanalysis. Later, Freud's biographer Ernest Jones toured the United States extensively and advocated the new treatment's value. Americans picked up the importance of childhood events in forming adult character and looked to psychoanalysis to undo the damage early experiences may have caused. To estimate the power of psychoanalysis so highly was characteristically American, and it reflected an optimism that any barrier, including the psychological, could be overcome with the right technology ( [Burnham, 1967](#)).

Representative of this sanguine outlook is William Healy, a Chicago neurologist who, in 1909, established the Juvenile Psychopathic Institute to advise the courts on the psychology of arrestees. Healy had been persuaded to undertake this innovative role by several prominent Chicago women: Jane Addams, later awarded the Nobel Prize for Peace; Julia Lathrop, later the first head of the U.S. Children's Bureau (1912); and Ethel S. Dummer, a philanthropic activist. Healy strongly opposed the organic explanation of antisocial behavior; he found the answer in the environment from which the child offender emerged. He was optimistic because he believed the environment could be fixed, whereas biological defects seemed irreversible.

In 1917, Healy and his associate (and later his spouse) Augusta F. Bronner, a psychologist, were lured away from Chicago to Boston where they were given treatment facilities by the new Judge Baker Foundation. The lack of such facilities in Chicago had frustrated Healy, who was not satisfied with just supplying the court with an offender's diagnosis.

Healy was one of several prominent scholars and therapists who believed that the new dynamic psychology, chiefly psychoanalysis, provided a powerful tool for attacking the root causes of criminal behavior. Using the term *orthopsychiatry* to describe what we would now call child psychiatry, Karl H. Menninger brought together in 1923 a group of psychiatrists concerned with youthful offenders. Thus in 1924 was born the American Orthopsychiatric Association, with William Healy as its first president. This association, with annual meetings, a journal, and active representation to other social institutions, further focused attention on treatment of childhood disorders and helped to lead to the separation of child psychiatry from general or adult psychiatry ( [Musto, 1975](#)).

Inspired by Healy's Chicago and Boston clinics, a new kind of institution, the child guidance clinic, came into being in 1921 with the support of the Commonwealth Fund. These clinics had at their core a team approach: a psychiatrist who dealt with the patient, a psychologist who tested the patient, and a social worker who worked with the remainder of the family. The importance attributed to this style of care is shown by the rules for membership in American Orthopsychiatric Association, which by 1930 mandated that a member had to work in a clinic with the team approach.

While the child guidance clinics multiplied in the 1920s, theories of child development, advocated by able partisans on all sides, clashed; parents tried to decide



which philosophy to follow. John B. Watson, a proponent of behaviorism, powerfully influenced the U.S. Children's Bureau which made available millions of pamphlets on child rearing. Parents learned from these publications that rigid feeding schedules conditioned the infants properly and could be part of a larger conditioning program that would bring out the best in the child. Watson had no use for concepts like the unconscious; to him, inculcation of proper behavior would come from unyielding conditioning, with the proper stimulus eliciting the proper response. In other words, the parents' job was to mold the child's malleable mind ( [Kessen, 1965](#)). Rejecting this assumption was Arnold Gesell, a student of G. Stanley Hall who established a psychoclinic at the Yale Dispensary in New Haven, Connecticut in 1911. Like Hall, Gesell saw human life unfolding from inborn biological givens. Parents should be supportive, but the child's development would progress in a regular pattern unless the parents interfered too much. There was no need for rigid conditioning. Elaborate still and motion pictures of infants and children helped Gesell to compile atlases of development. Parents used these guides to assure themselves that their children were progressing normally and to anticipate the next stage of growth ( [Gesell, 1952](#)).

Gesell's psychoclinic grew into the Yale Clinic of Child Development and eventually, under the leadership of Milton Senn, the Yale Child Study Center. The shift from Gesell to Senn, a psychoanalytically inclined pediatrician, illustrates the great change in attitude that followed the era of biological determinism. Senn and many like him stressed a more optimistic faith in environmental causes of childhood disorder. In the tradition of Healy, reform of the child's environment and social improvement generally, including the problems of racism, poverty, and lack of education, became targets. A new emphasis by society on childhood, even if this focus was late and fell short of need, attracted persons who would otherwise have become adult or general psychiatrists.

If one were to search for a key event in the rise of child psychiatry in the United States, it could well be Adolph Meyer's appointment of Leo Kanner to be liaison between pediatrics and psychiatry at the Johns Hopkins Hospital in Baltimore. Meyer's decision had a salutary effect on the attitude of each field toward the other. Kanner termed himself a child psychiatrist and within 5 years published the first American text on child psychiatry ( [Kanner, 1935](#)). He is also the pioneer observer of what he called autism ( [Kanner, 1952](#)).

Pediatrics proved an excellent field from which to draw child psychiatrists. The Commonwealth Fund in 1937 began awarding fellowships to pediatricians for the study of psychiatry. The training program had not been established without debate, but major child guidance clinics and a few medical schools provided what was then available for the new field. The choice of pediatricians followed more than a decade of supporting psychiatrists. In 1922, the Commonwealth Fund and, in 1924, the Rockefeller Foundation had begun providing fellowships for psychiatrists. It was not unusual for psychiatrists to enter psychoanalytic training as well as improve their pediatric skills.

Out of the clinics the first formal training program for child psychiatrists evolved. Clinic directors began discussions in 1940, shortly before the United States entered World War II, that led to a new organization. The war slowed their efforts, but in 1945 they established the American Association of Psychiatric Clinics for Children. In 1947, this association set out formal guidelines for training in child psychiatry. A person could complete the training and could practice as a child psychiatrist, but there was still no accreditation. The physician could be a board certified psychiatrist or pediatrician, but not a certified child psychiatrist. Achieving the high standards of certification became a goal of the slowly growing profession.

In 1953, the American Academy of Child Psychiatry was formed, at first composed of only the highest ranking academics. By 1957, the academy's existence encouraged the American Board of Psychiatry and Neurology to establish child psychiatry as a subspecialty and to establish training standards for child psychiatry ( [Schowalter, 2000](#)). Gradually, the Academy has expanded so now it counts as members most child psychiatrists.

Since the 1970s, child psychiatry has had a burst of accomplishment in epidemiology, genetics, and biochemical studies and the gradual improvement in definition and course of autism, Asperger's syndrome, and attention deficit disorder. An important event that allowed progress in epidemiology was the appearance of the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* and the 10th edition of the *International Classification of Diseases*, which established a common international language for child psychiatrists ( [Chapter 39](#)). New publications set forth the explosion in child psychiatric research. The American Academy of Child and Adolescent Psychiatry, for example, in 1962 inaugurated its journal to transmit this current and voluminous research to the profession.

Even, however, as advances in child psychiatry reached unprecedented levels, in the United States the actual delivery of services to child and families in need collided with the cost-cutting restrictions of for-profit health maintenance organizations. Under a managed-care system, these organizations have imposed limitations on the kind and extent of services at the very time that child psychiatry has more to offer than ever before.

## ORIGINS OF CHILD PSYCHIATRY IN SOME OTHER NATIONS

### United Kingdom

Children's psychological abnormalities attracted some attention in 19th-century England. As reflected in Maudsley's 1867 textbook *The Physiology and Pathology of Mind*, which devoted a chapter to "the insanity of early life" ( [Maudsley, 1867](#)), childhood study there grew more intensive and more common around 1900 with child study research similar to Hall's in the United States. With the advent of psychoanalysis, an opportunity arrived when the various strands and professions concerned with children could be drawn together in the Child Guidance Movement. Supported by the Commonwealth Fund, which had underwritten the American clinics, training was made available to British therapists in the 1920s. Emanuel Miller opened the first clinic in East London, and then the London County Council established one. By 1944, there were 95 clinics ( [Wardle, 1991](#)). According to W. Parry-Jones, "Child Guidance Clinics created a model of inter-disciplinary collaboration by psychologists, psychiatric social workers and psychiatrists" ( [Parry-Jones, 1989](#)). One area in which collaboration failed was the creation of a common psychoanalytic training for child therapists: a theoretical split between Anna Freud and Melanie Klein had led to two separate training programs ( [King and Steiner, 1991](#)).

After World War II, the United Kingdom established the National Health Service, which mandated regional planning and the growth of inpatient services for children and adolescents. This system's influence on care was not without controversy. Public pressure mediated through the structures of the National Health System led Wardle to complain that "children and their services have been prey to *causes célèbres*, fashion and exaggerated fads and foibles of the media and politicians" ( [Wardle, 1991](#)). Although service policies may have been controversial, Britain significantly contributed to the understanding of childhood and its vicissitudes through the work of Bowlby, Rutter, Winnicott, and others ( [Bowlby, 1969-1977](#); [Rutter et al., 1970](#); [Winnicott, 1965](#)).

### Sweden

Sweden's early interest in childhood is exemplified by the 1845 appointment of the world's first university chair in pediatrics. During the period from 1900 to 1950, Sweden instituted mental health services for children in hospitals, public schools, and child guidance clinics, although services leaned more toward pediatrics than psychiatry. As early as 1915, prominent pediatricians such as Isak Jundell argued for the creation of a new discipline that would deal with the behavioral and emotional problems of children. In 1951, child and adolescent psychiatry became a separate discipline. Sven Ahnsjö, who came from a background in genetics, held the first chair of child and adolescent psychiatry ( [Rydellius, 1995](#)).

More recently, attention has focused on a program of eugenic sterilization conducted between 1935 and 1975. More than 60,000 Swedes (mostly women) were sterilized to improve the quality of Swedish society. This biological basis for dealing with extreme and condemned behavior had its origins going back as early as 1921, when the Institute of Racial Biology was established. In 2000, one of the physicians who had performed sterilizing operations explained: "We were thinking about the children," a demonstration of childhood's symbolic power to justify extreme measures ( [Boyes, 2000](#)).

### Germany

In general, German approaches toward childhood disorders have been organically based since the 19th century and have never been so widely accepting of psychoanalytic theory as in the United States. Serious attention to what is now embraced in child psychiatry can be found in such pioneering works as *Die Psychischen Störungen des Kindersalters* (Psychic Disturbances in Childhood) by H. Emminghaus in 1887. Perhaps because of the emphasis on organic causation, the eugenics movement, as in Sweden, pre-World War II United States, and other nations, had a great appeal. A desire to improve the quality of citizens stimulated legislation to sterilize criminals, feeble-minded persons, and the insane. U.S. Supreme Court Justice Oliver Wendell Holmes captured the attitude supporting eugenics when he declared, in a decision upholding the state's power to sterilize, "Three generations of imbeciles are enough" (*Buck v. Bell, 1927*) ( [Lewis et al., 1999](#)).

The rise of National Socialism and Adolf Hitler changed the German eugenics program into one that eliminated persons thought to be undesirable. It is estimated that 300,000 psychiatric patients were killed during the Nazi era ( [Peters, 1999](#)). Leading professors of psychiatry were replaced with Nazi adherents; anyone who was Jewish or who had a Jewish spouse was removed. Overall, the Nazi era interfered with investigations and progress in child psychiatry because appointments to head

departments and institutes had as their primary criterion an adherence to the racial ideology of National Socialism. Along with the rise of National Socialism came the exodus of German physicians and scientists, in most cases of Jewish origin, to other nations. In 1921, Prof. Karl Bonhoeffer had opened the Department of Child Neuropsychiatry at Berlin, and it is probably there that the most sustained research continued until his retirement in 1938. Since World War II, German child psychiatrists have formed new societies, reinvigorated research centers, and, once again, have achieved a high international standing.

## CONCLUSION

In many nations during the 20th century, child psychiatry alternated between two major phases: belief in the environment as the crucial determinant for child development and adult maturation, and a faith in an organic or biological basis for behavior. These positions have taken hold with astounding depths of conviction and have led to extreme measures such as massive sterilization and euthanasia in the hope of improving the next generation of children.

Once again, we have entered an era of growing faith in biological determinism. The human genome is in the process of being unraveled and characterized. As we move toward improving life through biological discovery, we must keep in mind the history of exclusive faith in biology and the temptation to act against individuals under the guise of purifying mankind. The early days of a new approach to understanding behavior have always been thrilling and filled with a sense of release from ties and restraints of the past. Such enthusiasm accepts few antidotes, but an appreciation of history tends to temper judgment.

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